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Abstract #1
BRAIN BIOPSY IN THE DOG: THE LAITINEN'S METHOD. *P. Moissonnier*. Service de Chirurgie, Ecole Nationale Vétérinaire d'Alfort, Maisons Alfort, France.

Abstract #2
NEW SURGICAL TREATMENTS OF BRACHIAL PLEXUS AVULSION. *P. Moissonnier*. Service de Chirurgie, Ecole Nationale Vétérinaire d'Alfort, Maisons Alfort, France.

Abstract #3
COMPLICATIONS OF MYELOGRAPHY. A.L. Hopkins. North Florida Neurology, Jacksonville, FL

Abstract #4
PATHOLOGICAL FINDINGS ASSOCIATED WITH OSSEOUS DURAL METAPLASIA, AN UNDERESTIMATED SPINAL SYNDROME. *M.T. Mandara**, E. Lepri*, M. Sforna*, E. Bellezza° *Department of Biopathological Veterinary Science, University of Perugia, Italy. °Department of Pathology, Diagnosis and Veterinary Clinic, University of Perugia, Italy.

Osseous Dural Metaplasia (ODM), also known as spinal ossifying pachimeningitis, is considered an age-related change in the dog. Though ODM is observed very frequently, the main functional and clinical implications of this dystrophic process merit further investigation.

The results obtained from the examination of the spinal cord or intracanal biopsy in 51 dogs are reported. Twenty-six dogs showed clinical signs of spinal deficits, associated with ODM diagnosed by gross and/or bioptic examination. The remaining 25 dogs did not show any neurological spinal signs, and for this reason were considered as the control group. Of these, eleven dogs, including six German Shepherds and ten male dogs, with a mean age of 7.5 years, showed an incidental ODM at gross examination. Of the 26 clinical cases, ODM affected mostly German Shepherds and in ten cases the animals were under 5 years of age (38.5%). In eight cases myelography showed spinal compression, nevertheless ODM was not identified. In ten cases the spinal syndrome had an acute onset (38.5%), while in 16 cases it was chronic and slowly progressive.

Spinal cord samples from 14 dogs with clinical spinal diseases were submitted to histological examination, routinely performed with H&E and Luxol fast blue. Spinal lesions ranging from myelin loss to a decreased number of axons and focal areas of leucomalacia, associated with islands of lamellar bone at times containing adipocytes and myeloid elements were found. In three cases, characterized by an acute onset of the spinal syndrome, the lesions consisted of necrosis and haemorrhages involving both the grey and white matter, while in four further cases, characterized by a slowly progressive spinal syndrome, a selective loss of spinal cord neurons was evident, along with white matter and spinal nerve root degeneration, as an expression of the compressive traumatic action of ODM.

Therefore, in the present study osseous dural metaplasia was a cause of spinal syndrome in both old and young animals. Indeed, it confirms the predisposition of large canine breeds and shows the high prevalence of this dystrophic event in male dogs. Finally, ODM caused varying degrees of lesions suggesting ODM should be considered in the differential diagnosis of both acute and chronic onset ischemic and/or compressive spinal diseases and surgery encouraged.

Abstract #5
CONGENITAL APLASIA OF VERTEBRAL ARTICULAR FACETS IN FOUR DOGS: RADIOGRAPHIC, MYELOGRAPHIC AND MR IMAGING FINDINGS. *Penderis J*[1], Schwarz T[2], McConnell F[1], Garosi LS[1], Thomson C[3], Dennis R[1] [1]Animal Health Trust, Newmarket, UK. [2]Department of Clinical Studies, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, USA. [3]Department of Small Animal Clinical Studies, Glasgow Veterinary School, Glasgow, UK.

Congenital anomalies of the vertebral column compromising vertebral column stability or impinging on neural structures are well described in the veterinary literature. The majority of these cases are associated with aberrations of one of the primary vertebral ossification centres. Clinically significant abnormalities of secondary vertebral ossification centres, particularly involving caudal articular processes, are much less frequently reported. In this study we describe three dogs with congenital aplasia of the caudal vertebral articular processes and one dog with congenital hypoplasia. Thoracolumbar spinal cord compression occurred in the cases of aplasia. In all three cases of aplasia, the animals presented with progressive pelvic limb ataxia and focal thoracolumbar pain occurred in two cases. No clinical signs were evident in the case with hypoplasia of the caudal vertebral articular processes. The radiographic appearance was similar in all cases, with aplasia or hypoplasia of the caudal articular facets at one or more intervertebral joints in the thoracolumbar region. The adjacent cranial articular facets were increased in size occupying the space of the absent caudal articular facets. Bone proliferation was evident secondary to an associated degenerative joint disease. This compensatory hyperplasia of the adjacent cranial articular facets protruded into the vertebral canal, resulting in a compressive myelopathy as demonstrated by myelography and MR imaging. The findings of this study indicate that congenital anomalies of the caudal articular facets occur in dogs, demonstrate typical imaging findings and that total absence of the caudal articular facets has a high association with neurological deficits.

Abstract #6
SUSPECTED SPINAL CORD INFARCTION IN TWO SMALL BREED DOGS: MAGNETIC RESONANCE IMAGING FINDINGS. *F. Gruenenfelder*[1], D. Weishaupt[2], F. Steffen[1] 1 Dept. for Small Animals, Neurology Services, University of Zurich, 2 Institute of Diagnostic Radiology, University Hospital Zurich, Switzerland

Abstract #7
ANTERIOR FORAMINOTOMY AS A NEW TREATMENT MODALITY IN DEGENERATIVE LUMBOSACRAL DISEASE. *T. Goedde*, Tierärztliche Gemeinschaftspraxis, Piding; Germany.

Abstract #8
STATUS EPILEPTICUS IN THE DOG: RETROSPECTIVE EVALUATION OF 41 CASES. *G. Gandini**, G. Fluehmann°, E. Brini*, S. Cizinauskas§, A. Jaggy ° *Department of Veterinary Clinical Sciences, University of Bologna, Ozzano Emilia (Bologna), Italy. °Department of Veterinary Clinical Medicine, Animal Neurology Section—University of Berne, Switzerland. §Department of Clinical Veterinary Sciences, Animal Neurology Service—University of Helsinki, Finland.

Status epilepticus (SE) is a well known life-threatening condition in the dog.

Nevertheless, retrospective studies on large populations of dogs are few in veterinary literature. Purpose of this study was a retrospective evaluation of epidemiological, clinical and pathological data of 41 dogs with the history of status epilepticus. All patients were admitted to the emergency service of Animal Neurology Section of University of Berne.

All the dogs underwent a complete physical and neurological examination as well as complete blood cell count and serum biochemical profile examinations. The clinical work-up included in selected cases additional blood parameters (i.e. bile acids and/or ammonia: 19 dogs), cerebrospinal fluid (CSF) examination (28 dogs) and brain imaging (CT/MRI) (6 dogs). Histopathologic examination of the brain was performed in 14 cases. Duration of SE was between 30 minutes to 24 hours in our dogs. There was no breed and gender predilection. Mean bodyweight was 22,6 ±13,4 kg. 22 dogs (group A) survived SE and 19 (group B) died or where euthanized because of poor condition. 19 dogs (46%) experienced seizures before the onset of SE; out of these, 12 dogs (63%) belong to the group B. Absence of previous seizures was more frequent in group A (15 dogs: 68%).

Epilepsies were classified as follows. Symptomatic epileptic seizures (SES) were suspected when data from the following work-up suggested structural brain disease. Re-active epileptic seizures (RES) were diagnosed if the findings in the history suggested intoxication or an extracranial metabolic condition which could have impaired brain function was found. Dogs were identified to have primary epileptic seizures (PES) when no pathological changes were found during the work-up of the patient.

The most frequent clinical diagnoses in group A dogs were RES (11 dogs; 50%), followed by PES (5 dogs; 23%) and SES in one dog. Unclassified seizures were diagnosed in the rest 5 dogs of group A. SES were diagnosed in 15 dogs (79%) of group B as well as PES and RES in one dog each. Unclassified seizures were diagnosed in two dogs in group B.

Results of our study show that SE is more frequent in dogs suffering from SES and RES than from primary epileptic seizures. It seems that dogs with SE and SES have a poor prognosis.

Abstract #9

CHARACTERISTICS OF BRAIN NEOPLSIA INDUCED SEISURES IN CATS. *Cizinauskas S.*[1,2], Gandini G.[3], Fatzer R.[1], Schenkel M. [1], Jaggy A.[1] [1]Department of Veterinary Clinical Studies, University of Bern, Switzerland; [2]Department of Clinical Veterinary Sciences, University of Helsinki, Finland; [3]Department of Veterinary Clinical Sciences, University of Bologna, Italy.

Etiological investigations of seizures in large cat populations are rare in veterinary medicine. The aim of this study was to describe the clinical and epidemiological findings in cats suffering seizures caused by brain neoplasia.

Clinical records of cats referred to the animal neurology section of University of Bern between 1985 and 2000 were evaluated retrospectively. From the total of 1258 patients, 177 cats with a history of seizure activity were identified (177/1258; 14%). 154 cats with comprehensive clinical information were further evaluated. In less than one tenth (8.4%) of our patients (13/154) the intracranial neoplasia was diagnosed. The diagnosis was confirmed by histopathological brain examination in 12/13 cats and included lymphoma (6/13), meningioma (3/13), astrocytoma (2/13), olfactory neuroblastoma (1/13) and bronchogenic squamous cell carcinoma (1/13).

Majority of cats were the domestic shorthair cats (10/13). No sex predilection was noticed. All cats were adult and the age range was 1 to 13 years. Both generalized (8/13) and complex focal (4/13) seizures were observed by the owners. In one cat both mentioned types of seizures were observed. Seizure frequency had a tendency to be highest in cats with astrocytoma, moderate in lymphoma patients and low in cats with meningioma. Neurological abnormalities such as changes in behavior or gait were the major complain by the owner in our patients. Majority of the cats showed marked neurological deficits pointing to lesion in the forebrain (8/13), brainstem (3/13) or both (1/13). Brain imaging showed a space occupying lesion in two cats. Cerebrospinal fluid examination was abnormal in 4 and normal in 3 cats. Long term prognosis was unfavorable in the majority of cases in spite of antiepileptic therapy. Surgery was performed in one cat.

It has been described in veterinary literature, that lymphoma and meningioma account for most brain neoplasia in cats. Our results support these findings also in the epileptic cat population.

Abstract #10

CANINE INTRACRANIAL MENINGIOMA OUTCOME FOLLOWING CORTICOSTEROIDS, HYPOFRACTIONATED RADIOTHERAPY OR MULTIMODALITY THERAPY; 60 CASES. *S.R.Platt*,[1] L.S. Garosi,[1] V. Adams,[2] S. Murphy,[1] C.J. Abramson[3] [1]Centre for Small Animal Studies, The Animal Health Trust, Newmarket, Suffolk, England; [2]Centre for Preventive Medicine, The Animal Health Trust, Newmarket, Suffolk, England; [3]Department of Veterinary Clinical Sciences, The Ohio State University. Columbus, Ohio, USA.

Surgical resection alone and surgery combined with hyperfractionated radiotherapy (RT) has been shown to improve survival times in dogs with intracranial (IC) meningiomas. Although the use of hypofractionated RT has been documented for the treatment of canine brain tumours, it has not been specifically compared as a sole therapy to the use of steroids alone or to combined surgery and RT. The purpose of this study was to evaluate the effect on survival of dogs diagnosed with IC meningiomas after treatment with corticosteroids, hypofractionated RT or combined surgery and RT.

Three groups of dogs were evaluated; all dogs were diagnosed with an IC extra-axial rostro-tentorial mass on MRI, with signal characteristics compatible with a meningioma, and confirmed histopathologically in groups I and III. Group I dogs (n = 10) were treated with anti-inflammatory doses of prednisone (0.5–1.0 mg/kg/day). Group II dogs (n = 28) were additionally treated with 5 once weekly doses of RT (7–9 Gy). Group III dogs (n = 22) were treated with surgical resection and RT. Survival was calculated from the time of diagnosis to the time of death related to the tumour. Survival analysis was carried out using the Kaplan-Meier procedure. Censored cases included dogs that were still alive or were lost to follow-up but were still alive at the last recorded follow-up. Survival rates among treatment groups were compared using log-rank tests with significance set at $P < 0.05$.

The following table shows the median survival times by treatment group. Dogs treated with combined RT and surgery survived significantly longer than dogs treated with RT alone ($P = 0.03$) or with steroids alone ($P < 0.0001$). Dogs treated with RT alone survived significantly longer than dogs treated with steroids alone ($P = 0.0025$).

				Survival time (days)	
Group	Treatment	Cases	Censored	Median	95% CI
I	Steroids	10	0	119	84–182
II	Steroids + RT	28	4	224	182–420
III	Steroids + RT + Surgery	22	8	448	378–616

This study was not a randomised controlled trial and therefore findings may be related to inherent bias in treatment selection groups. The study demonstrates that multimodality therapy may be more beneficial for the treatment of canine IC meningiomas than treatment with RT and steroids or steroids alone, although it does not compare favourably to historical data on hyperfractionated RT regimens.

Abstract #11

FUNCTIONAL $GABA_B$ RECEPTORS ARE LOST AFTER STATUS EPILEPTICUS. *K.E. Chandler*[1], A.P. Princivalle[2], R. Fabian-Fine[3], N.G. Bowery[2], D.M. Kullmann[1] and M.C. Walker[1] [1] Institute of Neurology, University College London, UK, [2]Department of Pharmacology, University of Birmingham, UK, [3]Department of Psychology and Neuroscience Institute, Dalhousie University, Canada.

The synapse between hippocampal mossy fibres and CA3 pyramidal neurons constitutes a major excitatory input to the hippocampus. The release of glutamate at this synapse is potently modulated by a number of inhibitory mechanisms that may prevent propagation of seizure activity into the hippocampus proper. One such mechanism is mediated by spillover of GABA onto mossy fibre presynaptic $GABA_B$ receptors. Thus, transmitter release from one group of mossy fibres can depress synaptic activity in an adjacent group of mossy fibres. We determined if $GABA_B$ receptor mediated heterosynaptic depression (HD) is altered after status epilepticus in two different rodent models—the pilocarpine and perforant path stimulation models

We measured HD by recording extracellular field excitatory post-synaptic potentials in acute hippocampal slices prepared from control rats and rats following status epilepticus. We induced HD by stimulating a test mossy fibre pathway at low frequency and applying a train of stimuli to an adjacent mossy fibre pathway.

We found that status epilepticus in both rodent models, was followed 24 hours later by a loss of $GABA_B$ receptor-mediated heterosynaptic depression of mossy fibre transmission. This was accompanied by a decrease in the sensitivity of mossy fibre transmission to the exogenous $GABA_B$ receptor agonist baclofen. Autoradiography revealed a reduction in $GABA_B$ receptor binding in the mossy fibre termination zone (stratum lucidum) after status epilepticus.

$GABA_B$ receptor mediated heterosynaptic depression is lost after SE and is associated with a reduction in functional $GABA_B$ receptors. Failure of $GABA_B$ receptor-mediated modulation of mossy fibre transmission may contribute to heightened excitation of the hippocampus and the development of spontaneous seizures after status epilepticus.

Abstract #12

GABAPENTIN AS ADJUNCTIVE THERAPY FOR REFRACTORY IDIOPATHIC EPILEPSY IN DOGS. *S.R.Platt*,[1] V. Adams,[2] L.S. Garosi,[1] C.J. Abram-

son,[3] J. Penderis.[1] [1]Centre for Small Animal Studies, The Animal Health Trust, Newmarket, Suffolk, England; [2]Centre for Preventive Medicine, The Animal Health Trust, Newmarket, Suffolk, England; [3]Department of Veterinary Clinical Sciences, The Ohio State University. Columbus, Ohio, USA.

Idiopathic epilepsy (IE) in dogs can be refractory to the appropriate and combined therapy of phenobarbitone (PB) and potassium bromide (KBr). Gabapentin (GBP) has been approved as add-on therapy in human patients with epilepsy. The purpose of this investigation was to evaluate the efficacy and side-effects of GBP as an add-on therapy in dogs with IE refractory to PB and KBr.

Eleven dogs diagnosed with refractory IE were administered oral GBP for a minimum of three months at an initial dose of 10 mg/kg q 8 hours. Each dog was experiencing generalised tonic-clonic seizure episodes and had been previously investigated for an underlying disease by means of cerebrospinal fluid and MRI assessments. All of the dogs had been chronically treated with a combination of PB and KBr at doses necessary to attain acceptable therapeutic serum levels without causing significant side-effects. The seizure frequency per week, average seizure duration and the number of days on which seizures occurred were compared in each patient for three months before and after GBP administration, using non-parametric Wilcoxon signed rank tests for paired data. Significance was set at $P < 0.05$ for all statistical tests. A positive response to GBP was interpreted as a minimum 50% reduction in the seizure frequency per week.

Five dogs (45%) exhibited a positive response to adjunctive GBP. There was no significant ($P > 0.05$) change in the duration of seizures. Seizure frequency was significantly reduced after the addition of GBP, both for number of seizures per week ($P = 0.005$) and number of days with any seizures in a two-week period ($P = 0.03$). When two cases which clustered daily, prior to the initiation of GBP, were excluded, seizure frequency for the remaining 9 cases remained significantly reduced ($P = 0.012$) but the number of days with any seizures in a two-week period was not ($P = 0.07$). Mild side-effects (ataxia and sedation) were noted in 5 dogs (45%) but were not severe enough to warrant discontinuation of the therapy. One dog exhibited a sterile panniculitis after 18 months of therapy, which responded to cessation of the GBP.

This small study indicates that gabapentin may be an effective adjunctive medication in reducing seizure frequency in some dogs with refractory IE; however, a larger scale study is warranted to further evaluate its role in veterinary medicine.

Abstract #13

INHERITANCE OF OCCIPITAL BONE HYPOPLASIA (CHIARI I MALFORMATION) IN CAVALIER KING CHARLES SPANIELS. *C. Rusbridge*, S.P. Knowler. Stone Lion Veterinary Centre, London

Occipital bone hypoplasia resulting in caudal fossa overcrowding, obstruction of the foramen magnum with secondary syringomyelia (SM) and ventricular dilatation is a common condition in the Cavalier King Charles spaniel (CKCS) and is a naturally occurring genetic model of human type 1 Chiari malformation

A family tree of over 4,200 related dogs was constructed from the pedigrees of 100 dogs with syringomyelia. For comparison a family tree from 40 CKCS with primary epilepsy and no clinical signs of syringomyelia was also constructed.

The study showed that from earliest known cases 15 years ago, 6 out 8 great grandparents of all affected cases could be traced back to 2 female ancestors so that all 8 were descended from one or the other or both. As a consequence it is suggested that the inheritance is likely to involve 2 or more genes. The disease appears to be more severe and have an earlier onset with increased inbreeding i.e. the disease seems to show genetic anticipation. This is especially apparent when breeding from affected dogs. There appears to be 3 forms of the disease based on severity and age of onset 1) neonatal form (less than 6 months) presenting with clinical signs relating to hydrocephalous 2) juvenile form (6–15 months) initially presenting with scoliosis secondary to syringomyelia and 3) adult form (1–10years) initially presenting with shoulder scratching and pain secondary to syringomyelia. The family tree of idiopathic epilepsy was a different subset of the CKCS population although there was some overlap. Epilepsy is particularly a problem in whole–coloured CKCS because of the relatively small gene pool for recessive ruby colouration. The disease is not linked to coat colour however selection for coat colour variation is believed to have influenced the development of the disease as certain significant ancestors for syringomyelia are also important in the inheritance of coat colour. Avoidance of some lines which carry certain disease e.g. heart and cataract disorders is also affecting the incidence of syringomyelia by narrowing the gene pool.

The aim for the next phase of the project is to collect DNA from extended 3 or 4 generation CKCS syringomyelia families with a view to genotyping, linkage analysis and positional gene cloning.

Abstract #14

SUBOCCIPITAL CRANIECTOMY AND CRANIAL DORSAL LAMINECTOMY AS A TREATMENT OPTION FOR CHIARI TYPE I MALFORMATION IN THE CAVALIER KING CHARLES SPANIEL. *K. Vermeersch*[1]; L. Van Ham[1]; J. Caemaert[2]; M. Tshamala[1]; O. Taeymans[1]; S. Bhatti[1]; I. Polis[1.1]Small Animal Department, Faculty of Veterinary Medicine, Ghent University, Merelbeke, Belgium. [2]Department of Neurosurgery, Faculty of Medicine, Ghent University Hospital, Gent, Belgium.

Syringohydromyelia combined with cerebellar tonsil herniation, known as Chiari type I malformation (CIM) is known to occur in Cavalier King Charles spaniels in many countries. The dogs either have progressive cranial (eg, facial deficits, seizures, vestibular syndrome) or spinal (eg, hyperesthesia with persistent scratching of shoulder and neck region) symptoms. Diagnosis is best made by magnetic resonance imaging (MRI): fluid-filled cavity within the spinal cord and cerebellar tonsil herniation. As therapeutic options medical treatment with corticosteroids, acetazolamide, NSAIDs or oral opioids may give an improvement but most often not a resolution of signs. It is thought that syrinx formation in humans and dogs with CIM occurs secondary to partial obstruction of CSF flow at the cranio-cervical junction. Therefore, in human medicine, a suboccipital craniectomy and cranial dorsal laminectomy with opening of the dura mater is the procedure of choice for surgical treatment of CIM.

This surgical technique was performed on 4 Cavalier King Charles spaniels diagnosed with Chiari type I malformation by symptoms (scratching of neck region) and by MRI. The dogs were evaluated neurologically 24 hours, 1 month and 3 months postoperatively. Control MRI took place 3 months postoperatively.

Three dogs recovered uneventfully from anesthesia. The fourth dog was euthanized within 24 hours after surgery at owners request due to progressive seizures and decreased capability of oxygen saturation. Neurologically, the 3 dogs did worse 24 hours after surgery (neck pain, neck weakness and head tilt), improved gradually and by 3 months postoperatively, achieved the same neurological state as before surgery. Control MRI of the 3 dogs at that time showed no regression of syrinx size.

The results of this study indicate that there is no improvement on short term basis in either syrinx size or clinical symptoms after surgical intervention with a suboccipital craniectomy and cranial dorsal laminectomy with opening of the dura. Seen the progressive nature of the disorder, follow-up over a longer period of time is necessary to see if this surgical technique may influence the progression of syrinx formation and clinical signs in Cavalier King Charles spaniels with Chiari type I malformation.

Abstract #15

INTRACRANIAL HAEMORRHAGE ASSOCIATED WITH *ANGIOSTRONGYLUS VASORUM* INFECTION IN THREE DOGS. *L.S. Garosi*, S.R. Platt, S.C. Shaw, F.J. McConnell & *K. Smith. Centre for Small Animal Studies, The Animal Health Trust, Newmarket, Suffolk, England. *Centre for Preventive Medicine, The Animal Health Trust, Newmarket, Suffolk, England.

The present report describes three dogs with intracranial haemorrhage secondary to severe coagulation defects associated with *Angiostrongylus vasorum* infection. The initial case was diagnosed at necropsy with two subsequent cases diagnosed ante mortem and successfully treated.

Three dogs with an age range of fourteen months to two and half years were presented for evaluation of a severe, subacute-onset of suspected brain disorders. Neuroanatomical localisation was consistent with a multifocal forebrain and brainstem disorder in each dog. Magnetic resonance imaging (MRI) performed on all three dogs was suggestive of multiple areas of intraparenchymal brain haemorrhage. Coagulation profiles (activated partial thromboplastin time, prothrombin time and thrombin clotting time and fibrin degradation products) were abnormal in all three dogs. No additional diagnostic tests were performed on the initial case as the owners elected for euthanasia. Post-mortem examination confirmed the presence of multiple intracranial and extracranial haemorrhages. An unexpected finding was that of a marked multifocal nematode infestation of the lungs with an associated vasculopathy. The parasites were confirmed to be *Angiostrongylus vasorum.* In two dogs, faecal examination by Baermann technique confirmed *Angiostrongylus vasorum* infestation. Thoracic radiographs in both dogs revealed broncho-interstitial lung pattern. Treatment in two dogs with Fenbendazole and plasma transfusion in one resulted in normalisation of the clotting profile within two days. Repeat neurological examination after six weeks was normal in both dogs.

This case series indicates that *Angiostrongylus vasorum* infection should be considered as possible aetiology of intracranial haemorrhage in dogs.

Abstract #16

A SPONTANEOUS CEREBRAL HAEMATOMA IN A DOG? *Y-P Chang*[1], A. Williams[2], V. Johnson[1], T.J. Anderson[1] [1]Department of Veterinary Clinical Studies and [2] Department of Veterinary Pathology, Institute of Comparative Medicine, University of Glasgow Veterinary School, Glasgow, Scotland.

A 14-year-old female neutered Jack Russell terrier was presented to the Uni-

versity of Glasgow Veterinary School (UGVS) with a two-week history of apparent neck pain followed by aggression behavioural changes. A grade five systolic heart murmur was auscultated on physical examination. Neurological examination was unremarkable.

Mild changes were identified on haematological, biochemical and urine analyses, consistent with a previous identified renal disease. Serology for *Toxoplasma, Neospora* and canine distemper virus was negative. Computed tomography (CT) (Exel 2400 elite®, Elscint Ltd) of the skull revealed a mass with a hypodense centre and mild contrast ring enhancement in the left caudal olfactory lobe, compressing the rostral horn of lateral ventricle. Cerebrospinal fluid (CSF) analysis revealed xanthochromia, increased protein and marked pleocytosis with predominance of neutrophils and RBCs. Bacteriology of CSF was negative.

Treatment was initiated with enrofloxacin, metronidazole and prednisolone. No improvement in the dog's behavior was noted over the subsequent two weeks. Magnetic resonance (MR) imaging (Philips Gyroscan ACS-NT 1.5 Tesla) was performed. A mass apparently within the left lateral ventricle was detected. It was hyperintense on T1-weighted images and hypointense to isointense on T2-weighted images. A signal void representing magnetic susceptibility artifact was seen on gradient echo sequences. The differential diagnoses at this stage included haematoma associated with vascular malformation or haemorrhage associated with neoplasia. A coagulation screen and systolic blood pressure measurements were normal. At this stage the owner elected to euthanise the dog due to the continuing aggression and the guarded to poor prognosis.

A post-mortem examination was performed. Pathological examination of the brain revealed a haematoma-like lesion in the left lateral ventricle, adjacent to and involving its medial wall. Microscopic examination revealed an organised sub-ependymal periventricular haematoma protruding into the left lateral ventricle.

The underlying cause of the cerebral haematoma remains unknown. No evidence of neoplasia and vascular malformation was detected. In general, cerebral haematoma is a rare condition and may mimic neoplastic disease.

Abstract #17

EXPERIMENTAL RECONSTRUCTION OF AUTONOMIC B- AND C-FIBERS IN RATS *Matiasek K*[1], May F[2], Weidner N[3], Caspers C[2], Mrva T[2], Gaensbacher B[4], Hartung R[2]; [1]Institute of Veterinary Pathology & Neuropathology, LMU Munich; [2]Dept. of Urology, TU Munich; [3]Dept. of Neurology, University of Regensburg; [4]Dept. of Exp. Oncology & Therapeutic Res., TU Munich; Germany

In order to evaluate new microsurgical strategies for B- and C-fiber reconstruction a panel of techniques had been applied to surgically transected cavernosal nerves in rats and compared to established regimen and sham operated controls. Despite its rather poor outcome autografting remains the golden standard of bridging autonomic nerve defects which indicates a lack of more specific therapies. Other trials that proved successful in sciatic nerve reconstruction are hampering from essential differences in regeneration biology which might be overcome by selective supplementation of neurotrophic sources and/or factors crucial for B- and C-fiber restoration. In the present study we checked and compared the therapeutic efficacy of 1. autografts, 2. empty silicon conduits, 3. silicon tubes seeded with autologous Schwann cells, and 4. silicon tubes filled with Schwann cells overexpressing the glial-derived neurotrophic factor GDNF on crossing 5 mm-gaps within cavernosal nerves of rats. Altogether 90 adolescent male Fisher rats were randomly distributed to the groups cited above or to sham operated controls. Cavernosal nerve function was assessed postoperatively after a period of 3 months by electrostimulation of the major pelvic ganglion and the proximal cavernosal nerves. Thereby, erectile function was evaluated by adspection and intracavernosal as well as systemic blood pressures were recorded simultaneously. After 3 months all animals were sacrificed and control nerves and interponates were inspected histologically and ultrastructurally at an equal distance from the pelvic ganglion. 3 months after nerve transection only 25% of graft recipients and 45% of rats with empty tubes recovered from erectile dysfunction successfully. Implantation of seeded guides, however, lead to a recovery rate of 95% with and 91% without GDNF-overexpression. In contrast to other groups ($P < 0.001$), both Schwann cell recipients did not differ significantly ($P = 0.5/ 0.3$) from sham operated controls in either visible erection or pressure rise. Moreover, pathological inspection revealed larger regenerates with enhanced differentiation of peri- and endoneurium within the seeded silicon tubes. These results demonstrate that supplementation with autologous Schwann cells is an excellent method to accelerate autonomic nerve healing and to improve restoration quantitatively by means of nerve fiber regrowth and target reinnervation. It proved superior to autografts which in this study were the least effective even far behind implantation of empty synthetic conduits. The use GDNF-overexpressing Schwann cells, moreover, adds a forceful neurotrophic component to the intralesional microenvironment that promotes regeneration of autonomic fibers quite selectively.

Abstract #18

ASSESSMENT OF FUNCTIONAL RECOVERY IN THE RAT SCIATIC NERVE MODEL. *A.S.P. Varejão*,[1] S. Geuna,[2] A.J. Ferreira,[3] R.C. Gabriel,[4] V.M. Filipe,[5] P.A. Couto,[5] P. Melo-Pinto[5]. [1]Department of Pathology and Veterinary Clinics, CETAV—University of Trás-os-Montes e Alto Douro, Vila Real, Portugal. [2]Department of Clinical and Biological Sciences, University of Turin, Italy. [3]Faculty of Veterinary Medicine, University of Lisbon, Portugal. [4]Department of Sport, CETAV—University of Trás-os-Montes e Alto Douro, Vila Real, Portugal. [5]Department of Engineering, CETAV—University of Trás-os-Montes e Alto Douro, Vila Real, Portugal.

The ability to assess recovery of motor and sensory functions after a nerve lesion is a very important part of peripheral nerve research. The rat sciatic nerve model is by far the most used model for the study of peripheral nerve regeneration after different types of injuries or repair techniques. It is not generally agreed which type of functional evaluation is the most useful descriptor of recovery. The goals of the present study were to compare, in the rat sciatic nerve model, the sequence of functional recovery in two experimental models of nerve regeneration: 1) reconstruction of a 10-mm gap with a biodegradable poly (DLLA-ϵ-CL) nerve guide; 2) acute nerve crush injury with a non-serrated clamp.

Follow-up post-operative functional assessment was carried out over a 24-week period in the nerve guide group, and 8-week period in the crush group. Traditional methods such as the sciatic functional index (SFI), the extensor postural thrust (EPT), the withdrawal reflex latency (WRL), the motor nerve conduction velocity (MNCV) and the gastrocnemius and soleus muscle weight were used to assess rat sciatic nerve recovery. Additionally, the kinematic behaviour of the foot and ankle was investigated during the stance phase of walking.

In the nerve guide group, the animals showed a functional recovery of approximately 60% after 24 weeks, while in the group of animals that suffered crush injury, a full functional recovery was predicted by several parameters at 8-week post-operatively, while others were still recovering their original values. Computerized gait analysis provided a tool for generating objective and reliable data in order to detect small biomechanical changes.

The results of this work emphasize the importance of using multiple methods of analysis for a comprehensive assessment of functional recovery in the rat sciatic nerve model.

Abstract #19

SPONGIFORM LEUKOENCEPHALOPATHY IN CRETAN HOUND PUPPIES. *Z.S. Polizopoulou*[1], C. Herden[2], A.F. Koutinas[1], W. Baumgaertner[2], N. Soubasis[1. 1] School of Veterinary Medicine, Aristotle University of Thessaloniki, Greece, [2] School of Veterinary Medicine, Hannover, Germany.

This report describes the clinical signs and histopathological findings of a neurological syndrome very similar to what is referred as ''shaker puppy syndrome'', in two litters of puppies belonging to a local hunting breed in the island of Crete. Efforts from local breeders to establish the desired phenotypic characteristics of this breed resulted in an intensive inbreeding where a limited number of dams and sires had been used. The parents of these two litters, though different, were closely related and had repeatedly produced affected puppies in the past.

All 6 puppies from both litters were affected and their neurological disease that appeared at the age of 2–3 weeks was characterized by pelvic limb bouncing and generalized tremors, which varied in severity. In every instance, the neurological signs worsened with excitement and subsided during rest or sleep. No other clinical abnormalities could be found and the results of routine clinicopathological examination, performed upon admission, were unremarkable. Five of the animals that presented moderate to severe neurological signs were eventually euthanized, while the least affected puppy was adopted, thus allowing a 9-month follow-up period during which it was showing a progressive improvement of hindlimb tremors, apparent from the age of 6 months.

Histopathology was limited only to the white matter of the brain and was consistent with spongiform lesions located in corpus callosum, fimbria, capsula interna, cerebellum, basal ganglia and the fiber tracts of hypothalamus and mesencephalon. Only some of the affected areas demonstrated mild to moderate hypomyelination. All these lesions were much more severe in the puppies of the second litter. Brain immunocytochemistry was negative for CDV and BSE prion protein but positive for CPV-2 antigen, apparently because those puppies had experienced parvoviral enteritis very soon after their arrival at our teaching hospital.

Abstract #20

EQUINE MOTOR NEURON DISEASE IN A HORSE FROM SPAIN—CASE REPORT. P. Montoliu, E. Vidal, D. Segura, S. Añor, L. Monreal, M. Pumarola. Department of Animal Medicine and Surgery, Veterinary School, Autonomous University of Barcelona. Bellaterra, Barcelona, Spain.

Abstract #21

EVALUATION OF CSF ANALYSIS AND PCR FOR THE DIAGNOSIS OF ENCEPHALITOZOONOSIS IN RABBITS. *A. Jass*, A. Fischer, H. Küchenhoff. Clinic of Veterinary Internal Medicine, Ludwig-Maximilians-University of Munich, Munich, Germany.

Symptoms of neurological disorder including vestibular disease and paresis/paralysis are a complaint, commonly presented in dwarf rabbits. Encephalitozoonosis is considered as the most common cause for these symptoms, with the main differential diagnosis being pasteurellosis, otitis media/interna and trauma. Other causes of infection as listeriosis, Borna disease and cerebral nematodiasis have also been reported in a view cases. In veterinary medicine definite diagnosis in most cases can only be made post mortem, as healthy rabbits may also be seropositive and excretion of the spores within the urine occurs discontinuously. On the other hand, while rabbits get more and more popular as pets *E. cuniculi* has also been proven to cause opportunistic infections in patients with AIDS and therefore is considered a potential zoonotic organism. Therefore it is of great importance to make a definite diagnosis, while considering prognosis, kind of treatment and potential risks for the owner.

CSF tap was performed in 20 healthy pet rabbits and 23 rabbits with neurological symptoms due to encephalitozoonosis. CSF tap was performed with the animal under general anaesthesia and the head flexed in a 90° angle to the spinal cord. All rabbits were intubated in order to enable easy ventilation without respiratory distress during CSF collection. PCR was performed following DNA extraction with a commercial kit from 200µl CSF and 200µl urine. For the first PCR, 1 µl each of 50µM solutions of the outer primers MSP-1 and MSP-2B, and for the second (nested) PCR, equal amounts and concentrations for the inner primers MSP-3 and MSP-4B were used. To verify PCR products as *E. cuniculi*, restriction enzyme Mnl I was used to generate RFLP patterns. *E. cuniculi* was identified with expected bands at 0,21 and 0,09bp (Katzwinkel-Wladarsch et al., 1996).

In healthy rabbits total nucleated cell count was 0–4/µl (1,7 +1,1) and protein was 0,13–0,31 g/l (0,23 +0,05). In contrast to this, CSF analysis of rabbits suffering from encephalitozoonosis revealed an increased nucleated cell count in 19/23 CSF samples (5–87/µl; 23,5 +23,7) and increased protein content in 23/23 CSF samples (0,31–1,54 g/l; 0,74 +0,35). Cytocentrifuge cell differentiation always showed distinct mononuclear pleocytosis with predominantly mono- and lymphocytes in rabbits with encephalitozoonosis. PCR-testing of CSF revealed positive results only in 2/21 CSF samples (9,5%), while for urine 16/44 samples tested positive (38,6%). Compared to trichrome stain with positive results only in 4 out of 44 urine samples (9%) that distinction is significant. All urine samples which revealed positive results by trichrome stain were also positive with PCR-testing.

It was concluded that mononuclear pleocytosis and elevated protein is a regular feature of rabbit encephalitozoonosis. PCR of urine may improve detection of the causative agent, but is useless in CSF.

Abstract #22

IS THIS NARCOLEPSY? *A. D'Angelo**, L.Bergamasco°, A. Bertuglia*, A. Tarducci*, G. Trucchi*, R. Zanatta* and A. Jaggy §. *Department of Animal Pathology and °Department of Veterinary Morphophysiology, University of Turin, Italy; §Department of Clinical Veterinary Medicine, Section of Animal Neurology, University of Bern, Switzerland.

Abstract #23

EXTENSIVE HEMILAMINECTOMY IN DISK EXTRUSION ASSOCIATED WITH EPIDURAL HEMORRHAGE: A RETROSPECTIVE STUDY IN 23 DOGS. *M. Baroni*, C. Tartarelli, M. Borghi. Clinica Veterinaria Valdinievole, Monsummano Terme, Pistoia, Italy

Abstract #24

BRAINSTEM AUDITORY-EVOKED POTENTIALS IN GREEN IGUANA (*IGUANA IGUANA*). *E. Bianchi*, F. Di Ianni, D. Callegari, L. De Risio, M. Dondi. Animal Health Department—University of Parma—Italy.

Brainstem auditory-evoked potentials (BAEPs) are electrical manifestations of the functioning of the auditory system used for neurologic and otologic investigations in humans and in other mammalian species. The purpose of this study was to assess BAEPs in adult green iguanas (*Iguana iguana*), determining normal tracings and normative values. In addition we tried to develop a clinically useful technique for performing this test in non-anesthetized subjects.

BAEPs were recorded from 10 awake healthy green iguanas of both sexes, ranging from 2 to 8 years of age. Room temperature was kept between 26 and 28° C. A 100 µs alternating acoustic click stimulus was delivered monaurally from a headphone transducer at a click rate of 10/second. The test was performed on animals restrained manually, by holding the headphone against the tympanic membrane being tested, and a sound absorption foam against the controlateral one. Recordings were taken from both sides in turn. They were amplified and averaged with an electrodiagnostic system, beginning with a stimulus intensity of 105 dB HL, and decreasing by 15 dB steps. Recording needle electrodes were placed in the subcutis, on the median plane rostral to the nuchal crest (positive electrode), on the ipsilateral dewlap just below the subtympanic shield (negative electrode), and on the neck (ground electrode).

BAEPs were recorded in every subject tested and showed a high degree of repeatability. Typical waveform was characterized by 4–8 positive peaks produced within the first 8 ms after the application of the stimulus, with 4 prominent peaks (P1, P2, P3, P4). Latencies of evaluated peaks are reported in table 1. Click hearing threshold was between 30 and 45 dB HL in all tested subjects.

Table 1. Mean absolute latencies (±SD) of positive peaks in left and right ear at a stimulus intensity of 105 dB HL.

Stimulated ear	P1	P2	P3	P4
Left	0.78 ms (±0.09)	1.61 ms (±0.20)	2.57 ms (±0.14)	3.40 ms (±0.19)
Right	0.76 ms (±0.10)	1.54 ms (±0.13)	2.49 ms (0.27)	3.44 ms (±0.26)

BAEPs of adult green iguanas are similar to those recorded in other species. Waveforms were reproducible in each iguana, with minor variability in latencies between subjects. The recording technique used in this study was easy to perform and produced clinically useful waveforms in awake iguanas. Despite largely different auditory system morphology and physiology in Iguanids, it is surprising to see how BAEPs overall aspect is similar to what is observed in mammals.

Abstract #25

COMPARATIVE EVALUATION OF CSF ANTI-CORONAVIRUS TITERS AND RESULTS OF POSTMORTEM EXAMINATION IN CATS. *I. C. Boettcher*[1], A. Fischer[1], T. Steinberg[1], K. Matiasek[2], H. Küchenhoff[3], C. E. Greene[4], K. Hartmann[1, 1]Clinic of Veterinary Internal Medicine, [2]Dept. of Pathology and Neuropathology, [3]Dept. of Statistics, Ludwig-Maximilians-University of Munich, Germany, [4]Dept. of Small Animal Medicine, University of Georgia, Athens, USA.

In the recent literature, determination of CSF coronavirus antibody titers in cats had been reported to be useful for the diagnosis of the neurologic form of Feline Infectious Peritonitis (FIP). The intention of the presented study was to critically re-evaluate previously published results in a large hospital-based patient base. The results of the postmortem examination of 67 cats were compared to the presence of anti-coronavirus-IgG-titers in the CSF.

Serum and CSF were taken from 67 cats before euthanasia for clinical reasons. IgG against coronaviruses was determined in CSF and serum using indirect immunofluorescence. All cats underwent gross and histopathologic examinations including the central nervous system (CNS). Ten cats were diagnosed with the neurologic form of FIP. In 13 other cases FIP typical lesions were found without involvement of the CNS. Twenty cats showed other CNS diseases than the neurologic form of FIP. The fourth group consisted of 24 cats that had neither FIP nor any other CNS disease.

Fifty-five of the 67 cats had no detectable antibodies against coronaviruses in the CSF (titer $< 1:32$). In 12 patients immunofluorescence for anti-coronavirus antibodies in the CSF was positive. Six of them were associated with the neurologic form of FIP, four with FIP without involvement of the CNS and two with other CNS diseases. CSF antibody titers ranged from 1:32 to 1:4096 with highest CSF titers in the neurologic form of FIP. However, no statistical differences concerning the titer range could be stated. All cats with anti-coronavirus titers in the CSF also revealed antibodies in the serum. These serum titers were in all cases within higher ranges from 1:4096 to 1:16384. There was a positive correlation of CSF titers with serum titers (spearman's rank test, $r = 0{,}655$, $P = 0{,}000$). Testing for anti-coronavirus titers in the CSF had a sensitivity of 60,0 % and a specificity of 89,5 % for the diagnosis of the neurologic form of FIP. The positive predictive value was 50,0 % and the negative predictive value 92,7 %.

We found antibodies against coronaviruses in the CSF to be a rare finding in cats. CSF titers were never present in seronegative cats while they correlated with serum titers in seropositive animals, so the local production of anti-coronavirus IgG in the CSF is questionable. The presented data suggest that determination of anti-coronavirus titers alone in CSF of cats does not confirm the diagnosis of the neurologic form of FIP. Future studies, however, should address to the value of specific antibody indexes.

Abstract #26

THE USE OF ISOELECTRIC FOCUSING TO DETECT OLIGOCLONAL IGG OF INTRATHECAL SYNTHESIS IN DOGS' CSF: PRELIMINARY RESULTS. *Callegari D.*[1], De Risio L.[1], Bianchi E.[1], Martelli P.[1], Cogato I. [2] 1) Animal Health Department, University of Parma, Italy, 2) Hospital of Fidenza, Parma, Italy

Isoelectric focusing (IEF) is a laboratory technique that separates proteic molecules based on their isoelectric point. In human medicine, IEF has been used to differentiate oligoclonal IgG of intrathecal synthesis (IS) from IgG of haematic derivation (HD) in CSF. The purpose of this study was to investigate the use of IEF in dogs with several neurological diseases.

Thirty-seven dogs have been included in the study. IEF has been performed on CSF and serum collected (at the same time) from each dog. Definitive diagnosis was reached by means of clinical and neurological examination, CBC, chemistry panel, urinalysis, serology, survey radiographs, CSF analysis, myelography, CT, histopathology.

Twenty-seven dogs (group I) had neurological diseases that do not determine local activation of the immune system: spinal cord compression (n = 20), fibrocartilaginous embolism (n = 1), cerebellar abiotrophy (n = 2), brain neoplasia (n = 3), idiopathic epilepsy (n = 1). The remaining 10 dogs (group II) had neurological diseases in which local activation of the immune system is possible: steroid responsive meningitis-arteritis (SRMA) (n = 2), granulomatous meningo-encephalitis (GME) (n = 1), viral meningo-encephalitis (VM) of unknown origin (n = 2), canine distemper virus (CDV) encephalitis (n = 1), probable degenerative myelopathy (DM) (n = 4). IEF showed no bands of IgG of IS in the CSF of all 27 dogs included in group I. IEF allowed to identify bands of IgG of IS in the CSF of the dog with GME, 1 of the 2 dogs with VM of unknown origin, the dog with CDV-encephalitis, and 2 of the 4 dogs with DM. In the CSF of the 2 dogs with SRMA IEF showed no bands of IS. These results seem to be in contrast with previous published data reporting an increased IgG Index in dogs with SRMA. This discrepancy could be explained hypothesising a generalised response of lymphocytes B with a polyclonal setting without oligoclonal bands. The 2 dogs with DM whose CSF showed bands of IS were both German shepherd. The 2 other dogs with DM were a Shetland sheepdog and a Caucasian sheepdog. These data on dogs with DM are opened to several speculations, as the etiopathogenesis of this disease is not well understood yet. The absence of IgG of IS in the CSF of 1 of the 2 dogs with VM could be related to the stage of the disease (CSF was normal, but pathology was strongly suggestive of VM).

IEF allowed to differentiate oligoclonal bands of IgG of IS from IgG of HD in all dogs included in our study. IEF results in dogs included in group I and in most dogs included in group II are consistent with the pathogenesis of these diseases. Further research is needed on the use of IEF in those neurological diseases whose etiopathogenesis remains unknown.

Abstract #27

SONOGRAPHIC EXAMINATION OF THE ISCHIADIC NERVE IN THE DOG. *A. Fischer*[1], S. Reese[2]. [1]Clinic of Veterinary Internal Medicine and [2]Department of Anatomy, Histology and Embryology, Ludwig-Maximilians-University of Munich, Germany.

Focal peripheral mononeuropathy may be due to peripheral nerve trauma (compression, transection), local infection (abscess) or neoplasia. Electrodiagnostic techniques are commonly used to confirm a clinical diagnosis of peripheral mononeuropathy and to establish the level of the lesion roughly. While electrodiagostic testing also provides prognostic information there is no information about the underlying cause. Sonography has the advantage of visualizing nerve and adjacent tissues. In the dog, sonography has been used for the diagnosis of peripheral nerve sheath tumors of the vagus nerve (Ruppert et al., 2000) and brachial plexus (Platt et al., 1999) and the normal sonographic anatomy of the vagus nerve has also been described (Reese and Ruppert, 2001). The intention of the following study was to describe the sonographic anatomy of the ischiadic nerve in the dog.

The ischiadic nerve was examined sonographically in both pelvic limbs of 29 dogs of various sizes. A Siemens Sonoline Elegra using a 9 MHz linear ultrasound transducer with a contact area of 4 x 1 cm was used. The nerve was visualized at three locations: (1) proximal just distal to the trochanter, and (2) halfway between the trochanter and the stifle, and (3) at the level of the division into tibial and peroneal nerve. The nerve was examined in a transverse and a longitudinal section at each location.

The nerve could be visualized at each location in all dogs. Longitudinally the nerve was characterized by two hyperechogenic linear structures with a median plane with the same or slightly less echogenicity than the muscle. The hyperechogenic linear structures were thought to reflect fat and connective tissue of the perineurium. The less echogenic median plane was thought to reflect bundles of nerve fibers and their respective nerve sheaths with associated endoneurium and epineurium. Transverse sections showed two ring-like structures in close association corresponding to the tibial and peroneal sections of the ischiatic nerve. Again, the hyperechogenic ring contained a less echogenic center.

Sonography proved to be useful to visualize the ischiadic nerve and surrounding muscles at the level of the femur in small and large breed dogs. In the future, sonography may be useful to further characterize traumatic, neoplastic or inflammatory lesions of the ischiadic nerve.

Abstract #28

A CASE OF POSSIBLE ACTH INDUCED TYPE 2 FIBER STROPHY. *G.B.Cherubini*[1], R.Cappello[1], G.D.Shelton[2]. The Queen Mother Hospital for Animals, the Royal Veterinary College, London UK[1]; Department of Pathology, University of California, San Diego, USA[2]

A 4 year old female spayed chocolate Labrador retriever was referred with a 6–8 month history of progressive panting, ataxia and weakness. The neurological examination was consistent with a LMN disorder. Routine laboratory evaluations including CBC, serum chemistries, T4, TSH, antibody titers for *Toxoplasma gondii* and *Neospora caninum*, and CSF analysis were unremarkable. Resting cortisol and cortisol post-ACTH were elevated. A LDDT did not support a diagnosis of Cushing's syndrome. ACTH concentration was markedly elevated at 278 (20–80 pmol/l). No abnormalities were detected on further investigations including abdominal ultrasound (adrenal glands within normal limits), brain MRI, electromyographic examination or measurement of motor nerve conduction velocity.

Generalized type 2 fiber atrophy was present in fresh frozen muscle biopsy sections. A mild depletion of large myelinated fibers and subperineurial edema was identified in resin embedded sections of the peroneal nerve. Unfortunately the dog developed complications following general anesthesia and expired. The owner declined a necropsy.

As either hyperadrenocorticism or hypothyroidism could explain the observed type 2 fiber atrophy, our hypothesis is that the myopathic effects in this case may be secondary to the increased blood ACTH concentration. Myopathy has been confirmed in humans after adrenalectomy. In addition, elevated blood concentrations of ACTH may impair neuromuscular transmission by decreasing the quantal content of the end-plate potential. Based on our findings, further evaluation of ACTH concentrations in cases of type 2 fiber atrophy with weakness is warranted.

Abstract #29

CELL PROLIFERATION MARKERS IN NEUROONCOLOGY: PROLIFERATING CELL NUCLEAR ANTIGEN (PCNA) AND KI-67 LABELING INDICES IN INTRACRANIAL MENINGIOMAS OF THE DOG. D. Janik[1], B. Stierstorfer, *K. Matiasek*, W. Schmahl. [1]Institute of Veterinary Pathology, Chair of Pathology and Neuropathology, Ludwig-Maximilians-University, Munich, Germany.

Abstract #30

TOMACULOUS ALTERATIONS IN PERIPHERAL NERVES OF DOMESTIC ANIMALS. *Kuhne-Velte SM*, Matiasek K, Schmahl W. Institute of Veterinary Pathology, Chair of General Pathology & Neuropathology, LMU Munich, Germany.

In order to assess their frequency and relevance in diseased nerves of companion animals a pool of nerve probes had been re-screened for presence of tomaculous myelin sheath alterations in histological and teased-fiber specimen.

Sausage-like swellings of the myelin sheath, so-called tomacula, are a feature of demyelinating nerve disease. Soon after their first description in human nerves as globules in 1968 their occurrence could be correlated with an increased susceptibility of peripheral nerves to mechanical damage. Up to now, different authors recognized tomacula in several sub-primate species without a proven pathogenetic link. Most authors considered them to be a result of genetic or immunologic perturbations while some cases had been associated with paraneoplastic neuropathy.

In the present study we found tomacula in peripheral nerves of 15 animals out of a group of 116 individuals with severe systemic disease. Among affected animals 8 suffered from non-neurogenic neoplastic disease, 4 from neoplasia plus endocrine disturbance, and 3 from peripheral neuropathy. In all but one case, peripheral nerve alterations were marked to severe while in 7 of these tomacula contributed just a little to the whole pathological picture. In general, they were not restricted to demyelinating neuropathy but occurred in 1 case with axonal disease and in 6 cases with mixed patterns. Thereby, tomacula had been accompanied by several additional findings: in demyelinating and mixed situations, nerve fibers consistently revealed paranodal and/or segmetal de- and remyelination. Moreover, 9 samples showed multifocal myelin clefts and cystic

myelin sheath edema. Exaggerant myelin sheath proliferation—other than tomaculous—could be assessed in 6 cases whereas myelin breakdown was found in 7 nerves.

Despite a lack of reports about tomacula in veterinary neuropathology they can be considered as a rather frequent finding in demyelinating and mixed neuropathies. Since there is a coincidence with other signs indicative for myelin instability, compaction of myelin sheath may be the primary target even in acquired neuropathies with tomacula formation. In axonal disorders loss of axon-derived factors essential for maintenance of myelin sheath integrity could explain the development of these redundant and disoriented figures. To further address the question of dyscompaction, perturbations of myelin sheath proteins have to be investigated at the molecular level.

Abstract #31

LACTATE AND PYRUVATE LEVELS IN BLOOD AND CEREBROSPINAL FLUID IN DOGS. *V. Löbert*, R. Mischke and A. Tipold. Dept. Small Animal Medicine and Surgery, School of Veterinary Medicine, Hannover, Germany.

In dogs the main clinical manifestations of mitochondriopathies are myopathies and in some breeds encephalopathies, which are accompanied by necrosis or calcification. Lactate acidosis can frequently be diagnosed in mitochondriopathies because of a disturbed oxygen metabolism accompanied by an increased consumption of pyruvate in the Krebs cycle. The purpose of the present study was to determine reference values for pyruvate, lactate and the pyruvate/lactate ratio in the blood. In addition these values were measured also in the blood and CSF of dogs with defined diseases of the central nervous system. 104 dogs with different diseases of the central nervous system (CNS), 20 neurologically healthy dogs and 6 dogs with exercise induced weakness were included. Parameters such as the number of leukocytes and the content of glucose in blood and CSF as well as blood gas analysis from venous blood were correlated with lactate and pyruvate levels. Pyruvate levels had a high variation. The time point to remove proteins from blood samples is of importance for pyruvate measurement. A delay of 5 minutes reduces statistically significantly the concentration of pyruvate (median 0.047 mmol/l). Freezing of blood samples is not to recommend, since pyruvate- and lactate concentrations drop distinctly. However, CSF samples can be kept frozen at −20°C. Pyruvate and lactate levels measured before or during anaesthesia did not differ significantly. The high variation of pyruvate values could not be explained by influence of the total number of leukocytes and glucose in blood and CSF, of blood gas analysis or the age and weight of the animals. Different CNS diseases did not differ statistically significantly, but differed significantly from neurologically healthy dogs. Neurologically healthy dogs and patients with idiopathic epilepsy displayed nearly the same values. The highest lactate concentrations were measured in dogs with disc protrusion and inflammatory CNS diseases. In summary, only pyruvate concentrations higher than 0,42 mmol/l in blood samples and higher than 0,4 mmol/l in the CSF, lactate concentrations higher than 9.5 mmol/l in blood samples and higher than 3.7 mmol/l in the CSF are suspicious for a mitochondriopathy.

Abstract #32

ELECTRONEUROGRAPHIC EXAMINATION OF THE FACIAL NERVE IN DOGS AND CATS. S. Lobo-Roth, *A. Fischer*. Clinic of Veterinary Internal Medicine, Ludwig-Maximilians-University of Munich, Germany.

Facial nerve paresis in the dog can either occur as an idiopathic syndrome or secondary to otitis media/interna, peripheral nerve compression, brainstem disease or be a feature of a polyneuropathy. Bilateral facial nerve weakness is also a common feature of focal and generalized myasthenia gravis in the dog. It is our experience that many cases of acute facial paresis in the dog represent an idiopathic syndrome similar to acute idiopathic facial paresis also called Bell's palsy in humans. In previous reports electrodiagnostic examination of the facial nerve has been confined to the recording of spontaneous electric activity. Only recently a technique for recording of the blink reflex was described (Anor et al, 2000). The intention of this investigation was to establish a technique for electroneurographic examination of the facial nerve in dogs and cats.

The facial nerve was stimulated below the base of the ear at the level of the stylomastoid foramen with two monopolar needle electrodes. The compound muscle action potential (CMAP) was recorded from the M. orbicularis oris with a concentric needle electrode. Latency, duration, amplitude and area of the CMAP were measured. In a second part of the study, a method for repetitive stimulation of the facial nerve was established. Amplitudes and areas of the negative components of the 3rd, 5th and 10th CMAP were measured and expressed as a percent decrement of the 1st CMAP. 33 nerves from 17 neurologically healthy dogs and ten nerves from five healthy cats provided reference values for the CMAP evoked by direct facial nerve stimulation. Reference values for repetitive stimulation (RNS) of the facial nerve were calculated from 22 nerves from 12 dogs and six nerves from four cats. Subsequently, electrical stimulation of the facial nerve was done in neurologically diseased dogs with and without facial nerve paresis.

The CMAP presented as a stable mono- or biphasic potential with a predominant negative phase. In the dog, latency was 4.8±1.0 msec, duration 2.9±1.0 msec, amplitude 1.1±0.7 mV, and area 1.5±0.8 mVmsec. In the cat, latency was 1.7±0,2 msec, duration 2.1±0.4 msec, amplitude 1.5±0.7 mV, and area 1.5±0.6 mVmsec. Latency and duration increased and amplitude decreased with increasing distance between stimulating and recording electrodes. In dogs and cats, RNS of the facial nerve could be done with a minimal decrement (< 10 %) of amplitude and area.

The results of this study provide reference values for the electrodiagnostic evaluation of the facial nerve. In future these technique could prove useful to further characterize idiopathic facial nerve paresis and focal myasthenia gravis in dogs and cats.

Abstract #33

IMPAIRED REMYELINATION AND DEPLETION OF OLIGODENDROCYTE PROGENITORS DOES NOT OCCUR FOLLOWING REPEATED EPISODES OF FOCAL DEMYELINATION IN THE RAT CENTRAL NERVOUS SYSTEM. *Penderis J*[1,2], Shields SA[1], Franklin RJM[1] [1]Cambridge Centre for Brain Repair and Department of Clinical Veterinary Medicine, University of Cambridge, Cambridge, UK. [2]Animal Health Trust, Newmarket, UK.

It has been hypothesized that the progressive failure of remyelination in chronic multiple sclerosis is, in part, the consequence of repeated episodes of demyelination at the same site, eventually depleting oligodendrocyte progenitor cells (OPCs) and exhausting the remyelinating capacity. We investigated the effect of previous focal, ethidium bromide-induced demyelination of brain stem white matter (with intervening recovery) on the efficiency of the remyelination process during second and third subsequent episodes of demyelination, and the OPC response during a second episode of demyelination. Previous focal demyelinating lesions followed by recovery did not result in any retardation of the remyelination process, nor did they alter the proportion of Schwann cell versus oligodendrocyte remyelination. The OPC response during remyelination was quantified by in situ hybridization using a probe to platelet-derived growth factor-alpha receptor (PDGFαR), an OPC-expressed mRNA. Following recovery from focal, toxin-induced CNS demyelination, the OPC density returned to levels equivalent to those in normal white matter. Furthermore, there was no depletion of OPCs following repeated episodes of focal, toxin-induced CNS demyelination at the same site. These results indicate that repeated CNS demyelination, which has the opportunity to repair in the intervening period, is not characterized by impaired remyelination or depletion of OPCs.

Abstract #34

COMPARATIVE EVALUATION OF CSF PROTEIN, IGG INDEX AND ALBUMIN QUOTIENT IN DOGS WITH NEUROLOGIC DISEASE. *T. Schindler*, A. Fischer. Clinic of Veterinary Internal Medicine, Ludwig-Maximilians-University of Munich, Germany

The intention of the following study was to explore the hypothesis that IgG index (IgGind) and albumin quotient (AQ) would be a useful extension of routine protein analysis of the cerebrospinal fluid. The IgG index serves as an indicator for intrathecal IgG synthesis, the albumin quotient as an indicator for disturbances in the blood-brain barrier.

122 dogs were categorized into five groups: inflammatory disease (n = 33), tumor (n = 11), disc prolaps (n = 37), orthopedic disease (n = 20), and idiopathic epilepsy (n = 27). In addition to conventional CSF analysis which included quantitative protein measurement, the concentrations of albumin and IgG in CSF and serum were determined by a nephelometric method. AQ and IgGind were calculated for each dog. Reference values for AQ (< 3,5 x 10^{-3}) and IgGind (< 0,7) were obtained from 20 laboratory beagle dogs.

In 10% of the examined dogs, CSF pathology was only exposed by the IgGind or the AQ, with the CSF protein content being normal (< 0,3 g/L). This result was equivalent to 14% of the dogs with normal CSF protein content. Among these were dogs with white dog shaker disease (n = 3), distemper and choroid plexus carcinoma. On the other side, dogs with increased CSF total protein (n = 35) had a 97% chance of increased IgGind or AQ. 67% of the dogs with inflammatory diseases showed an increase in IgGind and 70% an increase in AQ. In 46% of these animals both parameters were elevated. Maximum changes were observed in dogs with central European tick-borne encephalitis. Dogs with white dog shaker disease were the only ones to exhibit an exclusive increase in the IgGind. 36% of the dogs with brain tumors showed an increase in IgGind and 64% an increase in AQ. In 18% of the animals both parameters were elevated. The highest level of all IgGind was obtained in a patient with an ependymoma. Patients with disc prolapse showed a mild increase in IgGind in 8% of the cases and a mild increase in AQ in 14% but never a combination of both. As expected, no changes occurred with idiopathic epilepsy.

It was concluded that in dogs with neurological symptoms without increased levels of protein, IgGind and AQ can help to identify CNS pathology. However, IgGind and AQ do not help to differentiate between inflammatory and neoplastic disease in general. Nevertheless, in the future the determination of IgG index and albumin quotient may be an useful extension of routine CSF protein analysis to identify or exclude structural CNS disease.

Abstract #35

NITRIC OXIDE PRODUCTION IN THE CANINE BRAIN—ARE MICROGLIAL CELLS INVOLVED? *V. Stein*, R. Carlson and A. Tipold. Dept. Small Animal Medicine and Surgery, School of Veterinary Medicine of Hannover, Germany.

Microglial cells are the major immune effector cells of the central nervous system (CNS) and respond to every kind of pathological event. Their role in host defense against invading microorganisms and neoplastic cells is crucial. Upon activation microglia and macrophages share most phenotypical markers and can exert similar effector functions such as phagocytosis, modulation of T cell response and production of reactive oxygen species (ROS). ROS production of canine microglia was enhanced in demyelinating lesions due to canine distemper virus infection as shown in earlier studies. Nitric oxide (NO) is one intermediate of ROS and was shown to be increased in listeric encephalitis by immunohistochemistry. Whether intrathecal NO production can also be shown in dogs and is due to microglial activation remains to be elucidated. Therefore, the aim of this study was to measure levels of NO in microglial culture supernatant and CSF in dogs with different kinds of intracranial diseases compared to dogs with normal CNS.

24 dogs euthanised on owners request suffering from different intra- and extracranial diseases were examined. CSF from each dog was collected during anaesthesia and kept frozen. Canine microglial cells were isolated by density gradient centrifugation protocol and cultured in RPMI 1640-medium with CPSR-1 (5%), 10 mmol HEPES and antibiotics. After cultivation in 5% CO_2 at 37°C and a humid atmosphere for 24 h, culture supernatant was collected. Supernatant, negative controls and CSF of each dog were examined by Griess-reaction.

In 7 dogs supernatant of cultivated microglial cells showed enhanced NO levels in comparison to pure medium cultivated under the same conditions. 4 of these dogs had seizures. Otherwise the NO production could not be correlated with a specific CNS disease. There was no correlation with values of CSF samples. Our results point to a contributing role of microglial cells in NO-production in the dog.

Abstract #36

APPROACHING MOTOR UNITS IN THE RAT *Wieczorek L*[1], Matiasek K[2], Adriany E[3], Fischer A[1]; [1]Clinics of Veterinary Internal Medicine and [3]Surgery, [2]Institute of Veterinary Pathology & Neuropathology, LMU Munich, Germany

In experimental trials concerning peripheral nerve injury and regeneration laboratory rats are the preferred animal model. In order to improve techniques for evaluation of motor functions in this species and to provide a reliable monitoring of motor nerve restoration after experimental trauma, 10 healthy Wistar rats underwent clinical and electrodiagnostic inspection. Clinical investigation consisted of ankle kinematics and foot print analysis. As an indicator for gastrocnemius-soleus function tibiometatarsal angles were measured during different phases of the gait by a 80 Hz SIMI®-high speed digicam/software system. In all healthy rats the angle ranged between 20° and 115° according to the different stance and swing phases. In contrast to former studies all sequences could be well documented using only three landmarks: 1. the distal third of the tibia, 3. the distal edge of the fifth metatarsal bone, and 3. the lateral malleolus as rotation point. Gait analysis was accomplished by the sciatic function index (SFI), modified by Bain-Mackinnon-Hunter (Bain et al., 1989). It is the best parameter for assessment of multisystemic functional compensation of unilateral motor deficits. Since our rats were unaffected and revealed similar gait patterns at both sides, SFI was about 0 ± 2. Protocols from previous studies could be improved by replacing the walking tracks by a treadmill with a constant velocity of 0.16 m per second. Thereby, cyclic movements could be recorded continuously without acceleration/ deceleration errors. Electrodiagnostic testing was performed under general anesthesia (medetomidine, midazolam, fentanyl) with a Nicolet IV®. Electromyography of all muscle groups of the hind limbs was inconspicuous. Nerve conduction velocity was measured from the tibial nerve with stimulating electrodes placed at the level of the trochanter and proximolateral to the tarsal joint. In all rats conduction velocity after proximal stimulation was greater 45 m/s (mean 55 ms; SD = 8). Duration of the M-wave was 1.2 ms at the tarsus and 1.4 ms at the trochanter region as maximum. The amplitudes varied tremendously among individuals, thereby, precluding assessment of a standard value. Independent of stimulation intensity each M-wave was followed by an H-reflex, which inconsistently was superimposed by F-waves. The latter easily could be identified by their polymorphism. Mean onset latency of late responses was 7,2 (SD = 0,49). We were able to demonstrate that H-reflexes were less contaminated by F-waves at low stimulation intensities applied at a rate of 1 per 5 seconds. A slight increase of stimulation intensity heightened H-reflex amplitude but also F-wave contamination. With raising stimulation intensities F-waves became more prominent while H-reflexes remained unaffected but above a individual threshold between 12 and 20 mA H-reflexes began to disappear. In conclusion, functional recovery of sciatic nerve in rats can be readily assessed and quantified by gait analysis and electroneuro/myography if species- and strain-specific variations are taken into account.

Abstract #37

NEUROPATHIC LESIONS IN CUSHING'S SYNDROME. *Wolff B*[1], Matiasek K[1], Kopp A[2], Fischer A[2], Schmahl W[1]. [1]Institute of Veterinary Pathology, Chair of General Pathology & Neuropathology and [2]Clinic of Veterinary Internal Medicine, Ludwig-Maximilians-University, Munich, Germany.

The purpose of the study was to elucidate whether there is a neuropathy or at least a neuropathic component contributing to neuromuscular changes in patients with hyperadrenocorticism in veterinary medicine.

Peripheral nerve samples of 11 dogs, 1 cat and 1 horse with spontaneous Cushing's syndrome were examined using histology, electronmicroscopy and teased fiber techniques. In 7 dogs muscle samples were examined as well. In all patients but the horse adrenocortical neoplasia was diagnosed while one dog had a basophilic pituitary gland adenoma as well. The latter holds true also for the equine patient. 9 patients suffered from additional diseases with possible effects on the peripheral nervous system. 3 of these patients had a history of hyperglycemia, 2 patients were non-hyperglycemic and for the others glycemic state was not assessed.

We found features of peripheral neuropathy in 12 of the 13 patients. Demyelinating neuropathy was seen in 7 patients, axonal neuropathy could be assessed in 3 animals and mixed forms in 2 patients. The degree of the neuropathy was mild for the axonal types but at least moderate up to severe for the mixed and demyelinating forms. We were unable to detect typical features of diabetic neuropathy rendering the possibility that the neuropathy in these patients is caused by diabetic conditions rather unlikely. Within the muscle samples examined, there was only one canine case of steroid-myopathy. The other probes revealed either signs of neurogenic muscular atrophy (3 cases) or no pathologic lesion at all (3 cases) leading to the conclusion that the neuropathies are not secondary to a primary myopathy.

From the results of this study it was concluded that a steroid-neuropathy exists besides the already known steroid-myopathy and that the type of the neuropathy is predominantly demyelinating. This finding is consistent with results of a previous study (J Vet Int Med 17(2), p 254), which demonstrated decreased motor nerve conduction velocities in dogs with hyperadrenocorticism compared to healthy age-matched controls.

Abstract #38

ENDOSCOPIC BRAIN SURGERY. *Caemaert J.* Department of Neurosurgery, Faculty of Medicine, Ghent University Hospital, Ghent, Belgium.

Abstract #39

SURGICAL TREATMENT OF IDIOPATHIC EPILEPSY IN HUMANS. *Caemaert* J. Department of Neurosurgery, Faculty of Medicine, Ghent University Hospital, Ghent, Belgium.