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PROCEEDINGS 26th Symposium ESVN-ECVN

PARIS, FRANCE
26th–28th September 2013
Selected research communications of the 26th Symposium of the ESVN-ECVN
Paris, France 26th to 28th September 2013

TIMETABLE OF THE SYMPOSIUM

FRIDAY 27TH SEPTEMBER

08.00: **Registration**

NEURO-EMERGENCY AND CRITICAL CARE

08.30: **Welcome to the meeting**

08.45: **INVITED SPEAKERS SESSION**

Professor O. Dulac

"STATUS EPILEPTICUS IN THE EMERGENCY ROOM"

09.30: Dr. P. Meyer

"HEAD TRAUMA MANAGEMENT"

10.15: Coffee break, exhibition and poster session

10.45: **FREE COMMUNICATIONS RESEARCH ABSTRACTS**

1) SIGNALMENT, CLINICAL SIGNS AND TREATMENT OF ATLANTOAXIAL SUBLUXATION IN DOGS: A SYSTEMATIC REVIEW OF THE 336 PUBLISHED CASES FROM 1967 TO 201

IN Plessas, HA Volk

2) DECOMPRESSIVE CRANIECTOMY FOR INCREASED INTRACRANIAL PRESSURE AFTER REMOVAL OF THE BRAIN TUMOUR IN TWO CATS

N Tanaka, M Kitagawa, D Ito, S Ohta, K Ono, H Yamada, T Watari

3) TWO CASES OF VENTRAL BRAINSTEM MENINGIOMA RESECTION BY TRANSBASIOCCIPITAL APPROACH THROUGH THE ORAL CAVITY

A Ijiri, K Yosiki, S Tsuboi1, A Sibahasi, T Sugie, H Akiyoshi, F Ohashi, T Nakade, ? Atsuki

4) CORPUS CALLOSAL ABNORMALITIES IN THE DOG

R Gonçalves, HA Volk, PM Smith, J Penderis, L Garosi, E MacKillop, A de Stefani, JF McConnell

5) VALIDATION OF A STEREOTACTIC ACCESS TO THE BRAINSTEM IN ANIMALS USING AN OVINE CADAVER HEAD MODEL

A Staudacher, A Oevermann, M Stoffel, D Gorgas

6) SURGICAL TREATMENT OF A CEREBELLOPONTINE EPENDYMAL CYST IN A DOG

MK Müller, N Bauer, E Ludewig, K Matiasek, G Oechtering, M Preuß, T Flegel

7) CERVICAL DISC ARTHROPLASTY USING THE ADAMO SPINAL DISC IN 30 DOGS AFFECTED BY DISC-ASSOCIATED WOBBLER SYNDROME AT SINGLE AND MULTIPLE LEVELS

PF Adamo, RC da Costa, R Kroll, C Giovannella, M Podell, P Brofman

12.30: Lunch exhibition and poster session (Posters attended by authors)

14.00: INVITED SPEAKERS SESSION

Dr. Simon Platt

"A NEW CLINICAL EVALUATION SYSTEM FOR HEAD TRAUMA"

L Clark

"NEW USES FOR OLD DRUGS - RE-INVENTING THE PHARMACOLOGY WHEEL"

15.30: Coffee break, exhibition and poster session

16.00: FREE COMMUNICATIONS RESEARCH ABSTRACTS

8) OUTCOME OF 96 DOGS TREATED MEDICALLY OR SURGICALLY FOR SPINAL ARACHNOID DIVERTICULA.

D Mauler, S De Decker, L De Risio, HA Volk, R Dennis, I Gielen, E Van der Vekens, K Goethals, L Van Ham

9) UNUSUAL 'PRAYING POSITION'-LIKE STANCE AND GAIT AS CLINICAL MANIFESTATION OF CAUDAL LUMBAR DISC DISEASE IN A DOG AND A CAT.

I Espadas, O Chai, S Lindley, A Wessmann

10) RECORDING OF CORD DORSUM POTENTIALS IN CALVES.

I Van Soens, C De Vlamynck, F Pille, L Vlaminck, L Van Ham

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

18:00: Closing of the day

SATURDAY 28TH SEPTEMBER

08.00: Registration

08.30: FREE COMMUNICATIONS RESIDENT RESEARCH ABSTRACTS

11) THE INFLUENCE OF ACUTE VAGUS NERVE STIMULATION ON THE PTZ SEIZURE THRESHOLD IN DOGS.

V Martlé, R Raedt, T Waelbers, L Van Ham, K Peremans, K Vonck, P Boon, L Duchateau, S Bhatti

12) MRI TRACTOGRAPHY OF THE MESOCEPHALIC CANINE BRAIN AND THE DEVELOPMENT OF A WHITE MATTER ATLAS.

R Mineo, B Moffat, S Long, M Le Chevoir

13) THIAMINE DEFICIENCY IN CATS: A RECENT OUTBREAK IN TAIWAN.

YP Chang, PY Chiu, CC Wu, CH Liu

14) EFFECT OF RADIOTHERAPY ON SEIZURE FREEDOM IN DOGS WITH BRAIN TUMOURS.

S Monforte-Monteiro, J Rossmeisl, J Russell, MA Holmes, A Wessmann, AE Vanhaesebrouck

15) RISK FACTORS ASSOCIATED WITH THE DEVELOPMENT OF SEIZURES IN DOGS WITH HEPATIC ENCEPHALOPATHY.

C Ricco, M Tivers, HA Volk

16) NON-CONVULSIVE STATUS EPILEPTICUS IN DOGS.

C Mariani

17) DEXMEDETOMIDINE IN THE MANAGEMENT OF STATUS EPILEPTICUS AND TREMORIGENIC MYCOTOXICOSIS.

C Rusbridge, V Rubasinska, S Griffiths, J Aguiar, A Foucault, J Uhrig, J Griffiths, R Elliot, A Bradnock, S Rodendas, R Bralow

10.15: Coffee break, exhibition and poster session

10.45: Dr. Simon Platt

"DELIVERY OPTIONS FOR BENZODIAZEPINS IN STATUS EPILEPTICUS"

11.30: FREE COMMUNICATIONS RESEARCH ABSTRACTS

18) RISK FACTORS AND OUTCOMES IN FELINE ACQUIRED MYASTHENIA GRAVIS (2001-2012).

DW Hague, HD Humphries, MA Mitchell, GD Shelton

19) CLINICAL, PATHOLOGICAL AND MOLECULAR CHARACTERISTICS OF INHERITED CEREBELLAR ATAXIA IN ITALIAN SPINONE DOGS.

L De Risio, O Forman, S Platt, C Mellersh, K Matiasek

20) ACQUIRED EQUINE POLYNEUROPATHY: A UNIFORM ENTITY?

S Hanche-Olsen, K Hultin Jäderlund, J Molin, G Gröndahl, K Matiasek

21) ACCURACY OF THE NEUROLOCALIATION IN COMPARISON TO FINDINGS IN FURTHER DIAGNOSTICS IN 214 DOGS: A PROSPECTIVE STUDY.

C Loeffler, K Truar, MK Mueller, IC Boettcher, G Oechtering, T Flegel

12.30: Lunch, exhibition and poster session (Posters attended by authors)

14.00: INVITED SPEAKERS SESSION

Daniel Chan

"FLUID BALANCE AND NUTRITIONAL SUPPORT OF THE CRITICAL NEUROLOGICAL PATIENT"

14.45: FREE COMMUNICATIONS RESEARCH ABSTRACTS

22) PROGNOSTIC VALUE OF MAGNETIC RESONANCE IMAGING IN DOGS AFTER TRAUMATIC BRAIN INJURY: 50 CASES.

E Beltran, SR Platt, F McConnell, R Dennis, D Keys, L De Risio

23) SURVIVAL AND FUNCTIONAL CAPACITY IN 32 DOGS WITH TRAUMATIC BRAIN INJURY: A PROSPECTIVE STUDY.

MV Bahr Arias, CG Vianna

24) OCCURRENCE OF HYPOTHALAMIC-ANTERIOR PITUITARY HORMONE DEFICIENCIES FOLLOWING HEAD TRAUMA IN DOGS.

K Murtagh, L Arrol, R Goncalves, A German, PM Smith

15.30: Coffee break, exhibition and poster session

16.00: FREE COMMUNICATIONS RESEARCH ABSTRACTS

25) IONIZING RADIATION FOR THE TREATMENT OF CANINE MENINGOENCEPHALITIS OF UNKNOWN ORIGIN: AN OBSERVATIONAL PILOT STUDY.

K Beckmann, I Carrera, F Steffen, P Kircher, U Schneider, V Meier, C Rohrer Bley

26) SOLE PREDNISOLONE THERAPY IN CANINE MENINGOENCEPHALITIS OF UNKNOWN ORIGIN: 45 CASES (2006-2012).

I Cornelis, L Van Ham, K Kromhout, K Goethals, I Gielen, S Bhatti

27) A RETROSPECTIVE STUDY OF 225 CASES OF IDIOPATHIC HEAD TREMOR IN DOGS.

L Shell, J Berezowski, B Nibblett, P Kelly, M Rishniw

28) IS ROUTINE ADMINISTRATION OF MRI CONTRAST MEDIA REQUIRED FOR EXCLUSION OF A BRAIN LESION IN DOGS AND CATS?

EJ Ives, N Rousset, N Heliczer, AE Vanhaesebrouck

17.00: John Presthus and Boehringer Ingelheim Awards

17.30: Closing remarks

ORAL PRESENTATIONS

SIGNALMENT, CLINICAL SIGNS AND TREATMENT OF ATLANTOAXIAL SUBLUXATION IN DOGS: A SYSTEMATIC REVIEW OF 336 PUBLISHED CASES FROM 1967 TO 2013.

I.N. Plessas, H.A. Volk, Department of Clinical Science and Services, The Royal Veterinary College, London, UK

Atlantoaxial subluxation (AAS) is a common condition in small breed dogs and a plethora of case series have been reported since 1967. The aim of this review was to analyse the signalment, clinical signs and treatment outcomes of the currently 336 reported cases.

Fifty-seven publications were reviewed from 1967 to 2013. Thirty-eight breeds have been reported with AAS, amongst them Yorkshire Terriers (28%), Toy Poodles (17%) and Chihuahuas (15%) were the most common. 127 (48%) dogs were male (10 neutered) and 136 (52%) female (24 neutered). Mean weight was 3.6 kg and mean age at onset of clinical signs 21.3 months. Clinical signs were assessed using the following former reported scale: (5) normal gait; (4) ataxia; (3) ambulatory paresis; (2) non-ambulatory paresis; and (1) tetraplegia. The mean score of clinical signs before treatment was 2.4 for those treated conservatively, 3 for those treated with ventral surgical techniques and 2.8 for those treated with dorsal techniques. Radiography revealed that odontoid process was aplastic in 24% and hypoplastic in 32% of affected dogs.

Thirty-nine dogs were treated conservatively and 284 underwent surgical stabilisation with various ventral (201) and dorsal (83) surgical techniques. From the dogs treated conservatively, 28 (72%) recovered successfully (grade 4-5). Successful outcome was more frequently reported with ventral (166 (82%)) compared to dorsal (54 (65%), $P = 0.0013$) stabilisation techniques. Fatality rate associated with surgical treatment was 5% with ventral and 8% with dorsal techniques. The mean score of clinical signs 1 year after treatment was 4.2, 4.8 and 4.5 for those treated conservatively, surgically with ventral or dorsal technique, respectively.

AAS is mainly reported in young toy breed dogs, odontoid process pathology plays an important role and ventral surgical techniques may have a more favourable outcome compared to other treatment options.

DECOMPRESSIVE CRANIECTOMY FOR INCREASED INTRACRANIAL PRESSURE AFTER REMOVAL OF THE BRAIN TUMOUR IN TWO CATS.

N. Tanaka[1,2], M. Kitagawa[1], D. Ito[1], S. Ohta[1], K. Ono[1], H. Yamada[1], T. Watari[1], [1]School of Veterinary Medicine, Nihon University, Kameino, Fujisawa, Kanagawa, Japan, [2]Grace Animal Hospital, Ogikubo, Suginamiku, Tokyo, Japan.

Intracranial pressure (ICP) is sometimes increased by brain hemorrhage and edema after extraction of a brain tumour. Therapies for elevated ICP include steroid administration, hyperosmolar therapy, and hyperventilation as medical treatments, and decompressive craniectomy and dural incision as surgical treatments. We report surgical treatment for increased ICP and changes in ICP in two cats showing elevated ICP after brain tumour removal.

Case 1: Brain tumour in the frontal lobe was identified on magnetic resonance imaging (MRI) (1.5-T) and removed surgically. Intracranial pressure sensor was monitored intermittently before brain tumour extraction. Intracranial pressure was 6 mmHg before craniotomy and 10 mmHg at the end of surgery, but increased rapidly to 35 mmHg by 4 h postoperatively. Spontaneous breathing was lost and cerebellar herniation was identified on MRI; therefore, we selected surgical decompression with right lateral rostrotentorial craniotomy. Intracranial pressure was 15 mmHg when the temporal bone was removed and 19 mmHg when the temporal muscle fascia was sutured. Intracranial pressure after this second operation increased to 40 mmHg, and decreased and remained at 10-20 mmHg. The cat died due to ventilatory insufficiency five days after completion of the second surgery. The histopathological diagnosis was lymphoma.

Case 2: Brain tumour located from the right parietal lobe to the occipital lobe was detected on MRI and removed surgically. Intracranial pressure was 19 mmHg before craniotomy, and 10 mmHg at cranioplasty. Intracranial pressure gradually increased to 30 mmHg by 17 h postoperatively, and the cat presented with depressed level of consciousness and anisocoria. In addition, cerebellar herniation was found on MRI, so surgical decompression was performed. Intracranial pressure decreased to 2 mmHg by removal of the skull piece removed by craniotomy in the first operation, but increased to 10 mmHg when the temporalis muscle was sutured. Therefore, we placed artificial dura mater between the bilateral temporalis muscle, and ICP was decreased to 4 mmHg. Intracranial pressure after this second operation increased to 20 mmHg, but reduced to 10 mmHg after two days and stabilized. The cat showed favorable recovery. Histopathological diagnosis was meningioma.

Intracranial pressure after the second operation was higher in Case 1 than in Case 2. We considered that the low ICP in Case 2 was due to decompression by incising the dura mater at the removal of the brain tumour and using artificial dura mater for duraplasty. In addition, greater decompression of the ICP by relaxation of the temporalis muscle using artificial dura mater during temporalis muscle suture appeared helpful in the second operation in Case 2. In Case 1, decompression therapy may have been incomplete. The cat in Case 1 needed ventilation for a long time because cerebellar herniation was not sufficiently recovered, due to insufficient decompression of ICP. We therefore consider that this cat died of ventilatory insufficiency with ventilator-associated pneumonia.

TWO CASES OF VENTRAL BRAIN STEM MENINGIOMA RESECTION BY TRANSBASIOCCIPITAL APPROACH THROUGH ORAL CAVITY.

A. Ijiri[1], K. Yosiki[1], S. Tsuboi[1], A. Sibahasi1, T. Sugie[1], H. Akiyoshi[2], F. Ohashi[2], T. Nakade[3], [1]Atsuki Animal Medical Center, Kusatsu, Shiga, [2]Department of Veterinary Surgery, School of Life and Environmental Sciences, Osaka Prefecture University, [3]Department of Small Animal Clinical Sciences, School of Veterinary Medicine, Rakuno Gakuen University, Rakuno Gakuen University Teaching Hospital, Ebetsu, Hokkaido, Japan.

Surgical resection of canine ventral brain stem meningioma is difficult. It is common to perform midline incision over atlanto-occipital joint for novel atlanto-transbasioccipital approach (TBA). However reported outcome of this surgical approach always have very high mortality. So we decided to explore the possibility to perform resection of ventral brain stem meningioma by TBA through oral cavity like commonly successful hypophysectomy by transsphenoidal approach through oral cavity. The resection was performed under surgical microscope and with ultrasonic aspirator machine. MRI was taken step-by-step during the surgery for accuracy. Case 1 was diagnosed as papillary meningioma. Case 2 was a meningothelial meningioma. Because meningioma is always rich in nourishing blood supply and surgical field is very limited in space, resection is risky and difficult. Both patients showed residual meningioma on MRI during surgery but our goal is to remove only up to 80% of it by hollowing out. Only symptomatic treatments were sufficient for smooth recovery so no radiation and chemotherapy were provided. Case 1 patient has been surviving for 5 months and case 2 patient has been past 1.5 years post-op and keeping good quality of life. Technical difficulty on TBA through oral cavity is about the same as with transsphenoidal approach. We believe that resection through oral cavity helped us to stabilize post-op condition of the patients easier.

CORPUS CALLOSAL ABNORMALITIES IN THE DOG.

R. Gonçalves[1], H. Volk[2], P.M. Smith[4], J. Penderis[3], L. Garosi[4], E. MacKillop[5], A.de Stefani[6], J.F. McConnell[1], [1]Department Veterinary Science, University of Liverpool, UK, [2]Dept of Veterinary Clinical Sciences, Royal Veterinary College, London, UK, [3]Faculty of Veterinary Medicine, University of Glasgow, UK, [4]Davies Veterinary Specialists, UK, [5]Pittsburgh Veterinary Specialty and Emergency Centre, USA, [6]The Animal Health Trust, UK.

Sporadic cases of corpus callosal abnormalities (CCA) in dogs have been reported in recent years due to the more widespread use of magnetic resonance imaging (MRI); nonetheless these malformations are still poorly understood in this species. The aims of this case series were therefore to describe the clinical presentation and the MRI findings of dogs with CCA.

The medical records of the contributing institutions were retrospectively reviewed to identify dogs that had been diagnosed with intracranial malformations affecting the corpus callosum. Cases in which the CCA was thought to be secondary (e.g. cases of hydrocephalus, porencephaly or hydranencephaly) were excluded. Data collected for each case included signalment, clinical history, findings of the physical and neurological examinations, serum biochemistry results, MRI of the brain and long-term outcome.

Fourteen cases were identified. Median age at MRI was 11.5 months (range 3-81 months). The most represented breeds were Staffordshire bull terriers (5) and Miniature Schnauzers (3). The clinical signs most commonly reported were adipsia associated with hypernatremia (12), tremors (6) and seizures (5). Five dogs (of which 4 were Staffordshire bull terriers) were reported to have hyperkeratosis affecting the feet and nasal planum. Revision of the MR images revealed that 9 dogs had an absence of the rostral CC (in some cases a small remnant could be seen) and hypoplasia of the caudal portion whilst 5 dogs had a diffusely hypoplastic CC that was significantly reduced in length and thickness. In only 2 cases could a septum pellucidum be identified and only in 3 cases could a fornix be seen. In 13 cases, there was abnormal cortical development with fusion of the ventral frontal lobes and part of the diencephalon. In 7 dogs, a dilation of the suprapineal recess could also be identified. Four dogs were lost to follow-up and 3 dogs were euthanased. The remaining 7 dogs are still alive, with a median follow-up time of 20 months (range 10 – 77 months after diagnosis). Those dogs that presented with adipsia have been managed by adding water to their food and this has minimised the incidence of episodes of hypernatraemia.

This case series describes the clinical presentation of dogs with CCA; previous literature has mainly associated this malformation with adipsia but not all dogs affected appear to present this. There are different degrees of the malformation but in most dogs the cranial portion of the CC (the region of the rostrum and genu) seems to be most affected in dogs, unlike humans. Also, most dogs have simultaneous fusion of the midline structures rostral to the CC; this region has several structures involved in thirst regulation (including the AV3V) and may explain this derangement.

VALIDATION OF A STEREOTACTIC ACCESS TO THE BRAINSTEM IN ANIMALS USING AN OVINE CADAVER HEAD MODEL. A. Staudacher[1], A. Oevermann[2], M. Stoffel[3], D. Gorgas[1], [1]Division of Clinical Radiology, Department of Clinical Veterinary Medicine, [2]Divison of Neurological Sciences, Department of Clinical Research and Veterinary Public Health, and, [3]Institute of Veterinary Anatomy, Vetsuisse-Faculty Bern, Switzerland.

Due to their deep-seated location in a vitally important anatomical area, brainstem lesions are especially challenging to access. Therefore stereotactic biopsy is currently the gold standard to obtain tissue samples in human medicine, where either a transfrontal or a trancerebellar approach is used. Anatomical differences such as the horizontal brain axis, the large frontal sinuses and the lateral position of the cerebellar peduncles, prohibit the use of the described approaches in animals. Since sheep serve as a translational model for various types of brain disease in humans, the aim of this prospective study was to develop a safe and accurate stereotactic approach to the brainstem in sheep.

A transcerebellar approach with an entry point in the occipital bone above the vermis between the transverse sinus and the external occipital protuberance was assessed. Magnetic resonance (MR) guided stereotactic procedures were carried out in 18 ovine cadaver heads using the Brainsight™frameless stereotactic system. For accuracy testing, 0.5 µl of diluted gadolinium and toluidine blue were injected into midbrain, pons or obex. The needle placement error was calculated by comparing coordinates of the target and the injected gadolinium bloom. In order to confirm the gadolinium data, the position of the toluidine dot was assessed macroscopically.

The developed transcerebellar approach allowed access to the target site in all the heads. The overall mean needle placement error was 1.85 ± 1.22 mm.

The achieved needle placement error is comparable to that reported with the Brainsight™ system for targets in the canine rostral and middle cranial fossa, as well as to those reported with computed tomography-guided stereotactic systems in veterinary medicine and with frameless MR-guided stereotactic systems used in living humans. The developed transcerebellar route is short, provides accurate access to most of the structures within the posterior fossa of sheep and thus, is a promising approach to be used in other species such as dogs and cats. The safety of this approach has to be evaluated in live animals.

SURGICAL TREATMENT OF A CEREBELLOPONTINE EPENDYMAL CYST IN A DOG. M.K. Müller[1], N. Bauer[2], E. Ludewig[1], K. Matiasek[3], G. Oechtering[1], M. Preuß[4], T. Flegel[1], [1]Department of Small Animal Medicine, University of Leipzig, Leipzig, Germany, [2]Department of Veterinary Clinical Sciences, Clinical Pathology and Clinical Pathophysiology, Giessen, Germany, [3]Section of Clinical and Comparative Neuropathology, Institute of Veterinary Pathology, Ludwig Maximilians University, Munich, Germany, [4]Department of Neurosurgery, Pediatric Neurosurgery, University Hospital Leipzig, Leipzig, Germany.

Ependymal cysts are rare benign cysts lined by ependymal cells, which secret a clear serous fluid consistent with cerebrospinal fluid. Those cysts are suspected to develop during embryogenesis due to sequestration of neuroectoderm. To date, there is only one case report in veterinary medicine describing an ependymal cyst in a dog. We present a case of a 6-month-old intact male mix breed dog that was presented for a 4 week history of a left sighted head tilt and positional rotatory nystagmus. No other neurologic deficits were present on neurological examination. Hence a vestibular dysfunction, most likely of central origin, was suspected.

Magnetic resonance imaging, using a 0.5 T magnet, revealed a 2 x 2 cm large thin-walled fluid- filled ovoid structure located at the cerebellopontine angle predominantly on the right side of the caudal fossa. The cyst caused a lateral displacement of the brainstem and a dorsolateral displacement of the cerebellum to the left. The signal characteristics (FLAIR-, T2-, T1-weighted images) revealed that the cyst contains cerebrospinal fluid. Cyst content and wall did not show enhancement after contrast administration (0.2 mmol/kg gadopentetate dimeglumine). There was no evidence of an adjacent edema.

The cyst was fenestrated via a suboccipital craniectomy. A lateral extracerebellar approach was not feasible due to the elevated pressure of the cerebellum. Therefore a transcerebellar route was chosen. A corticotomy was performed over the right hemisphere of the cerebellum parallel to the folia. Thereafter the cerebellar surface was separated. The cyst was opened by blunt dissection of the cerebellum and its content collected for cytological examination. According to the histopathologic examination of a fragment of the cyst wall and cytological examination of its content, the diagnosis of an ependymal cyst was made.

One day after surgery, the patient presented with a high-grade ataxia and was unable to stand. He developed a pleurothotonus to the right, a positional vertical nystagmus and a medial strabismus in his right eye while the left showed a dorsolateral strabismus. All of this was suspected to be caused by invasive surgery to the cerebellum.

These symptoms gradually improved and the dog was able to stand and walk a few steps 3 days post surgery with the high-grade ataxia still being present. At the follow up examination five weeks after surgery the gait and neurological examination were unremarkable.

CERVICAL DISC ARTHROPLASTY USING THE ADAMO SPINAL DISC® IN 30 DOGS AFFECTED BY DISC ASSOCIATED WOBBLER SYNDROME AT SINGLE AND MULTIPLE LEVELS. P.F. Adamo[1], R.C. da Costa[3], R. Kroll[2], C. Giovannella[4], M. Podell[5], P. Brofman[6], [1]East Bay Area Veterinary Specialists, Walnut Creek, CA., [2]VCA Northwest Veterinary Specialists, Clackamas, OR., [3]Department of Veterinary Clinical Sciences, The Ohio State University, Columbus, OH., [4]Gulf Coast Veterinary Neurology and Neurosurgery, Houston, TX., [5]Chicago Veterinary Specialty Group, Chicago, IL., [6]Veterinary Specialty Care, Mt Pleasant, SC, USA.

The objective of this study was to evaluate the immediate postoperative recovery and the short- and intermediate-term follow-

up of dogs with disc-associated-wobbler-syndrome (DAWS) treated with cervical-disc-arthroplasty (CDA) using the Adamo-Spinal-Disc®. Thirty dogs with over 2 months' history of DAWS diagnosed by MRI or CT myelography were evaluated. Eighteen dogs were treated for a single lesion, 10 dogs for a double lesion and 2 dogs for a triple lesion.

The prosthesis and technique were similar to that described in the previous study [Adamo et al, Proc ACVIM, 2012]. For some cases, a redesigned thinner disc was employed in which the internal convex surface was replaced with PEEK (Polyether-Ether-Ketone). All dogs were evaluated neurologically shortly after surgery and with immediate and serial postoperative radiographs.

All dogs had uncomplicated recovery with good degree of distraction in the immediate postoperative radiographs. In the majority of dogs, the amount of distraction and the mobility at the treated sites decreased over time compared to the immediate post-operative status; this was less pronounced in the dogs treated with the thinner size implant. The loss of distraction (except for one dog) and the decreased mobility did not appear to be clinically significant. Median clinical follow-up was 24 months (1 – 40 months). All but one dog showed improvement in neurological status during the observation period.

Cervical-disc-arthroplasty using the Adamo-Spinal-Disc appears to be a well-tolerated surgical technique, and it might be a valuable method to treat DAWS in dogs. The preliminary results of this study are promising. Long-term follow-up studies are underway.

OUTCOME OF 96 DOGS TREATED MEDICALLY OR SURGICALLY FOR SPINAL ARACHNOID DIVERTICULA. D. Mauler[1], S.De Decker[2], L.De Risio[3], H.A. Volk[2], R. Dennis[3], T. Gielen[1], E.Van der Vekens[1], K. Goethals[1], L.Van Ham[1], [1]Faculty of Veterinary Medicine, Ghent University, Merelbeke, Belgium, [2]Royal Veterinary College, London, UK, [3]Centre for Small Animal Studies, Animal Health Trust, Newmarket, UK.

Little is known about the outcome of various treatment options for spinal arachnoid diverticula (SADs) in dogs. Although considered a surgical condition, only one medically-treated dog has been reported in veterinary literature. This retrospective study compared medical versus surgical treatment in a larger population of dogs with SADs.

Medical records of dogs with SADs between the year 2000 and 2012 were reviewed regarding information about clinical signs and treatment. To gain information about the outcome of these dogs, the referring veterinarians were contacted with a standardized questionnaire.

From the 96 dogs, 50 were treated medically: 44 received prednisolone, 3 physical therapy, 2 carprofen and 1 gabapentin. 46 dogs were managed surgically: a durectomy was performed in 24, a durotomy in 16, in 3 dogs the SAD was marsupialized and in 3 the SAD was removed. The median follow up was 16 months (range 1 to 90) in the medically-treated and 23 months (range 1 to 94) in the surgically-treated group, which was not significantly different ($P = 0.6420$).

The age at onset of clinical signs had a significant influence on the choice of treatment, with younger dogs undergoing surgery more often ($P = 0.0486$). All other variables, including severity of clinical signs and duration of signs did not have an influence.

Comparing the outcome between medical and surgical treatment, there was a significant difference between the improved, stable and deteriorated dogs ($P = 0.0005$). From 37 medically-treated dogs with available follow up, 11 improved, 11 remained stable and 15 deteriorated. From the 38 surgically-treated dogs with available follow up, 31 improved, 1 remained stable and 6 deteriorated. Significantly more medically-treated dogs deteriorated ($P = 0.0047$). Likewise, there were more surgically-treated dogs in the improved group ($P = 0.0074$).

The results suggest that surgical treatment is superior compared to medical treatment in the management of SADs in dogs. As the retrospective nature of this study is a major limitation, further studies are necessary to investigate differences between the treatment modalities.

UNUSUAL 'PRAYING POSITION'-LIKE STANCE AND GAIT AS CLINICAL MANIFESTATION OF CAUDAL LUMBAR DISC DISEASE IN A DOG AND A CAT. I. Espadas[1], O. Chai[2], S. Lindley[1], A. Wessmann[3], [1]School of Veterinary Medicine, College of Medical, Veterinary and Life Sciences, University of Glasgow, Glasgow, UK, [2]Koret School of Veterinary Medicine, Hebrew University of Jerusalem, Rehovot, Israel, [3]Pride Veterinary Centre, Derby, Derbyshire, UK.

A 10-year-old male Border collie presented with a one-year history of slowly progressive painful paraparesis adopting an abnormal self-limiting posture, and a 5-year-old male DSH with a one-week rapid progressive history of unusual stance and gait. Both animals placed the lumbosacral joint into hypoextension by walking in a 'praying'-like position lowering their torso with their elbows close to the ground and raising their pelvis. Additionally, the cat walked plantigrade with a dropped flaccid tail.

Neurological examination showed two paraparetic animals with reduced withdrawal reflexes in the hind limbs and lumbosacral discomfort. The cat had also an absent anal reflex and tail tone. Their neuroanatomical localisation was L4- S3 spinal cord segments.

MRI revealed a L7-S1 disc herniation in the dog and CT a L6-L7 disc herniation in the cat. The dog improved significantly to a mildly paraparetic gait with conservative treatment until he was euthanized six months later due to an unrelated condition. The cat underwent a decompressive dorsal laminectomy removing calcified disc material. Re-examination 8 weeks later showed complete resolution of his clinical signs.

Similarly to dogs adopting an arched back with a thoracolumbar disc herniation, it is suspected that the unusual 'praying'-like posture was chosen by both pets to alleviate the cauda equina compression by widening the caudal lumbar intervertebral disc spaces. The resolution of the unusual posture with focused treatment of the cauda equina compression supports this finding. This is the first description of a praying-like posture as clinical presentation for lumbosacral disc disease in pets.

RECORDING OF CORD DORSUM POTENTIALS IN CALVES. I.Van Soens[1], C.De Vlamynck[2], F. Pille[2], L. Vlaminck[2], L.Van Ham[1], [1]Department of Small Animal Medicine and Clinical Biology., [2]Department of Surgery and Anaesthesiology of Domestic Animals, Ghent University, Merelbeke, Belgium.

Cord dorsum potentials (CDP) are sensory evoked potentials that are being used to assess proximal sensory nerve, dorsal nerve root and spinal cord dorsal horn function. The CDP is a spinal cord field potential that arises in the region of the spinal cord segments receiving input from peripheral nerves. The purpose of the present pilot study was to establish normal values for CDP onset latency (OL), peak latency (PL) and peak-to-peak amplitude (PPA) after saphenous nerve stimulation.

CDP were recorded under general anaesthesia in 15 clinically and neurologically healthy calves of 8 ± 2 (mean $\pm$ SD) weeks of age. Bodyweight (65.6 ± 9.4 kg), height at the withers (84.9 ± 3.4 cm), limb length from trochanter major to calcaneus (43.4 ± 2.7 cm) and body length from T2 to S1 (50.9 ± 4.1 cm) were measured. Body temperature was maintained between 36 and 39°C.

The saphenous nerve was stimulated with 2 monopolar needle electrodes placed at the caudal border of the tibia at 2/3 its length, in close proximity and cranial to the saphenous vein. CDP were recorded from the interarcuate space L3-4, L4-5 and L5-6 with a monopolar needle electrode. OL (in ms) was measured as the shortest distance between the trigger point and the takeoff of the initial phase and PL (in ms) between the trigger point and the peak of the highest phase. PPA (in mV) was measured between the two largest peaks of opposite polarity.

CDP were easily recorded at the different recording sites. CDP consisted of a large negative peak (actual CDP) followed by a long latency positive phase; in some calves polarity was reversed. An initial small polyphasic wave was occasionally observed. Median OL (in ms) at L3-4, L4-5 and L5-6 was 8.13 (5-11), 7.65 (4-10) and 7.39 (4.05-9.90), respectively. Median PL (in ms) at L3-4, L4-5 and L5-6 was 11.6 (9.8-12.9), 11.25 (9.9-13.5) and 10.25 (8.95-13.10), respectively. Median PPA (in mV) at L3-4,

L4-5 and L5-6 was 2.89 (0.77-9.98), 6.54 (1.16-14) and 7.47 (3-14). Significant differences were observed between OL, PL and PPA recorded at different sites, except for OL between the L4-5 and L5-6 recording site.

Conclusion: CDP in response to saphenous nerve stimulation in calves were reproducibly recorded in calves at L3-4, L4-5 and L5-6 recording sites, with the largest responses at L5-6. Although the initial small peak was often not present in calves, the actual CDP and long latency waveform as described in other species were observed. A statistically significant increase was seen in PL after moving the recording needle cranially, indicating its clinical and predictive value.

THE INFLUENCE OF ACUTE VAGUS NERVE STIMULATION ON THE PTZ SEIZURE THRESHOLD IN DOGS. V. Martlé[1], R. Raedt[2], T. Waelbers[1], L.Van Ham[1], K. Peremans[3], K. Vonck[2], P. Boon[2], L. Duchateau[4], S. Bhatti[1], [1]Department of Small Animal Medicine and Clinical Biology, Faculty of Veterinary Medicine, Ghent University, Merelbeke, Belgium., [2]Laboratory for Clinical and Experimental Neurophysiology, Department of Neurology, Ghent University Hospital, Ghent, Belgium., [3]Department of Medical Imaging and Small Animal Orthopaedics, [4]Department of Physiology and Biometrics, Faculty of Veterinary Medicine, Ghent University, Merelbeke, Belgium.

Vagus nerve stimulation (VNS) is an effective adjunctive treatment for refractory epilepsy in humans, but the anticonvulsant efficacy of VNS in dogs has not been well established. This study investigates the influence of two acute VNS paradigms and phenobarbital on the pentylenetetrazole (PTZ) seizure threshold in dogs.

In 8 Beagle dogs, implanted with a VNS therapy system, the PTZ seizure threshold was determined after one hour of sham, standard and microburst VNS following a randomized blinded cross-over design with a wash-out period of one month. As a positive control, the PTZ seizure threshold after a single oral dose of phenobarbital (20 mg/kg) was determined. The dogs were infused with a 3% solution of PTZ in 0.9% NaCl at a rate of 3 ml/min IV. The PTZ seizure threshold was defined as the amount of PTZ (mg/kg) administered until the first whole-body myoclonic twitch was noticed. The exact threshold was determined by 3 observers on post experimental video analysis. Statistical analysis was based on a mixed model with dog as random effect; the two VNS paradigms were compared pair wise with sham using Dunnett's multiple comparisons technique at a global significance level of 5%.

Acute standard and microburst VNS did not cause significant changes in the PTZ seizure threshold compared to sham stimulation. Phenobarbital caused a significant increase of the PTZ seizure threshold in all dogs ($p < 0.001$).

In conclusion, an anticonvulsant effect of acute VNS could not be shown in this canine seizure model. The effect of acute VNS in other animal models and the effect of chronic VNS requires further study.

MRI TRACTOGRAPHY OF THE MESOCEPHALIC CANINE BRAIN AND THE DEVELOPMENT OF A WHITE MATTER ATLAS. R. Mineo[1,2], B. Moffat[3], S. Long[1], M.Le Chevoir[1], [1]Faculty of Veterinary Science, University of Melbourne, Werribee, VIC, Australia, [2]School of Medical Sciences, RMIT University, Bundoora, VIC, Australia, [3]Faculty of Medicine, University of Melbourne, Melbourne, VIC, Australia.

Diffusion weighted imaging (DWI) tractography is an advanced Magnetic Resonance Imaging (MRI) technique that allows delineation of the white matter micro-structural architecture of the canine brain. The purpose of this study was to develop the first 2D segmented and 3D representative canine brain atlases to demonstrate and delineate the normal canine white matter networks in disease free mesocephalic dogs using DWI.

White matter tractography was generated using a constrained spherical deconvolution method alongside a probabilistic algorithm approach from DWI data attained from 5 brains from mature mesocephalic breeds both *in vivo* and *ex vivo* on 1.5T and 3T systems. Prominent axonal fibre bundles were identified via whole brain fibre tacking and through *a priori* histological knowledge. Streamlined tracks via positioned seed points to illustrate the corticobulbar tract, the corticospinal tract, the thalamic radiations, association fibres, limbic system fibres and callosal fibres were generated using the MRtrix software package (Brain Research Institute, Melbourne, Australia). Each DWI dataset was registered (rotation and translation) to a template space using Statistical Parametric Mapping (SPM) and overlayed onto a T1 image depicting and correlating the position of the white matter tracts with the superficially visualised gyri and sulci. Morphologic evaluation of the shrinkage associated with the paraformaldehyde fixation on the ex vivo was investigated.

Results demonstrate mesocephalic subjects share common deep white matter pathways. These pathways have been defined by the developed white matter atlases. The diversity in the superficial white matter networks has been noted to occur on an individual basis and has been evaluated via qualitative visual inspection and quantitatively though evaluated though fractional anisotropy map analysis. MRI tractography derived white matter atlas of the normal axonal network of the disease free mesocephalic brain is an invaluable resource as a reference template for healthy control data that will be used to structurally delineate the white matter architecture of the deep matter association networks in a 3D context for research and clinical purposes.

THIAMINE DEFICIENCY IN CATS: A RECENT OUTBREAK IN TAIWAN. Y.P. Chang[1,2], P.Y. Chiu[1], C.C. Wu[1,2], C.H. Liu[1,2], [1]School of Veterinary Medicine, [2]National Taiwan University Veterinary Hospital, National Taiwan University, Taipei, Taiwan.

Dietary requirement for thiamine in cats is high. An outbreak of thiamine deficiency in cats occurred in Taiwan from December 2012 to February 2013 due to consuming a brand of dry food with insufficient thiamine content (0.35 mg/kg; AAFCO recommendation 5 mg/kg). The clinical presentation, MRI findings, and outcome from 14 cats in this outbreak were reviewed.

Female cats and domestic shorthair cats were overrepresented. The duration of consuming the specific diet could be traced in six cats and it ranged from one to six months. Prior to the acute development of neurological signs, most cats showed non-specific signs such as anorexia, lethargy or vomiting. Clinical signs consistent with bilateral vestibular and brainstem dysfunction were presented in the majority of cats (13/14). In one cat, seizures and blindness were the only clinical signs. Half of patients exhibited generalized or partial seizures. Magnetic resonance imaging (MRI) of the brain was performed in six cats. Bilateral symmetrical hyperintense changes in the lateral geniculate nuclei and caudal colliculi on T2-weighted images were noted in five cats; in one of these patients, similar changes were also detected in the cerebral cortex. MRI was performed in one cat who continued to show marked neurological deficits despite receiving treatment for five days; MRI at that stage, however, was unremarkable. Management included discontinuation of the specific diet, thiamine supplement, and supportive treatment. Significant improvement within one month was seen in eleven cats. However, two cats died from the condition, and one cat showed slow recovery and remained markedly ataxic two months after treatment was initiated.

Thiamine deficiency is an important differential diagnosis in cats with seizures and bilateral vestibular signs. Prognosis can be good if treated promptly, but slow recovery or death may occur.

EFFECT OF RADIOTHERAPY ON SEIZURE FREEDOM IN DOGS WITH BRAIN TUMOURS. S. Monforte-Monteiro[1], J. Rossmeisl[2], J. Russell[1], M.A. Holmes[1], A. Wessmann[3], A.E. Vanhaesebrouck[1], [1]Department of Veterinary Medicine, University of Cambridge, UK, [2]Virginia Maryland Regional College of Veterinary Medicine, USA, [3]School of Veterinary Medicine, University of Glasgow, UK

Seizures are a common presenting sign of brain tumours in dogs, which might lead to euthanasia. Total surgical resection and chemotherapy have been reported in humans to allow control of seizures, whereas the exact role of radiation therapy in seizure reduction is still unclear in both humans and animals.

This retrospective study aimed to investigate the efficacy of radiotherapy in controlling epileptic seizures in dogs with brain tumours.

Thirty-two dogs presented with seizures, resulting from a suspected or confirmed brain tumour. The study group received

radiotherapy (n = 18) and was compared with a control group (n = 14). All dogs received medical treatment, consisting of antiepileptic drugs with or without corticosteroids. The effect of radiation therapy on seizure freedom was analyzed, along with the role of clinical and MRI characteristics on seizure frequency following radiotherapy or palliative treatment. Minimum follow-up period was 12 months.

The period of seizure freedom was significantly increased in the radiotherapy group ($P < 0.0005$ using a log rank test), with a mean of 24.0 months (95% CI: 14.3-33.8) versus 1.7 months in the control group (95% CI: 0.5 – 2.9). Nearly halve (44%) of the radiotherapy group were still seizure free at the end of the study, compared with none of the dogs within the control group. In the radiotherapy group 5 dogs died during the study period, but without recurrence of seizures. In the control group, recurrence of seizures was observed before death in all dogs.

A Cox's proportional hazard analysis found no statistically significant association between clinical (i.e., frequency or severity of seizures) or MRI characteristics (i.e., tumour type, localization or size, edema, mass effect or contrast enhancement) and the length of time that dogs receiving radiotherapy were seizure free. .

A longer period of seizure freedom was observed in dogs with brain tumours following radiotherapy, compared with medical treatment only. Further studies are needed to investigate underlying pathophysiological mechanisms.

RISK FACTORS ASSOCIATED WITH DEVELOPMENT OF SEIZURES IN DOGS WITH HEPATIC ENCEPHALOPATHY. C. Ricco[1], M. Tivers[2], H.A. Volk[2], [1]Centre Hospitalier Vétérinaire Frégis, Arcueil, France, [2]Royal Veterinary College, London, UK.

Portosystemic shunt (PSS) is an anomalous portal venous circulation bypassing the liver leading to organ dysfunction and hepatic encephalopathy (HE). HE can be associated with the development of seizures. The exact pathophysiology of HE causing seizures remains unclear but it is believed that the impaired hepatic function leads to the build up of neurotoxins. To the authors' knowledge there are currently no studies analysing the risk factors for seizures in dogs with PSS prior to surgery.

In this cross-sectional retrospective study 64 dogs with PSS (32 of which had seizures prior to surgery) were randomly selected from 256 dogs diagnosed with PSS. Factors included in the analysis were: Age, sex, neuter status, body weight, length of time until diagnosis, interictal neurological deficits, changes in haematology and serum biochemistry, glucose, ammonium, dynamic bile acids. Data were analysed using generalised linear mixed models for binary outcomes (glmmML and glmmPQL, SPSS, Version 20). Model fit was assessed using the deviance and Akaike's information criterion. In all models breed was included as a random effect. A P value of less than 0.05 was considered significant.

On the basis of multi-variable analysis, factors significantly associated with seizures in dogs diagnosed with PSS was age (odds ratio [OR] 0.96; 95% confidence interval [CI] 0.92-1) and eosinophilia (OR 9.97; 95% CI 1.6-61). Eosinophilia was reported in 9/32 dogs with PSS and seizures and only in 3/32 without seizures. The age in the group with seizures had a median of 13.5 months (interquartile range [IQR] 6-33.5 months) versus 5 months (IQR 9.5-17 months) in the group without seizures.

This study shows that patients with PSS and seizures appear to be older in age and have more frequently eosinophilia than dogs which do not develop seizures with PSS.

NONCONVULSIVE STATUS EPILEPTICUS IN DOGS. C. Mariani, Department of Clinical Sciences, College of Veterinary Medicine, North Carolina State University, Raleigh, NC, USA,

Status epilepticus (SE) can be defined as continuous seizure activity for longer than 5 minutes or two or more seizures without an interictal return to normal baseline neurologic function. Although typically accompanied by tonic-clonic convulsions, SE may occur with minimal or absent motor activity, which is known as nonconvulsive SE (NCSE). NCSE is a well-recognized entity in humans, but has not been well defined in veterinary patients. The purpose of this report is to describe a group of canine patients with NCSE that presented to the Veterinary Teaching Hospital at North Carolina State University.

Electroencephalography (EEG) was performed using subdermal needle electrodes arranged in a combination referential-bipolar montage. Data was acquired using one of two commercially available EEG systems. A diagnosis of NCSE was made when patients had impaired mentation, the absence of overt convulsive activity, and EEG activity consistent with status epilepticus.

The most frequently identified underlying etiologies were encephalitis and intoxications. Electrographic seizure patterns included atypical spike-and-wave, multiple spike-and-wave, and rhythmic delta with intermittent spikes. A number of dogs responded to an intravenous midazolam bolus with cessation or marked improvement in the ictal EEG pattern and in some cases improvement in mentation. Several dogs did not respond to an initial midazolam bolus but showed improvement after a midazolam constant rate infusion. Although most dogs died or were euthanized, several survived to discharge. NCSE occurs in dogs and similar to humans, appears to carry a guarded prognosis. However, early recognition and aggressive intervention of this condition may improve outcome.

DEXMEDETOMIDINE IN THE MANAGEMENT OF STATUS EPILEPTICUS AND TREMORGENIC MYOTOXICOSIS. C. Rusbridge, V. Rubasinska, S. Griffiths, J. Aguiar, A. Foucault, J. Uhrig, J. Griffiths, R. Elliot, A. Bradnock, S. Rodendas, R. Bralow, Stone Lion Veterinary Hospital, Wimbledon, UK,

Dexmedetomidine (Dexdomitor; Zoetus) is highly specific central α2-adrenoreceptor agonist which has been shown to have an anticonvulsant effect on rodent models of self-sustaining status epilepticus (SE), and other causes of seizures. This retrospective study evaluated the clinical usefulness of dexmedetomidine in dogs and cats with SE due to variety of different causes.

Medical records between January 2012 and May 2013 from a primary and out-of-hours emergency veterinary clinic were searched for dexmedetomidine administration and SE. The cause of the seizures, dose and response to medication and the outcome were analyzed.

26 cases (22 dogs and 4 cats) were identified. The most common cause of SE was toxicity (12 cases) by metaldehyde (5 dogs, 4 of which were Labrador Retrievers); tremorgenic mycotoxicosis (2 dogs); permethrin (1 cat); caffeine tablets (1 dog) and unknown (3 dogs observed to be eating something before presentation). Idiopathic (5 dogs) or acquired (1 dog) epilepsy was the second most common cause of SE. Seizures secondary to intracranial neoplasia caused SE in 4 dogs. The cause of SE was not determined in 3 cats and 1 dog. The dose of dexmedetomidine varied but mostly commonly was 375 mcg/m2 intramuscularly (IM) and according to the SE protocol for this practice was usually the second line agent. In 16 animals there had been a poor or unsustained response to intravenous (IV) diazepam before dexmedetomidine administration. However 10 animals received dexmedetomidine as first line therapy. For 3 animals this was because the seizure was so violent that intravenous access was not possible; IM dexmedetomidine in these animals resulted in temporary seizure cessation. For the other animals receiving dexmedetomidine as first line therapy, 5 cases did not require other drugs for management of SE; 2 also received propofol. Dexmedetomidine was most successful for SE associated with pre-existing epilepsy with all of the 6 dogs discharged the following day, usually with adjusted oral therapy. All 6 dogs had at least 2 injections 4 hours apart. The most difficult SE to manage was that caused by toxins with the majority of cases requiring polypharmacy including multiple injections of dexmedetomidine (9 of 12) in addition to propofol constant rate infusion (CRI) (7 of 12); methocarbanol (3 per rectum, 1 oral); ketamine CRI (2 of 12); midazolam CRI (2 of 12) and phenobarbital (1 of 12). However all cases survived, typically discharged 2 days later. The four cases with intracranial neoplasia were ultimately euthanatized. For 2 of 4 cases dexmedetomidine was only partially effective with other drugs including propofol and ketamine CRI required to manage the seizures.

Dexmedetomidine is potentially useful for management of SE. It has the advantage that it is relatively cheap, can be combined with other drugs thus reducing the dosage and potential adverse effects of those drugs and can be administered IM when IV access is not possible.

RISK FACTORS AND OUTCOMES IN FELINE ACQUIRED MYASTHENIA GRAVIS (2001-2012). D.W. Hague[1], H.D. Humphries[2], M.A. Mitchell[1], G.D. Shelton[2], [1]University of Illinois College of Veterinary Medicine, Urbana, IL., [2]University of California San Diego, San Diego, CA.

The diagnosis of acquired myasthenia gravis (MG) in cats is confirmed by an acetylcholine receptor antibody titre > 0.3 nmol/L. The purpose of this retrospective study was to extend the previous report of 2000 (J Am Vet Med Assoc 2000;215:55-57) and evaluate long-term outcome, medical versus surgical treatment, and presence or absence of spontaneous remission in cats with confirmed acquired MG. From 2001 to 2010, acquired MG was diagnosed in 235 cats at the Comparative Neuromuscular Laboratory, University of California San Diego. Referring veterinarians were contacted and medical record information obtained for 133 cats. Specific information included signalment, presence and type of mediastinal mass, treatment (surgical versus medical), survival time, and outcome including spontaneous remissions.

Males and females were equal in number and mixed breed cats (80.4%) were more common than purebred cats (19.6%). No significant difference in outcome by sex ($p = 0.7$) or breed ($p = 0.24$) was found. A mediastinal mass was observed in 52% of the cats; however, outcome was not associated with the presence of a mass ($p = 0.49$). The most common mass diagnosed was thymoma (97.7%).

Outcomes were reported in 92 cats: 35 cats (38%) were alive (range 11 days to 115 months from diagnosis), 4 (4%) died, and 53 (58%) were euthanized (range 1 day to 97 months from diagnosis). Repeat antibody titres were obtained post thymoma surgery in 17 cats and showed no differences. The titres increased above baseline in 7 cats (41%), decreased below baseline in 8 cats (47%) and initially decreased, but then increased above baseline in 2 cats (12%). There was no significant difference in outcome by medical treatment ($p = 0.67$) or surgery ($p = 0.09$), but a significant negative correlation ($r = -0.3$, $p = 0.004$) was present between titre and survival time. Kaplan-Meier survival curves were used to determine if treatment had an effect on survival. There was no significant difference in survival for cats receiving surgery ($p = 0.5$) or based on medical treatment ($p = 0.4$).

CLINICAL, PATHOLOGICAL AND MOLECULAR CHARACTERISTICS OF INHERITED CEREBELLAR ATAXIA IN ITALIAN SPINONE DOGS. L.De Risio[1], O. Forman[2], S. Platt[3], C. Mellersh[2], K. Matiasek[4], [1]Neurology Unit and [2]Kennel Club Genetics Centre, Animal Health Trust, Newmarket, UK, [3]Department of Small Animal Medicine and Surgery, University of Georgia, Athens, Georgia, USA, [4]Section of Clinical and Comparative Neuropathology and Neurology, Ludwig Maximilians University of Munich, Germany.

A progressive gait disorder of probable cerebellar origin has been reported in a brief clinical communication on 2 Italian spinoni (IS) in 1996 and anecdotally in a few others IS worldwide. A linkage based DNA test has been available since 2008 to enable breeders to identify heterozygous carriers in their breeding lines. However no candidate mutations were identified and the disease remained poorly characterised clinico-pathologically.

We have elucidated the clinical and pathological aspects of the disease in a case series of 6 IS of which samples could be harvested for genetic analysis. Genetic analysis involved homozygosity mapping using the six affected IS and 6 controls, exon resequencing of interval genes and subsequently target resequencing.

Neurological signs are recognised at four months of age and include a wide based stance, cerebellar ataxia, characterised by thoracic limb hypermetria, pelvic limb hyperflexion, truncal swaying and impaired balance, pendular nystagmus and bilaterally absent menace response. Neurological dysfunction progresses to the point that the dogs become unable to stand up and ambulate at one year of age on average, leading to euthanasia. Antemortem diagnostic investigations including haematology, serum biochemistry, urinalysis, BAER, MRI of the brain and cervical spine, and CSF analysis show no characteristic abnormalities. On histological examination, there are cerebellar cortical lesions, in particular affecting the rostral parts of the cerebellar vermis. Generally, these lesions are mild with occasional Purkinje cell (PC) gaps, rare PC degeneration, multifocal basket prominence and focal mild to moderate Bergmann gliosis. Subjectively the granular cell layer appears oligofocally thinner. Moreover a mild vacuolation of the olivary nuclei is seen. The mode of inheritance of the disease is autosomal recessive. Genetic testing has identified a common mutation in the ITPR1 gene of chromosome 20. Subsequent expression analysis has shown a subtotal reduction of ITPR1 immunoreactivity of the PC throughout the cerebellum and highlighted their abnormal dendrite configuration.

Inherited cerebellar ataxia of the IS dog is a combined developmental and neurodegenerative disorder of the cerebellum due to reduced ITPR1 availability in PC. ITPR1 mediates $Ca2+$ release from the rough endoplasmatic reticulum and is a key player in long term potentiation and cerebellar synaptic plasticity. The clinical phenotype corresponds to the spatial lesion gradient.

ACQUIRED EQUINE POLYNEUROPATHY: A UNIFORM ENTITY? S. Hanche-Olsen[1], K. Hultin Jäderlund[1], J. Molin[2], G. Gröndahl[3], K. Matiasek[2], [1]Norwegian School of Veterinary Science, Oslo, Norway, [2]Ludwig-Maximilians University, Munich, Germany, [3]National Veterinary Institute, Uppsala, Sweden.

Acquired Equine Polyneuropathy (AEP) is a neurologic disease observed in Scandinavian horses, characterized by knuckling of the pelvic limbs, sometimes leading to non-ambulatory paresis. Earlier studies infer no genetic trait, gender predilection or infection as part of the aetiology. Cases cluster in farms during winter/spring and a possibly forage-related common toxic factor is discussed. An elaborate neurohistopathological examination of one previous case revealed a demyelinating, mildly inflammatory peripheral neuropathy.

Aims of the present study were to detail the neurohistopathological lesions and distributions in horses euthanized due to AEP and thereby elucidate the aetiology of this disease. Seven horses with clinical signs present from 10 days to 12 months before euthanasia were included, age ranged from 1 to 18 years. Nerve samples from up to 30 sites (including proximal and distal parts of nerves from thoracic and pelvic limbs as well as cranial nerves), dorsal root ganglia and spinal cord segments were examined by nerve fibre teasing, paraffin and semithin histology and transmission electron microscopy. Immune cell phenotyping was made by immunohistochemistry.

All nerves investigated showed identical changes throughout the animals regardless of sampling sites, only with some variation regarding stage and severity in accordance to the clinical presentation. Degenerative fibre lesions comprised myelin sheath irregularities with conspicuous protrusions, myelin splitting, extensive Schwann cell hypertrophy with inclusions and Wallerian-like degeneration of mainly large A(alpha)-fibres. Fibre-invasive T-cells and macrophages were detected in all locations. Further lymphocytic infiltrates were seen in the associated spinal cord.

AEP was shown to be a pathologically uniform disease of the peripheral nervous system (PNS) with both degenerative and inflammatory features, without a significant proximo-distal gradient. The inflammatory response appears to involve the central nervous system (CNS) as well. The clinical features of AEP cases appear strikingly similar with knuckling of the pelvic limbs as pathognomonic sign of the disease, but CNS lesions do not translate into obvious clinical signs. Even though the histopathological findings did not reveal a certain causative agent in the present cases, the results point to immune mechanisms as a major trigger for the conspicuous changes in the PNS.

ACCURACY OF THE NEUROLOCALISATION IN COMPARISON TO FINDINGS IN FURTHER DIAGNOSTICS IN 214 DOGS: A PROSPECTIVE STUDY. C. Loeffler, K. Truar, M.-K. Mueller, I.C. Boettcher, G. Oechtering, T. Flegel, Department of Small Animal Medicine, University of Leipzig, Germany,

Neurological examination is the first diagnostic step in order to localize a neurological problem to a certain area of the ner-

vous system. The aim of this prospective study was to evaluate the accuracy of the neurolocalisation based on the neurological examination. Over a period of one year dogs that fulfilled the following criteria were included: 1. complete neurological examination by a veterinarian of the neuro-division (three doctoral candidates, two diplomates of the ECVN), 2. a neurolocalisation was determined and 3. further diagnostics (myelography, CT, MRI, CSF analysis, electrodiagnostics) were performed. Dogs were excluded if a complete neurological examination was not possible. Subsequently the agreement of neurolocalisation and lesion location was evaluated. First, the overall agreement was analyzed. Second, the agreement in different groups (brain, spinal cord) were determined and compared to each other. Third, it was investigated whether the clinical experience of the examiner had an influence on the accuracy of the neurolocalisation.

Two hundred and fourteen dogs met the inclusion criteria, 124 males and 90 females. The overall agreement of the neurolocalisation and the lesion location was 71%; in 13% of the patients the neurolocalisation was wrong. In 16% of the cases no definite lesion could be identified and therefore the accuracy could not be evaluated. The most common neurolocalisation was a T3-L3 spinal cord lesion (67/214): here the agreement between neurolocalisation and lesion location was 78% (52/67). Nineteen percent (6/32) of the dogs with a neurolocalisation at L4-S3 had a lesion at T3-L3. Twenty-one percent (11/52) of the dogs with an agreement between neurolocalisation and lesion location at T3-L3 had a reduced withdrawal reflex in the hind limbs. The highest error rate was observed in the lower cervical spine. In 40% (8/20) of the dogs with a neurolocalisation at C6-T2 a lesion at C1-C5 was detected. In contrast, only 9% (3/34) of the dogs with a neurolocalisation at C1-C5 had a lesion at C6-T2. The forebrain was the most common intracranial neurolocalisation (46/214); the agreement was 63% (29/46). There was no significant evidence of any influence of the investigator's clinical experience on the agreement of neurolocalisation and lesion location.

In conclusion, the neurolocalisation was wrong in 13% of the cases. Therefore further work up is indicated if no lesion can be found in the presumed neurolocalisation. Results show that a decreased withdrawal reflex in the thoracic limbs does not always indicate a lesion from C6 to T2. Similar applies for the hind limbs; patients with a T3-L3 lesion often show a reduced withdrawal reflex in the hind limbs. The clinical experience of the examiner seems to have no effect on the overall agreement of neurolocalisation and lesion location.

PROGNOSTIC VALUE OF MAGNETIC RESONANCE IMAGING IN DOGS AFTER TRAUMATIC BRAIN INJURY: 50 CASES. E. Beltran[1], S.R. Platt[2], F. McConnell[3], R. Dennis[1], D. Keys[4], L.De Risio[1], [1]Animal Health Trust, Newmarket, UK, [2]College of Veterinary Medicine, University of Georgia, USA, [3]Faculty of Veterinary Science, University of Liverpool, Liverpool, UK, [4]Independent Statistical Consultant in Athens, GA, USA,

The clinical benefit of early magnetic resonance imaging (MRI) in dogs after traumatic brain injury (TBI) is unclear. The aim of this study was to determine whether MRI findings are associated with prognosis after canine TBI.

Medical records from the Animal Health Trust (2000-2012) were searched to identify dogs with TBI that underwent 1.5T MRI within 14 days of a head trauma. Fifty client-owned dogs fulfilled the inclusion criteria. All images were blindly evaluated by three of the authors (EB, FMc and RD) who were not aware of the clinical presentation. A modified MRI grading system was applied: Grade I (normal brain parenchyma) to Grade VI (bilateral lesions affecting the brainstem with or without any of the lesions in the lesser grades). Skull fractures, % intraparenchymal lesions, degree of midline shift and type of brain herniation were also evaluated. Modified Glasgow coma scale (MGSC) was assessed at presentation. The presence of seizures at the time of the trauma or later in life was also recorded. Outcome was assessed by evaluation at 48 hours (alive or dead), and 3, 6, 12 and 24 months following the TBI (death, poor, good or excellent outcome). Follow-up information was obtained by telephone consultation with the owner and/or referring veterinarians and/or combined with information from medical records.

Sixty-six per cent of the dogs had an abnormal MRI. There was a significant negative correlation of MRI grade ($p < 0.0001$) with MGSC at presentation. There was a significant negative correlation of MRI grade, degree of midline shift, and percentage of intraparenchymal lesions with follow-up scores. The MGSC score at presentation was significantly lower in dogs with brain herniation than without ($p = 0.0321$). Follow-up scores at 3, 6, 12 and 24 months were significantly lower in dogs that had brain herniation or fractures. The possibility of having seizures was significantly associated with higher percentage of intraparenchymal lesions ($p = 0.0047$).

There are statistically significant correlations between the MRI findings and prognosis in dogs with TBI. MRI can help to predict prognosis in dogs with TBI.

SURVIVAL AND FUNCTIONAL CAPACITIY IN 32 DOGS WITH TRAUMATIC BRAIN INJURY: A PROSPECTIVE STUDY. M.V. Bahr Arias, C.G. Vianna, Department of Veterinary Clinics, Londrina State University, Londrina, Parana, Brazil,

Traumatic brain injury (TBI) in dogs and cats is a common cause of neurologic dysfunction in the veterinary clinic's routine. Modified Glasgow Coma Scale (MGCS) for dogs and cats has been the most reliable indicator of the severity of injury and probability of survival after 48 hours, but there are insufficient data available for long-term outcome in surviving animal.

A prospective study was designed to compare survival and outcome in 32 dogs with brain injury admitted in the veterinary hospital of LSU. Our observations focused on the clinical and neurologic signs, results of blood glucose measurement immediately after trauma and subsequent days, the presence of concomitant injuries, the evaluation of treatments results and the accuracy of Modified Glasgow Coma Scale in prognosis of these patients. In addition, after leaving the coma state, patients were evaluated in relation to the functional capacity as pets. We performed a statistical analysis using the proportions test chi-square and Fisher's exact test, with significance level of 5% to compare survival in respect to age, weight, site of injury, blood glucose in the initial care, use of mannitol, prior medication usage, presence of concomitant injuries and MGCS in the initial care.

We observed that MGCS initial was grave in nine dogs, guarded in 19 dogs and good in four dogs. The MGCS in the initial care significantly interfered with the patient survival, because among the group of survivors, most come with a good score in the initial care, and in the group of no survivors, half presented grave score in the initial care. The factors as weight, time between the occurrence of trauma and treatment, presence of associated injuries in other systems, medication administered by the owner prior to treatment, location of the lesion in the central nervous system, the presence of hyperglycemia in the initial care and use of mannitol did not predict survival of patients.. At the end of observation period, 18 dogs survived and 14 died. We observed that 22 patients showed a good score in the end of inpatients treatment, but of these only 14 had complete functional recovery as pets. We verified that two dogs from survivor group died, one of them drowned in a pool because of vestibular sequelae, and one because of trouble with feeding. Another two dogs were euthanized upon request of the owners, because they did not interact successfully.

Survival and good final score in ECGM did not meant functional capacity as pets, since many patients had significant neurological sequelae after brain injury.

OCCURRENCE OF HYPOTHALAMIC-ANTERIOR PITUITARY HORMONE DEFICIENCIES FOLLOWING HEAD TRAUMA IN DOGS. K. Murtagh[1], L. Arrol[2], R. Goncalves[1], A. German[1], P.M. Smith[3], [1]Small Animal Teaching Hospital SATH, University of Liverpool, Neston, UK, [2]Anderson Moores Veterinary Specialists, Winchester, Hampshire, UK, [3]Davies Veterinary Specialists, Higham Gobion, UK.

Head trauma can have a number of long term consequences that impact on quality of life. In humans, one important component of this is the development of hormonal abnormalities as a result of damage to the pituitary gland and hypothalamus. The primary study objective was to investigate whether

hypothalamic-anterior pituitary axis (HPA) hormone deficiencies occurred after head trauma in dogs. In this retrospective, observational, cohort study, records from dogs referred to three separate referral centres (University of Liverpool Small Animal Teaching Hospital, Davies Veterinary Specialists and Anderson Moores Veterinary Specialists) between April 2008 and February 2013 were reviewed. Dogs were included if they had suffered from non-fatal head trauma causing evidence of brain dysfunction at the time, and follow-up evaluation included hormone testing (insulin-like growth factor 1 [IGF-1], endogenous adrenocorticotrophic hormone [ACTH], basal cortisol, thyroid-stimulating hormone [TSH], total thyroxine [TT4] and, if appropriate, free thyroxine [FT4]).

During this study period thirteen client-owned dogs fitted the study criteria. These represented a range of breeds, ages, weights and genders. Of the hormones analysed, decreased IGF-1 concentration was most common (7/13, 53%; median 114 ng/mL, range <15 to 536). This was followed by decreased TT4 (2/13, 15%; median 22; range 9 to 49) and in both of these cases, TSH was concurrently depressed with a value of <0.1 ng/ml in each case. Basal cortisol was unmeasurable at <20 nmol/L in one case; this dog also had concurrent unmeasurable ACTH of <5 pg/ml and was confirmed to have hypoadrenocorticism by an ACTH stimulation test. Based upon these results, seven dogs had suspected partial or complete growth hormone deficiency, one dog had suspected secondary hypoadrenocorticism (1/13, 8%), two dogs had suspected secondary hypothyroidism and one dog (1/13, 8%) had suspected multiple endocrine abnormalities. Despite these abnormal findings, only the case with multiple endocrinopathies had clinical signs consistent with these deficiencies, with weight loss, nonunion of a fracture and lethargy.

In conclusion, dogs with a history of traumatic brain injury can develop hypothalamic-anterior pituitary dysfunction and this should be considered in the differential diagnosis for those dogs that fail to recover fully following an episode of head trauma.

IONIZING RADIATION FOR TREATMENT OF CANINE MENINGOENCEPHALITIS OF UNKNOWN ORIGIN: AN OBSERVATIONAL PILOT STUDY. K. Beckmann[1], I. Carrera[2], F. Steffen[1], P. Kircher[2], U. Schneider[3,4], V. Meier[3], C. Rohrer Bley[3], [1]Division of Neurology, Clinic for Small Animal Surgery, Vetsuisse Faculty, University of Zurich, Zurich, Switzerland, [2]Division of Diagnostic Imaging, Vetsuisse Faculty, University of Zurich, Zurich, Switzerland, [3]Division of Radiation Oncology, Vetsuisse Faculty, University of Zurich, Zurich, Switzerland, [4]Radiotherapy Hirslanden AG, Institute for Radiotherapy, Zurich, Switzerland.

A plethora of treatment options have been described for canine meningoencephalitis of unknown origin (MUE), yet, a gold standard has not been established. The aim of this pilot study was to observe the clinical course of dogs with MUE treated with moderate doses of ionizing radiation, additionally monitoring the response by conventional MRI and MR-spectroscopy.

Six dogs diagnosed with MUE were included in this prospective study. Neurological examination, CSF tap and MRI, including MR-spectroscopy were performed before, at the end and at 3 months later with additional clinical follow-up at 6, 9 and 12 months. Treatment was prescribed to the whole brain, delivered in 10 daily fractions of 3 Gy.

The neurological status of all six dogs improved rapidly and massively during radiation therapy, with 4 of 6 cases returning to normal. MRI follow-up at the last radiation session revealed massive reduction of the perilesional edema and mass effects in all cases, with complete resolution of lesions in 3 cases. Two dogs were euthanized early during follow-up (<3 weeks after therapy). The other 4 dogs are still alive without signs of relapse >6, 9, 12, 12 months after the end of radiation therapy, receiving no anti-inflammatory treatment.

Radiation therapy with moderate doses can provide an additional option for anti-inflammatory treatment of MUE. Advantages of this treatment are rapid improvements without systemic side effects. Radiation therapy provides an additional treatment option that can be preceded or followed with medical treatment, possibly yielding a better and longer control of the disease.

SOLE PREDNISOLONE THERAPY IN CANINE MENINGOENCEPHALITIS OF UNKNOWN ORIGIN: 45 CASES (2006-2012). I. Cornelis[1], L.Van Ham[1], K. Kromhout[2], K. Goethals[3], I. Gielen[2], S. Bhatti[1], [1]Department of Small Animal Medicine and Clinical Biology, [2]Department of Veterinary Medical Imaging and Small Animal Orthopaedics, [3]Department of Comparative Physiology and Biometrics, Faculty of Veterinary Medicine, Ghent University, Belgium.

Meningoencephalitis of unknown origin (MUO) causes many diagnostic, therapeutic and prognostic dilemmas to clinical investigators. Prognosis is considered to be poor if left untreated, and a lot of possible treatment protocols with different immunomodulatory drugs and different median survival times have been reported. Sole prednisolone therapy is described to give poor outcome, with reported median survival times between 28 and 375 days. The aim of this study was to compare the outcome of 3 treatment schedules of sole prednisolone therapy in dogs with MUO.

Forty-five dogs diagnosed with MUO were retrospectively reviewed regarding signalment, neurological signs and response to treatment with prednisolone. Three different prednisolone treatment schedules were compared. The length (3, 8 and 18 weeks), as well as the dose (tapering dose, starting with 1 mg/kg q24 h, 1 mg/kg q12 h, 1.5 mg/kg q12 h, respectively) of the schedules varied. Twenty, 18, and 7 dogs received the respective treatment schedules.

Significantly different survival times were observed in the 3 treatment groups ($P = 0.04$). Overall, no difference in survival time was observed between large, medium and small breed dogs, and female dogs were not significantly over represented. Dogs presenting with multifocal intracranial neurological signs had a significantly shorter survival time compared to dogs with focal intracranial signs ($P = 0.03$). In the latter group, dogs with focal forebrain signs had a significantly shorter survival time compared to dogs with focal brainstem signs ($P = 0.04$). Dogs that survived their initial treatment protocol had a significantly better outcome than those who died or were euthanized during this period ($P < 0.01$). Twenty-three dogs were alive at time of data capture, 22 dogs died, with an overall survival time ranging from 2-2065 days. The surviving dogs had survival times ranging from 120-2605 days.

An 18 week schedule of sole prednisolone therapy can be used as a valuable treatment protocol in dogs with MUO, with the best prognosis for dogs with focal brainstem signs surviving their initial treatment protocol.

A RETROSPECTIVE STUDY OF 225 CASES OF IDIOPATHIC HEAD TREMOR IN DOGS. L. Shell[1], J. Berezowski[2], B. Nibblett[1], P Kelly[1], M. Rishniw[3], [1]Department of Clinical Sciences, [2]Department of Biomedical Sciences, Ross University School of Veterinary Medicine, St. Kitts, [3]Veterinary Information Network, Davis, CA, USA.

Idiopathic head tremor syndrome (IHTS) is considered a benign condition of dogs involving uncontrolled movements of the head, usually in a 'yes' or a 'no' motion. Despite multiple anecdotal reports of IHTS in various dog breeds, only one study has been published (Wolf 2011) which characterized the syndrome in Doberman pinschers. Our retrospective study sought to further characterize IHTS using cases reported on the Veterinary Information Network's (VIN) message boards (VMB) and a survey (VS) of 47,000 VIN members.

There were 225 cases reported (137 from VMB between January 1999 and April 2013 and 88 from VS in April 2013) that satisfied inclusion criteria. In the case of the VMB these criteria included either a submission of a video that demonstrated IHTS to the authors, or the use of key descriptive words (head tremors, head bobs, head bobbing, bobble head, bobble doll) to describe the event and the absence of limb or truncal tremors, an intermittent occurrence of the head tremors, and no mental changes or other neurological signs that could suggest another possible cause of the head tremor. For the VS data, veterinarians were given a description of IHTS signs, including a rotational tremor, and shown 2 videos of cases before taking a survey in which they provided data on suspected cases.

Data from the VMB showed 90% of cases occurred in dogs under 48 months with the age of onset varying from 3 to 120 months (median 24 months). Combined VMB and VS data showed ITHS occurred more frequently in males (60% for VMB; 55% VS) than in females (40% VMB and 45% VS) with the

most commonly affected breed being the Bulldog (37.5% VMB; 39% VS) followed by the Boxer (11% VMB; 14% VS), Labrador retriever (8% VMB; 15% VS), and Doberman pinscher (7% VMB; 10% VS). Direction of head tremor was recorded in 88 VMB cases and in 83 of VS cases with side-to-side ('no') tremor in 46% and 45.4% respectively; up and down ('yes') in 30% and 35% respectively; and rotational (both directions) in 25% and 14%, respectively. Mentation was recorded or observed to be normal in 118 VMB dogs (85.5%).

In summary, our study shows IHTS is often first seen in dogs under 48 months of age. In addition to Doberman pinschers, the condition is also commonly seen in Bulldogs, Boxers and Labrador retrievers. 'No" directional tremors are most common followed by 'yes' and 'rotational tremors'. Most dogs have no other neurological or behavioral abnormalities.

IS ROUTINE ADMINISTRATION OF MRI CONTRAST MEDIA REQUIRED FOR EXCLUSION OF A BRAIN LESION IN DOGS AND CATS? E.J. Ives, N. Rousset, N. Heliczer, A.E. Vanhaesebrouck, The Queen's Veterinary School Hospital, Department of Veterinary Medicine, University of Cambridge, UK.

There are currently no evidence-based guidelines as to the administration of intravenous gadolinium-based contrast media to veterinary patients and their routine use has been questioned for brain magnetic resonance imaging (MRI) of human patients.

The aim of this study was to investigate whether administration of intravenous contrast media alters the likelihood of identifying an MRI-detectable brain lesion in dogs and cats.

Two reviewers independently analysed transverse and sagittal T1-weighted, T2-weighted and fluid-attenuated inversion recovery low-field MRI sequences from 433 dogs and 54 cats for the presence or absence of a clinically-significant brain lesion. All sequences were subsequently reviewed in the same manner with additional access to transverse and sagittal T1-weighted images acquired immediately after administration of intravenous gadolinium-based contrast media.

A previously occult lesion was identified in 6 cases (1.2% of total) by at least one observer due to changes observed only on post-contrast sequences. Central nervous system (CNS) inflammatory disease was the most common diagnosis in these cases (4/6 cases). Inter-reviewer agreement on lesion presence/absence increased from 93% to 96% following access to post-contrast sequences. Interestingly, a total of 14 cases (2.9% of total) changed from the presence to absence of a lesion after inclusion of post-contrast sequences.

The authors conclude that following negative pre-contrast imaging, the administration of intravenous gadolinium-based contrast media is highly unlikely to reveal a previously occult lesion for low-field brain MRI in dogs and cats. However, administration should be considered if there is high clinical suspicion of CNS inflammatory disease and may also aid in the exclusion of a suspected lesion.

POSTER PRESENTATIONS

HORNER'S SYNDROME ASSOCIATED WITH A FRONTAL SINUSITIS IN A DOG. D. Mauler[1], H.de Rooster[1], K. Kromhout[2], A. Rubio-Guzman[1], I. Gielen[2], L.Van Ham[1], [1]Department of Small Animal Medicine and Clinical Biology and, [2]Department of Medical Imaging, Faculty of Veterinary Medicine, Ghent University, Merelbeke, Belgium.

A 9-year-old male entire Weimaraner was examined for a unilateral Horner's syndrome. Three months before presentation the owner noticed a protrusion of the left third eyelid, which progressed gradually to a complete Horner's syndrome with a protruded third eyelid, ptosis, severe miosis and a mild enophthalmos.

Neurologic examination revealed no abnormalities except for a Horner's syndrome on the left side and mild resistance to manipulation of the neck. A cervical spinal cord lesion was suspected. Possible differential diagnoses included cervical intervertebral disk disease, neoplasia and myelitis.

Routine preanesthetic blood examination including complete blood count, serum biochemistry and electrolytes was normal. On MRI including sagittal and dorsal T1-, T2-weighted and FLAIR sequences of the cervical region and the brachial plexi, no abnormalities were detected.

Because of the prolonged course of the disease and the age of the dog, an underlying neoplasia compressing the sympathetic trunk was suspected. Imaging of the whole track of the sympathetic nerve supplying the eye was performed, consisting of a MRI of the head and a CT scan of the thorax. There were no abnormalities detected, except for a ventral hyperintensity in the left frontal sinus on T2 weighted and FLAIR images. The lesion was isointense on T1 weighted images and showed heterogeneous contrast enhancement after IV gadolinium administration. These findings were most compatible with an infectious, inflammatory or neoplastic lesion in the frontal sinus.

A trepanation of the left frontal sinus was performed, and a rigid scope was inserted. At the lateral aspect mucus and tissue flakes were encountered. Biopsies and samples were taken. The frontal sinus was cleared from all debris by flushing. Histopathology, bacteriology and mycology results revealed a sterile inflammation of the frontal sinus. The Horner's syndrome resolved completely 2 days after surgery and did not recur until the last recheck examination 4 months later.

In human medicine, there has been a report about Horner's syndrome associated with ethmoid sinusitis. To the author's knowledge, this report describes the first canine case with frontal sinusitis causing a Horner's syndrome.

SPINAL EPIDURAL EMPYEMA IN THREE CATS: DIAGNOSTIC IMAGING, LABORATORY RESULTS, TREATMENT AND OUTCOME. D.D.A. Lu[1], S.S. Guo[1], J.J.Y. Hui[2], [1]Peace Avenue Veterinary Clinic, Hong Kong, [2]PathLab Medical Laboratories Ltd.

Spinal epidural empyema is rarely reported in cats. Three cases were presented. Case 1 is a 3 month old female entire Exotic cat, presented with a 2-week history of transient pyrexia and non-ambulatory paraparesis. Haematology showed leukocytosis with lymphocytosis. Lumbar myelography demonstrated extradural compression over L1-3 vertebral bodies. CSF Coronavirus PCR was negative. Histopathological examination revealed pyogranulomatous inflammation and reactive new bone formation. Eubacterial FISH (fluorescence *in situ* hybridization) could not identify any bacteria. Case 2 is a 3 year old male neutered Persian cat with progressive paraparesis to non-ambulation over 3 weeks. Skin abscesses were removed in the past. Radiography revealed shortened and fused T13-L1 vertebral bodies resulted from an old spinal injury. Lumbar myelography demonstrated extensive extradural compression from T10 to L2 vertebral bodies. CSF was mildly inflammatory. Surgery revealed extradural purulent material which yielded heavy growth of *Pasteurella multocida*. Case 3 is a 2 year old female neutered DSH cat presented with a tail base wound, paraplegia, equivocal deep pain perception and a flaccid tail. MRI revealed significant cord swelling and extradural compression in the cauda equina, and evidence of connection between the wound and the spine. A scanty growth of *Sphingomonas paucimobilis* was yielded from the wound. Urine, blood and CSF cultures were negative. Repeated MRI 6 days later showed reduction of the extradural lesion and cord swelling. Outcome was excellent in all cases following surgical decompression (Cases 1 and 2) and appropriate antibiosis based on culture and sensitivity tests (Cases 2 and 3). Spinal epidural empyema has to be considered as one of the differential diagnoses in young cats presented with paraparesis, especially with evidence of infection in the past history or on clinical examination.

EVALUATION OF OUTCOME AFTER SURGICAL TREATMENT OF THORACIC HEMIVERTEBRAE WITH VERTEBRAL COLUMN ANGULATION BY SPINAL STAPLING, WITH OR WITHOUT PARTIAL LATERAL CORPECTOMY. M. Charalambous[1], N.D. Jeffery[2], A. Barker[2], G. Hayes[1], E. Ives[1], A.E. Vanhaesebrouck[1], [1]Queen's Veterinary School Hospital, Faculty of Veterinary Medicine, University of Cambridge, Cambridge, UK, [2]Hixson-Lied Small Animal Hospital, College of Veterinary Medicine, Iowa State University, USA.

A retrospective study evaluated the effectiveness of spinal stapling in 7 dogs with clinically significant hemivertebra(e).

All dogs were young, screw-tailed, small breeds with clinical signs ranging from progressive moderate hind limb ataxia to non-ambulatory paraparesis. Four dogs also showed urinary and faecal incontinence. In each dog, one or two thoracic hemivertebra(e) were detected by radiography and MRI. In all dogs hemivertebra(e) were associated with vertebral column angulation and a reduction in spinal canal diameter. All dogs were surgically managed using a spinal stapling technique, spanning over a minimum of ten vertebrae, combined with partial lateral corpectomy in 2 dogs.

All dogs showed gait improvement following surgery, with resolution of incontinence if present. Two to three years later, only a residual, subtle ataxia could be detected on examination for all dogs. In one dog, clinical signs re-occurred three years post-operatively due to pressure necrosis of the dorsal spinous processes and implant loosening.

In conclusion, this is the first case series to describe the long-term outcome following the use of spinal stapling in small dogs with hemivertebra(e) and vertebral column angulation. Spinal stapling is a relatively simple surgical procedure, with minimal risk of iatrogenic damage to the spinal cord, and may act as an internal brace to minimize further angulation in young dogs or further spinal cord damage due to chronic instability in mature dogs. The presence of residual, mild neurological deficits is similar to previous reports and could be the result of associated spinal cord malformations or pre-operative chronic spinal cord compression. Although these initial findings are promising, further adaptations may be required to avoid implant loosening in the long-term, or to allow continued growth if used in immature dogs.

FOREIGN BODY REACTION CAUSED BY SURGICAL USE OF POLYMETHYLMETHACRYLATE (PMMA) IN A DOG. D. Mauler[1], M. Tshamala[1], L. Bosseler[2], V. Volckaert[3], L.Van Ham[1], [1]Department of Small Animal Medicine and Clinical Biology, [2]Department of Pathology, Bacteriology and Poultry Diseases, [3]Department of Medical Imaging, Faculty of Veterinary Medicine, Ghent University, Merelbeke, Belgium.

An 8 year old male entire Magyar Vizsla was presented for an acute soft tissue swelling dorsal to the spine which occurred 4 days after vigorous exercise. The dog had a history of a vertebral stabilization surgery with pins and polymethylmethacrylate (PMMA) for a vertebral fracture of L1 and L2 seven years ago with an uneventful recovery and normal neurologic status post-operatively.

On physical examination a large, fluctuating, normothermic swelling dorsally to the cranial lumbar spine that was painful on palpation was detected. Neurologic examination was normal. On spinal radiographs, the vertebral PMMA construction had a similar appearance compared to the postoperative images seven years ago. A large soft tissue swelling was visible at the level of T13-L3. A fine needle aspiration was performed and cytology revealed a foreign body reaction with large mononuclear cells without signs of bacteria or neoplastic cells.

The region was surgically explored and a large amount of serous fluid was aspirated. The PMMA was removed, biopsy samples from the surrounding soft tissues were taken and the area was vigorously flushed. The dog made a full recovery over the following week.

Histopathology showed a pyogranulomatous inflammatory reaction characterized by the presence of neutrophils, lymphocytes, plasma cells and macrophages, some of which were multinucleated. Additional stainings could not identify any bacteria, fungi or protozoa. A bacteriologic culture was not performed.

Infections after insertion of PMMA have been reported, but to our knowledge this is the first report of a foreign body reaction associated with PMMA. We hypothesize that due to the exercise a small part of the PMMA loosened and induced an inflammatory response. The long time between stabilization surgery and occurrence of the swelling, in combination with the absence of infectious agents on histological examinations, makes a bacterial infection very unlikely. However, because a bacterial culture is lacking, we cannot completely exclude this cause.

PMMA is widely used in vertebral stabilization surgeries, but if signs of a foreign body reaction occur, it might be indicated to remove it.

SCIATIC T-CELL NEUROLYMPHOMATOSIS IN A DOG. A. Rupp, E. Ives, H. Rudorf, A.C. Palmer, F. Constantino-Casas, Department of Veterinary Medicine, University of Cambridge, Cambridge, UK.

Neurolymphomatosis is rarely reported in dogs and cats, and in the majority of cases the lymphocytic infiltrates are found within the cranial nerves and nerve roots. A recent report documents B-cell infiltration of the femoral nerve in a dog, whilst sciatic B-cell infiltration has been reported in one cat.

A 4.5 year-old female cross-breed dog presented with a one week history of anorexia, weight loss, tenesmus, tail flaccidity and progressive monoparesis of the right hind limb. Ultrasound imaging revealed a thickened gastric wall with loss of its normal layering and a large mass at the root of the mesentery. MRI showed an enlargement of the right L7 spinal nerve root and thickening of the proximal portion of the right sciatic nerve. Incisional biopsies of the gastric wall and mesenteric lymph nodes were consistent with malignant lymphoma.

At necropsy, multiple thickened areas were found within the gastric wall and the mesenteric lymph nodes were confluent and enlarged. The right L7 spinal nerve root and the proximal portion of the right sciatic nerve were also enlarged and reddened. Histology of stomach, mesenteric lymph nodes, right L7 spinal nerve root and right sciatic nerve revealed marked infiltration with sheets of CD3-positive cells, consistent with a T-cell lymphoma. Mild to moderate bilateral lymphocytic infiltrates were also found in more caudal spinal nerve roots, adrenal gland, ganglion coeliacum and associated nerves within the mesentery. The spinal cord appeared to be free from lymphocytes.

The possibility of T-cell lymphoma should be included as a differential diagnosis in cases of canine peripheral nerve dysfunction and enlargement.

SEQUENTIAL MAGNETIC RESONANCE IMAGING AND CEREBROSPINAL FLUID ANALYSIS IN A DOG WITH SUSPECTED ACUTE DISSEMINATED ENCEPHALOMYELITIS. H. Ueno[1], S. Suwa[2], K. Miyoshi[1], [1]Division of Small Animal Clinical Sciences, Department of Veterinary Medicine, Rakuno Gakuen University, Ebetsu, Hokkaido, Japan., [2]Hokkaido Guide Dog Association, Sapporo, Hokkaido, Japan.

A neutered male 1-year-old, 25 kg, Labrador Retriever was presented with the central nervous system (CNS) symptoms such as generalized seizures, reduced consciousness mind level, ataxia and partial seizures of left forelimb (day 1). The dog was vaccinated twenty-nine days ago. Until day 15, these symptoms gradually improved and completely disappeared.

On day 15, magnetic resonance imaging (MRI) of brain, there were increased intensity of the lesions on T2-weighted (T2W) and fluid-attenuated inversion recovery (FLAIR) images in frontal and parietal lobe. In cerebrospinal fluid (CSF) analysis, although pleocytosis and increased total protein concentration (TP) were not revealed, anti-glial fibrillary acidic protein (GFAP) autoantibody increased. Clinically, although anti-GFAP autoantibody increased, both necrotising meningoencephalitis (NME) and brain tumour were not guessed, because of a single and non-progressive episode of clinical symptoms, MRI findings and CSF findings. Therefore, the dog was observed without any medications. Hence, follow-up CSF analysis and MRI were performed on days 50 and 190. In CSF analysis, slight mononuclear pleocytosis (7.67 cells/μL), the increase of TP concentration (32.7 g/L) and anti-GFAP autoantibody showed on days 190. On the other hand, visible lesions were reduced on serial MRI and the clinical course was satisfactory without any medication. At the time of writing (on day 370), the dog remains alive without any clinical problems.

Clinically, post-vaccination acute disseminated encephalomyelitis (ADEM) in human is guessed in the following case; (1) The period from a vaccination to symptoms; post-vaccination ADEM may occur up to three months after the vaccination. (2) Acute onset and a single attack or episode of CNS symptoms. (3) Characteristic MRI finding; the increasing intensity of the lesion on T2W and FLAIR images, which were suggesting demyelination. This report describes a dog with an acute onset and a single episode of CNS symptoms. The symptoms occurred on twenty-nine days after the vaccination. On MRI, there were increased inten-

sity of the lesions on T2W and FLAIR images in frontal and parietal lobe. Therefore, the present dog was suspected of having post-vaccination ADEM clinically. To the authors' knowledge, post-vaccination ADEM has not been previously reported in the dog. In light of this case, post-vaccination ADEM should be considered as a differential diagnosis for cases with clinical findings that may suggest neuropathy with characteristic clinical course and MRI finding after vaccination.

BRAINSTEM AUDITORY EVOKED RESPONSE IN NORMAL HEARING CLIENT-OWNED FERRETS. S. Piazza, L. Cauzinille, FREGIS Veterinary Hospital, Arcueil, France.

Congenital pigment-associated deafness is a condition currently being debated by pet ferret breeders, but scientific data are lacking.

The objective of this study was to describe a method to evaluate Brainstem Auditory Evoked Response (BAER) of the normal ferret and establish baseline data for future investigations of deafness in this species.

BAER measurements were recorded from 28 clinically normal ferrets under general anaesthesia with subcutaneously located needle electrodes for both ears. A "click" sequence applied by earphone with an intensity of 90 dB was used. The final BAER curve represents an average of 500 successive stimulations.

The BAER curve of the normal ferret included 4 reproducible waves named I, II, III and V, as established in dogs and cats. The mean amplitude ± SD, expressed in microvolts (μV), was 1.1 ± 0.09; 1.9 ± 0.11; 2.9 ± 0.20; 3.8 ± 0.21, respectively for peak I, II, III and V. The mean latency ± SD, expressed in milliseconds (ms), was 1.6 ± 0.36 for peak I.

In the present study, we established a reliable method for clinical evaluation of the hearing function in the pet ferret. It can be easily performed in a clinical setting, as early as 8 weeks of age. The congenital deafness in ferrets appears to be an emerging problem, especially in ferrets with a white coat. This audiometric method has a definite interest in screening for congenital deafness and will be used in the near future to determine the incidence of this defect in the pet ferret population.

DO FEMALE DOGS SUFFER CATAMENIAL EPILEPSY? S.Van Meervenne[1,2], H. Volk[3], L.Van Ham[2], [1]Läckeby Djursjukhus, Läckeby, Sweden, [2]Ghent University, Faculty of Veterinary Medicine, Department of Small Animal Medicine and Clinical Biology, Merelbeke, Belgium, [3]Department of Clinical Sciences and Services, Royal Veterinary College, London, UK.

Catamenial epilepsy in humans is defined as changes in seizure frequency over the course of the menstrual cycle. Approximately one third of women with intractable complex partial seizures may have catamenial epilepsy. Three hormonally based patterns of seizure exacerbation have been recognized. The aim of this study was to evaluate whether there might be a similar clinical pattern in intact bitches with idiopathic epilepsy.

Clinical records of intact bitches with a diagnosis of seizures or idiopathic epilepsy were collected from 1st of October 2008 till 31st of December 2012 at Läckeby Djursjukhus, a small animal hospital in Sweden. Dogs fulfilling the following criteria were included: suffering recurrent seizures, onset between 6 months and 6 years of age with a normal interictal general clinical examination and neurological examination, normal laboratory blood results including complete blood cell count and routine biochemistry (including electrolytes and ammonia). Magnetic resonance imaging (MRI) of the brain to exclude symptomatic seizures was desired but not required as inclusion criteria. Patients being older than 6 years had to survive more than one year without developing additional neurological clinical signs to be included. The stage of the estrous cycle reported by the owner and/or recorded by the veterinarian at the time of the first seizure was noted.

A total of 109 dogs presented with seizures, 49 dogs were diagnosed as suffering idiopathic epilepsy, 25 dogs had symptomatic or reactive seizures, 16 dogs were lost in follow up and 19 were wrongly diagnosed. Of the 49 intact bitches with idiopathic epilepsy, 18 (37%) had their first seizure when in heat, but only one patient in the symptomatic/reactive seizures group was in heat during the first seizure episode ($P = 0.002$). Ten of the bitches with idiopathic epilepsy had seizures recurring in relation to their estrous cycle.

These findings suggest an association between estrus and onset of seizures in intact bitches with idiopathic epilepsy, not only in the beginning of the disease, but in more than half of the patients also during the further course of their epilepsy. This could be caused by the pro-convulsive effects of estrogen. Early castration, however, might not be as beneficial to these patients, as the female dogs might then be deprived of the protective effect of progestagens. Although, dogs with idiopathic epilepsy are more likely to have their first seizure associated with estrus compared to patients with symptomatic and reactive seizures, further prospective, longitudinal studies are needed to monitor intact bitches with idiopathic epilepsy over time, including regular measurements of serum sex hormones concentration.

ANTIEPILEPTIC DRUG WITHDRAWAL IN DOGS WITH IDIOPATHIC EPILEPSY. F.K. Gesell[1], S. Steinmetz[1], W. Löscher[2], A. Tipold[1], [1]Department of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover, Germany, [2]Department of Pharmacology, Toxicology and Pharmacy, University of Veterinary Medicine Hannover, Germany.

Epilepsy is one of the most common neurological disorders in dogs and is treated by chronic administration of antiepileptic drugs (AEDs). In humans with epilepsy, it is common clinical practice to consider drug withdrawal after a patient has been in remission (seizure free) for three or more years, but withdrawal is associated with the risk of relapse.

In the present study, the consequences of AED withdrawal were studied in dogs with epilepsy. Therefore, 200 owners of dogs with idiopathic epilepsy were contacted by telephone interview. In 11 cases the therapy had been stopped after the dogs had become seizure free for a median time of 1 year. Reasons for AED withdrawal were appearance or fear of adverse side effects, financial aspects and the idea that the medication could be unnecessary. Following AED withdrawal, 4 of these dogs remained seizure free, 7 dogs suffered from seizure recurrence, of which only 3 dogs could regain seizure freedom after resuming AED therapy.

These results show that different outcomes after drug withdrawal in dogs with idiopathic epilepsy are to be expected. Our study gives a hint that similar numbers as in human patients are found, and the data can help owners of epileptic dogs and the responsible clinician to decide when and why to stop antiepileptic medication. Seizure recurrence was observed in the withdrawal period itself and the immediate months afterwards. Owners should control their dogs very carefully in this time period in order to recognise seizure recurrence.

DEVELOPMENT OF A DOG MANIKIN-BASED SIMULATOR FOR EPIDURAL PUNCTURE AND ATLANTOOCCIPITAL CSF COLLECTION. Y.W. Lin[1], M. Lüpke[2], A. Tipold[1], J.P. Ehlers[3], M. Dilly[4], [1]Department of Small Animal Medicine and Surgery, [2]Institute for General Radiology and Medical Physics, [3]Competence Centre for e-Learning, Didactics and Educational Research in Veterinary Medicine, [4]Clinical Skills Lab, University of Veterinary Medicine Hannover, Germany.

Cerebrospinal fluid (CSF) analysis is considered as one of the 10 most important job competencies for diplomates/residents of the European College of Veterinary Neurology. Certain risks are associated with epidural and atlantooccipital CSF puncture. Epidural simulators have been developed since 30 years in human medicine and are also used in veterinary medicine training. However, simulators for atlantooccipital CSF collection training are with one exception not well established. To improve teaching of special skills, a canine simulator for epidural and CSF puncture was created at the Clinical Skills Lab of the University of Veterinary Medicine Hannover.

A commercial stuffed dog toy was used as the external layer of the simulator. The main structures comprised artificial vertebrae made of polyurethane casting resins from a CT scan and 3D-printed model. A multilayer pad mimic skin, fat tissues and muscles and a double walled silicone tube were inserted in the spinal canal of the atlantooccipital region rep-

resenting dura mater, subarachnoidal space and spinal cord and providing tactile feedback during puncture. Two compartments in the double walled tube can be filled with clear and coloured liquid to provide feedback, if CSF was gained or central nervous tissue penetrated. In the lumbar region, a mono-walled silicone tube connected to a syringe to produce partial vacuum was used. Students get feedback reaching the epidural space, when a drop of saline disappears above the needle. Besides, the atlantooccipital joint and the limbs of the dog are flexible that trainees are able to learn the correct positioning of the animal.

This manikin-based simulator for veterinary medicine was tested by ECVN diplomates and residents and can be considered to be a useful prototype for clinical skills labs.

CONGENITAL MALFORMATIONS RESEMBLING VACTERL ASSOCIATION IN A GOLDEN RETRIEVER. A. Gamito-Gomez[1,2], R Gutierrez-Quintana[2], A Wessmann[2,3], [1]Teaching Veterinary Hospital, University of Córdoba, Córdoba, Spain, [2]School of Veterinary Medicine, College of Medicine, Veterinary Medicine and Life Sciences, University of Glasgow, Glasgow, UK, [3]Neurology Service, Pride Veterinary Centre, Derby, UK.

A two-month-old Golden retriever presented with malformation of the left thoracic limb and a small circular indentation of the skin in the cranial thoracic spine. Radiographs showed a cleft between the second and fifth metacarpal bones of the left thoracic limb compatible with ectrodactyly and spina bifida affecting T4 and T5 vertebrae. Magnetic resonance imaging of the thoracic spine showed dorsal reposition of the spinal cord and a tract connecting from the dura mater to the skin. No other malformations were detected. Surgical excision of the tract was performed and histopathological examination diagnosed a dermoid sinus type IV.

Dermoid sinus and spina bifida are well-recognised congenital spinal and spinal cord defects, yet association with other congenital malformation are rarely reported in dogs. The here reported dog had spinal and spinal cord abnormalities with concurrent limb malformation, which are two components of a group, described in people as VACTERL association, a non-random association of birth defects. To the authors knowledge this is the first report describing concurrent dermoid sinus type IV, spina bifida and ectrodactyly in a dog and highlights the importance of patient examination for occurrence of multiple malformations to provide an appropriate prognosis for an owner.

IMMUNOCYTOCHEMISTRY OF CEREBROSPINAL FLUID IN THE ANTE-MORTEM DIAGNOSIS OF FELINE INFECTIOUS PERITONITIS INVOLVING THE CENTRAL NERVOUS SYSTEM. E.J. Ives, F. Cian, A.E. Vanhaesebrouck, The Queen's Veterinary School Hospital, Department of Veterinary Medicine, University of Cambridge, UK.

A 4-month-old female entire domestic shorthair cat presented with an acute onset of blindness, tetraparesis and subsequent generalised seizure activity.

Haematology and serum biochemistry demonstrated a moderate, poorly regenerative anaemia, hypoalbuminaemia and hyperglobulinaemia with a low albumin:globulin ratio. Serology for feline coronavirus antibody was positive with an elevated alpha-1 acid glycoprotein. Analysis of cisternal cerebrospinal fluid (CSF) demonstrated a markedly elevated protein and a mixed, predominately neutrophilic pleocytosis. Immunocytochemistry for feline coronavirus was performed on the CSF with positive staining observed inside macrophages. The cat was subsequently euthanized, and both histopathology and immunohistochemistry were consistent with a diagnosis of feline infectious peritonitis.

This is the first reported use of immunocytochemistry for detection of feline coronavirus within CSF macrophages. If this test proves highly specific, as for the identification of feline coronavirus within tissue or effusion macrophages, it would be strongly supportive of an ante-mortem diagnosis of feline infectious peritonitis in cats with central nervous system involvement, without the need for biopsy.

REVERSIBLE PARALYSIS AND LOSS OF DEEP PAIN SENSATION AFTER TOPICAL INTRATHECAL MORPHINE ADMINISTRATION. Y. Chamisha, M. Shamir, Y. Merbl, O. Chai, Department of Neurology and Neurosurgery, Koret School of Veterinary Medicine, the Hebrew University of Jerusalem, Israel.

Direct extradural administration of morphine has been shown to provide effective and safe postoperative analgesia in dogs undergoing thoracolumbar spinal decompressive surgery for intervertebral disc extrusion. The analgesic effect of local opiate administration in the spinal cord is mediated by opioid receptors located in the dorsal gray column. In intrathecal administration of morphine, dose-dependent side effects including respiratory depression, pruritus, lethargy and urinary retention have been reported. No information is currently available about adverse effects of topical intrathecal morphine in dogs.

Here, we describe two cases of dogs undergoing laminectomy and durotomy that were treated with morphine-soaked gel foam (0.5 mg/kg) placed directly over the dural defect. A five-year-old castrated male French bulldog was operated for subarachnoid diverticulum at T9-T10 and a 9.5-year-old castrated male Belgian Shepherd dog underwent hemilaminectomy at T12-T13 for herniated disc with intradural component. During recovery, both dogs exhibited paralysis and loss of deep pain sensation in the pelvic limbs. Following intravenous naloxone administration (0.02 mg/kg), both dogs immediately regained ambulation and normal pain sensation. This effect was temporary and lasted for a few hours. Permanent resolution of the paralysis and loss of deep pain sensation was achieved in both dogs.

Since in both dogs the clinical signs were resolved by administration of the specific opiate antagonist naloxone, the adverse effect was attributed to morphine overdose rather than to the additive in the morphine solution or to a neurotoxicity effect. Although the adverse effect was reversible, topical intrathecal morphine administration (0.5 mg/kg) should be avoided until the efficacy and adverse effects of this treatment on the motor and nociceptive-related pathways are determined.

FELINE TEMPORAL LOBE EPILEPSY. WHAT CAN WE LEARN FROM EARLY EXPERIMENTAL RESEARCH? A. Pakozdy[1], A. Klang[2], S. Kitz[1], M. Wrzosek[3], P. Halasz[4], [1]Clinic for Internal Medicine, University of Veterinary Medicine, Vienna, Austria, [2]Institute of Pathology and Forensic Veterinary Medicine, University of Veterinary Medicine, Vienna, Austria, [3]Department of Internal Disease with Clinic of Horses, Dogs and Cats, Faculty of Veterinary Medicine, Wroclaw University of Environment and Life Science, Poland, [4]Institute of Experimental Medicine, Budapest, Hungary.

There is growing evidence that epileptic seizures originating from temporal lobe occur in cats. Such cats show clinically complex partial seizures (CPS) with orofacial automatism: salivation, facial twitching, lip smacking, chewing, licking, swallowing and motor arrest, vegetative and emotional signs also may occur[1]. However, with electroencephalography (EEG), it is theoretically possible to detect epileptiform discharges it is currently not a routine procedure for cats and no consensus exists regarding electrode placement, restrain technique and interpretation. The consequence is that the "abnormal excessive or synchronous neuronal activity in the brain" usually cannot be confirmed, so whether an episodic event is epileptic or not, can only be suspected based on clinical, laboratory and neuroimaging findings.

For a period of time cats were frequently used for neurophysiological experiments and electrophysiological studies. From this research area originates important basic knowledge about epilepsy, but this knowledge was rarely cited in the clinical veterinary studies. The aim of our study was to summarize earlier experimental studies on feline temporal lobe epilepsy.

Kaada (1951) found that after electrical stimulation of different rhinencephalic structures, rhythmic chewing movement can be produced by the stimulation of rostral pyriform cortex and amygdala, swallowing and licking movement originate from olfactory tubercle. Using alumina cream injection into amygdaloid complex, hippocampus and pyriform cortex Gastaut et al (1959) observed that 3-5 weeks later animals started with

psychomotor seizures. Typical psychomotor seizure included: pupillary dilatation, clonic jerking of vibrissae and facial muscles, contralateral head turn accompanied by mastication and salivation.

Delgado and Sevillano (1961) found that the feline hippocampus after discharge (HAD) following electrical stimulation does not elicit any motor signs. When HAD spread to the amygdala than facial twitching could be observed which was usually localized ipsilaterally to the side of stimulation. When the amygdala was stimulated similarly ipsilateral face movement could be observed. The authors concluded that the facial motor effect, such as twitching of the eyes and movement of the vibrissae, lips and face, often observed during hippocampal seizure, are not depend on the hippocampal stimulation, but rather on the active participation of the amygdala, as fundamental part of the limbic system. Staring and looking around was related to hippocampal involvement. Majkowski et al. (1976) described seizures in an experiment with alumna cream induced epileptic focus on cats after callosotomy. When discharges reached the temporal lobe, animals developed clinically partial facial motor seizures.

For temporal lobe epileptic seizures staging system was established by Wada et al. (1974) for cats regarding of the seizure spreading. Stage 1 - represents ipsilateral facial twitching, stage 2 - bilateral facial twitching, stage 3 - head nodding, stage 4 - tonic extension of contralateral forelimb or contralateral head turning with rapid circling, stage 5 - clonic jumping while standing, and stage 6 – falling down with generalized convulsion.

Experimental research on cats results suggest that ictal clinical signs originating from the temporal lobe can be divided into autonomic, somatic and behavioural symptoms. The main ictal signs include mydriasis, salivation, urination, defecation, piloerection (autonomic); licking, chewing, facial twitching, tonic adversion, tonic stiffening (somatic); and alerting, fear, and rage (behavioural).

Experimental research data are in agreement with clinical observation regarding ictal clinical signs of epileptic temporal lobe seizures in cats. Based on clinical and experimental data seems that temporal lobe epilepsy representing a special clinical entity with unique seizure type.

ELECTROENCEPHALOGRAPHY IN FOUR EPILEPTIC AND IN EIGHT NON-EPILEPTIC HORSES UNDER DETOMIDINE SEDATION. S. Steinmetz[1], J.M. Cavallerib, K. Feige[2], A. Tipold[1], [1]Department of Small Animal Medicine and Surgery, [2]Department of Equine Medicine and Surgery, University of Veterinary Medicine Hannover, Hannover, Germany.

In the current study electroencephalographic abnormalities in three horses with presumed idiopathic epilepsy and in one horse with posttraumatic epilepsy (PTE) were evaluated and compared to values of eight non-epileptic horses. The horses with presumed IE had interictally no abnormalities in physical, neurological and laboratory examinations, MRI (n = 3), CT (n = 1) and CSF analysis (n = 3) were unremarkable in the horses with presumed IE, in PTE a skull lesion and corresponding brain haemorrhage was detected. The four horses with epilepsy [(#1) PTE, (#2,#3,#4) IE] and eight non-epileptic horses underwent EEG examination (NicoletOne nEEG, Viasys healthcare Inc.) under Detomidine sedation. Qualitative (visual) and quantitative (Fast Fourier Transformation) EEG analysis were accomplished.

The horses were sedated with 0.01 mg/kg i.v. alpha-2-adrenergic agonist Detomidine. Five subdermal EEG electrodes (O1, O2, Cz, F3, F4), a ground electrode and a reference electrode were inserted as described by Lewin (1998). The recordings were performed with a sensitivity of 70 μV/cm; time constant 0.3 seconds; Hf 70 Hz, Lf 0.5 Hz; notch filter inserted; impedance of all electrodes < 10 kΩ. After three minutes of recording, a "four minute photic stimulation" followed. The photic stimulator was placed 20 cm in front of the forehead and every five seconds the stimulation was performed for 8 seconds (flash interval). The flash frequency was increased from 5 Hz to 50 Hz and decreased from 50 Hz to 5 Hz. After the photic stimulation a "three minute post stimulating phase" was recorded. For the qualitative analysis monopolar and bipolar montages were used. For the quantitative analysis, thirty times two-second artefact free periods were visually chosen.

In horse #1 (PTE) isolated sharp waves, slow waves and spike-wave complexes were visually detected. Although the MRI examination revealed a cerebral haemorrhagic defect of the right parietal lobe due to an impression fracture, EEG abnormalities did not represent this focus. Horses #2-4 displayed changes in wave frequency and amplitudes, sleep spindles and rarely spikes and slow wave complexes. The background activity was dominated by delta activity. The eight non-epileptic horses showed frequently sleep spindles and single spontaneously occurring changes in wave amplitudes and wave frequency superimposed on background activity and single spikes. In one non-epileptic horse photic driving was detected. In conclusion, no significant differences between EEGs in epileptic and non-epileptic horses could be detected in this study under Detomidine sedation despite photic stimulation.

LYMPHOSARCOMA INVOLVING MULTIPLE NERVE ROOTS IN A CAT. S. Piazza, E. Gomes, L. Cauzinille, FREGIS Veterinary Hospital, Arcueil, France.

A 7-years-old, neutered female domestic shorthair had a 5-weeks history of progressive right pelvic limb lameness, non responsive to non-steroidal anti-inflammatory therapy.

At the time of referral, proprioception, voluntary movements and nociception were absent on the right pelvic limb and withdraw reflex was absent with severe flexor muscles atrophy. The cat had slightly reduced placing and hopping responses on the left thoracic limb, temporal muscle atrophy, facial sensory loss, and Horner syndrome, all left sided. This was consistent with multifocal lesions, at least involving the right sciatic nerve and the left part of the brainstem or associated cranial nerves.

Magnetic resonance imaging (MRI) of the brain revealed a severe enlargement and contrast enhancement of the left trigeminal nerve as it enters the trigeminal canal, with important mass effect on the brainstem and the diencephalon. Fluid material was present within the left tympanic bulla, with peripheral contrast enhancement, consistent with otitis media. MRI of the lumbar spine revealed an enhancing tumefaction of the right nerve roots L6-L7 and L7-S1 associated with mass effect into the vertebral canal.

Cerebrospinal-fluid analysis demonstrated moderate lymphoid pleocytosis with 20% lymphoblasts.

Fine-needle aspirate cytology of the enlarged sciatic nerve was suggestive of lymphosarcoma.

The cat was euthanized at the owner request and histopathology confirmed the diagnostic of lymphosarcoma.

CONGENITAL INTERNAL AND EXTERNAL HYDROCEPHALUS IN A DOG. L. Espino, G. Santamarina, M.L. Suarez, A. Goicoa, N. Miño, J.D. Barreiro, Hospital Universitario Veterinario Rof Codina, Universidad de Santiago de Compostela, Lugo, Spain.

Hydrocephalus is defined as an abnormal accumulation of cerebrospinal fluid (CSF) within the cranium with subsequent dilation of the ventricular system. There are numerous classification schemes for hydrocephalus and depending on the location of the accumulated CSF, hydrocephalus is classified as internal or external. Internal hydrocephalus is the most congenital anomaly of the nervous system in dogs; however, external hydrocephalus is occasionally described in humans but it was only reported in one dog and two cats. The purpose of this report is to describe the historical, physical, diagnostic findings, and treatment of a case of congenital internal and external hydrocephalus in a dog.

A 3-month-old, male non-castrated, Yorkshire terrier was examined because of a history of abnormal behavior and visual impairment since the dog was adopted from an animal protection society. Upon physical examination, it was noted that the dog was smaller than the average, he had a large dome-shaped calvarium with several open fontanelles and showed ventrolateral bilateral strabismus. Abnormal findings on neurologic examination included, altered mental status ranging from depression to hyperexcitability, reduced postural reactions in all four limbs and blindness with absent menace response in both eyes. The neuroanatomic diagnosis was a forebrain lesion. An ultrasonographic study was performed through a persistent dorsal midline fontanelle. The lateral ventricles appeared enlarged and fused as a single cavity, the cortex was narrow and ring shaped and the brain

was surrounded by a large quantity of anechoic fluid. Computed tomography revealed a severe dilation of lateral ventricles, widened, open fontanels were evident and between the skull and the neocortex, there was a moderate accumulation of fluid isodense to CSF considered to be an enlarged subarachnoid space. CSF analysis did not show any abnormality. Based on these findings, a presumptive diagnosis of congenital internal and external hydrocephalus was made. The owner declined surgical placement of a ventriculoperitoneal shunt and medical therapy with oral prednisone was started.

The term external hydrocephalus is used to describe enlargement of the subarachnoid space with mild to moderate or not ventricular dilatation. External hydrocephalus as a primary entity has been only reported in a dog and two cats and bacterial meningoencephalitis was suspected to be the cause in the dog and may have played a role in one cat. However, the simultaneous presentation of internal and external hydrocephalus has not been previously described. The fluid dynamics responsible for the development of external hydrocephalus are poorly understood. Agenesis or occlusion of the arachnoid villi due to meningitis or developmental defect can result in dilatation of the ventricles and the subarachnoid space. In our case, a congenital origin was supposed due to the absence of an underlying cause.

SYRINGOMYELYA SECONDARY TO BRAIN MASSES IN 19 DOGS; CLINICAL AND MAGNETIC RESONANCE IMAGING FINDINGS. S. Ródenas[1,2,3], C.de la Fuente[1], K. Marioni-Henry[3], S. Añor[1], J Ezquerra[4], J.R.E.del Castillo[2], [1]Departament de Medicina i Cirurgia Animal. Facultat de Veterinària. Universitat Autònoma de Barcelona, Bellaterra, Spain, [2]Faculté de Médicine Vétérinaire, Université de Montréal, Quebec, Canada., [3]Southern Counties Veterinary Specialists, [4]Facultad de Veterinaria de Caceres, UEX.

Syringomyelia (SM) associated with posterior fossa tumours has a reported incidence of 5-21% in people, with only sporadic reports of SM associated with supratentorial masses. In veterinary medicine, only 4 single case reports of SM secondary to intracranial masses in dogs have been published. In both, humans and dogs with SM secondary to brain masses, clinical signs associated solely with SM are uncommonly reported.

The purpose of this study was to describe the magnetic resonance imaging (MRI) and clinical findings in a population of dogs with SM associated with brain masses. Medical records from 4 institutions were retrospectively reviewed from 2006-2012, and 19 affected dogs were identified. Medical records of 22 dogs with brain masses and no SM were used as controls. We hypothesized that: (1) cerebellar vermis herniation predisposes to the development of SM, (2) size and localization of the brain mass, severity of edema, presence of hydrocephalus and mass effect predispose to the development of foramen magnum (FM) herniation and SM, and (3) size of the syrinx predisposes to the development of symptomatic SM. The following MRI features were assessed: presence of FM and caudal transtentorial (CTT) herniation, location of the mass, skull and mass volume, edema, mass effect and hydrocephalus. Clinical signs were scored as: (0) clinical signs referable to brain alone, (1) predominance of brain signs versus spinal cord signs (2) predominance of spinal cord signs, and (3) spinal cord signs alone. An exploratory analysis was performed to identify potential risk factors and stepwise multivariate logistic regressions were performed to confirm the variables that contributed to the risk of the positives outcomes. Foramen magnum herniation was present in 78% of dogs in the SM group and in 22% of dogs in the control group. Dogs with FM herniation had significantly increased odds of SM (odds ratio (OR=16.093, 95% C.I. = [2.690, 96.262]) and dogs with dilated 3rd ventricle had increased odds of SM (OR = 20.872, 95% C.I. = [1.556, 279.90]). Presence of CTT herniation increased the odds of FM herniation (OR = 11.225, 95% C.I. = [2.066, 60.992]). There was no association between mass location and development of SM (cranial rostral fossa, 52.3%; CCF, 47.3% of dogs with SM). Presence of moderate to severe mass effect was associated with decreased odds of spinal cord signs (OR = 0.114, 95% C.I. = [0.010, 1.356]). This study showed that the main risk factor for developing SM secondary to a brain mass is the presence of FM herniation. Presence of a dilated 3rd ventricle was also associated with development of SM in dogs with brain masses. Finally, development of SM appeared to be independent of the mass location within cranial or caudal fossa.

DOES MONOCYTE RECRUITMENT TO THE CNS DURING ACUTE CANINE DISTEMPER VIRUS INFECTION INFLUENCE THE PATHOGENESIS OF THE DISEASE? M. Martin[1], R. Carlson[1], A. Tipold[1], W. Baumgärtner[2], V.M. Stein[1], [1]Department of Small Animal Medicine and Surgery, [2]Institute of Pathology, University of Veterinary Medicine Hannover, Hannover, Germany.

In acute canine distemper virus (CDV) infection microglia cells are activated and change their immunophenotype and functionality. They can release reactive oxygen species (ROS) which is in low concentrations necessary for repair and defense mechanisms. However, in high concentrations ROS are cytotoxic, particularly to oligodendrocytes which are damaged as innocent bystanders resulting in demyelination. During this phase of infection recruitment of peripheral blood monocytes into the central nervous system is reported. However, the function of these cells is not known. To prove the hypothesis that CDV-infected monocytes may contribute to the demyelination by their potential to generate ROS *ex vivo* isolated monocytes were infected with different CDV strains and ROS generation was measured by flow cytometry.

Density gradient centrifugation of 10 ml whole blood from 10 healthy Beagles was performed to isolate monocytes. Either a CDV-vaccination strain (Onderstepoort, Ond) or a virulent CDV-strain (R252) was used to infect the monocytes. Non-infected monocytes served as a negative control (Ctr). Measurements evaluating the infection rate, ROS generation (+/- stimulation with PMA) and CD14 expression were performed via flow cytometry directly and 3 hours post infection (p.i.).

Monocytes could be infected with both CDV-strains but the percentage of infected monocytes was twice as high in the R252- (mean value, mv = 61.2%) compared to the Ond- (mv = 32.5%) infected monocytes. Moreover, the virus load was higher with R252 (mv = 247.7) compared to the Ond infection (mv = 108.2). The expression of the surface molecule CD14 was up-regulated in the infected monocytes compared to the Ctr and the expression intensity of CD14 was 2.5-fold higher directly p.i. (mv Ctr mv = 659; OND mv = 1624; R252 mv = 1648) and 4.8-fold higher 3 hours p.i. (Ctr mv = 289; OND mv = 1424; R252 mv = 1379) compared to the Ctr. ROS was generated by infected and non-infected monocytes, and enhanced in the triggered groups at both time points (directly p.i. < 3 h p.i.). Intriguingly, the R252-infected monocytes generated less ROS than the OND-infected and non-infected monocytes.

In conclusion, both CDV-strains were capable to infect a high proportion of isolated monocytes thereby activating the innate immune system through the CD14/TLR4-dependent pathway. The hypothesis could be proven that CDV infection stimulates monocytic ROS generation and therefore might contribute to demyelination in CDV infection. However, the ROS generation intensity was lower in R252-infected monocytes compared to their potential becoming evident in the OND- and non-infected monocytes. This might reflect a viral strategy to remain undetected and therefore escape the immune response which might be one mechanism for viral persistence.

ANAPLASIC OLIGODENDROGLIOMA IN THE OPTIC NERVE WITH METASTASIS TO A REGIONAL LYMPH NODE. J.R. Pedregosa Morales[1], F. Fernández Flores[2], F. Rodríguez Fernández[1], J.J. Mínguez Molina[3], M. Pumarola[2], [1]Hospital Veterinario Al Sur. Granada, Spain., [2]Department of Animal Medicine and Surgery. Veterinary Faculty. Universidad Autónoma de Barcelona, Barcelona, Spain., [3]Hospital Veterinario Guadiamar. Sanlucar La Mayor. Sevilla. Spain.

Gliomas are tumours of the non-neuronal tissue of the Central Nervous System (CNS). Depending on their origin they are classified as astrocytomas, oligodendrogliomas, mixed gliomas (oligoastrocytomas), ependymomas and choroid plexus papillomas. Metastases of these tumours outside the CNS are very rare. Primary neoplasms of the retina and optic nerve are quite infrequent, with meningiomas being the most common primary tumours; gliomas are rare.

A 2 year-old, male German Shepherd dog was assessed for a superficial corneal ulcer 1 x 0.2 cm. in the left eye. The rest of the clinical examination was normal. Three days later, the animal manifested a unilateral exophthalmos, slightly painful with resistance to retropulsion, chemosis, pain when opening the mouth and dorsal strabismus. The differential diagnosis included orbital cellulitis and/or retrobulbar abscess. Three days later, with topical and systemic treatment, there was an improvement in the ulcer size and exophthalmos disappeared. After one week the ulcer healed, and there was no exophthalmos or strabismus. Four days later the clinical symptom reappeared. Ocular ultrasonography was performed in the left eye, which showed a retrobulbar cyst (0.35 x 0.65 cm). After three days the dog presented blindness with bilateral mydriasis, an absent bilateral menace response, and absent direct and consensual papillary light reflexes in both eyes. The rest of the neurological examination was normal. The location of the lesion was the left optic nerve affecting the optic chiasm. The ERG was normal in both eyes. An MRI study of the neurocranium and retrobulbar space revealed a lesion on the left orbital cone. This lesion was isointense on T1-weighted sequence compared with the ocular muscles and showed heterogeneous intensity on T2-weighted sequences and FLAIR. The left optic tract and chiasm were hyperintense, enlarged and they produced a slight mass effect in the olfactory tubercle and piriform lobe. An increase in the muscle signal in the rostral portion of the left temporal muscle was found and a small mucus collection in the frontal sinus was also present. A severe, peripheral, irregular-shaped contrast uptake was observed in the left extraconal zone. The intraconal space showed a moderate, patched, cavitary-pattern contrast uptake. A moderate, homogeneous contrast enhancement pattern and irregular hallmarks were found in the brain lesions. An intense contrast uptake was seen in the muscle injuries and fronto-temporal bone periosteum. The differential diagnosis included a retrobulbar abscess and/or neoplasia. The CSF analysis was normal. A fine needle cytology of the retrobulbar space showed polygonal cells with round nuclei, acidophilic cytoplasm and anisokaryosis. Five days later there was lymphadenopathy in the left submandibular lymph node. The owner decided to euthanize the animal. The necropsy confirmed the presence of a mass in the left optic nerve affecting the optic chiasm and adjacent ventral brain stem structures and piriform lobe. The mass had progressed toward the left orbital cavity infiltrating the extraocular muscles and compressing the globe eyepiece. The histological study determined the presence of a neoplastic population of an undifferentiated and highly infiltrative polygonal cell population with glial characteristics, showing malignant features, and associated with vascular proliferation phenomena. The cervical lymph node was also invaded by these neoplastic cells as described in the primary tumour. The immunohistochemistry confirmed that the majority of tumour cells were highly positive for Olig2 marker. The definitive diagnosis was anaplastic oligodendroglioma.

Optic nerve gliomas are very rare in the dog. Most of the published canine optic nerve gliomas are astrocytomas, or lack a histological description. Metastasis from an optic nerve glioma has never been reported previously in animals. In humans, primary malignant gliomas of the optic nerve and chiasm are rare (4% of intracranial gliomas): the most frequently described are astrocytomas, and optic nerve oligodendrogliomas are very rare. They are most common in children, and in adults are usually rapidly fatal; tumours involving the optic nerve alone are associated with a better prognosis than intracranial gliomas affecting the optic chiasm and tracts. To our knowledge, this is the first case of optic nerve oligondendroglioma in a dog affecting the optic chiasm and piriform lobe and with metastases to the regional lymph node.

MAGNETIC RESONANCE IMAGING FINDINGS IN A DOG WITH SENSORY NEURONOPATHY. N. Hamzianpour, P.J. Kenny, R.F. Sanchez, C. Dawson, C.J. Driver, H.A. Volk, S.De Decker, Department of Veterinary Clinical Sciences, Royal Veterinary College, London, UK.

Sensory neuronopathies are a specific rare subgroup of peripheral nervous system diseases characterized by inflammation within the peripheral ganglia and spinal nerve roots. Although Magnetic resonance imaging (MRI) has been used in people with sensory neuronopathies this information is not yet available for dogs.

A 3 year old, male-neutered, Miniature Dachshund was presented for evaluation of a 2 month history of acute onset slowly progressive generalized ataxia. The dog demonstrated a bouncy generalized ataxia with exaggerated movements of all limbs and head. Neurological examination demonstrated widespread proprioceptive and nociceptive deficits. These findings were consistent with a generalized sensory ataxia resembling canine ganglioradiculitis.

Magnetic resonance imaging (MRI) of the cervical spinal cord demonstrated T2-weighted hyperintensity of the dorsal funiculus. CSF-analysis was within normal limits. The dog's clinical condition slowly progressed to a non-ambulatory state, visual impairment and difficulties prehending food. A second MRI, 14 months after the first MRI study, confirmed the previous findings, but demonstrated additional T2-weighted hyperintensity delineating the caudal cerebellar peduncles and cerebral cortical atrophy. Electromyography and a motor nerve conduction study were within normal limits. A sensory nerve conduction study revealed a decreased velocity. Electro-retinography demonstrated lack of retinal sensory function. Treatment with ciclosporin (Atopica) did not result in sustained clinical improvement.

The cervical spinal MRI findings in the dog presented here correspond well with pathological studies of dogs with ganglioradiculitis and MRI studies of people with sensory neuronopathies. The findings of the second MRI study, specifically the hyperintensity of the caudal cerebellar peduncles and cerebral cortical atrophy are possibly explained by trans-synaptic degeneration of the ascending proprioceptive tracts.

This is the first report indicating the potential role for MRI in the ante-mortem diagnosis and for monitoring progression of dogs with sensory neuronopathies such as ganglioradiculitis. Further studies are indicated to compare these MRI findings with pathological and histopathological abnormalities.

MRI FINDINGS IN HEREDITARY NECROTIZING MYELOPATHY IN A KOOIKER DOG. K. Truar[1], T. Flegel[1], E. Ludewig[1], P. Mandigers[2], G. Oechtering[1], [1]Department of Small Animal Medicine, University of Leipzig, Leipzig, Germany, [2]University Clinic for Companion Animals, University of Utrecht, Utrecht, Netherlands.

Hereditary necrotizing myelopathy of Kooiker dogs is a progressive degenerative disease of the central nervous system in young dogs of this breed. Affected dogs exhibit progressive paraparesis. The diagnosis is based on a gene test, although the exact mutation has not yet been found yet. We describe magnetic resonance imaging (MRI) findings of an affected dog exemplary for alterations caused by a degenerative spinal cord diseases.

This case report presents for the first time magnetic resonance images and pathological examination of the spinal cord of a 1-year-old Kooiker dog suffering from this disease. The dog showed a 6-week-history of progressive moderate pelvic limb ataxia with normal paw replacement test in all limb and mild bilateral deficits on hemiwalking. The neurological lesion was localized to T3-L3 spinal cord segments. Myelography and computed tomography were normal. Cerebrospinal fluid examination revealed an increase of total protein (0. 43 mg/dl) combined with a normal cell count (1.3 cells/ µl). The differential cell count displayed 70% monocytes, 20% activated lymphocytes and 10% lymphocytes. MRI of the caudal cervical und cranial thoracic spinal cord were performed. A hypertintense area in the ventral portion of the white matter at C5 to Th7 was present on T2 and isointense on T1 weighted images. The area was compared to the unaffected grey matter signal and no contrast enhancement was detectable. On transverse images the shape of this area was similar to an upside down "seagull". Due to these findings the ventral, lateral and dorsolateral funiculli seemed to be affected.

The histopathological examination revealed a symmetrical, well demarcated area of normal architecture loss in the cranial cervical segments. The physiological architecture of the caudal segments was lost due to malacia with extensive swelling of myelin sheaths and mild inflammation of the surrounding tissue. In addition, a moderate gliosis was present. The grey matter revealed a mild hemorrhage. In the thoracic spine similar alterations were present. Malacia and gliosis in this region extended over the ventral, lateral and laterodorsal funiculli. The caudal cervical and cranial thoracic segments showed no malacia, but mild signs of demyelination. Histopathological changes correlated

with alterations on the magnetic resonance images. The dog was positive for the aforementioned gene mutation.

There are few data about MRI findings of degenerative disease of the spinal cord. Although gene tests are available for some neurodegenerative diseases, histopathological examination is needed for a definite diagnosis in most cases. Since we found a correlation between MRI and the histopathological findings, MRI may be of diagnostic value for similar diseases of other breeds.

CEREBRAL HIGH-GRADE OLIGODENDROGLIOMA WITH SARCOMATOUS METAPLASIA ("OLIGOSARCOMA") IN A BOXER DOG. A. Fadda[1], I. Vajtai[2], J. Lang[3], D. Henke[1], A. Oevermann[4], [1]Division of Neurological Sciences, [2]Department of Clinical Veterinary Medicine, Vetsuisse Faculty, University of Bern, [3]Institute of Pathology, University of Bern, [4]Department of Veterinary Radiology, Department of Clinical Research and Veterinary Public Health, Vetsuisse Faculty, University of Bern, [4]Department of Clinical Research and Veterinary Public Health, Vetsuisse Faculty, University of Bern, Bern, Switzerland.

A 9-year-old neutered female boxer was referred to the veterinary teaching hospital of the Vetsuisse Faculty, University of Bern for severe cervical pain and tetraparesis, which rapidly progressed to lateral recumbency. At neurological examination, the dog showed obtunded mentation along with non-ambulatory tetraparesis. Postural reactions were decreased to absent in all four limbs. Cranial nerve examination revealed a bilaterally reduced menace response as well as spontaneous vertical nystagmus. Severe pain was elicited during lateral and dorsal flexion of the neck. Based on these findings the lesion was localized multifocal intracranially. Magnetic resonance imaging (MRI) of the head and neck revealed a space occupying intraaxial lesion in the left piriform lobe, which was connected to a second mass with identical MRI features in the left cerebellopontine angle. The mass was hyperintense on T2-weighted and hypointense on T1-weighted images, displayed a strong contrast uptake, and presented a bilobated, fluid-filled, cystic center that did not suppress completely on FLAIR images.

Post mortem microscopic study of the critical areas revealed a biphasic malignant neoplasm composed of an admixture of neuroepithelial and mesenchymal elements. The former displayed characteristics of conventional anaplastic oligodendroglioma (WHO grade III) involving brisk mitotic activity and glomeruloid microvascular proliferation on a background of afibrillary round cells with "honeycomb-like" perinuclear vacuolation.

Conversely, the sarcomatous moiety – one widely encroaching upon the subarachnoid space – exhibited haphazard fascicles of spindle cells amidst an intricate mesh of pericellular basal lamina and broad bands of collagen. Both tumour cell populations immunoreacted for Olig-2 and to a lesser extent for GFAP. In addition, the sarcomatous areas focally expressed vimentin, muscular actin, and smooth muscle actin. Irrespective of architectural variations, the MIB1 labelling index averaged 5%. An exquisitely uncommon pattern of oligodendroglial malignancy in humans, "oligosarcoma" has not previously been reported to affect dogs, although oligodendroglioma is a common CNS tumour in this species. In humans, oligodendroglial differentiation tends to confer significant clinical advantage with respect to prognosis and adjuvant treatment options. The awareness of such hallmarks and the investigation of their impact on prognosis are crucial for improved therapeutical strategies in dogs.

ENDOCANNABINOIDS MAY INFLUENCE THE CELL POPULATION IN CANINE CEREBROSPINAL FLUID. J. Freundt Revilla[1], A. Zörner[2], F. Gesell[1], M.H. Shamir[3], A. Tipold[1], [1]Small Animal Clinic, University of Veterinary Medicine Hannover, Germany, [2]Institute for Clinical Pharmacology, Hannover Medical School, Germany, [3]Department of Neurology and Neurosurgery, The Hebrew University of Jerusalem, Israel.

Endocannabinoids (ECs) are involved in immunomodulation, neuroprotection and control of inflammation in the central nervous system (CNS). Activation of cannabinoid receptors (CB) is known to diminish the release of pro-inflammatory factors and enhance the secretion of anti-inflammatory cytokines. 2-arachidonoyl glycerol (2AG) induced the migration of eosinophils in a CB2 receptor-dependent manner in human peripheral blood and activated human neutrophils independently of CB activation. Therefore, we hypothesized that the influx and/or activation of different cell populations in canine cerebrospinal fluid (CSF) might be influenced by the ECs, anandamide (AEA) and 2AG, and quantified these ECs in CSF and serum samples of dogs suffering from Steroid Responsive Meningitis-Arteritis (SRMA) and intraspinal Spirocercosis.

AEA and 2AG were determined in samples of dogs affected with Spirocercosis, SRMA in the acute phase of the disease (SRMA A), SRMA under treatment with prednisolone (SRMA Tr) and in healthy dogs (Healthy). Dogs with SRMA A showed neutrophilic pleocytosis while those affected with Spirocercosis displayed an eosinophilic pleocytosis. Liquid chromatography combined with tandem mass spectrometry (LC-MS/MS) was performed using internal standards of d4-AEA and d5-2AG. Total AG (AG) was calculated using the sum of 1AG and 2AG.

Dogs with SRMA A showed a significantly higher concentration of total AG (median 193.92 nM; range 80.39- 976.15 nM) and AEA (median 0.44 pM; range 0.21-0.91 pM) in serum in comparison to healthy controls (total AG: median 61.84 nM; range 50.17-272.9 nM and AEA median 0.23 pM; range 0.17-0.34 pM) ($p > 0.05$). Levels of ECs in CSF where significantly elevated in SRMA A (total AG: median 4.05 nM; range 0.86-17.59 nM and AEA: median 7.75 pM; range 2.3-48.2 pM) compared to SRMA Tr (total AG: median 1.01 nM; AEA: median 5.4 pM) ($p > 0.05$). Furthermore, dogs affected with Spirocercosis displayed the highest ECs concentrations in serum (total AG: median 500.10 nM; range 186.35-827.85 nM and AEA: median 0.69 nM; range 0.41 -0.84 nM) and CSF (total AG: median 16.13 nM; range 2.46-94.38 nM and AEA: median 39.4 pM; range 3.5 -237.9 pM).

In conclusion, levels of ECs in CSF of patients with inflammatory CNS diseases were increased when compared with healthy dogs, and those under treatment. Such an over expression could be a physiological attempt to control CNS inflammation and could explain the waxing and waning course of SRMA. Furthermore, patients with eosinophilic pleocytosis showed a higher level of ECs than those with neutrophilic pleocytosis, leading to the assumption that ECs have a major effect on migration of eosinophils in the CSF.

DIAGNOSTIC IMAGING FINDINGS AND OUTCOME IN A CALF WITH TRAUMATIC BRAIN INJURY. M.V. Hoffmann[1], S. Janßen[2], F.X. Liebel[3], J. Tünsmeyer[1], J. Rehage[2], A. Tipold[1], [1]Department Small Animal Medicine and Surgery, [2]Clinic for Cattle, University of Veterinary Medicine Hannover, Foundation, Hannover, Germany, [3]Department of Clinical Neurology, Davies Veterinary Specialists, Higham Gobion, UK.

Traumatic brain injury is rarely observed in cattle and information in the literature mainly focuses on bolt stunning injuries at slaughter. The use of magnetic resonance imaging (MRI) to examine the bovine brain is uncommon. Therefore, diagnosis is based primarily on a compatible history and clinical sings of intracranial neurological dysfunction. In this case report, we describe the clinical presentation in correlation to MRI findings and outcome in a calf with acute traumatic brain injury and skull fracture.

A 2-weeks-old healthy male calf was presented to the Bovine Clinic due to acute trauma of the mother cow. During hospitalization the calf suffered a traumatic brain injury while jumping under the feeding trough. Directly after the traumatic event, the calf was unconscious for several seconds. An injection of 0.4 mg/kg dexamethasone was given intravenously from the referring veterinarian. Fifteen minutes later the calf was ambulatory, had a left sided epistaxis, reduced tongue tone and seemed apathetic. It had a bold round area in the region of the left os frontale. Haematological and biochemical analyses were within normal limits. Antibiotic and analgesic treatment was started. Two days after the trauma and initial treatment the calf was referred to the neurology unit. In the neurological examination the calf was still mildly apathetic, had a normal gait, a reduced tongue tone but was able to suckle properly. It had a mild head turn to the left and mild proprioceptive deficits on the right side. These findings were consistent with a left forebrain lesion with possible brainstem involvement. MRI obtained 6 days after the trauma was chosen over computed tomography (CT) due to the suspected caudal fossa involvement, which does not image well with CT

due to a beam-hardening artefact and the expected superior soft tissue detail findings in MRI. MRI revealed a closed comminuted depressed skull fracture at the level of the left os frontale, a hyperintense lesion (T2W, FLAIR and GRASE) at the level of the left frontal cortex which was heterogenous in appearance and had focal areas of hypointensity. The lesion was hypo- to isointense in T1W. These findings were consistent with an intracranial hemorrhage and closed comminuted depressed skull fracture. The calf improved without further medication and was discharged with the mother cow after one week. A 3-month-follow-up by telephone call revealed, that the calf was in good body condition, but still showed a slower behaviour than other calves at the same age. No seizures or other neurological deterioration were noted.

To the author's knowledge this is the first published case of a calf with acute traumatic brain injury including MRI findings and a three month follow up. Findings may help defining a diagnostic base of MR images for bovine cephalic diseases.

DYSTROPHIN DEFICIENT MUSCULAR DYSTROPHY IN A NORFOLK TERRIER. E. Beltran[1], G.D. Shelton[2], D. Sanchez-Masian[1], D. Robinson[3], L.De Risio[1], [1]Animal Health Trust, Newmarket, UK, [2]Comparative Neuromuscular Laboratory, University of California San Diego, USA, [3]Kingston Veterinary Group Ltd, UK.

Dystrophin deficient muscular dystrophy is a hereditary, X-linked, recessive, degenerative myopathy. The human form of dystrophin deficiency is known as Duchenne muscular dystrophy. At least 16 canine breeds with muscular dystrophy have been characterised phenotypically. This report describes the clinical presentation, magnetic resonance imaging, electrodiagnostic and histopathological findings with immunohistochemical analysis in a Norfolk terrier with dystrophin deficient muscular dystrophy.

A six month old male entire Norfolk terrier was presented with three month history of poor development, reluctance to exercise and progressive and diffuse muscle atrophy. Physical examination revealed poor muscle mass, in particular affecting the pelvic limbs. The gait was mildly stiff with mild abduction on the pelvic limbs and bunny hopping when trying to run. He also showed mild plantigradism. The rest of the neurological examination was unremarkable. Serum creatine kinase was markedly elevated. *Toxoplasma gondii* and *Neospora caninum* titres were negative. MRI of the epaxial muscles revealed asymmetrical streaky signal changes aligned within the muscle fibers (hyperintense on T2-weighted images and STIR with moderate contrast enhancement on T1 weighted images). Electromyography revealed pseudomyotonic discharges and fibrillation potentials localised at the level of the supraspinatus, epaxial muscles and tibial cranialis muscles. Sciatic motor nerve conduction velocity was normal. Cardiac ultrasound was unremarkable. Cardiac troponin I serum levels were within normal limits. Muscle biopsies obtained from the left lumbar epaxial muscles and left cranial tibial muscle revealed a degenerative and regenerative myopathy that was dystrophic in nature. Cryosections of the muscles were immunostained using a selection of monoclonal and polyclonal antibodies against dystrophy associated proteins. Compared to control tissue, staining was absent for both the rod and carboxy terminus of dystrophin and decreased for β and γ-sarcoglycans.

The dog remains stable two months after diagnosis with coenzyme Q10 and L-carnitine. Another male littermate has been reported with similar clinical signs; however diagnostic investigations have not been performed yet. The dam is clinically normal and no abnormalities have been reported in the 2 female littermates. Investigations to identify the genetic mutation leading to dystrophin deficient muscle dystrophy in Norfolk Terriers are currently underway.

PITUITARY NEOPLASM AND SKIN FRAGILITY SYNDROME IN A CAT: A CASE REPORT. B. Parzefall[1], N. Herbach[2], A. Blutke[2], [1]Neurology and Neurosurgery Service of the Queen Mother Hospital for Animals, Royal Veterinary College, University of London, UK, [2]Institute of Veterinary Pathology at the Centre for Clinical Veterinary Medicine, Ludwig-Maximilians University, Munich, Germany.

Feline Acquired Skin Fragility Syndrome (FASFS) is a very rare syndrome of unknown pathogenesis, characterized by fragile and thin skin with extensive tearing and shedding upon minimal trauma. FASFS has been described in association with endocrine disorders as hyperglucocorticoidism, diabetes mellitus, or with hepatic disease.

The present report describes the clinical and necropsy findings in a 14 year old male European-short-hair cat with FASFS. Clinically, the cat presented with obesity, general weakness, and elevated blood glucose levels. At the neck and the dorsum of the trunk, multiple extensive skin lacerations were present, readily appearing with normal handling. At necropsy, a 2x3x2.5 cm hypophyseal tumour with dorsal expansion into the adjacent brain parenchyma, necrosis and haemorrhage was present. The adrenal glands were bilaterally enlarged. Histopathological, immunohistochemical and ultrastructural examination of the pituitary gland revealed an adenoma of the adenohypophysis. The tumour cells displayed a strong immunohistochemical α-MSH and ACTH staining and characteristic electron-dense granules. The skin was severely atrophic and showed a marked attenuation and disarray of collagen fibers, as evidenced by light- and electron- microscopy. The adrenal gland displayed bilateral adrenocortical hyperplasia. Additionally, an invasive carcinoma in the pancreas and a cystic biliary duct adenoma in the liver were diagnosed.

Consequently, the skin lesions in the case at issue were most likely caused by hyperglucocorticoidism due to an ACTH-secreting adenoma of the pituitary gland with subsequent adrenocortical hyperplasia (Morbus Cushing). The present report therefore provides a detailed illustration of a wide spectrum of alterations typically associated with rare FASFS.

A CROSS-SECTIONAL STUDY OF PREVALENCE AND LONG-TERM OUTCOME IN SYMPTOMATIC AND ASYMPTOMATIC CAVALIER KING CHARLES SPANIELS WITH SYRINGOMYELIA. M.S. Thofner, A.A. Madry, C.S. Stougaard, C.S. Knudsen, H. Berg, C.S.E. Jensen, R.M.L. Handby, M. Berendt, Department of Veterinary Clinical and Animal Science, University of Copenhagen.

Syringomyelia (SM) is a hereditary and incurable condition affecting the spinal cord. SM manifests clinically with various degrees of chronic neuropathic pain and in some cases neurological deficits. The prevalence of SM in dogs is sparsely documented. In a study of 555 asymptomatic dogs 46% were diagnosed SM positive on MRI. The aim of the present study was to investigate the prevalence of SM and long-term outcome in symptomatic and asymptomatic SM-affected Danish Cavalier King Charles Spaniels (CKCS).

The study was initiated in 2007 and consisted of three phases. In phase I a cross-sectional study was conducted to estimate the prevalence of SM in dogs older than six years of age where clinical signs are expected to have arisen. The study population consisted of all 240 CKCS born / registered in the Danish Kennel Club in 2001. The 240 owners were initially contacted by mail and asked to answer a standardized screening questionnaire addressing six clinical key signs of syringomyelia (e.g. phantom scratching). Of 240 owners 134 (56%) responded. Eleven answers were excluded from the study because of invalid information leaving 123 dogs (61 females and 62 males) to be included in the investigation. After evaluation of all answers 19 dogs were reported to express one or more clinical key signs (positive responders) and were evaluated as possibly positive cases. For 104 dogs no clinical signs of SM were reported (negative responders). The owners of the 19 possibly SM positive dogs were subsequently contacted by phone and enrolled in an extensive interview in order to validate the answers given in the initial mailed questionnaire. Dogs considered positive after this interview were invited to participate in a clinical investigation including clinical and neurological examination, standard haematological, biochemical and thyroid profiles. Based on the clinical signs reported and the results of the clinical investigation (excluding possible differential diagnosis), 19 dogs were finally evaluated as positive SM cases.

In phase II the association of clinical findings and MRI findings in symptomatic and asymptomatic siblings was investigated. The phase I prevalence study identified several litters with symptomatic and asymptomatic siblings. Eight litters with one or more siblings with clinical signs of SM representing 35 dogs were invited for clinical evaluation and MRI investigation. The hypothesis of no association between the presence of syrinx(es)

and the expression of clinical signs was tested using Fishers exact test. Data was analysed using the statistical software in Excel.

In 2012 a five-year follow-up investigation (phase III) of the litters investigated in phase II was carried out. To assess the long-term outcome in symptomatic and asymptomatic dogs with SM, the owners were contacted by phone. The interview was based on an extensive structured questionnaire addressing the status of the dog including symptomatic / asymptomatic, alive / dead, and cause of dead if not alive. The estimated prevalence of clinically symptomatic syringomyelia in Danish CKCS born in 2001 (> six years of age) was 15.4% (CI95%: 9% - 22%).

In study phase II 22 dog owners accepted to let their dog participate in a full clinical work-up including MRI. Thirteen of the 22 dogs (59%) were clinically classified as SM positive whereas the

MRI scans revealed a syrinx in the cervical spinal cord in 21 of 22 dogs (95%). No statistical association between clinical signs and the presence of syrinx(es) could be established ($p = 0,41$). Of the 13 dogs where the owners rejected the MRI examination, five dogs (38%) expressed clinical signs of SM. Ninety-two percent (32/35) of the owners participated in the 2012 follow-up. It was found that after five years (by the age of 11) eleven of the 31 dogs (35%) were alive while 20 (65%) had been euthanized. In four dogs (20%) euthanasia was directly related to severe signs of SM. One dog that was asymptomatic in the phase II investigation in 2007 did develop signs of SM after the age of six.

This study found a high prevalence (15.4%) of symptomatic SM in the Danish CKCS population and revealed that despite positive SM findings on MRI, affected dogs may be clinically silent. Asymptomatic dogs may develop clinical signs rather late in life (in this study after the age of six). Despite the high number of affected dogs, euthanasia motivated by SM is relatively moderate. From a clinical point of view our results necessitate further examination of the progression of the disease and assessment of the threshold of outbreak of clinical symptoms.

ACCURACY OF NEUROLOGICAL EXAMINATION AND CLINICAL COURSE FOR DETERMINING MOST LIKELY DIFFERENTIAL DIAGNOSIS. T. Cardy, H.A. Volk, Royal Veterinary College, London, UK.

Spinal diseases are common neurological conditions and advanced imaging (CT, MRI) is heavily relied upon for its investigation. However a thorough case history and systematic neurological examination should allow accurate neurolocalisation and suggest differential diagnoses in the first instance. The primary objective of this study was to describe the frequencies and localisations of spinal lesions in dogs as described by the neurological examination compared with advanced imaging findings. In addition we aimed to identify simple algorithms among presenting neurological signs to assess if diagnoses and localisations could be predicted.

This retrospective study included 500 dogs presented for spinal disease that had a neurological examination and advanced imaging and other diagnostics to confirm localisation and diagnosis. Details regarding signalment, progression, mentation, gait and posture, cranial nerve deficits, postural reactions, spinal reflexes, spinal hyperaesthesia, deep pain, management and imaging were obtained from clinical records. Data were treated as categorical, except for age, which was continuous. Univariate analyses of potential explanatory variables for each condition were carried out (SPSS, Version 20.0). Variables were considered for inclusion in multiple linear regression if $P < 0.30$. Variables were retained in the final model if $P < 0.05$, based on the likelihood ratio test. Results are odds ratios (OR) and 95 per cent confidence intervals (CI) for significant findings for each condition versus the spinal disease population.

Dogs included 191 females (162 neutered) and 309 males (227 neutered) with mean age of 7.3 ± 3.2 years (Mean±SEM). Leading diagnoses were intervertebral disc disease (IVDD, n = 242), ischaemic myelopathy (IM, n = 48) and neoplasia (n = 44). Dachshunds (15%), cross-breeds (14%) and Cocker Spaniels (9%) were over-represented in the IVDD population. Almost 39% of IVDD lesions occurred at T12-L2 intervertebral spaces. IM and high velocity low volume discs were per-acute conditions with a median time to presentation (TTP) of one day. IVDD had a median TTP of 18 ± 32.5 days reflecting the diversity of Type I versus Type II disease. Anatomic localisation from neurological exam was confirmed by imaging in 90% of all cases. IM was the only non-deteriorating (OR: 0.1, 95%CI: 0.03-0.28), non-painful (OR 0.11, 95%CI 0.05-0.24) and lateralised (OR 4.5, 95%CI 2.1-9.6) condition. In contrast IVDD was painful (OR 3.8, 95%CI 2.3-6.2) with marked postural reaction deficits (OR 7.1, 95%CI 2.4-21.1). A simple algorithm based on five parameters identified multiple diagnoses with high positive predictive value (PPV, IVDD:66%, IM:61%, meningoencephalitis of unknown aetiology:65%). This is the first study to show that spinal diseases have well defined characteristics on neurological examination that can be used to predict a diagnosis (as confirmed with imaging) with statistical significance.

CLINICAL, IMAGING, ELECTROPHYSIOLOGICAL, HISTOPATHOLOGICAL AND ULTRASTRUCTURAL FINDINGS OF CENTRAL NERVOUS SYSTEM HYPOMYELINATION IN RABBITS. C. Ros, C.de la Fuente, M. Pumarola, S. Añor, Departamento de medicina y cirugía animal, Universidad Autónoma de Barcelona, Barcelona, Spain.

Hypomyelinating disorders affecting the central nervous system (CNS) have been described in humans and in a wide variety of domestic mammals usually as a consequence of mutations in the genes responsible for myelin formation. Myelin proteolipid protein (PLP) is the major protein involved in the formation and maintenance of compact myelin in the CNS, and its deficiency causes a decrease or absence of myelin in CNS axons. Pelizaeus Merzchbacher disease (PMD) in humans and paralytic tremor in rabbits are congenital hypomyelinating disorders caused by a derangement in the PLP gen. Paralytic tremor in rabbits is considered the analog of PMD and has been proposed as an animal model of PLP mutations in human. The aim of this study is to describe the clinical, imaging, electrophysiological and histopathological findings in a group of rabbits with congenital CNS hypomyelination suspected to be caused by a PLP mutation.

A few cases of CNS hypomyelination had been detected in the affected farm since 2010. Although the prevalence was initially low, it increased to affect currently 0.1% of the population. Fifteen of 46 rabbits belonging to 5 litters from the same progenitors showed similar neurological signs since birth, and males seemed to be more affected (9/15). The affected rabbits had a poor body condition, and were unable to walk due to severe, generalized tremors. The tremors were classified as action tremors, as they worsened with external stimuli or excitement. No abnormalities were detected in magnetic resonance imaging of the brain performed with a low field magnet. Electromyography did not show spontaneous electrical activity in any appendicular muscle. Peripheral motor and sensory nerve conduction velocities were considered normal compared with control littermates. Brainstem auditory evoked responses (BAER) revealed absence of waves III to V. Histopathology, immunochemistry (Olig2, GFAP) and ultrastructural analysis showed severe and diffuse hypomyelination in the CNS, with a normal number of oligodendrocytes. In contrast, the peripheral nervous system (PNS) was correctly myelinated. These findings were consistent with a PLP related disorder.

This study describes the clinical, imaging, electrophysiological, and histopathological characteristics of a CNS hypomyelinating disease without PNS involvement in a group of rabbits suspected to be caused by a PLP mutation. These findings are similar to those described in PMD.

ELECTROCARDIOGRAPHIC CHANGES INDUCED BY DIFFERENT NEUROPATHIES IN DOGS. D. Mocanu, M. Armasu, A. Zbangu, G. Solcan, M. Musteata, Department of Clinical Science, Internal Medicine, Faculty of Veterinary Medicine Iasi, Romania.

The goal of this study was to evaluate the impact of neurologic disorders on heart electrophysiology in dog.

Standard electrocardiograms (5 min ECG tracing of 6 periferal leads) were performed on 27 dogs, diagnosed with different neurological syndromes: epilepsy (8/27), cervicothoracic syndrome (2/27), thoracolumbar syndrome (6/27), brain tumour (4/27), myelomalacia (1/27), brainstem pathology (1/27), idiopathic polyradiculoneuritis (3/27) and encephalitis (2/27). Dogs were different breeds, of various weights 15.48 ± 13 kg and ages 5.77 ± 2.99 years, free of primary cardiovascular disorders (no

signs of cardiac remodeling on echocardiography and radiology). The ECG tracings were registered and interpreted in specialized software (Poly Spectrum 8VET). The following parameters were studied and statistically analyzed: durations of P wave, PR interval, QRS complex, QT interval and QT corrected (Bazzet's formula), time domain heart rate variability parameters SDNN, RMSSD, pNN50% and heart rhythm.

No significant correlations were found between a specific neuropathy and cardiac electrophysiological patterns. Rhythm disturbances were identified in 25/27 (92.5%) dogs, single or in combination: bradycardia 12%, sinus tachycardia 8%, atrial fibrillation 4%, irregular sinus arrhythmia 52%, regular sinus rhythm with normal heart rate 24%, ventricular premature complexes 24% and atrioventricular block 28% (first degree 12% and second degree 16%). P wave duration was significantly increased ($p < 0.001$) in comparison with normal values. For 33% of patients QT (0.32 ± 0.06 ms) and QTc (0.41 ± 0.1) were significantly increased ($p < 0.001$) from the normal values. No significant statistical differences were observed when HRV parameters were analyzed compared with normal dogs.

Since now, there are few experimental studies which describe these electrocardiographic changes in animals with induced neurological pathology. In humans with either encephalic or spinal diseases, these changes are well documented, and a specific attention is given to prevent and control the occurrence of life-threatening cardiac arrhythmias (e.g. atrial fibrillation, ventricular tachycardia, bradycardia). The similarities between these findings and those from human medicine, suggest that same therapy and follow up investigation might be needed for a neurological dog. Moreover, even if specific interspecies (human/dog) differences of cardiac electrophysiology are well known, these results underline that dogs can be used as a translational model for human neurocardiology research.

CLINICAL PRESENTATION AND OUTCOME FOR DOGS TREATED MEDICALLY FOR DEGENERATIVE LUMBOSACRAL STENOSIS. SDe Decker, LA Wawrzenski, HA Volk, Royal Veterinary College, London, UK.

Little is known about the role of medical management for the treatment dogs with degenerative lumbosacral stenosis (DLSS). The aims of this retrospective study were to compare the clinical presentation of dogs treated medically and surgically for DLSS and assess the outcome after medical treatment.

Dogs were included if they had clinical signs, clinical examination findings and magnetic resonance imaging abnormalities consistent with DLSS. Information retrieved from the medical records included signalment, duration of clinical signs, presence of neurological deficits, presence of urinary and/or fecal incontinence, presence of concurrent medical conditions, and presence and outcome of medical management before a diagnosis of DLSS was made. Medical management consisted of restricted exercise in combination with anti-inflammatory and analgesic drugs. Surgical management consisted of a dorsal lumbosacral laminectomy. Outcome for the medically treated dogs was obtained from the owners and/or referring veterinary surgeons by using a standardized questionnaire.

Ninety-eight dogs were included; 49 were treated medically and 49 surgically. Neurological deficits were significantly more observed in the surgically treated dogs ($P = 0.03$). Surgically treated dogs underwent significantly more unsuccessful medical management ($P < 0.0001$), while medically treated dogs underwent significantly more successful medical management ($P < 0.0001$) before a diagnosis of DLSS was made. No significant differences between medically and surgically treated dogs were observed for the remaining assessed variables. Thirty-one of the 49 medically treated dogs were available for follow up; 17 dogs (55%) were managed successfully with medical treatment; 10 (32%) failed medical treatment and underwent surgical decompression; 3 (10%) were euthanized due to progression of their clinical signs and one dog (3%) was still alive, but had more severe clinical signs after medical management for DLSS.

The present study suggests that dogs treated medically and surgically for DLSS differ in their severity of clinical signs and the outcome of medical treatment before a diagnosis of DLSS is made. Medical management for dogs with DLSS is associated with only a fair prognosis.

RADIOGRAPHIC CLASSIFICATION OF CONGENITAL THORACIC VERTEBRAL MALFORMATIONS IN BRACHYCEPHALIC "SCREW-TAILED" DOG BREEDS. R. Gutierrez-Quintana, J. Guevar, C. Stalin, K. Faller, C. Yeamens, J Penderis, School of Veterinary Medicine, College of Medical, Veterinary and Life Sciences, University of Glasgow, Glasgow, UK.

Congenital vertebral malformations are relatively common in brachycephalic "screw-tailed" dog breeds. The majority of affected dogs do not show any neurological signs, but in some cases secondary spinal cord compression can occur. The aims of this study were to propose and apply a radiographic classification scheme for congenital vertebral malformations affecting the thoracic vertebral column in a group of brachycephalic dogs, and to investigate the relationship between the type of vertebral malformation and the presence of neurological deficits.

The medical records of the University of Glasgow Small Animal Hospital were reviewed from September 2009 to April 2013 to identify French Bulldogs, English Bulldogs, Boston Terriers and Pugs that had well positioned lateral and ventro-dorsal radiographs of the thoracic spine with at least one vertebral malformation present. The age, sex, breed and the presence/absence of neurological deficits were recorded. The radiographs were reviewed by two observers and the type of vertebral malformation was determined through consensus. Vertebral malformations were classified as defects of segmentation if adjacent vertebral elements fail to divide (block vertebra) or defects of formation if a portion of the vertebra was deficient. Defects of formation of the vertebral body were then sub-classified into ventral agenesis (dorsal hemivertebra), ventro-lateral agenesis (dorso-lateral hemivertebra), ventral and median agenesis (butterfly vertebra), and ventral and/or lateral hypoplasia (wedge shape vertebra) on the basis of a similar classification scheme in human patients.

28 Dogs were included in the study, 11 English Bulldogs, 11 Pugs, 3 French Bulldogs and 3 Boston Terriers. 72.5% were males and the mean age was 30 months. 12 Dogs (42.8%) demonstrated neurological deficits associated with the vertebral malformation. 75% of the dogs with neurological deficits were Pugs. A total of 362 vertebrae were evaluated as some dogs had 12 thoracic vertebrae. 13.8% of the vertebrae showed a malformation. The vertebral malformations identified included butterfly vertebra (6.6%), wedge shape vertebra (5.5%), dorsal hemivertebra (0.8%), dorsolateral hemivertebra (0.5%) and block vertebra (0.3%). 50% of dogs had multiple vertebral malformations. The most commonly malformed vertebra was T7 (11dogs), followed by T8 (8 dogs) and T12 (8 dogs). Dorsal and dorso-lateral hemivertebrae were always associated with neurological deficits.

The present study demonstrated that vertebral malformations are common in the brachycephalic "screw-tailed" breeds. More severe defects of the vertebral body, including ventral or ventrolateral agenesis, tended to be associated with neurological deficits, while less severe malformations such as ventral or lateral hypoplasia and ventral and median agenesis may or not be associated with neurological deficits.

INCIDENCE OF EPILEPSY IN A LARGE POPULATION OF SWEDISH INSURED DOGS. L. Heske[1], A. Egenvall[2], M. Berendt[3], A. Nødtvedt[4], Karin Hultin Jäderlund[1], [1]Department of Companion Animal Clinical Sciences, Norwegian School of Veterinary Science, Oslo, Norway, [2]Department of Clinical Sciences, Faculty of Veterinary Medicine and Animal Husbandry, Swedish University of Agricultural Sciences, Uppsala, Sweden, [3]Department of Veterinary Clinical and Animal Sciences, Faculty of Health and Medical Sciences, University of Copenhagen, Frederiksberg, Denmark, [4]Department of Production Animal Clinical Sciences, Norwegian School of Veterinary Science, Oslo, Norway.

A Swedish animal insurance database (Agria) has proven useful for the study of disease occurrence in dogs. In this database, correctness of the diagnostic codes for canine epilepsy has been shown to be of acceptable quality for research purposes. In Sweden, 80% of the entire dog population are insured and Agria covers (2012) 50% of the insured dogs. Most commonly, dogs are covered by both veterinary care- and life insurance but it is also possible to choose one of the insurance plans separately.

The main objective of this cohort study was to use insurance data from 1995-2006 to estimate the incidence rate of canine epilepsy in dogs with respect to breed, sex and geographic region. A secondary objective was to analyse survival after an epilepsy diagnosis and to present the age distribution of the cases.

In total, 5,013 dogs had at least one claim for veterinary care for epilepsy and 2,327 dogs were euthanized or died because of epilepsy during the observation period. Overall there were > 2,000,000 dog-years at-risk (YAR) for life and veterinary care insurance claims. The incidence rate of canine epilepsy (only the first claim in each case counted; based on veterinary care insurance) was estimated to be 18 cases per 10,000 YAR. Among the 35 most common breeds in Sweden, the boxer breed was at the highest risk for a veterinary care-claim with 60.3 cases per 10,000 YAR. The Boxer was also found to have the highest mortality (based on life insurance) among the 35 most common breeds in Sweden, with a mortality rate of 46.7 deaths per 10,000 YAR. There was a difference in the rates between the sexes with males at a higher risk. The age distribution was similar in cases with veterinary care insurance and life-insurance settlements because of epilepsy. Marked breed differences in incidence rate exist, perceived to reflect a genetic basis for a breed predisposition to develop canine epilepsy. Furthermore, breed differences in survival after diagnosis were observed with dogs belonging to hunting breeds having high mortality rates.

UNCOMMON PRESENTATION OF MULTIPLE INTRASPINAL EXTRADURAL CYSTS IN THE CERVICAL SPINE OF A DOG. M.I.de Freitas[1], H. Rudorf[2], A. Rupp[2], E. Ives[2], P. Freeman[2], [1]Department of Veterinary Sciences, CECAV, University of Trás-os-Montes e Alto Douro, Vila Real, Portugal, [2]Department of Veterinary Medicine, University of Cambridge, Cambridge, UK.

A six-year-old, male, castrated Labrador retriever was referred for investigation of acute, persistent cervical hyperaesthesia. No neurological deficits were found on examination and radiographs of the cervical spine were unremarkable. MRI of the cervical spine revealed an intraspinal cystic structure surrounding and compressing the spinal cord at the level of the C6 vertebral body. An arachnoid diverticulum was suspected given the apparent intradural location. A smaller cystic structure was also visible at the level of the C7 vertebral body merging with an irregularly thickened C7 ventral nerve root on the right side. The dog was subsequently euthanized at the owner's request. Necropsy revealed the presence of two intraspinal extradural cystic lesions. Definitive determination of cyst origin proved difficult. Histopathology was most consistent with either a synovial or ganglion cyst compressing the spinal cord at the level of C6. The location and histopathological examination of the second cyst were most consistent with a perineurial (Tarlov) cyst compressing the right C7 nerve root.

To the author's knowledge, this is the first report of multiple intraspinal extradural cysts in a middle aged, large breed dog without evidence of degenerative joint disease or concurrent neurological deficits.

SPATIAL PATTERN OF HIPPOCAMPAL SCLEROSIS IN EPILEPTIC CATS. E. Wagner[1], M. Rosati[1], J. Molin[1], A. Fischer[2], L. Matiasek[2], T. Flegel[3], K. Matiasek[1], [1]Sections of Clinical and Comparative Neuropathology and, [2]Neurology, Ludwig-Maximilians University, Munich, Munich, Germany, [3]Section of Neurology, Department of Small Animal Medicine, University of Leipzig, Leipzig, Germany.

Hippocampal sclerosis (HS) is the most common histopathologic abnormality found in adult human patients with drug-resistant temporal lobe epilepsy. It refers to the combination of neuronal loss and astrogliosis affecting mainly the pyramidal cell band of the hippocampus. Thereby, the different segments of the cornu ammonis (CA) are involved at various degrees. According to their involvement, HS in humans presents as either CA1 predominant, CA4 predominant or combined CA1/4 type with variable CA2 and CA3 affection. CA2, in general, is considered most resistant.

In a previous study we documented the occurrence of HS in altogether 41 hippocampi of epileptic cats. The present investigation was conducted to clarify the species-specific segmental pattern of HS in cats. HS diagnosis was based on histopathological evaluation of brain sections stained by haematoxylin-eosin and glial fibrillary acidic protein immunohistochemistry, and the morphometric evidence of significant pyramidal cell loss in CA segments 1 through 4.

HS in cats presented as monosegmental (17/41) and multisegmental (24/41) subtypes. In monosegmental HS, the CA segments were involved at following frequencies: CA1 3/17 (17.6%), CA2 2/17 (11.8%), CA3 6/17 (35.3%) and CA4 6/17 (35.3%). With multiple segment involvement, the frequencies shifted towards CA1 17/24 (70.8%), CA2 16/24 (66.7%), CA3 20/24 (83.3%), CA4 16/24 (66.7%). Hence, CA1 and CA4 indeed are frequently involved but the most frequently HS-affected segment in cats is CA3. Moreover, CA2 does not fall far behind the other segments. It ranges fourth in monosegmental and third in multisegmental feline HS.

Similar to other reported species, HS is a frequent complication in epileptic cats. The segmental pattern, however, differs significantly in that CA2 is not as resistant as in humans, dogs and rodents, and CA3 appears to be the main target of feline HS. This may be due to the unique situation that only in cats the entorhinal cortex layer III projects into CA2 and CA3, while in other species they only address the subiculum and CA1.

RADIOGRAPHIC CALCULATION OF SCOLIOTIC AND KYPHOTIC ANGLES IN BRACHYCEPHALIC DOG BREEDS WITH CONGENITAL THORACIC VERTEBRAL MALFORMATIONS. J. Guevar, R. Gutierrez-Quintana, C. Stalin, K. Faller, C. Yeamens, J. Penderis, School of Veterinary Medicine, College of Medical, Veterinary and Life Sciences, University of Glasgow, Glasgow, UK.

Congenital spinal deformities are common in many breeds of dogs. In human patients, the Cobb angle is widely accepted as the standard method to quantify the degree of spinal curvature. The Cobb angle is an objective measure that can be used to monitor progression of kyphosis and scoliosis, as well informing on the requirement for and success of treatment. The aim of the present study was to investigate the use of Cobb angle measurements in dogs with congenital thoracic vertebral malformations in order to objectively quantify the degree of spinal curvature using an open-access, computer-assisted, digital radiographic measurement system.

The medical records of the University of Glasgow Small Animal Hospital were reviewed from September 2009 to April 2013 to identify French Bulldogs, English Bulldogs, Boston Terriers and Pugs that had well positioned lateral and ventro-dorsal radiographs of the thoracic spine with at least one vertebral malformation present. The kyphotic and scoliotic angles were calculated using an open-access, commercially available DICOM viewer containing a Cobb angle plug-in. The end vertebrae were defined as the cranial end plate of the vertebra cranial to the malformed vertebra and the caudal end plate of the vertebra caudal to the malformed vertebra. Two observers independently performed each measurement set. The paired T-student test was used to compare the two sets of data and the inter-observer variability was estimated using intra-class correlation coefficients and the coefficients of variance.

28 Dogs were included in the study, 11 English Bulldogs, 11 Pugs, 3 French Bulldogs and 3 Boston Terriers. 72.5% were males and the mean age was 30 months. The mean Cobb angle measured for kyphosis and scoliosis were respectively 19.5° (range: 0-64.2°) and 7.6° (range: 0-41.2°) for observer 1 and 20.2° (range: 1.3-66°) and 6.93° (range: 0-42.3°) for observer 2. The coefficients of variance were 6.3% and 14.7% and the intra-class correlation coefficients (ICC) were 0.995 and 0.987 for the kyphotic and scoliotic angles respectively.

In conclusion, computer-assisted, digital radiographic measurement can be used to calculate the Cobb angle in dogs. The low coefficient of variance and high ICC values suggest that this is a valid method for calculation of the Cobb angle in brachycephalic dog breeds with congenital thoracic vertebral malformations.

LESION MAPPING IN FELINE INTRACRANIAL HYPERTENSIVE ANGIOPATHY. S. Bertram[1,2], L. Matiasek[1], M. Rosati[2], E. Wagner[2], H.A. Volk[3], K. Matiasek[2], [1]Sections of Neurology and, [2]Clinical and Comparative Neuropathology, Ludwig-Maximilians University of Munich, Germany, [3]Clinical Science and Services, Royal Veterinary College, London, UK

Systemic arterial hypertension (SAH) is a frequent finding in adult cats suffering from renal diseases or hyperthyroidism. Even though the brain circulation underlies a sophisticated autoregulation, SAH is known to impact on structure and function of brain arteries and thereby can give rise to neurological complications. Concerning the high prevalence of SAH in cats, there is little scientific data on the spatiotemporal characteristics of the associated brain changes.

In order to clarify the pattern of SAH-associated lesions and to identify key areas for diagnostic imaging, this study focused to highlight SAH intracranial histopathology and to screen for vascular segments, territories and brain foci that might be at increased risk of hypertensive damage.

Sixteen brains of cats with clinical and/or post-mortem confirmation of hypertensive angiopathy were retrieved from the pathology archive. Transverse sections were reviewed and lesions of both blood vessels and brain tissue were mapped, categorised and graded.

All major meningeal arteries apart from the large cerebellar arteries were affected at similar frequency. The more ventrally located brain vessels (ventral branches of middle cerebral artery, communicating arteries, basilar artery) exhibited the most severe lesions. The second most affected areas were the dorsolateral convexities and median fissure. Extra-axial vascular pathologies comprised necrotising arteriopathy in 13 and vaso-occlusive arteriosclerosis in 10 animals. In 12 cases fibrinoid necrosis of intra-axial microvessels was identified. Fibrinoid changes were most frequent in the Purkinje cell layer and superficial granular layer of the lateral cerebellar hemispheres, while they were most extensive in the dorsal juxtacapsular caudate nucleus and in the marginal / suprasplenial and splenial cortex. Parenchymal sequelae of vascular defects were seen in 15 cats, resembling vasogenic oedema (12), infarcts (7 ischaemic, 2 haemorrhagic) and bland focal haemorrhage (6) in the named areas. In 5/9 infarcts a direct association with the blood vessel damage was seen. All cats with brain oedema had an asymmetric involvement of the corona radiata and on close inspection a majority of these showed segregation with fibrinoid necroses in the neighbouring cortex.

In summary, cerebellar hemispheres, caudate nucleus and marginal / suprasplenial cortex resemble hot spots of SAH-associated brain lesions. Seventy-five of affected animals also had unilateral or asymmetric oedema of the parietal corona radiata. Both distribution pattern and histological type of lesions are highly indicative of elevated blood pressure being the pathogenetic trigger of the brain damage in terms of a hypertensive angiopathy-encephalopathy complex.

CNS CELLS UNDERCOVER: COVERSLIPPING PROTECTS IMMUNOGENICITY OF STAINED CYTOLOGICAL SAMPLES. S. Gründl[1], L. Matiasek[1], S. Felten[1], K. Hartmann[1], K. Matiasek[2], [1]Sections of Neurology and, [2]Clinical and Comparative Neuropathology, Ludwig-Maximilians University Munich, Munich, Germany.

Applicability of ancillary diagnostic tests, such as immunocytochemistry (ICC), frequently is limited by low volumes of CSF and tissue samples, harvested from living animals with CNS disorders. The diagnostician, hence, has to choose standard cytology as the most sensitive method over more specific techniques. Panoptic staining, though, has been reported to allow for a range of immunocytological procedures upon destaining of specimens. For some reasons, however, this approach has not gained broad entrance in neurocytological laboratories.

Thus, the present study investigated the immunolabeling profiles of unstained and destained smear preparations and fine needle aspirates of the CNS and lymph node. Primary antibodies were directed at antigens bound to the cell membrane, cytoplasm or nucleus in lymphocytes, glial cells, and neurons. The investigation employed microwave-based antigen demasking procedures. A total of 180 slides was processed and evaluated microscopically.

ICC for all markers provided good, reliable and reproducible results, in terms of high intensity, high specificity and low background, in unstained specimens. After modified Wright staining specific labeling still was obtainable after hydrochloric acid pretreatment if the slides had been cover slipped by a xylene-based medium immediately after the cytological staining was applied. Delays of just a few days abolished the immunodetection for all used markers. This was the same for CNS smears as well as for lymphocyte preparations.

Immunolabeling of specific pathogens or tissue markers remains an option even after a cell slide was stained in order to allow for proper cytological evaluation. Post-staining ICC, however, only offers adequate and reproducible results if the slides are being cover slipped in between the procedures. This sequential approach is particularly useful for single-slide situations such as in neurocytology and CSF diagnostics.

ATLANTO AXIAL SUBLUXATION IN TWO DOMESTIC FERRETS (*MUSTELA PUTORIOUS FURO*). M. Orioles[1], I. Mateo[2], R. Dennis[1], S. Jayson[2], D. Sanchez-Masian[2], [1]Animal Health Trust, Kentford, UK, [2]Hospital Clínico Veterinario de la Universidad Alfonso X el Sabio, Madrid, Spain.

Atlantoaxial subluxation refers to instability of the atlantoaxial joint characterized by dorsal displacement of the axis in relation to the atlas with subsequent spinal cord compression. This condition typically arises from developmental defects, such as malformation of the dens or surrounding ligaments, but it may also occur as a result of trauma.

An 18 month-old female entire domestic ferret was presented for investigation of chronic progressive ataxia and ambulatory tetraparesis. Neurological examination revealed vestibular ataxia, intermittent right head turn, decreased postural reactions on pelvic and thoracic limbs and moderate hyperesthesia on manipulation of the cervical spine. Radiographic, MRI and CT studies revealed a complex congenital atlantoaxial malformation, characterized by distortion of the atlas, which seems to override the foramen magnum, and absence of the dens with consequent subluxation and spinal cord compression and gliosis.

A 5 month old male neutered domestic ferret presented with acute onset of non-ambulatory tetraparesis and obtunded mental status. No previous history of trauma was reported. Radiographic and CT studies revealed atlantoaxial instability and subluxation with spinal cord compression.

Strict cage rest and corticosteroid therapy was initiated and a neck brace applied in both ferrets. Although there was initial improvement in motor function in the second ferret, both cases showed deterioration in neurological signs and the owners opted for euthanasia.

To the authors' knowledge these are the first two cases reported of atlantoaxial subluxation in domestic ferrets (*Mustela putorius furo*).

MENINGITIS AND MENINGOENCEPHALITIS OF UNKNOWN ETIOLOGY IN DOGS – ARE CANINE VECTOR BORNE DISEASES (CVBD) TRIGGERING THE DISEASES? K. Lazzerini[1], A. Tipold[2], M. Kornberg[3], C. Silaghi[4], A. Mietze[5], A. Lübke-Becker[6], A. Balling[7], M. Pfeffer[7], L.H. Wieler[6], K. Pfister[4], B. Kohn[1], [1]Small Animal Clinic, Freie Universität Berlin, [2]Department of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover, [3]Small Animal Clinic, Trier, [4]Institute of Comparative Tropical Medicine and Parasitology, LM-University, Munich, [5]Institute for Microbiology, University of Veterinary Medicine, Hannover, [6]Institute of Microbiology and Epizootics, Freie Universität Berlin and , [7]Institute of Animal Hygiene and Veterinary Public Health, University of Leipzig, Germany.

In most cases of inflammatory central nervous system diseases in dogs, etiological infectious agents cannot be detected. Immunopathological studies suggest that an antigen may trigger an autoimmune response ("Hit-and-Run-hypothesis") in some patients. In order to define the role of CVBD agents in the etiology of meningitis/meningoencephalitis of unknown etiology in dogs in Germany, blood and cerebrospinal fluid (CSF) of dogs were analysed for such pathogens.

366 client-owned dogs were included in the prospective multicenter study between 12/2009 and 11/2011. They were classi-

fied into 3 groups: 1) control group: dogs with non-inflammatory CNS diseases (e.g., intervertebral disc disease, n = 21), 2) dogs with meningoencephalitis of unknown etiology (n = 22), 3) dogs with steroid-responsive meningitis/arteritis (SRMA, n = 23). PCR was performed in blood and CSF to detect DNA of *A. phagocytophilum*, *E. canis* (n = 28) and *Bartonella* spp. Serum antibodies against *E. canis*, *Bartonella* spp., Tick-borne encephalitis virus (TBEV) and *Borrelia burgdorferi* sensu lato were analysed by IFAT or ELISA. A qualitative eubacterial PCR was performed in CSF. Group comparison was performed with univariate unifactorial variance analysis (SPSS 17.0 for windows, SPSS Inc., USA). A p-value < 0.05 was considered significant.

DNA of *A. phagocytophilum* was found in EDTA-blood of 4 dogs (SRMA group). Serological and PCR analyses for *E. canis* were negative in blood and serum of all dogs. *B. henselae* DNA was detected in blood of 1 dog (SRMA group). There were no significant differences between the 3 groups regarding the seroprevalences of *Bartonella* spp. and *B. burgdorferi* sensu lato. No antibodies against TBEV were detected. No DNA of CVBD agents was found in CSF. *Pasteurellaceae* spp. DNA was detected in 3 dogs of the control group, suggesting contamination.

There was no correlation detected between the presence of *E. canis* or *B. henselae* DNA or elevated antibody titers against *E. canis, Bartonella* spp., TBEV or *B. burgdorferi* sensu lato and inflammatory CNS diseases. *A. phagocytophilum* may play a role as a trigger of a secondary immunopathy which requires further investigation.

DIFFERENCIES IN INTEROBSERVER AGREEMENT OF THE BICIPITAL REFLEX EVALUATION IN DOGS BASED ON LEVEL OF EXPERTISE. F. Giebels, B. Kohn, S. Loderstedt, Clinic of Small Animals, Faculty of Veterinary Medicine, Freie Universität Berlin, Berlin, Germany.

The evaluation of reflex excitability and briskness is an essential part of the neurological examination in veterinary medicine. The biceps reflex in dogs is assumed to have a low reliability. In human medicine it is well known that interobserver agreement increases with training. The goal of this prospective study is to compare the evaluation of the canine biceps reflex between 3 groups of 4 observers each, with different level of expertise: veterinary neurologists (group 1), veterinary surgeons without special affinity to neurology and 3-4 years work experience (group 2) and last year veterinary students (group 3).

Sixty-one thoracic limbs of clinically and neurologically unremarkable dogs were examined by two examiners (S.L., F.G.). The examinations were videoed in a standardized manner, anonymized and evaluated blinded by 12 observers including the two examiners. The observers had to evaluate the presence of the reflex (0;1) and the reflex briskness using an accepted scoring system (0 (absent) to 4 (clonic)). The interobserver agreement for reflex presence was evaluated using Fleiss-Kappa (FK) for each group and Kappa (K) for each pair of observers within each group. The interobserver agreement for reflex-briskness was evaluated using FK for each group and weighted Kappa (K_W) for each pair of observers within each group. K compares an observed agreement with an agreement by chance, FK is an extension on more than two observers and K_w considers the degree of disagreement by weighting the results. K-, FK- and K_w-values were interpreted as follows: ($K < 0.0$), slight ($0.0 \leq K \leq 0.2$), fair ($0.21 \leq K \leq 0.4$), moderate ($0.41 \leq K \leq 0.6$), substantial ($0.61 \leq K \leq 0.8$), and near perfect to perfect ($0.81 \leq K \leq 1.0$).

The interobserver agreement for the reflex presence was substantial (FK: 0.606; mean K: 0.628) for group 1, fair (FK: 0.380; mean K: 0.4) for group 2 and 3 (FK: 0.262; mean K: 0.315). The agreement for the reflex presence for FK was significantly different between the three groups. For the reflex briskness mean K_w-values were within the upper range of the substantial level (0.551) for group 1, substantial (0.482) for group 2 and slight to moderate for group 3 (0.286). FK values for reflex briskness were as follows: group 1 (0.329), group 2 (0.313), and group 3 (0.158).

The level of expertise is an influencing factor on interobserver agreement of canine biceps reflex evaluation. In healthy dogs the biceps reflex can be reliably assessed by veterinary neurologists.

POLYNEUROPATHY WITH SPONTANEOUS RESOLUTION IN AN ABYSSINIAN KITTEN. A. Jeandel[1], M. Carlus[2], S. Blot[1], [1]Neurobiology Unit, [2]Pathology Unit, Université Paris-Est, Ecole Nationale Vétérinaire d'Alfort, Maisons-Alfort, France.

Polyneuropathy in kittens is a rare condition. Unusual courses with spontaneous resolution have been reported in Bengal and Snowshoe kittens.

A 6-months-old intact female Abyssinian cat was presented after a sudden onset of gait disorder one week before, with reluctance to walk and to jump and slow evolution to paraparesis. General physical examination was normal. Neurological examination revealed a paraparesis with weak pelvic limbs. Plantigrade stance and absence of flexion of the tarses were obvious. Only mild muscle atrophy was detected. Postural reactions and spinal appendicular reflexes were decreased to absent. Skin sensation was normal. There were no cranial nerves abnormalities. Vocalization was unchanged. A generalized neuromuscular disease was suspected. Electrophysiological investigation showed abnormal spontaneous electrical activities diffusely observed in the appendicular muscles, polyphasic muscular action potentials amplitudes with low amplitudes, and decreased sensory and motor nerve conduction velocities (compared to reference ranges published in kittens). A generalized polyneuropathy was strongly suspected. CSF cytological examination and proteinorachy were normal. Histological examination of the right peroneal nerve revealed no abnormalities. Muscle biopsies showed angular fibers suggesting denervation and intramuscular nerve branches myelinated fiber loss. A diagnosis of polyneuropathy with a more important distal component was established. Thoracic radiographs and abdominal ultrasounds were unremarkable. Serum biochemistry was normal but slightly elevated CK. Serum protein electrophoresis revealed an alpha-2 globulin increase. Serum FIV antibody and FeLV antigen tests were negative. *Toxoplasma* IgM titer was negative, IgG titer was slightly positive suggesting past exposure. Spontaneous gait improvement was observed a week later, and two weeks later, the owner considered the gait as normal.

This is the first description of a polyneuropathy with spontaneous resolution in an Abyssinian kitten. Relapsing courses of similar diseases are described in Bengal, Snowshoe kittens and in an adult domestic shorthair. Current follow-up is too short to compare this case to the others.

IMPACT OF SEIZURE NOSOLOGY AND SEMIOLOGY ON THE PREVALENCE OF HIPPOCAMPAL SCLEROSIS IN FELINE EPILEPSY. E. Wagner[1], J. Molin[1], M. Rosati[1], A. Fischer[2], L. Matiasek[2], T. Flegel[3], K. Matiasek[1], [1]Sections of Clinical and Comparative Neuropathology and [2]Neurology, Ludwig-Maximilians University, Munich, Munich, Germany, [3]Section of Neurology, Department of Small Animal Medicine, University of Leipzig, Leipzig, Germany.

Hippocampal sclerosis (HS) is a convergent tissue reaction to seizures in people and resembles a major cause of therapy resistance, disease perpetuation and progression. HS has been reported anecdotally in epileptic cats but systematic studies on the frequency and its clinical implications are missing.

This study was launched to elucidate the occurrence of HS in feline epilepsy patients and to investigate its association to the clinical presentation and aetiopathogenesis. Altogether, 93 archived brains from cats with clinically documented seizures were retrieved and underwent a detailed histopathological revision with regards to structural brain lesions and HS, in accordance to standard algorithms. The microscopic approach was accomplished by digital image analysis for calculation of neuronal cell density throughout the different cornu ammonis (CA) segments 1 through 4.

HS was identified in 31 cats, 21 with unilateral and 10 with bilateral manifestation (41 hippocampi in total). All but three HS-affected cats had structural brain lesions other than HS, which involved 25/41 hippocampi directly. Concerning seizure nosology, animals with inflammatory brain diseases were most likely to exhibit HS (51.7%). Also more than 40% of cats with intracranial mass lesions and metabolic/degenerative diseases (41.7% and 42.9% respectively) developed HS. With 12.5% each, epilepsy of unknown cause (EUC) and vascular forebrain

lesions resulted in HS. Individual HS cases were seen in all clinical seizure types. However, partial seizures with secondary generalisation and generalised seizures progressing into status epilepticus had the highest prevalence (50% and 70%). With no detectable bias from seizure type or aetiology, male cats were more frequently affected by HS (39.5%) than female patients (29%).

In summary, HS was histologically confirmed in one third of a non-selected population of epileptic cats. It may occur in all aetiopathogenic classes of epilepsy but it is particularly prevalent in inflammatory brain diseases, metabolic and degenerative disorders and tumours. Irrespective of the seizure nosology, HS is associated with a progression from partial seizures to generalised seizures and from the latter to status epilepticus. This interdependence suggests that HS may resemble a complicating factor in feline epilepsy.

SISPA – A SIMPLE APPROACH TO PCR DIAGNOSIS OF UNKNOWN AND UNEXPECTED NEUROTROPIC DNA VIRUSES. E. Dittberner[1], R. Fux[2], J. Molin[1], L. Derisio[3], K. Matiasek[1], [1]Section of Clinical and Comparative Neuropathology, Ludwig-Maximilians University of Munich, Germany, [2]Institute of Virology, Ludwig-Maximilians University of Munich, Germany, [3]Neurology Unit, Animal Health Trust, Newmarket, UK.

The aetiological trigger of inflammatory CNS diseases all too often remains undetermined due to either lack of specific diagnostic tools or the wrong expectation on the underlying pathogen. Identification of unknown neurotropic viruses more recently has been successfully achieved by high throughput polymerase chain reaction (PCR) methods. These technologies, however, are rarely available and very cost extensive. Hence, their application in clinical settings is limited.

This investigation was carried out to explore the possibility to isolate and identify neurotropic DNA viruses by cheap and technologically simple sequence independent single primer amplification (SISPA). In separate trials, Equine Herpes Virus (EHV) 1 containing supernatant and infected cells underwent genomic DNA degradation, proteinase treatment and labelling with sequence-independent double-stranded adapter molecules, following PCR amplification. The PCR products were cloned, sequenced and BLASTed against virus-specific sequences available via GenBank.

In all trials, the enzymatic pre-treatment allowed for obtainment of an adequate amount of DNA, feasible for subsequent PCR, cloning and sequencing. Adapter molecules in supernatants bound to viral DNA after nucleocapsid degradation and the cloned PCR product in all tests revealed 100% EHV1 homology. Positive detection rate and specificity were not measurably compromised when virus was accompanied by cellular proteins, RNA and genomic DNA of infected cells.

SISPA is an easy to perform, non-selective DNA amplification method with four days turnaround that allowed for straightforward detection of cellular EHV1 infection. Cellular components do not seem to have compromised the avidity of DNA binding adaptor molecules. Hence, SISPA appears promising for CSF analysis in animals with active CNS infection by DNA viruses. Further studies will focus on the minimal detection levels and the applicability on CNS tissue samples harvested from animals with clinical infection.

SYRINGOMYELIA IN HONG KONG PET POPULATION: 10-YEAR DATA FROM A PRIVATE VETERINARY CLINIC (2003-2013). S. Guo, D. Lu, Peace Avenue Veterinary Clinic, Hong Kong.

Syringomyelia (SM) is more readily diagnosed in veterinary medicine due to increased availability of magnetic resonance imaging (MRI). Pet breeds vary geographically worldwide. In Hong Kong, information on breeds predisposed to or conditions associated with SM is lacking. This retrospective study is the first novel report revealing these facts in a private referral practice in Hong Kong, based on the cases seen from 2003 to 2013. The MR system used is 0.2 Tesla, open permanent magnet.

Medical records were retrieved from the computer system and cases with an MRI diagnosis including "syringomyelia" or "syringohydromyelia" as part of the features were reviewed. Following an initial review, the cases were allocated into the following categories: SM associated with ventriculomegaly (SMven), neoplasia (SMneo), intervertebral disc prolapse (SMivd), inflammatory CNS disease (SMinf), trauma (SMtra), malformation (SMmal), fibrocartilaginous embolism (SMfce) and SM with undetermined cause (SMund).

A total of 189 cases were included. There were 182 dogs and 7 cats. Of these cases, the distribution is as follows: SMven (61 cases, 32.3%), SMneo (23 cases, 12.2%), SMivd (47 cases, 24.9%), SMinf (41 cases, 21.7%), SMtra (3 cases, 1.6%), SMmal (6 cases, 3.2%), SMfce (1 case, 0.5%) and SMund (7 cases, 3.7%). The most common breed is Pomeranian (62 cases) followed by Chihuahua (36 cases) and Yorkshire Terrier (20 cases). There are only 4 cases of Cavalier King Charles Spaniel, which is a breed commonly seen with the presence of syringomyelia, indicating a geographical distribution difference of the breeds affected. The SMven cases are likely to be related to Chiari malformation. However, due to the variable skull shapes of the breeds included, further study of the "normal variant" ventricular size in Hong Kong pet population is necessary. In conclusion, MR imaging of the brain should be included as part of the investigation if SM is found in the spinal cord as a large proportion of the spinal SM is associated with ventriculomegaly.

NIEMANN PICK TYPE C DISEASE DUE TO A MUTATION IN THE NPC2 GENE IN TWO RELATED CATS. E. Bianchi[1], S. Zampieri[2], C. Cantile[3], R. Saleri[1], G. Gandini[4], G. Bevilacqua[5], M. Dondi[1], A. Dardis[2], [1]Department of Veterinary Medical Sciences, University of Parma, Italy, [2]Centre for Rare Diseases, University Hospital, Udine, Italy, [3]Department of Veterinary Sciences, University of Pisa, Italy, [4]Department of Veterinary Medical Sciences, University of Bologna, Italy, [5]"I Portoni Rossi" Veterinary Hospital, Zola Predosa, Italy.

Niemann-Pick disease type C (NPC) is an autosomal recessive neurovisceral lysosomal storage disease, due to mutation in NPC1 or NPC2 genes and characterized by the progressive accumulation of cholesterol and other lipids within the late endosomes/lysosomes. NPC1 mutations account for more than 95% of the human cases and are the only one described in cats. This study describes two cats from the same litter affected by NPC caused by a mutation in NPC2 gene.

Two 6 months old kittens (1 male and 1 female) from a litter of 5 were evaluated for progressive neurologic signs, which started to be appreciated at the age of 3 months and were characterized by intention tremors and truncal ataxia. On neurological examination, the female (cat 1) showed a prayer-like posture, head and neck intention tremors and was still ambulatory with hypermetria of the hind limbs. The male (cat 2) was unable to stand without assistance because of the severe cerebellar dysfunction and had bilaterally reduced menace response. Neuroanatomical localisation was consistent with a cerebellar disorder.

3CBC, serum biochemistry and urine analyses of cat 1 showed a mild increase in ALT and CK. The same cat was treated with Clindamycin due to high titre positivity for anti-*Toxoplasma gondii* IgG. She also had a mild hepato- and splenomegaly revealed by ultrasonography and normal Magnetic Resonance of the brain. A lysosomal storage disease was suspected. Staining of cultured fibroblasts, derived from a skin biopsy of cat 1, with filipin, a polyene antibiotic that binds specifically to unesterified cholesterol (filipin stain), showed a massive perinuclear storage of this lipid which is consistent with the biochemical phenotype of NPC disease. Molecular analysis of NPC1 and NPC2 genes identified a homozygous intronic mutation (c.84 + 5G>A) in NPC2 gene.

Cat 2 died at the age of 10 months, cat 1 was euthanized when she had 21 months. Histological examination of cat 2 showed diffuse neuronal ballooning with Nissl substance and nuclei displacement, associated with moderate gliosis. Axonal spheroids were prominent in the cerebellar cortex. Neurons throughout the CNS, hepatocytes, and macrophages of lung and lymphoid tissue were distended by intracytoplasmic accumulation of faintly eosinophilic vacuolar material which did not stain with periodic acid-Schiff and Luxol fast blue.

Feline NPC caused by a mutation in NPC2 gene, described for the first time in this report, may represent a valuable model for understanding the molecular basis of NPC disorder.

ORIGINAL SURGICAL TREATMENT OF THORACOLUMBAR SUBARACHNOID CYSTS IN 6 CHONDRODYSTROPHIC DOGS. C.Bismuth, M. Millet, FX. Ferrand, D. Fau, T. Cachon, E. Viguier, C. Escriou, C. Carozzo, VetAgro Sup, Marcy-l'étoile, France.

Subarachnoid cyst (SAC) is a rare cause of spinal cord compression in veterinary medicine but has been increasingly noted with almost 100 cases. It is defined as a localized enlargement of the subarachnoid space with accumulation of cerebrospinal fluid. In humans, most SACs are found on the posterior aspect of the spinal cord, but very rarely develop in the cervical area whereas the cervical area (C2-C4) is well described particularly in Rottweilers. The distribution of the canine spinal thoracolumbar SACs, less observed, is mainly restricted to areas of high spinal mobility with some breeds described as Rhodesian Ridgebacks, Weimaraners, Shih Tzus, Shar Peis and Pugs. We carried out a retrospective study between May 2003 and April 2012 on dogs with spinal thoracolumbar subarachnoid enlargement which underwent surgical treatment. The main objectives of our study are to describe the original surgical treatment resulting from the observations made on the reported cases i.e. leptomeningeal adhesions and disk protrusion.

The dogs were 5 Pugs and 1 French bulldog. Myelography (2/6) and myeloscan (4/6) provided images compatible with a "classic" subarachnoid cyst. A hemilaminectomy was done first in all dogs. The disk was then always assessed with a dental spatula and a disk protrusion was characterized by a protrusion of the dorsal part of the annulosus fibrosus above the level of the floor of the two adjacent vertebrae. A disk protrusion was thus identified in 5/6 dogs and was addressed in a second time with a lateral corpectomy. For one pug, no disk protrusion was found, however, a small 'step' was felt with the dental spatula in between the two vertebrae compared with the SAC. A durotomy with a dural suspension were finally done in all dogs on the side of the hemilaminectomy. The spinal cord appeared depressed. Ventral adhesions of the dura mater to the pia mater and the arachnoid were observed in all cases. Those adhesions were not seen dorsally. Dissection of the dura mater and the arachnoid from the pia mater was done under optical magnification over the half or the 2/3 of the ventral circumference of the spinal cord until no more impediments was felt with a dental spatula all over the area. This part of the dura mater and the dura mater over the cyst were removed by durectomy. The surgery was done in a mean time of 2 hours (2-3 hours). The mean hospitalization time was 3 days (ranging from 2 to 4 days) with no or mild neurologic status degradation (ataxia to paraparesis). Follow-ups carried out from 7 months to 4 years postoperatively revealed that there was a good outcome in 5/6 dogs with persistence of a mild ataxia in all cases.

The subarachnoid cysts described in our 6 chondrodystrophic dogs can be considered as already described thoracolumbar subarachnoid cysts in their presentation. However, they are all associated with ventral leptomeningeal adhesions, as previously described in Rottweilers but in the cervical spine, and for 5 dogs, associated with a disk protrusion.

In conclusion, it seems that chondrodystrophic breeds and especially Pugs, can present original subarachnoid cysts with ventral leptomeningeal adhesions possibly induced by meningeal microtraumas due to a chronic disk protrusion. Consequently, we think that the surgical treatment has to address both lesions at the same time with a dissection and a removal of the ventral leptomeningeal adhesions and a treatment of the associated disk protrusion via a lateral corpectomy. The surgery appeared feasible and brought good results in our 6 dogs with no or mild immediate postoperative degradation, a long term improvement of the neurological status and no recurrence.

HISTOPATHOLOGY OF THE INTRACRANIAL CENTRAL NERVOUS SYSTEM IN 58 CATS WITH NEUROLOGIC SIGNS, CLINICAL, LABORATORY FINDINGS AND DIAGNOSTIC IMAGING DATA CONSISTENT WITH FELINE INFECTIOUS PERITONITIS. L. Mandrioli[1], E. Bianchi[2], L. Aloisi[1], R. Sirri[1], M. Morini[1], G. Rocchigiani[3], C. Cantile[3], C. Benazzi[1], G. Gandini[1], [1]Department of Veterinary Medical Sciences, University of Bologna, Italy, [2]Department of Veterinary Medical Sciences, University of Parma, Italy, [3]Department of Veterinary Sciences, University of Pisa, Italy.

Fifty-eight cats referred in the period 2000-2013 have been included in this retrospective study. Nineteen were DSH, 2 Main Coon and 37 of unknown breed; 26 were male, 21 female and for 11 cats the sex was not reported in the database; the mean age was 11.6 months. They had history, neurological signs, laboratory findings and diagnostic imaging data consistent with a progressive multifocal to diffuse involvement of the Central Nervous System (CNS) compatible with FIP. The inclusion criteria were based on the histopathologic diagnosis (gold standard) performed on CNS tissues.

Transverse sections of CNS from telencephalon, diencephalon, mesencephalon, cerebellum and pons-medulla oblongata were cut and routinely processed for histology and the elementary lesions, referable to inflammation and circulatory disturbances, were registered and resulted as follows: pyogranulomatous leptomeningitis (82.8%), pyogranulomatous chorioiditis (75.9%), perivascular cuffings (44.8%), meningeal vessels hyperemia (70.7%), fibrinoid necrosis of the vessels wall (43.1%), pyogranulomatous ependymitis (74.1%), periventriculitis (56.9%), hemorrhages (13.8%), neuronal degeneration and/or malacic foci (27.6%), thrombosis (15.5%), intraventricular proteinaceous exudate (34.5%), choroid plexus hyperemia (22.4%), vasogenic edema (44.8%).

Histopathologic findings were similar to those reported in literature: surfaces and meningeal layers were constantly involved by inflammation, with a typical perivenular pattern, choroid plexi (especially of the IV ventricle) were constantly involved with the inflammation and lateral ventricles showed a variable dilation. The fact that the pyogranulomatous lesions appear more severe in some regions is subject of debate.

CEREBELLAR FUNGAL GRANULOMA MIMICKING A NEOPLASTIC LESION IN A CAT. I.C. Böttcher[1], K. Truar[1], E. Ludewig[1], K. Matiasek[2], T. Flegel[1], [1]Department of Small Animal Medicine, University of Leipzig, Germany., [2]Section of Clinical and Comparative Neuropathology, Institute of Veterinary Pathology, Ludwig-Maximilians-University, Munich, Germany.

A 7-year-old male castrated domestic shorthair cat was presented due to progressive ataxia developing over 2 days, with mild apathy and anorexia. The cat had access to outdoor environment and was regularly vaccinated and dewormed. Physical examination revealed mild hypothermia, tachypnea without dyspnea and moderate pain at the thoracolumbar junction. Neurological examination showed generalised ataxia, swaying while standing or sitting and a slight head tilt to the right. The neurolocalisation was multifocal involving the cerebellum and the spine at the thoracolumbar region. Blood work revealed a mild lymphopenia with a normal total leukocyte count, mild normochromic normocytic anemia, mild azotemia and mild hypokalemia. Urine was only available after infusion therapy had been started and showed mild proteinuria and mild pyuria. Urine culture was negative. Spinal radiographs, abdominal ultrasound and blood pressure were unremarkable.

MRI of the brain exhibited a round, strongly contrast enhancing, well demarcated intraparenchymal lesion located midline in the cerebellar vermis. The lesion was largely surrounded by a T2 and FLAIR hyperintense signal compared to brain grey matter which was addressed as perilesional edema that involved both cerebellar hemispheres. There was a discrete pleocytosis in the CSF (9 cells/µl, ref. 0-3) with considerably elevated protein

(0.66 g/l, ref. <0.25). The pleocytosis was of a mixed cell type with 29% neutrophils, 67% monocytes, 3% macrophages and 1% lymphocytes. Taking into account that the lesion was well demarcated, strongly contrast enhancing and that the CSF was more indicative for blood brain barrier dysfunction than inflammation, a neoplastic lesion was considered most likely. After the diagnostic imaging the cat showed marked spasticity and was unable to stand or walk. The hypothermia persisted despite treatment and bradycardia occurred. After 3 days the cat showed reduced level of consciousness and vertical nystagmus and was euthanized. Histopathological examination of the brain revealed a focal necrotising pyogranulomatous meningoencephalitis (cerebellitis) with intralesional pigmented, branching, septated hyphae. The final diagnosis was therefore fungal meningoencephalitis.

As primary pathogenic fungi are not common in Germany, the most likely cause in the presented case is an opportunistic fungal infection. However, neither immunosuppressive diseases nor treatment were apparent that usually promote the opportunistic infection. Therefore the aetiology remains unknown. This case also nicely demonstrates that inflammatory lesions do not need to present as multifocal lesions on MRI.

INFLUENCE OF BODY WEIGHT ON CAUDA EQUINA SYNDROME PATTERNS IN DOGS – A RETROSPECTIVE CT EVALUATION. C. Daraban[1], V. Vulpe[1], M. Musteata[1], D. Mocanu[1], L. Meomartino[2], [1]Faculty of Veterinary Medicine, Iasi, Romania, [2]Faculty of Veterinary Medicine, Naples, Italy.

The aim of the study was to investigate if body weight (BW) influences the lesional pattern of the lumbosacral junction (LSJ) in dogs with cauda equina syndrome (CES).

The CT lesional patterns of 60 dogs with CES were retrospectively evaluated. There were 45 males and 15 females, with a mean age 7.56 ± 3.15 years (range 1-15 years) and mean weight 29.5 ± 13.93 kg (range 4 - 66 kg). The dogs were divided into three groups according to BW: Group 1: 0 - 15 kg (9 dogs), Group 2: 15 - 30 kg (24 dogs), Group 3: $\geq$ 30 kg (27 dogs). The lesion associations were analyzed for each group. Data were statistically analyzed with SPSS 20.0 software. Spearman's test was used for analyzing correlations with a $p < 0.05$ significance level.

In the majority of CESs, the pathogenesis includes one or more different degenerative lesions. No specific correlation was found between the lesional pattern and a specific breed. Regarding the lesional pattern, no significant correlations were observed for group 1. In group 2 a strong correlation was observed between the vacuum phenomenon and ventral sacral subluxation (spondylolisthesis) ($r = 0.79$; $p < 0.001$) and with Schmorl's node ($r = 0.44$; $p = 0.04$). Foraminal stenosis was found in association with endplate sclerosis ($r = 0.53$; $p = 0.01$) and arthrosis of the articular processes ($r = 0.47$; $p = 0.03$). Discospondylitis was correlated with Schmorl's node ($r = 0.795$; $p < 0.001$) and endplate sclerosis ($r = 0.575$; $p = 0.006$). For group 3, we observed a significant correlation between spondylosis deformans and foraminal stenosis ($r = 0.55$; $p < 0.001$), endplate sclerosis with spondylosis deformans ($r = 0.613$; $p < 0.001$) and arthrosis of the articular processes ($r = 0.48$; $p = 0.006$), spondylosis deformans with arthrosis of the articular processes ($r = 0.562$; $p = 0.001$) and foraminal stenosis ($r = 0.558$; $p = 0.001$), Hansen I and arthrosis of the articular processes ($r = 0.38$; $p = 0.038$), Negative correlations were observed between spondylolisthesis and endplate sclerosis ($r = -0.47$; $p < 0.001$). Intervertebral disc extrusion and protrusion were found to be negatively correlated ($r = -0.447$; $p = 0.042$ and $r = -0.468$; $p = 0.009$ respectively), for both group 2 and 3.

Even though the LSJ has been extensively studied, to our knowledge there are no retrospective reports which describe the influence of BW in the development of a lesional pattern in CES. Due to the specific association of different LSJ degenerative lesions we observed in medium and large breed dogs, we hypothesize that in the development of a CES lesional pattern, BW has a determinant role.

NEUTROPHILIC INFILTRATION IN CANINE AND FELINE MENINGIOMAS. N. Jungwirth[1], A. Oevermann[2], M. Vandevelde[1], D. Gorgas[5], P. Kuhnert[4], M.G. Doherr[5], K. Raith[1], D. Henke[1], [1,2]Division of Neurological Sciences, [1,3]Department of Clinical Veterinary Medicine, [2,5]Department of Clinical Research and Veterinary Public Health, [3]Clinical Radiology Division, [4]Bacteriology Division, Vetsuisse Faculty, University of Bern, Switzerland.

Neutrophils in meningiomas have been sporadically described either in the cerebrospinal fluid (CSF) or in histological sections, but this phenomenon has not been investigated in depth so far.

The aim of this study was to evaluate the frequency rate of neutrophilic infiltration of meningiomas and to investigate relations to secondary infection, tumour grade, or histological subtype.

In this retrospective study 64 canine and 28 feline meningiomas have been evaluated histopathologically. Age, breed, gender, magnetic resonance imaging (MRI) findings, localization, results of CSF analysis, and histopathological findings including tumour grade, histological subtype, grade of neutrophilic infiltration, presence of abscess formation, necrosis and edema were evaluated, and analyzed statistically. Gram and Grocott's Methenamine Silver stainings, and 16S rRNA gene sequencing was performed on selected cases.

Canine meningiomas were more often atypical (Grade II) than benign (Grade I) compared to feline meningiomas ($P < 0.001$). Neutrophilic infiltration was evident in 75% of canine and 82% of feline meningiomas. Dogs had more often abscess formation than cats ($P = 0.015$). On MRI, abscesses were visible as cavitary/ cystic compartments. Evidence of bacterial infections was not present. In both dogs and cats, there was no correlation between neutrophilic infiltration and tumour grade ($P = 0.357$/ 0.862) or subtype ($P = 0.122$/ 0.573).

Suppuration of meningiomas is a frequent phenomenon in dogs and cats. Bacterial infection and ischemic necrosis were excluded as causes of suppuration. The histopathological analysis suggests that neutrophil invasion may precede tumour necrosis; and may represent an anti tumoural response. Further studies are needed to explain the mechanism of suppurative reactions in meningiomas, and its clinical relevance.

ODONTOID PROCESS FRACTURE IN A CALF – CLINICAL PRESENTATION AND IMAGING FINDINGS. V. Hülsmeyer[1], K. Flatz[2], K. Putschbach[1], M. Bechter[3], S.Weiler[3], M. Feist[3], [1]Section of Neurology, Clinic of Small Animal Medicine, Ludwig-Maximilians-University, Munich, [2]Clinic for Small Animal Surgery and Reproduction, Ludwig-Maximilians-University, Munich, [3]Clinic for Ruminants with Ambulatory and Herd Health Services, Center of Clinical Veterinary Medicine, Ludwig-Maximilians-University, Munich, Germany.

A 6-week-old female Simmental calf was referred to the Clinic for Ruminants for further work-up of an acute onset of lateral recumbency.

Physical and laboratory examination showed no significant abnormalities. On neurological examination the calf presented with non-ambulatory tetraparesis and lateral recumbency. The calf appeared conscious and responsive to stimuli and cranial nerve function was normal. With support, the calf was able to stand but showed postural reaction deficits of all four limbs besides normal spinal reflexes and increased muscle tone. A pronounced cervical hyperesthesia was evident. The neuroanatomical localization was cervical spinal cord segments C1-C5. Based on history and signalement, the most likely differentials were trauma, inflammation or anomaly. Radiographs of the cervical vertebral column were suspicious for fracture of the odontoid process with vertebral subluxation at this level. During transportation for MRI investigation the calf showed deterioration of neurological signs and was euthanized. Post-mortem MRI showed a severe compression of the spinal cord at the level of C2 due to fracture of the odontoid process and subsequent dorsal displacement of the C2 vertebral body. Cranial and caudal to the compression site the spinal cord appeared swollen and showed a

T2-hyperintense intramedullary signal compatible with spinal cord edema/contusion. CT images and post-mortem examination confirmed an odontoid process fracture.

Odontoid fracture with subsequent atlantoaxial instability should be considered as a differential in acutely recumbent calves, and clinical manifestation and imaging findings may be comparable to dogs diagnosed with dens fracture and subsequent atlantoaxial instability.

NEURONAL VERSUS INTERSTITIAL AND VASCULAR CHANGES IN DORSAL ROOT GANGLIA OF ENTRAPPED NERVE ROOTS– IMPACTS ON PATHOPHYSIOLOGY. U. Foitzik[1], T. Gödde[2], M. Rosati[1], F. Steffen[3], H.A. Volk[4], T. Flegel[5], K. Matiasek[2], [1]Section of Clinical and Comparative Neuropathology, Ludwig-Maximilians University, Munich, Germany, [2]Neurology Referral Service, Tierarztpraxis Stauffeneck, Piding, Germany, [3]Neurology Unit, Tierspital, Vetsuisse Faculty, University of Zurich, Switzerland, [4]Clinical Science and Services, Royal Veterinary College, London, UK, [5]Section of Neurology, Department of Small Animal Medicine, University of Leipzig, Leipzig, Germany.

Nerve root entrapment uniformly results in thickening of nerve root fascicles and their associated dorsal root ganglia (DRG), even if the latter are positioned proximal to the compression site. The pathomechanisms leading to ascending changes and their pathophysiological consequences are poorly understood. Hence, we performed a systematic histological study in DRG of clinically affected dogs using brightfield techniques, immunohistochemistry, polarised light studies and digital image analysis.

Twenty-two DRG (1 cervical, 2 thoracic, 12 lumbar, 7 sacral) were obtained from 14 dogs with spinal nerve root entrapment. Low level neuronal drop-out was seen in 12 DRG, while 7 samples showed significant cell loss. Residual neurons only occasionally presented with degenerative features and triggered satellite cell hyperplasia. All entrapped DRG showed a significant increase in neuronal expression frequency and intensity of calcium channel subunit alpha2delta. This gain was particularly evident in neurons co-expressing pain peptides SP and CGRP. Inflammatory infiltrates were seen in 3 DRG (of different dogs), 2 of which were lymphohistiocytic and 1 was polymorphnuclear. The endoneurial matrix of 10 DRG showed a diffuse accumulation of glycosaminoclycans. Special stains revealed a type I-predominant endoneurial collagenosis in 17 DRG. Nineteen animals presented with a perineurial fibrocollagenous hypertrophy. Vascular abnormalities were seen in 14 DRG, resembling venous dilation (7), arterial media hyperplasia (3) and endothelial prominence (5).

Compression of DRG and farther distal nerve root segments lead to sensory neuron hyperfunction and/or excitatory synaptogenesis as reflected by the surrogate marker and voltage gated calcium channel subunit alpha2delta. In part, redundant and aberrant synaptic connections may result from deafferentiation of the dorsal horn as a consequence to neuronal losses in the DRG. Neither the compact DRG nor its diffuse part shows significant inflammatory changes that would explain sensitisation and allodynia. Similar to the entrapped nerve segments degenerative changes of the DRG result from direct pressure, ascending perineurial constriction and increased endoneurial vascular resistance. The perineurial hypertrophy and endoneurial collagenosis are type I fibre predominant, incompatible with spontaneous remission.

MEGAOESOPHAGUS: A CLINICAL SIGN OF UNDERLYING NEUROLOGICAL DISEASE? A RETROSPECTIVE STUDY OF 100 DOGS (2001-2011). S. Gomes[1], L.Van Ham[2], A.Van Ham[2], E. Ives[1], A. Vanhaesebrouck[1], [1]The Queen's Veterinary School Hospital, Department of Veterinary Medicine, University of Cambridge, UK, [2]Department of Medicine and Clinical Biology of Small Animals, Faculty of Veterinary Medicine, University of Ghent, Belgium.

Most clinical survey studies of canine megaoesophagus date from over 15 years ago. Recent advances in both knowledge and diagnostic techniques means that more dogs with megaoesophagus may be diagnosed with an underlying neurological disorder, as has recently been suggested for canine laryngeal paralysis.

Medical records of 100 dogs diagnosed with megaoesophagus between 2001 and 2011 were reviewed. Inclusion criteria were the presence of clinical signs attributable to generalized oesophageal dilation or hypomotility recognized on thoracic radiographs or fluoroscopy, excluding structural causes. Further diagnostic tests included adrenal and thyroid function testing, acetylcholine-receptor antibody levels, edrophonium-response test, electrodiagnostics, muscle and/or nerve biopsies, neuroimaging and cerebrospinal fluid analysis, as appropriate.

The most common clinical sign was regurgitation (88%), followed by neurological deficits (51%) and respiratory signs (49%). Generalized weakness was present in 43% of total cases, laryngeal paralysis in 18%, dysphagia in 9%, facial weakness in 5%, and intracranial signs in 3%.

Idiopathic congenital megaoesophagus was presumed in 10 cases with an immature age at onset (10%). The other 90 cases were considered acquired, with neurological disease confirmed in 51 dogs (51% of total). Neurological diagnoses included myasthenia gravis (36%), polyneuropathies (7%), polymyopathies (5%) and brain or cranial nerve disorders (3%).

Outcome data was available for 76 dogs. Death or euthanasia directly related to megaoesophagus occurred in 46% of cases. Clinical signs resolved in 31% of cases and persisted in 22%. A diagnosis of myasthenia gravis was associated with a better outcome, with resolution of clinical signs in 56% of cases.

This study identified a significantly higher percentage of dogs with acquired megaoesophagus and an underlying neurological disease (57%) than has been previously reported (28-32%). As the canine oesophagus consists of a larger proportion of skeletal muscles than in other animals and is innervated by one of the longest nerves, the (para) recurrent laryngeal nerve, it is prone to be affected in neuromuscular disorders. The detection of an associated neurological disorder is important, as this may allow institution of the correct treatment with a subsequently better outcome.

EVALUATION OF MATRIX METALLOPROTEINASE-9 AS A BIOMARKER OF THE PROGNOSIS IN DOGS WITH THORACOLUMBAR INTERVERTEBRAL DISC HERNIATION. H. Ueno, Y. Fujisaki, T. Matsunagi, S. Isoda, H. Soma, K. Fujita, Division of Small Animal Clinical Sciences, Department of Veterinary Medicine, Rakuno Gakuen University, Ebetsu, Hokkaido, Japan.

The aim of this study is to describe the utility of Matrix Metalloproteinase-9 (MMP-9) in serum for a diagnosis of canine acute thoracolumbar intervertebral disc hernia (IVDH), especially progressive myelomalacia.

[Study 1] MMP-9 activity in the serum of healthy dogs without IVDH: 18 beagles (young age: n = 6, middle age: n = 5, advanced age: n = 7), 14 miniature Dachshunds (MD, middle age: n = 6, advanced age: n = 8), and Labrador Retriever (LR, young age: n = 6, middle age: n = 10, advanced age: n = 9) were evaluated. Gelatin zymography (GZ) was used. To measure MMP-9 activity in the serum, we used the SensoLyte® 490 Fluorometric Assay Kit (AnaSpec, Fremont, CA, US). In GZ, activity of pro-MMP-2 and MMP-9 was detected in all non-IVDH dogs. The MMP-9 activity value of advanced age beagles (9.1 ± 1.2) was higher significantly than that of young age (5.5 ± 1.1) and middle age (5.7 ± 1.4), respectively. In the MMP-9 activity of MD, there was not the significant difference between middle age (6.0 ± 1.8) and advanced age (5.8 ± 3.0). MMP-9 activity of LR was decrease by aging as young age (10.5 ± 0.7), middle age (8.8 ± 1.4) and advanced age (6.9 ± 1.1). [Study 2] MMP-9 activity in serum of the dogs with IVDH: Twenty-seven dogs with acute thoracolumbar IVDH diagnosed by CT or MRI were included. Serum specimens were collected at initial presentation from all cases. These IVDH dogs were graded at initial evaluation by the scale by Windsor et al. as grade (G) 1: spinal pain, G2: ambulatory paraparesis and/ or ataxia and/ or proprioceptive deficits, G3: non-ambulatory paraparesis, G4: paraplegia with intact deep-pain perception and G5: paraplegia with no deep-pain perception. In our study, regarding spinal cord injury scale, 7 dogs had G3, 12 dogs had G4, and 8 dogs had G5. The measurement of MMP-9 activity in the serum, we also used the SensoLyte® 490 Fluorometric Assay Kit. In the MMP-9 activity, there was not the significant difference among G3 (6.9 ± 1.9), G4 (10.5 ± 5.6) and G5 (5.4 ± 1.5). [Study 3] MMP-9 activity in the serum of the dogs with Progressive myelomalacia: Progressive myelomalacia (PM) was diagnosis clinically in 6 dogs. The

MMP-9 activity in the dogs with PM (11.4 ± 4.5) was higher significantly compared with that of G5 IVDH (5.4 ± 1.5).

From these studies, (1) In GZ, activity of MMP-9 was detected in all non-IVDH dogs. Therefore, it would be impossible to use as the prognostic value of the dogs with IVDH of testing for serum MMP-9 activity by GM. (2) The results suggest the utility of the quantitative evaluation for MMP-9 activity in serum in the diagnosis of progressive myelomalacia.

DIENCEPHALIC IMPACT OF BRAIN CONTUSION IN DOGS – A CASE SERIES. L. Nagel, J. Molin, M. Rosati, K. Matiasek, Section of Clinical and Comparative Neuropathology, Institute of Veterinary Pathology, Ludwig Maximilians University Munich, Munich, Germany.

Diencephalic lesions comprise a major cause of post-impact morbidity and mortality in people with contusive brain injury. Apart from interference with diencephalic neuronal networks, lesions to the hypothalamus may compromise central regulation of homeostasis and cause pituitary malfunction. Little is known about the involvement of the diencephalon in traumatic brain injury (TBI) in dogs. Hence, we elucidated the possibility of thalamic and hypothalamic lesions in a series of dogs affected by traumatic brain injury (TBI).

The study involved five dogs subjected to post-mortem examination after contusive (4/5) and combined contusive-lacerative types (1/5) of TBI. The sampling scheme was adapted to forensic neuropathology guidelines in humans. Routine haematoxylin-eosin stain was accompanied by special stains for neuronal cell death, diffuse axonal injury (DAI) and astroglial proliferation.

In all dogs the impact was localised to the cerebral convexities. In only two animals a couple of neurons stained positive for the cell death marker in mid thalamic nuclei, ipsilateral to the primary lesion. All five dogs stained positive for axonal damage, compatible with DAI. In two dogs however, the staining was restricted to the capsula interna and thalamic radiation. In the remaining three dogs DAI extended bilaterally towards thalamus and hypothalamus with an ipsilateral pronounciation in absence of colocalising parenchymal lesions. All but one animal exhibited a far more widespread labelling of diencephalic nerve cell perikarya with amyloid precursor protein (APP). The two brains with neuronal damage also showed a mild astrogliosis.

TBI in dogs may result in bilaterally asymmetric thalamic and hypothalamic damage through coup and contrecoup contusion. Thereby TBI impacts mainly on the fibre projections in terms of a DAI. More severe TBI also appears to involve and damage the diencephalic neurons at the level of the perikaryon. Both lesions may impair the function of the diencephalon, in general, and of the hypothalamus, in particular. The consequences regarding body homeostasis and pituitary gland function in canine TBI patients remain to be determined.

EFFECT OF MYELOGRAPHY ON LOW-FIELD MAGNETIC RESONANCE IMAGING THORACOLUMBAR STUDY TIME IN 70 DOGS. J. Ribeiro, A. Ribeiro, L. Rocha, Referência Veterinária, Alcabideche, Portugal.

Low-field magnetic resonance imaging (LF-MRI) studies are time consuming due to the length of the sequences needed to acquire images with diagnostic quality. Many LF-MRI units operated by veterinarians also have a small maximum field of view (FOV). Anatomic regions larger than the maximum FOV of these machines need repeating the study as many times as to fully include the region to be investigated. This is particularly true in thoracolumbar (TL) region of large dogs. This study proposes to investigate the effect of combined myelography and LF-MRI on the MRI study time.

70 dogs with neurological deficits localized to the T3-L3 spinal cord region underwent direct digital myelography followed immediately by low-field (0,18T) short-FOV (14 cm) MRI (VetMR, Esaote Genoa, Italy). Myelography study time was calculated subtracting the time recorded on the first radiograph obtained after the contrast injection from the time on the last radiography. MRI study time was calculated adding the times of the studied FOVs. The time of each studied FOV was calculated subtracting the scout end time from the last sequence end time. The sequence number per FOV (Seq/FOV) was also recorded. The distance between the cranial border of vertebra T2 to the caudal border of vertebra L3 (TLd) was measured in each dog from ventrodorsal radiographs (av.28,8 cm, 15,5-45). This distance was divided by 14 to determine 37 dogs with 2FOV, 30 dogs with 3FOV and 3dogs with 4FOV. The necessary time to study all FOVs of each dog with MRI alone was calculated adding its MRI time to 35 min for each additional FOV needed to fully study its TL region. This was designated estimated MRI time (ET-MRI) presuming localizers and at least one sequence in each plane (sagittal, dorsal and transverse).

Myelography av. time was 10 min (1-36). In 35 (50%) dogs myelography located the lesion(s) and provided diagnostic information, in 28 (40%) dogs it only located the lesion(s) and in 7 (10%) dogs it was inconclusive. MRI post myelography studied one FOV in 54 dogs (77.14%), two FOV in 13 dogs (18.57%), three FOV in 1 dog (1.43%) and four FOV in 2 dogs (2.86%). The av. time was 60 min (25-104) for the first FOV, 26 min (8-48) for the second FOV, 30 min for the third FOV (9-60) and 13 min for the fourth FOV (9-18). MRI av. total time was 67 min (31-121). ET-MRI av. total time was 111 min (66-179). Myelography contributed to reduce the MRI study time (av. 39 min, 6-67) and the number of studied FOVs (av. 1,23) as compared to the ET-MRI particularly in larger dogs (TLd>29 cm). In 7 dogs (10%) the MRI time was longer av.15 min (1-26) explained by multiple lesions, inconclusive myelography, and two of these were smaller dogs (Tld<17 cm) also with an increase in the total number of Seq/FOV. One explanation could be the difficulty in obtaining diagnostic information in smaller patients due to poor resolution in low field MRI. More studies are needed to determine the effect of combined myelography and LF-MRI in study time, safety, diagnostic yield and outcome.

CASE REPORT – INTRAMEDULLARY SPINAL GANGLIOGLIOMA IN A DOG. P. Karli[1], M. Vandevelde[2], K. Gendron[3], F. Forterre[4], D. Henke[1], [1,2]Division of Neurological Sciences, [1,3]Department of Clinical Veterinary Medicine, , [2]Department of Clinical Research and Veterinary Public Health, [3]Division of Clinical Radiology, [4]Department of Surgery and Orthopaedics, Vetsuisse Faculty, University of Bern, Switzerland.

Canine spinal tumours are relatively uncommon and classified based on their neuroanatomical location as either extradural, intradural-extramedullary or intramedullary.

A 9-year-old male intact mixed breed dog was presented because of a progressive left pelvic limb paresis. Neurological examination showed an ambulatory tetraparesis with left sided lateralization, and reduced segmental spinal reflexes in the left thoracic limb. The lesion was localized to the C6-Th2 spinal cord segments being more pronounced on the left side.

MRI revealed an intramedullary lesion on the left side immediately dorsal to the C7/Th1 intervertebral disc space, which caused an increase in cord diameter resulting in a focal interruption of the subarachnoidal fluid column. The lesion appeared isointense when compared to spinal cord with a thin hyperintense rim on T2w images. This rim was mildly hyperintense on T1w images as well. Strong contrast uptake was observed peripherally broader than the T2w and T1w hyperintense ring. The radiologic diagnosis was intramedullary, left-sided space- occupying lesion with severe reduction of the cervical spinal cord parenchyma at the level of C7.

The dog underwent a C7/Th1 hemilaminectomy. The spinal cord appeared mildly discoloured. After durotomy and subsequent myelotomy the tumour could been visualized and removed en masse. The patient showed marked, but transient, post surgical deterioration with progressive improvement and recovery with mild persisting neurological deficits. One year after surgery the dog's neurological state was unchanged and he didn't show any signs of recurrence.

Histopathological assessment of the tumour showed a well defined, densely packed mass. In two regions, microscopic fragments of normal spinal cord consisting of white matter attached to the mass could be identified reflecting the intramedullary location. The neoplasia consisted of three different regions with a center of necrosis, an inner ring of cells frequently organized in strands or palisades and an outer ring containing numerous vascular structures surrounded by tumour cells. This region also contained several blood vessels surrounded by large mononuclear inflammatory cuffs. The neoplastic cells often showed a triangular, bi- to multipolar cell body with big open faced, prominent

nuclei. The morphology of the cells was often reminiscent of neurons. Immunohistochemistry revealed marked positivity for neuronal specific enolase (NSE), moderate positivity for synaptophysin and mild positivity for neurofilaments in many tumour cells. In addition the tumour stained markedly positive for glial fibrillary acid protein (GFAP), vimentin and cytokeratin. The GFAP positive cells showed clearly neoplastic characteristics. The histopathological diagnosis was a mixed glioneuronal tumour, consistent with a ganglioglioma.

The MRI characteristics with ring-like T2w and T1w hyperintense signal are striking and correlate well with the histopathological findings. Such tumours are extremely rare in animals. To our knowledge there are no reports of surgically treated glioneuronal tumours in pets. The good outcome with no recurrence for more than one year after surgery suggests that despite the intramedullary localization of such tumours, surgical treatment should be considered

SPONTANEOUS INTRAMEDULLARY AND SUBARACHNOIDAL HAEMORRHAGES IN TWO DOGS PRESUMABLY ASSOCIATED WITH VASCULITIS. A. Maiolini, A. Röthlisberger, A. Tipold, V.M. Stein, Department of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover, Germany.

Spontaneous intramedullary and subarachnoidal haemorrhages are rare events in veterinary medicine. Most cases are secondary to spinal cord trauma, other causes include coagulopathies, hypertension, platelet disorders and vasculopathies. This report describes spontaneous haemorrhage presumably associated with steroid responsive meningitis-arteritis (SRMA) in two female dogs, a one-year-old Nova Scotia Duck Tolling Retriever (dog 1) and a 10-months-old Labrador Retriever (dog 2).

Dog 1 was evaluated for a one-week history of stiff gait and pyrexia, which suddenly progressed to tetraparesis. The neurological examination was consistent with a C6-T2 myelopathy. Haematology showed a moderate leucocytosis. Magnetic resonance imaging (MRI) revealed a heterogeneous (hyperintense to isointense in T2) intramedullary lesion at the level of C4 to C6. In the HEMO sequence the T2-hypointense areas appeared hypointense suggesting an acute haemorrhagic component. The cerebrospinal fluid (CSF) was reddish and turbid and its analysis revealed a severe neutrophilic pleocytosis (1536 cells/μL) and increased total protein.

Dog 2 had a two-week history of lameness and pyrexia, partially improving after treatment with antibiotics and a combination of steroidal and non-steroidal anti-inflammatory drugs. At presentation the dog was non-ambulatory tetraparetic. The neurological findings suggested a C6-T2 myelopathy. Complete blood cell count showed a mild leucocytosis and slight anaemia. MRI demonstrated a focal well-circumscribed extra-medullary lesion ventrally compressing the spinal cord at the level of C6-7. The lesion appeared isointense in T1 and hypointense in T2, SPAIR and GRASE, findings compatible with an acute subarachnoidal haemorrhage. The CSF was xanthochromic and its analysis revealed a severe mixed pleocytosis (5120 cells/μL) and increased total protein values. IgA levels were elevated in CSF and serum in both dogs. Based on these findings a tentative diagnosis of SRMA associated with haemorrhage was made in both cases and an immunosuppressive glucocorticosteroid therapy was initiated. The neurological status improved rapidly in both cases and they were clinically normal at follow-up examinations.

In this case report neither coagulopathies, hypertension or platelet disorders have been detected. SRMA was suspected particularly based on the severe pleocytosis and elevated IgA levels. We hypothesise that a systemic vasculitis was responsible for the spontaneous haemorrhages in these dogs. Gradient echo pulse sequences and CSF analysis were essential to diagnose this unusual presentation.