DEAR FRIENDS AND COLLEAGUES,

Dear ESVN/ECVN family,

It is my great honour and pleasure to welcome you at this year's conference as your current President of eCVN. ESVN/ECVN have been shaping the Veterinary Neurology community across Europe and the World for multiple decades now. The climax of the society’s and the college's annual cycle is our scientific conference. Every year its quality is going from better to better, from strength to strength. The topic of this year’s conference is Movement Disorders – “Moving on”. Movement disorders remain an underdeveloped area in our specialism. I am therefore especially grateful to Paul Mandiger, chairman of the 28th ESVN/eCVN congress, and his colleague Niklas Bergknut to have taken on this challenging topic and come up with a stellate scientific programme, which will help us to develop our understanding further. We are very fortunate to have so many outstanding speakers sharing their expertise and knowledge from human and veterinary medicine alike. Please make them feel welcomed. Please also join me in thanking the scientific committee (Rita Gonçalves, Peter Smith, Nicholas Jeffery, Ane Uriarte; headed by Nicolas Granger) for ensuring the high standard of the scientific programme

Welcome and enjoy all the scientific and social aspects of this conference.

Yours as always

Holger Volk
ECVN President

---

DEAR COLLEAGUES AND FRIENDS,

It is a great pleasure to welcome you all to the 28th ESVN-ECVN congress. Theme of this year is the challenging subject of Movement Disorders.

From VIn I copied a text we will all recognise:

7 month old FI pitbull mix presented peracutely with the behavior demonstrated in this video. 2 days prior to onset there was a brief period of diarrhea that resolved spontaneously. The dog has been doing this for an entire week except when she sleeps and for 1 hour when she was at the dog park. Normal CBC, chem, and UA. No meds. No recent vaccines. Have not imaged yet or obtained CSF. Certainly seems behavioral but I have never seen it come on so acutely. Of course could be a structural problem but was just curious what other’s thoughts were before proceeding. Given the history of diarrhea and the cost of imaging the owner prefers empirical medical treatment so I have started Prilosec, Flagyl, Xanax, and oral acepromazine. During the appointment I gave IV acepromazine and she very quickly settled down and the behavior stopped.

As veterinary neurologists we all struggle, like (so I am told) our human counterparts, with the broad clinical presentation in paroxysmal disorders, tremors, and epilepsy. I am convinced that despite the invention of the smart phone, which has made our life much easier, we all see at least once a week a case that’s not that straightforward. Not only do they offer an intellectual challenge to us they are excellent examples of the need to constantly strive for the best of ourselves.

We have invited an excellent group of world leading experts, MD’s and DVM’s, actively working in the field of epilepsy and movement disorders to shed their ‘State of the Art’ views on movement disorders (and epilepsy). We will discuss movement disorders in humans and how, if possible at all, to differentiate from epilepsy. We will have insights on the disorders we are facing in our animals and discuss how we could diagnose and treat them. And to test ourselves three of the invited speakers will guide us, during a 1.5 hours ‘smart phone film lecture’ through the foggy broad presentations owners can send us. If we succeed we can submit it for publication with the first veterinary publication mentioning all delegates as contributors. It would not be Amsterdam if we would not have somebody present to tell you something about the artificially induced movement disorders you can see (and experience) in the city centre.

The venue is the prestigious Royal Tropical Institute (www.royaltropicalinstitute.nl/en/) created in 1926. We will meet each other, in a lecture hall where once the greatest scientists of their time, presented their global explorations. And although the building is old, the lecture room is truly ‘State of the Art’.

As far as our socials are concerned we will start of on Thursday the 17th in the Amsterdam Zoo ‘Artis’, next we will have a gala dinner in the city centre and finish it off with one of the best live band with a mixture of (veterinary) musicians. And if you want you can take a boat trip (offered to you by the city of Amsterdam) or visit one of the many musea Amsterdam has.

Good to have you over. Please enjoy!

Paul Mandigers
Chairman of the 28th ESVN-ECVN congress

---

Holger Volk
ECVN President

Paul Mandigers
Chairman of the 28th ESVN-ECVN congress
Executive Committee ESVN-ECVN Congress 2015

Holger Volk
President

Veronika Stein
Secretary

Thomas Flegel
Vice-president

Laurent Garosi
Past President

Valentina Lorenzo
Treasurer

Lara Matiasek
Head of Examination Committee

Sam Long
Head of Education Committee

Julien Guevar
ESVN Representative

Paul Mandigers
Chairman

Niklas Bergknut

Susanne Pauwels

Local Organising Committee

TABLE OF CONTENTS

1. Foreword of the president of the ESVN-ECVN College 2
2. Foreword of the chairman of the ESVN-ECVN Congress 3
3. Members of the Executive Committee and local organizing committee 4
4. Table of contents 5
5. Platinum sponsors and sponsors of the congress 7
6. Who’s who 9
9. Useful information 9-10
10. Layout of the exhibition room 11
12. Congress programme 12-13
13. Invited speakers 15
14. Oral abstracts 37
15. Poster abstracts 55
This is the One

The One doet een compleet biochemisch onderzoek in enkele minuten
The One maakt betere veterinaire zorg mogelijk
The One is zeer accuraat en betrouwbaar
The One is uitermate gebruiksvriendelijk
The One brengt de kwaliteit van een referentielab in uw praktijk

IDEXX Catalyst One™

IDEXX Laboratories

De IDEXX Catalyst One™ biedt de veterinaire praktijk snelle en accurate in huis diagnostiek met de kwaliteit van een referentielaboratorium. Met de 'One' doet u sneller en efficiënter biochemisch onderzoek en kunt u uw patiënten nog completeer en hoogwaardigere veterinaire zorg bieden.

Meer informatie? Bel ons op telefoonnummer 00800 1234 3399 of ga naar www.idexxcatalyestone.nl

Bekijk de video om te zien hoe gebruiksvriendelijk de Catalyst One™ is.
WHO IS WHO?

ESVN-ECVN BOARD
Holger Volk (President)
Thomas Fliegel (Vice-president)
Veronika Stein (secretary)
Valentina Lorenzo (treasurer)
Lara Matiasek (Examination committee)
Sam Long (Education committee)
Julien Guevar (ESVN Representative)
Laurent Garosi (past-president)

EXAMINATION COMMITTEE
Lara Matiasek (chair)
Mike Targett
Christine Thomson
Sebastien Behr
Tom Harcourt-Brown (examination observer)
Tarja Joniken (examination observer)
Iris Van Soens (examination observer)

EDUCATION COMMITTEE
Sam Long (chair)
Elsa Beltran
Caroline Hahn
Nick Jeffery
Merav Shamir
Jean Laurent Thibaut
Andrea Tipold

SCIENTIFIC COMMITTEE
Nicolas Granger (chair)
Laurent Garosi
Peter Smith

SYMPOSIUM COMMITTEE
Paul Mandigers (chair)
Niklas Bergknut

PCO SYMPOSIUM
Susanne Pauwels

USEFUL INFORMATION

VENUE
The congress will take place in:
The Royal Tropical Institute (KIT)
Mauritskade 65, 1092 AD Amsterdam
The Netherlands
T: +31 20 56 88 711, F: +31 20 66 84 579
www.kit.nl, communication@kit.nl

ACCESSIBILITY THE ROYAL TROPICAL INSTITUTE
By car
Leave the ring road A10 via the exit
Watergraafsmeer/ Diemen (S113): follow the
Middenweg (direction Centrum/ Watergraafsmeer),
which turns into the Linnaeusstraat. KIT is at the
corner of the Linnaeusstraat and the Mauritskade.
There are paid parking facilities in the vicinity.

By public transport
– From Amsterdam Central Station: tram 9
– From Amsterdam Muiderpoort Station:
  tram 3 or 7
– From Dam square: tram 9 or 14
– From Leidseplein: tram 7 or 10
– From Museumplein: tram 3

PARKING
There are enough on-street parking areas near
KIT. Please note that all parking in the centre of
Amsterdam is metered from Monday through
Saturday, 09.00 to 19.00 hrs. For rates and
other parking information, please visit
www.bereikbaaramsterdam.nl.
Parking meters take coins and Chipknip smart cards.

REGISTRATION DESK
The registration area in the congress centre
will be open for registration:
Thursday 17 September: 16.00 – 18.00 hrs.
Friday 18 September: 07.30 – 18.00 hrs.
Saturday 19 September: 07.30 – 18.00 hrs.

CERTIFICATE OF ATTENDANCE
All participants will receive a certificate of
attendance by email after the congress.
LANGUAGE
The official language of the congress is English.

CONGRESS BADGES
All participants, accompanying persons and exhibitors must wear the Congress identification badges. Entrance to meeting halls, poster and exhibition area will not be permitted to any person without badge.

CURRENCY
The Dutch currency is Euro. Most hotels, restaurants and shops accept international credit cards.

VENUE WELCOME RECEPTION:
Thursday 17 September 19.00 hrs.
Artis
Natura Artis Magistra
Plantage Kerklaan 38-40 Amsterdam
The Netherlands

VENUE DINNER
NH Grand Hotel Krasnapolsky
Dam 9 Amsterdam
The Netherlands
nhkrasnapolsky@nh-hotels.com

VENUE DANCE
Olofskapel
Zeedijk 2 Amsterdam

MOBILE PHONE MANNERS
As a courtesy to other attendees, please observe good mobile phone manners. When attending sessions, please turn off your mobile phone and other wireless communications or use the silent notifications options. If you must take a call, please step out of the room rather than disrupting the event. Thank you for your cooperation.

LIABILITY
Upon registration, participants agree that neither the Organizing Committee nor the Congress Secretariat assume any liability. Participants should, therefore, organize their own health and travel insurance.

We reject all liability for direct or indirect damages, of whatever nature, resulting from or connected in any way with the use of our website, or from the temporary inability to be able to consult our website. Nor are we liable for direct or indirect damages arising from the use of information taken from our website.

CONGRESS SECRETARIAT
Pauwels Congress Organisers
Alexander Battalaan 7
6221 CA Maastricht
The Netherlands
Contact person: Susanne Pauwels
T +31 (0) 43 321 81 80
s.pauwels@pauwelspco.nl

ESVN-ECVN 2015
Royal Tropical Institute,
Amsterdam, The Netherlands
### Time table ESVN/ECVN congress

#### THURSDAY 17-9-2015

**Residents and Practitioners day at UU-Utrecht (from 10 to 16 hrs)**

<table>
<thead>
<tr>
<th>SPEAKER</th>
<th>SUBJECT</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>young neurologist in training / resident</em></td>
<td>Welcome party - Artis Zoo with guided tours from 18.30 to 20 hrs (registration is required!)</td>
</tr>
</tbody>
</table>

#### FRIDAY 18-9-2015

<table>
<thead>
<tr>
<th>Chair</th>
<th>SPEAKER</th>
<th>SUBJECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>8.30-8.40</td>
<td>Paul Mandigers</td>
<td>Welcome</td>
</tr>
<tr>
<td>8.40-9.30</td>
<td>Hans Stroink</td>
<td>Epilepsy in children, classification and diagnostics, when is it epilepsy?</td>
</tr>
<tr>
<td>9.30-9.45</td>
<td>Luisa De Risio</td>
<td>Epileptoid cramping syndrome in the norwich terrier: clinical characterisation and prevalence in the uk</td>
</tr>
<tr>
<td>9.45-10.00</td>
<td>Emi Nigare Barker</td>
<td>Degenerative encephalopathy of nova scotia duck tolling retrievers</td>
</tr>
<tr>
<td>10.00-10.15</td>
<td>Fiona James</td>
<td>Epileptogenic electroencephalographic findings in canine episodic tremor syndrome</td>
</tr>
<tr>
<td>10.15-10.45</td>
<td></td>
<td>Coffeebreak</td>
</tr>
<tr>
<td>10.45-11.00</td>
<td>Franziska Wieländer*</td>
<td>Myoclonic epilepsy with photosensitivity in rhodesian ridgebacks</td>
</tr>
<tr>
<td>11.00-11.15</td>
<td>Tarek Bouzourra</td>
<td>Long-term treatment of canine paroxysmal dyskinesias with fluoxetine: 6 cases</td>
</tr>
<tr>
<td>11.15-11.30</td>
<td>Mihai Mustetea</td>
<td>Interictal cardiac autonomic nervous system disturbances in dogs with idiopathic epilepsy</td>
</tr>
<tr>
<td>11.30-11.45</td>
<td>Mark Lowie</td>
<td>Canine epileptoid cramping syndrome: a gluten sensitive paroxysmal movement disorder - more than a gut feeling</td>
</tr>
<tr>
<td>11.45-12.30</td>
<td>Ronald Corbee (sponsored by Hills)</td>
<td>Influence of supplements, nutrition on epilepsy and movement disorders</td>
</tr>
<tr>
<td>12.30-13.45</td>
<td></td>
<td>Lunchbreak</td>
</tr>
<tr>
<td>12.45-14.15</td>
<td>Sofie Bhatti</td>
<td>Alternative non-medical treatments vagal stimulation, transcranial magnetic stimulation in epilepsy</td>
</tr>
<tr>
<td>14.00-14.15</td>
<td>Kenny Bossens*</td>
<td>The effect of imipetoin, a recently developed antiepileptic drug, on thyroid function test parameters and fat metabolism in healthy beagle dogs</td>
</tr>
<tr>
<td>14.15-14.30</td>
<td>Gualtiero Gandini</td>
<td>Efficacy of imipetoin as first choice drug in the treatment of S3 naive dogs affected by idiopathic epilepsy</td>
</tr>
<tr>
<td>14.30-15.15</td>
<td>Bas Bloem</td>
<td>Recognition, classification and treatment of neurological movement disorders in humans</td>
</tr>
<tr>
<td>15.30-16.00</td>
<td></td>
<td>Teabreak</td>
</tr>
<tr>
<td>16.00-16.30</td>
<td>Bas Bloem</td>
<td>Recognition, classification and treatment of neurological movement disorders in humans</td>
</tr>
<tr>
<td>16.30-16.45</td>
<td>Ana Maria Tomaz Coelho</td>
<td>Value of cerebrospinal fluid analysis in epileptic dogs that lack interictal neurological abnormalities and have unremarkable magnetic resonance imaging of the brain</td>
</tr>
<tr>
<td>16.45-17.10</td>
<td>Dennis O’Brien</td>
<td>Movement disorders in animals: terminology, classification part 1</td>
</tr>
<tr>
<td>17.10-19.00</td>
<td></td>
<td>ESVN/ECVN AGM</td>
</tr>
<tr>
<td></td>
<td>19.00-22.00</td>
<td>ESVN/ECVN Congress dinner - Wintertuin, Hotel Krasnapolsky (seated dinner)</td>
</tr>
<tr>
<td></td>
<td>22.00-02.00</td>
<td>Live music - band - Dolfkapel</td>
</tr>
</tbody>
</table>

* young neurologist in training / resident

#### SATURDAY 19-9-2015

<table>
<thead>
<tr>
<th>SPEAKER</th>
<th>SUBJECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>8.30-9.30</td>
<td>Dennis O’Brien</td>
</tr>
<tr>
<td>9.30-9.45</td>
<td>Catherine Escrion</td>
</tr>
<tr>
<td>9.45-10.00</td>
<td>Andrea Fischer</td>
</tr>
<tr>
<td>10.00-10.15</td>
<td>Caroline Hahn</td>
</tr>
<tr>
<td>10.15-10.45</td>
<td></td>
</tr>
<tr>
<td>10.45-11.45</td>
<td>Angelika Richter &amp; Franziska Richter</td>
</tr>
<tr>
<td>11.45-12.00</td>
<td>Clare Rusbridge</td>
</tr>
<tr>
<td>12.00-12.15</td>
<td>Clare Rusbridge</td>
</tr>
<tr>
<td>12.15-12.30</td>
<td>Gemma Elizabeth Langson</td>
</tr>
<tr>
<td>12.30-14.00</td>
<td></td>
</tr>
<tr>
<td>14.00-14.45</td>
<td>Hans Stroink &amp; Dennis O’Brien &amp; Angelika Richter</td>
</tr>
<tr>
<td>14.45-15.00</td>
<td>Jessica Frount &amp; Reville*</td>
</tr>
<tr>
<td>15.00-15.15</td>
<td>Simone Grass*</td>
</tr>
<tr>
<td>15.15-15.30</td>
<td>Florian Salger*</td>
</tr>
<tr>
<td>15.30-16.00</td>
<td></td>
</tr>
<tr>
<td>16.00-16.15</td>
<td>Kali Tulle Lasserin*</td>
</tr>
<tr>
<td>16.15-16.30</td>
<td>Sarah Hanemann*</td>
</tr>
<tr>
<td>16.30-16.45</td>
<td>Cristoforesco Francesco Richo*</td>
</tr>
<tr>
<td>16.45-17.00</td>
<td>Antonella Gallucci*</td>
</tr>
<tr>
<td>17.00-17.15</td>
<td>Dominik Faissler</td>
</tr>
<tr>
<td>17.15-17.30</td>
<td>Patricia Montoliu</td>
</tr>
<tr>
<td>17.30-17.45</td>
<td>Paul Mandigers &amp; Niklas Bergknud</td>
</tr>
<tr>
<td>17.10-18.00</td>
<td></td>
</tr>
</tbody>
</table>

* young neurologist in training / resident

---

**Welcome party - Artis Zoo with guided tours from 18.30 to 20 hrs (registration is required!)**

**Chair:** Sofie Bhatti

**SPEAKER:**
- Alternative non-medical treatments vagal stimulation, transcranial magnetic stimulation in epilepsy
- The effect of imipetoin, a recently developed antiepileptic drug, on thyroid function test parameters and fat metabolism in healthy beagle dogs
- Efficacy of imipetoin as first choice drug in the treatment of S3 naive dogs affected by idiopathic epilepsy
- Recognition, classification and treatment of neurological movement disorders in humans

**Coffeebreak**

**Chair:**

**SPEAKER:**
- Myoclonic epilepsy with photosensitivity in rhodesian ridgebacks
- Long-term treatment of canine paroxysmal dyskinesias with fluoxetine: 6 cases
- Interictal cardiac autonomic nervous system disturbances in dogs with idiopathic epilepsy
- Canine epileptoid cramping syndrome: a gluten sensitive paroxysmal movement disorder - more than a gut feeling
- Influence of supplements, nutrition on epilepsy and movement disorders

**Lunchbreak**

**Chair:**

**SPEAKER:**
- Alternative non-medical treatments vagal stimulation, transcranial magnetic stimulation in epilepsy
- The effect of imipetoin, a recently developed antiepileptic drug, on thyroid function test parameters and fat metabolism in healthy beagle dogs
- Myoclonic epilepsy with photosensitivity in rhodesian ridgebacks
- Long-term treatment of canine paroxysmal dyskinesias with fluoxetine: 6 cases
- Interictal cardiac autonomic nervous system disturbances in dogs with idiopathic epilepsy
- Canine epileptoid cramping syndrome: a gluten sensitive paroxysmal movement disorder - more than a gut feeling
- Influence of supplements, nutrition on epilepsy and movement disorders

**Teabreak**

**Chair:**

**SPEAKER:**
- Alternative non-medical treatments vagal stimulation, transcranial magnetic stimulation in epilepsy
- The effect of imipetoin, a recently developed antiepileptic drug, on thyroid function test parameters and fat metabolism in healthy beagle dogs
- Myoclonic epilepsy with photosensitivity in rhodesian ridgebacks
- Long-term treatment of canine paroxysmal dyskinesias with fluoxetine: 6 cases
- Interictal cardiac autonomic nervous system disturbances in dogs with idiopathic epilepsy
- Canine epileptoid cramping syndrome: a gluten sensitive paroxysmal movement disorder - more than a gut feeling
- Influence of supplements, nutrition on epilepsy and movement disorders

**Farewell**

---

For more information, visit [www.pauwelspco.nl](http://www.pauwelspco.nl) or call Mrs. Susanne Pauwels at 31 (0) 43 321 81 80.
Introducing PEXION® for canine epilepsy.

The first and only targeted therapy that lets dogs and their owners live in the moment instead of living with side effects. With PEXION®, you can change the conversation about epilepsy without changing your patients’ personalities.

Be a part of the change.


There are a million reasons why owners love their dogs
NONE OF THEM ARE WORTH GIVING UP

Reason 16: Duets are always more fun.

Introducing PEXION® for canine epilepsy.

The first and only targeted therapy that lets dogs and their owners live in the moment instead of living with side effects. With PEXION®, you can change the conversation about epilepsy without changing your patients’ personalities.

Be a part of the change.


There are a million reasons why owners love their dogs
NONE OF THEM ARE WORTH GIVING UP

Reason 16: Duets are always more fun.

Introducing PEXION® for canine epilepsy.

The first and only targeted therapy that lets dogs and their owners live in the moment instead of living with side effects. With PEXION®, you can change the conversation about epilepsy without changing your patients’ personalities.

Be a part of the change.


There are a million reasons why owners love their dogs
NONE OF THEM ARE WORTH GIVING UP

Reason 16: Duets are always more fun.

Introducing PEXION® for canine epilepsy.

The first and only targeted therapy that lets dogs and their owners live in the moment instead of living with side effects. With PEXION®, you can change the conversation about epilepsy without changing your patients’ personalities.

Be a part of the change.


There are a million reasons why owners love their dogs
NONE OF THEM ARE WORTH GIVING UP

Reason 16: Duets are always more fun.

Introducing PEXION® for canine epilepsy.

The first and only targeted therapy that lets dogs and their owners live in the moment instead of living with side effects. With PEXION®, you can change the conversation about epilepsy without changing your patients’ personalities.

Be a part of the change.


There are a million reasons why owners love their dogs
NONE OF THEM ARE WORTH GIVING UP

Reason 16: Duets are always more fun.

Introducing PEXION® for canine epilepsy.

The first and only targeted therapy that lets dogs and their owners live in the moment instead of living with side effects. With PEXION®, you can change the conversation about epilepsy without changing your patients’ personalities.

Be a part of the change.


There are a million reasons why owners love their dogs
NONE OF THEM ARE WORTH GIVING UP

Reason 16: Duets are always more fun.

Introducing PEXION® for canine epilepsy.

The first and only targeted therapy that lets dogs and their owners live in the moment instead of living with side effects. With PEXION®, you can change the conversation about epilepsy without changing your patients’ personalities.

Be a part of the change.


There are a million reasons why owners love their dogs
NONE OF THEM ARE WORTH GIVING UP

Reason 16: Duets are always more fun.
There are a million reasons why owners love their dogs

NONE OF THEM ARE WORTH GIVING UP

Introducing PEXION® for canine epilepsy.

The first and only targeted therapy that lets dogs and their owners live in the moment instead of living with side effects.1 With PEXION®, you can change the conversation about epilepsy without changing your patients’ personalities.

Be a part of the change.

---

PEXION® is a registered trademark of Boehringer Ingelheim Vetmedica GmbH.

© 2015 Boehringer Ingelheim Vetmedica GmbH.

---

**EPILEPSY IN CHILDREN, CLASSIFICATION AND DIAGNOSTICS, WHEN IS IT EPILEPSY?**

Hans Stroink (MD, PhD), Canisius Hospital, Nijmegen, The Netherlands

In childhood many non-epileptic paroxysmal events occur making a correct diagnosis difficult. The first question is: are the events epileptic or not? An eyewitness account is important, but not always clear. A home video may be helpful. The EEG can be misleading. 3.5% of healthy children, who will never develop epilepsy, show epileptiform discharges during a standard EEG. In children with for example ADHD or autism the incidence is even much higher. Artefacts and normal variants can simulate epileptiform discharges in all ages. This may result in a misdiagnosis. Moreover, many epilepsy patients don’t show epileptiform discharges.

In humans the onset of idiopathic (genetic) epilepsy is almost always before the age of 20. Most human idiopathic epilepsy syndromes are not diagnosed by excluding a cause. They are clinically easily recognisable, characterised by specific age of onset, semiology (generalised as well as focal) and moment of time of the seizures, and eventually the EEG. For example rolandic epilepsy with centrotemporal spikes starts during school age with specific partial seizures during sleep; juvenile myoclonic epilepsy and awakening epilepsy start between 10-20 years with generalised seizures shortly after awakening. During the presentation diagnosis and classification will be discussed extensively.

---

**INFLUENCE OF SUPPLEMENTS / NUTRITION ON EPILEPSY AND MOVEMENT DISORDERS**

Ronald Jan Corbee, DVM, PhD, Dipl. ECVCN. Department of Clinical Sciences of Companion Animals, Faculty of Veterinary Medicine, Utrecht University, Utrecht, The Netherlands.

**Introduction**

Nutrition affects health and disease, and nutritional therapy is common practice for many years. As Hippocrates stated: Let thy food be thy medicine and medicine be thy food. Nutritionists deal with nutrition induced disorders (i.e. disorders caused by nutrient deficiencies) and with nutrition related disorders (i.e. disorders that are affected by nutrition like the beneficial effect of phosphorus restriction on the progression of renal failure).

**Nutrition induced disorders regarding epilepsy and movement disorders**

Nutrition induced disorders regarding epilepsy and movement disorders are: deficiencies in nutrients that are needed for development of brain tissue and myelin sheaths, deficiencies in minerals and electrolytes interfering with neurotransmission, hypoglycemia, hypocalcaemia, arginine deficiency in cats causing hepatic encephalopathy, and thiamine deficiency. Most of these deficiencies are seldom seen due to the fact that most cat and dog owners are feeding complete and balanced diets. The effects of several nutrient deficiencies are described in NRC 2006 (1). Toxicities that can cause epilepsy and movement disorders are: protein excess causing hepatic encephalopathy, lead poisoning, pathogens from raw food, and contaminants like salinomycin (2,3) and mycotoxins (4). Guidelines for early diagnosis and treatment are given in the article by Barker et al. 2013 (5).

**Nutrition related disorders regarding epilepsy**

The ketogenic diet has been effective in cases of epilepsy in young children (6), however, dogs do not get ketotic so this is not an effective treatment option for dogs (7). Because no studies have been done in cats,
Potassium-bromide is used as mono-treatment and in multimodal-treatment of epilepsy. The working mechanism is well described in the article by Pusch et al. 1999 (11). Because bromide acts on chloride channels, there are several interactions with dietary chloride. It is important to keep dietary chloride intake as constant as possible during potassium-bromide treatment as increased intake of chloride (common source are human foods and treats) decreases the elimination half-life of bromide, whereas a decreased intake of chloride may lead to bromism (12).

Several micro-nutrients have been mentioned to play a role in epilepsy. Taurine (100-500mg per cat per day) has been reported to reduce seizure frequency in a small number (n=6) of cats (13,14) and (relative) deficiencies of several micronutrients have been reported to increase seizure frequency in humans (15). Dietary long-chain polyunsaturated fatty acids eicosapentaenoic acid and docosahexaenoic acid (EPA+DHA 106mg per kg of metabolic body weight) have been reported to reduce seizure frequency in a Great Dane (15), however a clinical trial of EPA+DHA (115mg per kg metabolic body weight) in 15 dogs failed to demonstrate an effect (16). The authors explained this difference due to the fact that early onset of treatment may be effective, whereas initiation of treatment in refractory cases is not (16). Medium chain triglycerides (MCT) have been reported to be effective in mice (17). A recent study in 21 dogs demonstrated a reduction in seizure frequency on a MCT supplemented diet compared to a control diet, but unfortunately the diet composition was not given (18).

**Nutrition related disorders regarding movement disorders**

Movement disorders may alter energy requirements. Furthermore, the medication used in movement disorders can affect taste and smell. In some movement disorders the gastrointestinal tract is involved and may require alterations in feeding management (i.e. megoaesophagus requires feeding from a height, swallowing disorders may require a different texture of the food). Movement disorders are associated with cobalamin deficiency (19). Evidence for nutritional therapy for movement disorders in dogs and cats is lacking, and in humans there is a lot of conflicting data (20).

Movement disorders can be associated with mycotoxicosis as described earlier (4,5), however these cases are usually self-limiting (4,5).

There is some evidence that supports nutritional therapy in dogs with canine epileptoid cramping syndrome (CECS) (21,22). One study on the phenotypic characterization of CECS in 29 Border terriers reported that in 14 out of 26 dogs that switched from their own diet to a "hypoallergenic diet" there was a reduction in frequency of episodes (21). One case report in a 9 month old yorkshire terrier reported that in 14 out of 26 dogs that switched from their own diet to a "hypoallergenic diet" there was a reduction in seizure frequency on a MCt supplemented diet compared to a control diet, whereas initiation of treatment in refractory cases is not (16). Medium chain triglycerides (MCT) have been reported to be effective in mice (17). A recent study in 21 dogs demonstrated a reduction in seizure frequency on a MCT supplemented diet compared to a control diet, but unfortunately the diet composition was not given (18).

**Brain diet: effects of nutrition on cognition**

The effects of antioxidants and l-carnitine on cognition in dogs have been studied (25,26). The effects were demonstrated on the long-term (2 years) in 48 Beagle dogs, and the dogs that received additional environmental enrichment performed best, indicating that a multimodal treatment (including diet fortification) is the best approach (26).

---

**References**

A broad battery of ancillary studies (the “scatter gun” approach) is generally unrewarding, because of judgement) is often limited, especially in early stages of the disease. Hiding clinical uncertainty behind time-consuming and sometimes invasive. Moreover, their diagnostic value (over and above clinical aged children and adolescents is as high as 21%.4

Background

Movement disorders such as Parkinson’s disease (PD), tremor, tics and dystonia are common. For example, the overall prevalence of PD is 1% in people in the ages 65-85 years, and this increases to 4.3% above the age of 85 years.1 The prevalence of essential tremor – the most common form of tremor – is 4% in the ages >40 years, and increases up to 14% in people over the age of 65 years.2,3 The prevalence of tics in school aged children and adolescents is as high as 21%.4

The clinical presentation of movement disorders is complex, often variable, and sometimes even bizarre. Establishing the correct diagnosis can therefore be difficult, even in the hands of experienced movement disorder specialists. However, accurate recognition based on clinical acumen is important, for several reasons.

First, a correct classification of the type of movement disorder(s) forms the basis for the subsequent diagnostic process. For most disorders, there is no specific biological marker that can unambiguously diagnose the underlying disease. Many diagnostic tests are available,1,6 but these are often expensive, time-consuming and sometimes invasive. Moreover, their diagnostic value (over and above clinical judgement) is often limited, especially in early stages of the disease. Hiding clinical uncertainty behind a broad battery of ancillary studies (the “scatter gun” approach) is generally unrewarding, because of the large range of potential diagnoses. The investigational work-up can be greatly simplified once the type of movement disorder has been defined properly, because the approach to each type of movement disorder is more focused. For example, the work-up for dystonia is very different from that for, say, chorea. Second, adequate classification – as a means to establish the correct diagnosis – often has prognostic implications. For example, essential tremor is sometimes mistaken for early PD, but the prognosis is clearly different. Furthermore, since several movement disorders are genetically determined (for example, Huntington’s disease), accurate classification leading to the proper diagnosis may also have implications for the patient’s family. Finally, differentiating between the different types of movement disorder can have important consequences for treatment.

Unfortunately, the diagnostic process is commonly perceived as being difficult, is frequently protracted, and misdiagnoses are common. Because of their unusual presentation, patients with movement disorders may be diagnosed as having a psychogenic disease (although the converse is also true).

Here, we provide a viewpoint, as a practical approach to help clinicians in the “pattern recognition” of movement disorders, and in the process of translating a particular movement disorder syndrome – once it has been classified clinically – into an etiological diagnosis. This is not a review of the literature, is not meant to be exhaustive, and will only briefly touch upon ancillary investigations, which are not within its scope. What we concentrate on is the most important step in the diagnostic process, i.e. the clinical approach. An unambiguous diagnostic process begins with the recognition of the type(s) of movement disorder present in the patient as the first crucial step. In the remainder of this viewpoint, we first highlight the salient features of these different types of movement disorder, attaching to each of them one or more specific keywords for ease of recognition. We then propose a practical approach, using the identified movement disorder(s) as the starting point for a stepwise diagnostic work-up.

General classification principles & phenomenology

Generally speaking, two main categories of movement disorder phenomena can be distinguished, with several specific subdivisions (Table 1). The first category corresponds broadly to akinetic-rigid disorders, the second to hyperkinetic disorders. The hyperkinetic disorders are usually perceived as being more difficult diagnostically. It helps tremendously to separate this group into two main subdivisions: one where the movements have a jerky character, and a second where this jerky character is absent. Not that many disorders feature a combination of both categories.

Akinetic-rigid syndromes

The literature uses the terms akinnesia, bradykinesia and hypokinesia inconsistently. We define akinnesia as an umbrella term for a symptom complex that can include bradykinesia (slowness of movement) and hypokinesia (poverty of movement, and movements that are smaller than intended), but also – crucially and fundamentally – the progressive fatiguing and decrement of repetitive alternating movements seen during finger or foot tapping. We ask the patient to make large, regular repetitive alternating movements of each extremity in turn: opposition of the thumb to the crease between the terminal phalanges of the index and third fingers, and repeatedly tapping the forefoot on the floor, keeping the heel on the ground. It is easy to see early progressive reduction in amplitude or speed of the movements (or, at the ankle, to hear it). Sometimes, however, the clinical question is not whether akinnesia/bradykinesia is present, but whether it is absent. It takes a bit more time to demonstrate, and in order to be certain of this we recommend asking the patient to do up to 64 repetitions in each extremity, if necessary. Sometimes severe tremor can intervene to “hijack” the movements, and make this assessment difficult or impossible. In the widely used Queen Square Brain Bank Criteria1 for the diagnosis of parkinsonism, bradykinesia is defined as including these last two elements, which we would consider under the broader rubric of akinnesia. This variability in terminology is not in itself important as long as, whatever name one gives, fatiguing and decrement are defining features for (untreated) parkinsonism. Note that signs of...
myoclonus may be subdivided into focal, multifocal, segmental or generalized. Etiologically, myoclonus can be focal, multifocal, or generalized.

Increased muscle tone across a joint due to rigidity or spasticity can be differentiated while examining the full range of motion of a joint with varying speeds. In rigidity the resistance is more or less stable, and equal between flexion and extension movements, during the whole trajectory. In spasticity the tone is preferentially increased in arm flexors and leg extensors, and sudden decreases of muscle resistance (“clasp knife phenomenon”) may be felt.

“Jerky” hyperkinetic syndromes

This category includes myoclonus (together with excessive startle), chorea and tics. Jerky movements may be seen in isolation or in combination with non-jerky movements.

Myoclonus

Myoclonic movements are sudden, brief, shock-like involuntary movements caused by muscular contractions, which are usually positive, but sometimes negative (due to brief loss, or inhibition of muscular tonus, as in “liver flap” or “asterixis” in hepatic or uremic encephalopathy). Negative myoclonus can also be seen while walking, producing a typical veering gait pattern, or the sudden postural lapses (“bouncy gait”) seen in post-anoxic myoclonus. Myoclonic muscle contractions are mostly accompanied by some movement of the affected body segment, in contrast to, for example, fasciculations or myokymia, where the twitches remain within the affected body segment. Myoclonus is best likened to the effect seen after stimulating the nerve supplying the muscle with a single electric shock (or with a train of shocks, because the myoclonic jerks may occur repetitively within the same muscle). So the key word in identifying myoclonus is shock-like movements.

When myoclonus occurs in series, the timing of the jerks can be either rhythmic or irregular. Sometimes rhythmic myoclonus may be mistaken for tremor (e.g. spinal segmental myoclonus; or hereditary cortical myoclonus, which has also been erroneously labelled “cortical tremor”). If myoclonus is repetitive but more arrhythmic (as in “polyminimyoclonus”, which consists of fine myoclonic individual finger jerks seen in the outstretched hands in, for example, patients with multiple system atrophy), the movements can be mistaken for irregular tremor. However, isolated tremor lacks the defining abrupt and shock-like character of myoclonus. What was called palatal myoclonus is now termed palatal tremor because of its rhythmic nature and lack of abrupt jerky movements.

There are several ways to describe and classify myoclonus. The distribution of myoclonus can be focal, multifocal, segmental or generalized. Etiologically myoclonus can be subdivided into physiological myoclonus (e.g. hypnic jerks), essential myoclonus (idiopathic or hereditary), epileptic myoclonus, or symptomatic myoclonus in case of an underlying disorder. Physiologically myoclonus is subdivided into cortical, subcortical, spinal and peripheral. Finally, it is important to carefully look for the specific moments of occurrence for myoclonus. Thus, myoclonus can occur spontaneously (at rest), but is also often present, and usually worsened, during movement (action myoclonus) or provoked by external tactile or acoustic stimuli (reflex myoclonus). Cortical myoclonus is more often action – or stimulus-sensitive, mostly to distal touch or stretch, and occasionally to visual stimuli. Brainstem myoclonus is more commonly provoked by auditory stimuli, or tactile stimuli around the face or snout. It is therefore important to look for stimulus sensitivity when assessing suspected myoclonus. Cortical myoclonus is often more focal, and subcortical myoclonus more often generalised. Certain types of myoclonus have different neurophysiological characteristics.

A clinically difficult category is propriospinal myoclonus, i.e. myoclonus generated within the spinal cord with subsequent upward and downward spread, as these movements are perceived as being too slow and lacking the jerky character. Often polymyographic recordings are needed to prove the myoclonic nature of these axial movements. In some cases spinal pathology can be demonstrated, but in most it is not. A proportion of this latter group are psychogenic, but their semiology and physiology can mimic organic cases, so diagnosis of pathogenesis is difficult.

Startle reactions, which are part of the spectrum of myoclonus, are also provoked by external stimuli, most often by auditory triggers, but also by surprise, alarm or acute pain. The startle reaction is characterised by a bilaterally synchronous shock-like set of movements.

Chorea

Chorea is perhaps not immediately appreciated as being a “jerky” movement disorder, perhaps because the word Chorea (Greek for dance) suggests a certain grace rather than abrupt, jerky movements. However, if one carefully observes a patient with chorea, it immediately becomes evident that the “choreography” includes a constellation of randomly flowing movements, which are, individually, jerky in nature. Thus, chorea can be defined as involuntary movements that are abrupt, unpredictable and non-rhythmic, resulting from a continuous random flow of muscle contractions. A key difference from myoclonus is that the pattern of movements randomly changes from one body part to another, conveying the impression of “fidgeting” to the observer. So if we were to attach a key description to chorea, it would be random, flowing jerks. Other typical signs of chorea are the motor persistence, such as seen in the fluctuating strength of the grip (so-called ‘milkmaid’s grip’), or hung-up reflexes (sustained contractions and choreomatosic movements of the leg after the knee jerk reflex). Some patients with chorea, e.g. in Huntington’s disease, may have additional brief (<100 msec) muscle jerks that are myoclonic, and/or longer (>500 msec) co-contracting muscle spasms that are dystonic.

Mild chorea may be subtle, but can usually be detected if the clinician carefully observes the patient with this possibility in mind. Finger chorea is best brought out by the subject counting backwards with eyes closed and arms outstretched, or when walking. Identifying chorea is sometimes hampered by the fact that patients frequently try to mask their chorea by incorporating the jerks into voluntary movements. Another pitfall is that choreatic patients themselves often have relatively few subjective complaints, certainly in early stages, when it is usually the partner who complains about the movements. This is not uncommon in for example Huntington’s disease.

Ballism is typically considered under the rubric of chorea because it shares the same pathophysiology and treatment. Ballistic movements are uncontrollable, severe, mainly proximal, large amplitude chorea. They are usually unilateral (hemiballism) and are classically described after an acute lesion in the region of the contralateral subthalamic nucleus. The term hemichorea can also be used when the amplitude of the movement is small. Sometimes the movements involve only one limb (monoballism). Bilateral ballistic movements are rare and mostly due to metabolic abnormalities.
Tics

Tics are the third category of sudden and jerky movements, but now the keyword for recognition is the stereotyped character of the recurrent movements. Another fundamental difference from myoclonus and chorea is that patients report that their tics are preceded by rising discomfort or urge (“sensory tic”) that is relieved by the actual movement (“itch and scratch” analogy). Another important feature is that tics can usually be largely suppressed for short periods by an effort of will. However, suppression of tics typically comes at the expense of mounting inner tension, leading to a “rebound” of tics afterwards. Because of their stereotyped character, tics can usually be mimicked easily.

Tics usually predominate in the face, upper arms and neck. They can be divided into simple tics (e.g. eye blinking, nose wrinkling, shoulder shrugging, throat clearing) or complex tics (e.g. touching things, smelling objects, echopraxia, jumping). Another subdivision is into motor tics (such as stereotyped head jerks) or phonic tics (repetitive sniffs or sounds, words or even sentences). A notorious diagnostic pitfall is that tics are often less prominent or even absent in the clinical examination room, apparently because the anxiety associated with being examined suppresses the phenomenon. Videotaping the patient, without an examiner present, can therefore be very helpful. Occasionally, a motor tic can have an abrupt onset but the subsequent movement or posture might be slow or prolonged rather than jerky. This is referred to as a dystonic tic, but the suppressibility and stereotyped nature are the clinical clues to classifying such movements as a tic. Dystonic tics can occur in conjunction with other non-dystonic tics.

Stereotypic movements are like tics, but the actions consist of a complex set of movements that are longer lasting, patterned, repetitive, purposeless or ritualistic. These stereotypies are less paroxysmal than tics, but occur over and over again in a more continuous fashion. The movements may be simple (composed of a few manoeuvres, e.g. rocking or head banging) or more complex (composed of multiple simple manoeuvres performed together or in sequence). Stereotypies are typically seen in patients with autism, mental retardation, Rett syndrome, psychosis, or congenital blindness and deafness.15 16

**“Non-jerky” hyperkinetic syndromes**

This category includes tremor and dystonia. Although dystonia can have a jerky nature, its core feature is prolonged muscle spasms. Therefore, dystonia is placed here in the non-jerky category.

**Tremor**

By definition, tremor is characterised by involuntary, rhythmic and sinusoidal alternating movements of one or more body parts. This does not necessarily involve a limb, as tremor can affect almost any body part (e.g. the head, chin or soft palate). The keyword in identifying tremor is rhythmicity, i.e. the oscillations occur at a regular frequency. However, identifying rhythmicity with the naked eye is not always easy, because tremors – despite having a fixed frequency – often have a variable amplitude.

Such changes in amplitude with time can occur spontaneously, but may also result from movements or changes in posture assumed by the patient, or from emotions and fatigue. Despite the amplitude change, tremor frequency remains unchanged. In such patients, objective and quantitative tremor registration, using electromyography and accelerometry, can confirm rhythmicity.

Tremors can be classified in various ways. One important classification system is according to the characteristic moment or situation of occurrence (Table 2).17 A resting tremor can only be definitively identified when the affected body part is not actively moving, and when the effect of gravity is removed completely. Resting tremor usually disappears during voluntary actions. Sometimes eye closure or distraction is needed to provoke the resting tremor (for example, asking the patient to count backwards whilst sitting with the arms resting on the arms of a chair). Occasionally it is seen in the arm when the subject is walking (“dependent tremor”). The rest tremor may be very focal. For example, tremor in PD may begin in a single digit. A diagnostic pitfall is failure to recognize that resting tremors can occur in any posture assumed by the affected body part, even when this involves a posture that is actively maintained against gravity (and thereby mimic a postural tremor). For example, the typical resting tremor in the hands of patients with PD can also be observed when the arms are stretched out in front. In this case, the distinction between postural tremor (as in essential tremor) and true resting tremor is possible by carefully examining how rapidly the tremor becomes manifest after the new posture has been assumed: immediately in case of postural tremor, but after a delay of several seconds in case of resting tremor (a phenomenon termed “resetting” or “re-emergent tremor”). Also, the frequency of a resetting tremor is the same as that in rest position.18

**Kinetic tremors** occur during volitional movements. A distinction is made between simple/action tremor (evident during a target-directed movement), terminal tremor (evident at the end of a target-directed movement) and intention tremor (which increases progressively in amplitude throughout the movement until it reaches the intended target). Isometric tremor occurs when muscles forcefully contract without shortening, e.g. while pushing against a wall. Finally, psychogenic tremor is characterized by a variable frequency, direction and amplitude, as well as by distractibility.18

Attempts have been made to classify tremor according to its frequency. However, it is rarely possible to establish a diagnosis based purely on this parameter, for two reasons: it is difficult to accurately assess the tremor frequency in the clinic, without neurophysiological equipment; and the frequency spectrum between different tremor types overlaps considerably. An exception is primary orthostatic tremor, a leg tremor that is present during standing, and which is characterised by an unusually high and pathognomonic tremor frequency of 14–18 Hz.20 This particular tremor is barely visible to the naked eye, although patients may manifest a discernable leg or trunk tremor with a lower frequency. Although commonly said to be mainly present during standing, EMG studies have shown that the high-frequency orthostatic tremor persists in the trunk and weight bearing leg during walking, and that this tremor can also arise in the upper extremities when patients support their weight with the arms.21

Many disorders are characterised by the presence of “mixed” tremors. Thus, PD patients not only have resting tremor, but commonly also a postural tremor with a higher frequency. Another example is Holmes tremor (also known as midbrain or rubral tremor), which typically has resting, postural and intention components, often at an unusually low frequency of around 2 to 3 Hz.

When tremor involves a body part already affected by dystonia (see below - most commonly this association is seen in patients with spasmodic torticollis), it is classified according to the MDS Consensus Statement as “dystonic tremor”. Many patients with spasmodic torticollis also have a postural tremor of one or both arms.22 In the Consensus Statement this is called “tremor associated with dystonia”. However, this term is a bit of a mouthful, and since most believe that this tremor is in fact part of the patient’s dystonia, we personally prefer to also call this “dystonic tremor”, provided no other cause is identified. Dystonic tremor can mimic the tremor of PD, especially when it precedes overt dystonia or when the dystonia is subtle, leading to a misdiagnosis of PD. To confuse matters further, arm swing is often reduced in patients with dystonia, even in torticollis patients with no other arm involvement. However, people with dystonic tremor do not have true akinesia, and also have normal dopamine transporter imaging, in contrast to patients with PD.22 Many dystonic tremors are also misclassified as essential tremor.24 To complicate matters further, postural tremors similar to essential tremor are frequently present in patients with dystonia. The presence of (often subtle) dystonic postures should distinguish between these two diagnoses (for example, “dinner-forking” posture of the outstretched hand, or a tendency for the ulnar fingers or thumb to point downwards with the arms held out).
Some tremors are easier to feel than to observe. Superficial palpation of involved muscles can suffice, sometimes at rest, but in particular during passive movements of the affected muscles. “Cogwheeling” simply reflects the sensation of feeling an underlying tremor, irrespective of its cause. It can be felt in patients with essential or dystonic tremor, and in patients with PD. However, in the first two examples there is no additional rigidity (in which case one speaks of “cogwheel rigidity”) or akinesia, whereas these are present in PD. Orthostatic tremor can also be palpated as a kind of “rhythmic shivering” of the legs, or heard through the stethoscope (thumping sound like a helicopter) in cases where it is not obviously visible.

Dystonia

One definition is “an involuntary abnormal co-contraction of antagonistic muscles, which may cause sustained abnormal postures or twisting and repetitive movements”. Another definition is “abnormal characteristic postures and movements, produced by slow sustained muscle contraction which distort limbs, trunk, neck, face or mouth.” Both definitions underscore that an important keyword in identifying dystonia is abnormal posture. As such, dystonia is the only movement disorder that can be visualised in a static image, even though additional rhythmic, irregular or paroxysmal jerky involuntary movements frequently can accompany the abnormal postures. An example of such involuntary associated movements is athetosis, defined as “distal mobile dystonia”: slow, writhing and irregular movements of the distal extremities, with abnormal posturing. The term choreoathetosis used to be applied to describe a mixture of chorea and dystonia (as in L-dopa induced dyskinesias in some PD patients), but today we tend to call these mobile choreodystonic movements.

Dystonias can be classified in several ways, based on their distribution (focal, segmental, multifocal, generalized, or hemi-dystonia), age at onset (early, i.e. ≤ 26 years; or late, i.e. ≥ 26 years), or based on cause (primary, dystonia-plus, degenerative or secondary).

Clinical features are helpful in distinguishing primary from secondary dystonia. Primary dystonia is characterized by the presence of dystonia only (except for tremor). Dystonia-plus syndromes present with a second and relevant neurological feature, such as parkinsonism (as in dopa-responsive dystonia) or sometimes – as has recently become apparent – ataxia. The term “myoclonus dystonia” crept into usage in relation to the very brisk, brusque, lightning-like tic-tac jerks that are typical of patients with hereditary alcohol-responsive myoclonus with dystonia (DYT-11); this syndrome is often due to an epsilon-sarcoglycan mutations. In secondary dystonias, other clinical features are usually associated, and an identifiable cause can often be found. These secondary dystonias are mostly associated with additional clinical features.

Several characteristics support the presence of dystonia. The following features are especially applicable for primary dystonias. The abnormal posturing typically has a consistent directionality (a torticollis with rightward head rotation will not usually suddenly change to a leftward torticollis). The abnormal movements are patterned and repeatedly involve the same muscle groups. In early stages, the dystonia is typically “mobile” (i.e. the patient can still actively or passively move the affected body part), but the dystonia might become more fixed with further disease progression. Note that fixed dystonia may be a relatively early feature in patients with corticobasal degeneration, while fixed posturing that is present immediately at onset of the disease is often felt to reflect a psychogenic cause. A further typical feature of dystonia is the presence of a sensory trick or “geste antagoniste”, which is a mechanism (usually identified and used by the patient) to reduce dystonia, for example, gently touching the cheek to correct torticollis, or chewing gum to reduce oromandibular dystonia. Dystonia is commonly brought out by action or activity (note that this is not the same thing as paroxysmal dystonia). Often this may take the form of an element of task-specificity, i.e. the movements or postures are predominantly or even exclusively present under specific circumstances. Examples include writer’s cramp or the various forms of musician’s dystonia. The task-specificity may lead to diagnostic confusion, for example in patients with leg dystonia who may have severe difficulty walking forward, but can walk backwards or run normally. If no problem is apparent and the complaint is highly task-specific, it can be helpful to ask the person to bring along the relevant musical instrument or golf club, to demonstrate the problem. Failing this, asking the patient to bring in a home video segment to highlight the symptom can also be very revealing.

“Lookalikes” of movement disorders

A range of conditions, both neurological and non-neurological, can mimic various movement disorders. It is obviously important not to miss these lookalikes, and several common examples are shown in Table 3.

Diagnostic levels

A systematic approach is recommended when clinicians see patients who present with one or more types of movement disorder (Flow Diagram). The work-up we use in every patient consists of four key questions that need to be addressed consecutively in order to establish the correct diagnosis. Of course, not every question can always be answered unambiguously in each patient.

1. Which different types of movement disorder are present in this patient?

Some movement disorders can occur almost in pure isolation. One example is essential tremor, where affected patients typically present with a symmetrical action and postural tremor in the arms but, by definition, without other neurological abnormalities, except for perhaps a mildly unsteady gait that might only become apparent during the tandem walk test. However, many clinical syndromes are characterised by the presence of several different types of movement disorders that occur in the same patient: the “mixed movement disorder”. For example, a patient with multiple system atrophy can present with a combination of akinesia, rigidity, tremor, ataxia, and fine polymyoclonus in the outstretched hands. If one looks carefully, such overlap is more often the rule than the exception in patients with movement disorders. The nature of this overlap differs between different disorders, between individual patients with the same disorder, and even within a given patient depending on their disease stage.

In order to differentiate between clinical syndromes (which relies heavily upon pattern recognition, i.e. specific combinations of symptoms and signs), it is important to precisely classify the type of movement disorder that occurs in individual patients. Some combinations immediately raise a specific diagnostic suspicion, such as the combination of dystonia and “lightning” myoclonic jerks which are characteristic of myoclonus dystonia (DYT-11).

Importantly, whenever patients present with a mixed movement disorder, one should always consider the possibility of adverse effects of medication (most commonly dopamine D2 receptor blocking agents such as neuroleptics). Drug induced movement disorders are frequently encountered in patients with a known movement disorder, but also in patients without a history of movement disorders. For example, the presence of chorea in a patient with a previous diagnosis of primary dystonia could be due to the use of anticholinergics, and should not lead to an extensive work-up for secondary dystonia. Patients without a known history of movement disorders and who use antipsychotics can develop tremor, a hypokinetic rigid syndrome, or orofacial dyskinesias. The risk increases with prolonged medication use, but even single doses can be responsible. It may be necessary to request a comprehensive list of previous medications from the general practitioner, as the effects of an offending agent can persist for months following discontinuation.

2. What is the dominant type of movement disorder?

Even when the clinical syndrome is characterized by the simultaneous presence of different types of movement disorder, usually one type will dominate. For example, most adult patients with Huntington’s disease not only have the characteristic chorea, but also display bradykinesia when this is carefully
sought. However, in the typical early-to-middle stage case the clinician will usually have little difficulty in identifying chorea as the dominant type of movement disorder. This distinction is important because the specific diagnostic work-up for chorea is different from that for bradykinesia. Thus, determining the dominant movement disorder syndrome is an essential step, as it steers the differential diagnosis and determines the subsequent diagnostic trajectory.

Recognising the dominant type of movement disorder is often easier in earlier stages of the disease. In patients with more advanced disease, the originally dominant signs may become masked due to secondary disease complications or newly emerging movement disorders. For example, in advanced stages of Huntington’s disease, chorea is often no longer prominent, but akinesia, rigidity and dystonia may predominate. Another example includes levodopa induced dyskinesias in patients with PD. Sometimes parkinsonian patients can simultaneously have tremor in some part of the body and levodopa induced dyskinesias-dystonia in other body parts. However, levodopa induced dyskinesias can also dominate the clinical picture and overshadow tremor and clinically resemble choreoathetosis. The solution in such cases lies in a detailed medical history, and familiarity with all stages of the disease.

1. What are the associated features? The complexity of the clinical picture increases when patients exhibit additional neurological or non-neurological symptoms or signs. Clinicians can, however, take advantage of this, as these associated features may provide important clues about the underlying aetiology. For example, examining the eyes for oculomotor apraxia and telangiectasia in patients with chorea and ataxia may lead to a diagnosis of autosomal recessive ataxia telangiectasia. Similarly, finding Kayser-Fleischer rings in the cornea in a patient with dystonia would indicate a diagnosis of Wilson’s disease, while early and prominent autonomic dysfuntion in a patient with parkinsonism should raise the possibility of multiple system atrophy.

Sometimes, elements of the history provide important clues, such as specific factors that exacerbate or relieve the abnormal movements. For example, involuntary movements that present in frequent, brief attacks that are induced by sudden movements (such as rising from a chair) suggest a diagnosis of paroxysmal kinesigenic dyskinesias. When patients with torticollis report that their head jerks improve dramatically with alcohol, a diagnosis of myoclonus dystonia (DYT-11) should be suspected. Associated non-neurological clues are important, e.g. chorea in a woman with migraine, recurrent venous thrombosis or multiple spontaneous abortions suggests antiphospholipid syndrome. The presence of associated neurological and non-neurological features can thus help to narrow the differential diagnosis that was initially based on the dominant movement disorder syndrome.

Family history and ethnicity may also be critical for the diagnosis. Parental consanguinity, a positive family history and ethnicity in otherwise classical idiopathic parkinsonism may raise the possibility of a monogenic cause of PD. A dominant family history of tremor in patients with a postural tremor suggests essential tremor or dystonic tremor. In dystonia, many inherited forms are known. A positive family history of dystonia combined with Filipino ethnicity raises the possibility of the X-linked DYT-3 (“Lubag”).

2. What is the differential diagnosis and diagnostic work-up? Taken together, an overall clinical syndrome is determined from the specific combination of one (dominant) movement disorder, plus perhaps several concurrent types of movement disorder, plus a set of associated neurological and non-neurological abnormalities. This clinical syndrome should in turn lead to a differential diagnosis. Sometimes simple pattern recognition will suffice and lead directly to the diagnosis, but often ancillary investigations are required. In such cases, the diagnostic process will be guided by the dominant movement disorder.

Note that some specific types of movement disorder always influence clinical decision-making, even when present in a subtle way and not as the “dominant” movement disorder. For example, when patients present with predominant dystonia but also with mild signs of ataxia, the work-up should include, and perhaps even primarily focus on, a search for causes of (hereditary) ataxia.

Details of the diagnostic work-up largely depend on the dominant type of movement disorder and the residual clinical uncertainties with respect to the differential diagnosis. For example, in patients with unexplained chorea that looks like Huntington’s disease, the initial diagnostic step may often simply involve genetic testing for Huntington’s disease, after appropriate counselling. When this is negative, the diagnostic work-up may then be expanded. Each movement disorder and each clinical syndrome thus has its own specific diagnostic approach. A detailed discussion of these diagnostic trajectories themselves is not within the scope of this review.

Conclusion We have outlined a suggested clinical approach to the patient with a movement disorder. As Supplementary Material, we provide examples of how this method might work for patients presenting predominantly with myoclonus (Table 5), chorea (Table 6) or dystonia (Table 7). We hope that application of the proposed serial diagnostic steps may help clinicians to identifying overall clinical syndromes, which in turn will help to guide the diagnostic process.

Acknowledgement This clinical approach was handed down to us by the late Prof. David Marsden.

Conflict of interest statement This work was supported by NWO VIDI research grant #016.076.352 to Professor B.R. Bloem. Dr. W.F. Abdo was supported by a research grant from the Stichting Internationaal Parkinson Fonds. Dr. B.P.C. van de Warrenburg, Professors D.J. Burn and N.P. Quinn have no conflict of interest to declare regarding the submission of this manuscript.

References
consciousness and post-ictal behavior changes. Focal seizures, however, may not affect consciousness, abnormal electrical activity in the cerebral cortex seen on EEG, and are typically accompanied by loss of function. They technically fall under this definition, but they are classified separately. Seizures are characterized by a disruption in the normal flow of electrical activity in the brain, which can cause sudden, brief episodes of altered consciousness, movement, or sensation. These episodes are not fully understood yet, but they are not always associated with loss of consciousness or other serious neurological consequences. Understanding the nature and causes of seizures is crucial for developing effective treatment strategies.

DVM PhD DACVIM-Neurology, Columbia, Missouri
Dennis P. O'Brien

MOVED DISORDERS

Dennis P. O'Brien

Movement disorders are a group of condition characterized by involuntary movements. Seizures would technically fall under this definition, but they are classified separately. Seizures are characterized by abnormal electrical activity in the cerebral cortex seen on EEG, and are typically accompanied by loss of consciousness and post-ictal behavior changes. Focal seizures, however, may not affect consciousness, and some movement disorders are also episodic. Thus the border between episodic movement disorders and seizure disorders are often unclear. Diseases that cause visible contractions of muscle fibers, such as fasciculations, but do not cause movement of the body part are not classified as movement disorders.

TREMORS

Tremor is defined as a rhythmic, oscillatory movement of body parts. They are further broken down by the frequency of the oscillations and when the tremors occur. Feedback systems have a natural tendency to oscillate and in muscles such feedback systems produce a physiologic tremor. Under most conditions, physiologic tremor is barely perceptible, but stress, fear, fatigue, weakness, and drugs such as caffeine can produce an exaggerated physiologic tremor. These tremors are typically fast and small amplitude and tend to occur when standing still, diminishing with movement. Disease processes such peripheral nerve disease, pheochromocytoma, hypoglycemia will exaggerate physiologic tremors. Tremor can also be incidental in aged dogs. Orthostatic tremors occur when standing and affect primarily the hind limbs. They tend to be a little slower and coarser than physiologic tremor. The dogs often hesitant to sit or stand, and thus appear weak. When the dog walks, however, the movements show no evidence of weakness and the tremors resolve. They also resolve when the dog is sitting or lying down. The cause is not known but they have been reported most commonly in Great Danes. Bulldogs and Doberman Pinschers show benign head tremors that appear to be hereditary. These may be a variation on orthostatic tremors since the head must also be supported against gravity. Both appear to be benign conditions. Intention tremors are a hallmark of cerebellar disease of any cause. The term intention tremor may not be appropriate for animals since we cannot know what they intend to do, but it captures the key feature of the tremor so the term is still used. These tremors only occur when the animal is making a goal directed movement. They disappear completely at rest. They are most apparent in the head but can involve the entire body or limbs. The head tremors can become dramatic when the animal eats and the limb tremors when they try to walk down stairs. The tremor is very slow and coarse.

MYOCLONUS

Myoclonus is a brief, sharp contraction of a muscle, group of muscles or the entire body. It can be single or repeated rhythmically. Rhythmic myoclonus lacks the smooth, oscillatory character of a tremor. Focal myoclonus is a common sequelae to canine distemper infections. Generalized myoclonus is often a sign of a more generalized neurodegenerative disease. Sometimes myoclonus can be elicited by stimulation. A hereditary myoclonus in Irish Wolfhounds called starele disease or hyperkplexia is caused by a dysfunction of the inhibitory neurotransmitter glycine. Without this inhibition, the normal startle response is exaggerated. In other conditions the jerks occur spontaneously. The latter is sometimes called myoclonic epilepsy but is often not clear if the cortical epileptic discharges that would classify it as a seizure disorder are present or not. Negative myoclonus looks similar clinically, but is actually caused by brief loss of tone in a muscle followed by a jerk as the animal catches itself. Opsoclonus-myoclonus is an unusual form of myoclonus affecting the eyes as well as the body. The body appears more like a generalized tremor, but the eyes make very rapid, uncontrollable darting movements. It is thought to be an autoimmune disease.

CEREBELLAR ATAXIA

Cerebellar ataxia is the most common movement disorder diagnosed in veterinary practice. The cerebellum is responsible for the fine-tuning of movement. When an animal is learning to walk or perform any motor task, the cerebellum monitors commands coming from the forebrain motor systems and receives feedback from the vestibular system and proprioception sensors in muscles about balance and execution of the movement respectively. Motor learning occurs in the cerebellum as synapses are strengthened or weakened to perfect the movement. Coton de Tulears with Bandera's neonatal ataxia
have a glutamate in the gene for the metabotropic glutamate receptor 1 (MGlur1), the neurotransmitter receptor that mediates the weakening of synaptic connections on Purkinje cells in the cerebellum. As a result, motor learning is abolished and they can never walk. Interestingly, the menace response is also a learned motor response and not a reflex. Thus it does not develop until after weaning age and it is often lost in cerebellar disease. Once the movement has been learned, the cerebellum utilizes what has been learned to adjust the motor program in real time in response to sensory feedback so that movements are smooth. Animals with cerebellar ataxia are not weak and have no proprioception deficits. Instead the range and force of movement are not regulated properly, the result is an exaggerated, goose-stepping gait. This is most apparent when the animal is moving quickly or trying to do a more skilled movement like walking down stairs. In severe cases, the animal may have all four feet off the ground simultaneously or flip over backwards. Because the cerebellum works intimately with the vestibular system, affected animals may lose their balance with sudden movements or develop nystagmus which may be vertical. Occasionally, affected dogs will fall and adopt a contorted posture which is sometimes mistaken for a seizure. Many acquired diseases including infections such as Neospora caninum encephalitis, tumors, and strokes commonly affect the cerebellum and produce cerebellar ataxia and intention tremors. An idiopathic tremor syndrome called idiopathic tremors or idiopathic cerebellar ataxia has been recognized. It is thought to be an immune mediated disease. Affected dogs develop an acute onset of severe generalized tremors and cerebellar ataxia. Most dogs improve with anti-inflammatory drugs and diazepam. Maltese and other white dogs are most commonly affected and the condition was originally called “little white shakers” but we now know it can affect any breed.

Developing the circuitry of the cerebellum requires interaction of a variety of developmental signals and the process of regulating movement in real time once the cerebellum has developed is very complex. Thus mutations in a variety of genes can disrupt cerebellar development or function, and hereditary cerebellar ataxias are common. Developmental disorders are typically apparent neonates. For example, in Eurasier dogs, mutations in a gene that is part of the signaling pathway that regulates brain development cause Dandy-Walker syndrome, a failure of the midline of the cerebellum to develop, and cerebellar ataxia. Functional disruptions will also cause cerebellar ataxia. Potassium channels are critical for regulating neuronal excitability. Jack Russell Terriers with mutations in one of these channels that is found in high density in the cerebellum have severe spinocerebellar ataxia because the increased excitability disrupts the timing of impulses essential for coordinating movement. The dogs show other signs of excessive excitability of neurons such as seizures and myokymia, uncontrollable, rippling contractions of the muscles at rest. Hereditary ataxia in other breeds have been associated with genes involved in a variety of processes including autophagy (ATG4D in Old English Sheepdog) degradation of proteins in the endoplasmic reticulum (SEL1 in Finnish Hounds) and structural proteins (SPTBN2 in beagles).

PARKINSONISM

Parkinson’s disease is a common neurodegenerative disease of older people. It is caused by degeneration of the substantia nigra. Neurons in this area that use dopamine as a neurotransmitter project forward to synapse on the basal nuclei. This system functions the gate-keeper for movement. Dopamine is an important component of the “reward system” in the brain. Based on past experience, this system gives either a “go” or a “no-go” signal to the motor programs. Without the dopamine input, the system is stuck in “no-go” and movement cannot be initiated. Although the resting tremor is the sign of Parkinson’s disease that everyone recognizes, it is the inability to initiate movement that devastates patients with Parkinson’s disease. These signs of difficulty initiating movement and resting tremors are called parkinsonism and can occur in other hereditary, toxic, and degenerative diseases in people.

Parkinson’s disease per se has not be identified in dogs or cats, but the signs of parkinsonism are seen in some hereditary movement disorders of dogs. Chinese Crested dogs and Kerry Blue Terriers have a juvenile-onset, hereditary disease called canine multiple system degeneration. As the name implies, multiple parts of the motor system in the brain degenerate beginning about 4 months of age.

The cerebellum is the first to degenerate and the dogs have classic cerebellar ataxia. Affected dogs have tremors, but they are intention tremors associated with movement in contrast to the tremors of parkinsonism which occur at rest. Except for primates, none of the experimental or hereditary forms of parkinsonism in animals show a resting tremor. Beginning at about 6-8 months of age, affected dogs begin having difficulty initiating movement. They shift their weight forward until they finally propel themselves forward in a gait called festination. With further progression of the disease, they are unable to initiate voluntary movements. They adopt a hunched-up posture when they try to walk and rock until they lose their balance and fall. In addition to degeneration of the cerebellum, affected dogs have degeneration of the substantia nigra and basal nuclei.

DYSKINESIA AND DYSTONIA

Parkinsonism is considered a hypokinetic disease of the basal nuclei because there is difficulty initiating movement. Another group of diseases are called hyperkinetic movement disorders because the basal nuclei are unable to stop movements. Involuntary movements, (dyksinesia) or abnormal muscle tone (dystonia) then occur. Dyskinesias can be focal or generalized. Focal dyskinesia can involve the facial muscles or single limbs. In the limbs, proximal or distal muscles may be preferentially affected. The movements can either be slow or rapid and they may be continuous or episodic (paroxysmal). In humans, dyskinesias are divided into three general categories: athetosis, chorea, and ballism. Athetosis is a slow, continuous, writhing movement involving primarily the distal arm. The movements may be somewhat purposeful. Chorea (from the Greek “to dance”) also affects predominantly distal limb muscles, but the movements are totally involuntary. They are more rapid and fragmented. Ballism is also a rapid movement, but it affects predominantly proximal muscles. The result is a flailing or flinging movement. Dystonia is a sustained contraction of muscles. The axial muscle or limb muscles can be affected. The limbs can either be held in flexion or extension and sometimes a tremor of the affected muscles accompanies dystonia. As with dyskinesias, the episodes can last from minutes to hours but tend to be more infrequent. Often a mix of different dyskinesias and dystonia will occur in the same patient. Acquired dyskinesias can be drug induced or secondary to basal nuclei lesions from infections or stroke. The best characterized dyskinesias in veterinary medicine are paroxysmal and appear to be hereditary. Like seizures, the animals are normal between episodes. In contrast to generalized seizures, animals remain conscious during paroxysmal dyskinesias and they do not have the life threatening complications sometimes seen in seizures. Some dyskinesias have survival triggers such as stress or excitement while others do not. The episodes can occur infrequently to multiple times per day and they can last minutes to hours. The episodes can vary from simple flexion of one limb while walking which looks like a lameness, to frantic, alternating flexion and extension of multiple limbs. The paroxysmal dyskinesias will sometimes respond to antiepileptic drugs or the carbonic anhydrase inhibitor acetazolamide.

Chinook dogs have a paroxysmal dyskinesia with primarily dystonia that appears to be inherited as an autosomal recessive trait. Affected dogs have episodes of dystonia and tremors lasting up to an hour. They are conscious during the episodes and appear perfectly normal on recovery. Occasional flailing movements of a limb can occur also. In Cavalier King Charles Spaniels with episodic falling syndrome, a mutation has been identified in BCAN, a gene involved in connections between cells and fiber guidance in the brain development in dogs. Although the dogs do collapse during these episodes, they do not lose consciousness and they have increased tone in the limbs resulting in them adopting a “deer stalking” posture. A similar syndrome, Scottie cramp, has been recognized in Scottish Terriers since the 1940s but sequencing the BCAN gene did not reveal any mutations. Canine epileptoid cramping syndrome or Spike’s disease is a movement disorder that affects Border Terriers. As the name implies, the episodes resemble seizures, but the dog remains conscious throughout. Most episode last from 2-30 minutes but may last for hours. The episodes consist primarily of tremors and dystonia affecting the head and body. Excessive intestinal noise and vomiting or diarrhea may accompany the episodes leading some owners to suspect a dietary link. Finally a paroxysmal dyskinesia has recently been recognized in Soft Coated Wheaten Terriers.
Affected dogs have episodes of dyskinesia or dystonia which last minutes to hours and may occur multiple times per day and a mutation in gene involved in anchoring proteins to the cell surface has been identified. Some affected dogs have shown a dramatic response to acetazolamide therapy. Other breeds with reports of dyskinesia are Bichon Frise, Boxers, and Springer Spaniels.

CONCLUSION

The increased availability of video capture with cell phones has meant that movement disorders are being increasingly recognized in veterinary medicine. In the case of hereditary diseases, current gene discovery techniques permits us to identify the mutations responsible and eliminate the disease from affected breeds. Understanding the basis of these hereditary diseases can suggest new pathways to explore for effective therapies for acquired conditions. What we can learn from our patients can also shed light on the comparable human diseases.

REFERENCES


WHAT CAN WE LEARN FROM ANIMAL MODELS OF DYSTONIA/DYSKINESIAS IN VETERINARY AND HUMAN NEUROLOGY?

Prof. Dr. Angelika Richter and Dr. Franziska Richter, Institute of Pharmacology, Pharmacy and Toxicology, VMF, University of Leipzig

More than 3 million people worldwide suffer from dystonia, the third most common movement disorder in humans characterized by sustained or intermittent muscle contractions causing abnormal, often repetitive, movements, postures, or both. The term dystonia encompasses paroxysmal movement disorders which include dystonia, chorea and ballism in conscious individuals. Hence, dystonia/ dyskinesias are diagnosed based on the clinical picture, but heterogeneity in symptoms and etiology result in a large percentage of misdiagnoses and incorrect or insufficient treatment. Even in case of correct diagnosis, pharmacological treatment of the different clinical types of dystonia is insufficient or ineffective in most cases and largely based on empirical, rather than pathophysiological rationale. Novel treatment options are urgently warranted, but will require more knowledge of the etiology and pathophysiolog of dystonia. Animal models are pivotal for studies of pathogenesis and treatment of disorders of the central nervous system which in its complexity cannot yet be modeled in vitro or using computer simulations. The choice of a specific model should be based on validity of the model for the approach: does the model reflect symptoms, pathogenesis and treatment response present in human patients? For dystonia, prior to the availability of genetically engineered mice, spontaneous mutants were chosen based on expression of dystonic features, including abnormal muscle contraction, movements and postures. Interestingly, spontaneous occurrence of dystonia/dyskinesia is not only found in rodents. In fact, different hyperkinetic movement disorders, described in dogs, horses and cattle, show similarities to human types of these movement disorders. However, similar to the human condition there is a high rate of misdiagnosis due to lack of awareness that these movement disorders occur in animals. Recently, a number of causative genes and gene products were discovered in dystonia in humans, and some forms of hyperkinetic movement disorders in animals are well characterized and the causing mutations are known. For rodents, this initiated the creation of novel genetically modified models based on gene mutations which now greatly enhance the knowledge of pathophysiology of dystonia and may be used for preclinical drug testing. Clearly, much could also be learned by exchanging experience between human and veterinary medicine on specific cases and treatment options in clinical practice. It is challenging to compare the human condition to how it may present in the animal, requiring experience and solid characterization especially if the condition in animals will be used to develop and test novel drug treatment to be applied for the human condition. Here we present a review of current models of dystonia, with a focus on genetic rodent models, which will likely be first choice in the future either for pathophysiological or preclinical drug testing or both. We will first present models with episodic/ paroxysmal dystonia/dyskinesias, followed by models which express persistent motor dysfunction. We conclude that current models do replicate features of dystonia/dyskinesias and are useful tools to develop urgently demanded treatment for these debilitating disorders. Occurrence of hyperkinetic movement disorders in animals, including farm animals and pets, could provide a valuable resource to learn about etiology, pathophysiology and treatment. We therefore strongly encourage the ongoing exchange of experience between human and veterinary medicine on the clinical as well as scientific level.
ORALS
EPILEPTOID CRAMPING SYNDROME IN THE NORWICH TERRIER: CLINICAL CHARACTERISATION AND PREVALENCE IN THE UK

L De Riso, J Freeman
Neurology Unit, Animal Health Trust, Newmarket, UK

Episodic muscular hypertonicity in Norwich terriers (NT) was first reported in a brief letter in the Veterinary Record in 1984. Since then there have been anecdotal reports and the condition has remained poorly characterised. The aims of this study were to characterise clinically NT epileptoid cramping syndrome (ECS), and to estimate its prevalence in the UK.

The owners of NT born since 1 January 2000 were invited by the UK Kennel club and NT breed club to complete a specifically designed questionnaire aimed at identifying affected and unaffected NT, and clarifying the clinical characteristics of NTECS.

The questionnaire was returned for 198 NT. Of these, 26 (13%) NT were classified as affected by NTECS following revision of the questionnaires, videos of the episodes, veterinary medical records and telephone interview with the owners. All NT were clinically normal between episodes. No significant abnormalities were detected on diagnostic investigations, including electroencephalography (which was performed in 2 NT). Mean age at the first episode of NTECS was 3 years. The episodes were characterised by sustained muscular hypertonicity, dystonia of the pelvic or all 4 limbs, and difficulty or inability standing up and walking. Consciousness was normal. Episode frequency varied both between and within individuals. Stress, anxiety, excitement, and variation in daily routine were recognised as episode triggers in 13 NT. Episode duration was 2-5 minutes in the majority of NT (range <1-30 minutes).

Several affected NT were genetically related. Genetic investigations to identify causal mutations are in progress.

Ethical permission was not required for this study

DEGENERATIVE ENCEPHALOPATHY OF NOVA SCOTIA DUCK TOLLING RETRIEVERS

E. Barker1, D. O’Brien2, L. Dawson3, G. Johnson1, J. Rose1, S. Van Meeravenne1, K. Sörensen1, C. Rohdin3, A. Leijon1, O. Frykman1, N. Granger1

1University of Bristol, Bristol, UK; 2University of Missouri, Columbia, MO, USA; 3AniCura Läckeby Djursjukhus, 38 Proceedings 28st ESVN-ECVN Congress - Amsterdam 2015

Degenerative encephalopathy has been identified in young adult NSDTRs, with a worldwide distribution. MRI and histopathological lesions are characteristic. The prognosis is guarded due to progressive disease which is minimally responsive to empirical treatment.
A breed-specific syndrome characterized by frequent myoclonic jerks has been observed in Rhodesian Ridgebacks. The aim of the study was to provide a clinical and electroencephalographic description of this syndrome and to investigate its epileptic nature.

Seventeen dogs that shared a unique phenotype were identified. Owners were asked to provide a videotape and to complete an online-questionnaire. Extensive diagnostic work-up was offered and pedigrees were analyzed. Awake ambulatory wireless video-electroencephalography (EEG) was performed in 11 affected Rhodesian Ridgebacks and five controls of the same breed. One dog underwent an additional video-EEG with photic stimulation in a neuropaediatric epilepsy center. EEG was reviewed by a neurophysiologist, a neuropaediatrician, and three veterinarians, with consensus results reported. Violent myoclonic jerks occurred in a recumbent and relaxed, drowsy or asleep state (n=17) and occasionally also while standing (n=10). In five dogs myoclonic seizures could be triggered by flashing lights. Median age of onset was 6 months (9w - 1.5y). Over time seven dogs additionally developed other seizure types. Video-EEG confirmed the diagnosis of a myoclonic epilepsy in affected dogs. One dog showed photomyoclonic responses on video-EEG with photic stimulation. Control dogs displayed unremarkable EEG findings. None of the performed examinations (MRI, CSF, neurometabolic screening) demonstrated an underlying structural or metabolic cause. Pedigree analyses pointed to a hereditary disorder. Rhodesian Ridgebacks suffer from an idiopathic (genetic) myoclonic epilepsy with photosensitivity. Affected dogs show a characteristic EEG pattern. This is proposed as the definition of a peculiar electroclinical syndrome whose genetic base is currently under investigation.

LONG-TERM TREATMENT OF CANINE PAROXYSMAL DYSKINESIAS WITH FLUOXETINE: 6 CASES

T. Bouzouroua1, C. Escriou2
1Internal Medicine; 2Neurology, VetAgro Sup, Lyon, France

It is only recently that both clinical features and genetic basis of canine paroxysmal dyskinesias (CPDs) have been documented. Their management remains challenging though phenothiazine, benzodiazepines even anticonvulsants yielded unsatisfactory results. Fluoxetine, a selective serotonin reuptake inhibitor, seems promising.

MYOCLOCNIC EPILEPSY WITH PHOTOSENSITIVITY IN RHODESIAN RIDGEBACKS

F. Wielaender1, F. James2, M. A. Cortez2, G. Kluger3, M. Kornberg3, A. Bathen-Noethen4, T. Flegel1, S. Bhatti4, V. Hulsmeyer5, H. Lohi5, A. Fisher1
1Centre for Clinical Veterinary Medicine, LMU University of Munich, Munich, Germany, 2Department of Clinical Studies, Ontario Veterinary College, University of Guelph, Ontario, Canada, 3Neurosciences & Mental Health Program, Peter Gilgan Centre for Research and Learning, SickKids Research Institute, Ontario, Canada, 4Department of Neuropediatrics, Epilepsy Center, Vogtareuth, Germany and Paracelsus Medical University, Salzburg, Austria, 5Veterinary Hospital Trier, Trier, Germany, 6Veterinary Practice Bathen-Noethen, Cologne, Germany, 7Centre for Clinical Veterinary Medicine, LMU University of Munich, Munich, Germany, 8Department of Small Animal Medicine, University of Leipzig, Leipzig, Germany, 9Research Programs Unit, Molecular Studies, Ontario Veterinary College, University of Guelph, Ontario, Canada, 3Neurosciences & Mental Health Program, Peter Gilgan Centre for Research and Learning, SickKids Research Institute, Ontario, Canada

Three Scottish terriers (STs), 2 Cavalier King Charles Spaniels (CKCSSs) suffering from Scottie Cramp (SC) and Episodic Falling, respectively, then an English Setter with a dyskinesia of unknown origin, displayed many daily episodes, triggered by exercise and/or excitement that definitely prevented them from living normally. All dogs received fluoxetine as a single treatment (2-4 mg/kg q24h). Four dogs displayed early and complete remissions that persisted over long periods (1 year for 1 CKCSS then 2, 6 and 7 years for the 3 STs). The remaining 2 cases showed a significant improvement with a decline to approximately one single episode every 2 months for both dogs. Transient relapses occurred when treatment was interrupted or tapered in 3 dogs. No treatment resistance or side effect was observed. Fluoxetine may be a good and safe option for the long-term management of CPDs, allowing dogs for resuming to normal activity and lifestyle. It can directly correct serotoninergic transmission imbalance suspected in SC. Its activity remains unclear in other CPDs; direct serotoninergic action is likely but indirect effect through a reduction of behavioral reactivity to triggering factors like stress, excitation or environmental stimulations is also plausible.

INTERICLAL CARDIAC AUTONOMIC NERVOUS SYSTEM DISTURBANCES IN DOGS WITH IDIOPATHIC EPILEPSY

D. Mocanu1, M. Musteata1, G.D. Stanciu1, M. Armasu1, A. Baisan1, G. Solcan1
1Dept. of Clinical Science – Internal Medicine, Faculty of Veterinary Medicine Iaşi, Romania

Up to 65% of dogs with idiopathic epilepsy (IE) have an abnormal EEG in the interictal phase, but no neurological deficits or other positive findings on imagistic and blood examination are observed at that moment. In humans, an impaired activity of autonomic nervous system (ANS) is seen in the interictal phase of IE patients and appears to have an important role in long term prognosis of the cases. Until now there are no reports in which ANS is analyzed in dogs with IE. To assess ANS we used time domain and spectral power analysis of heart rate variability (HRV) on 5 minutes of all dogs. We demonstrated an underlying structural or metabolic cause. Pedigree analyses pointed to a hereditary disorder. Rhodesian Ridgebacks suffer from an idiopathic (genetic) myoclonic epilepsy with photosensitivity. Affected dogs show a characteristic EEG pattern. This is proposed as the definition of a peculiar electroclinical syndrome whose genetic base is currently under investigation.

LONG-TERM TREATMENT OF CANINE PAROXYSMAL DYSKINESIAS WITH FLUOXETINE: 6 CASES

T. Bouzouroua1, C. Escriou2
1Internal Medicine; 2Neurology, VetAgro Sup, Lyon, France

It is only recently that both clinical features and genetic basis of canine paroxysmal dyskinesias (CPDs) have been documented. Their management remains challenging though phenothiazine, benzodiazepines even anticonvulsants yielded unsatisfactory results. Fluoxetine, a selective serotonin reuptake inhibitor, seems promising.
**CANINE EPILEPTOID CRAMPING SYNDROME: A GLUTEN SENSITIVE PAROXYSMAL MOVEMENT DISORDER – MORE THAN A GUT FEELING**

**M Lowrie**1, O Garden2, M Hadjivassiliou3, R Harvey4, D Sanders1, R Powell1, L Garosi1
1Davies Veterinary Specialists, Higham Gobion, Hitchin, UK; 2Department of Clinical Sciences and Services, Royal Veterinary College, Hatfield, UK; 3Department of Neurology, Royal Hallamshire Hospital, Sheffield, UK; 4Department of Pharmacology, UCL School of Pharmacy, London, UK; 5Department of Gastroenterology, Royal Hallamshire Hospital, Sheffield, UK; 6Powell Torrance Diagnostic Services, Higham Gobion, Hitchin, UK

Canine epileptoid cramping syndrome (CECS) is a paroxysmal movement disorder of Border terriers (Bts). Previous work has suggested these dogs may respond to a gluten-free diet. The objective of this study was to investigate the possibility of an immune response to gluten and to study the effect of a gluten-free diet in Bts with CECS. Our hypothesis was that CECS is a manifestation of gluten sensitivity.

Six Bts with clinically confirmed CECS were tested for anti-transglutaminase 2 (TG2 IgA) and anti-gliadin (AGA IgG) antibodies in the serum at presentation, and subsequently three, six and nine months after the introduction of a gluten-free diet. Duodenal biopsies were performed in one patient. Serum samples were also collected from five Bts having medical investigations for conditions unrelated to neurological or gastrointestinal disease to serve as healthy controls.

Serum TG2 IgA titers were increased in 6/6 Bts and AGA IgG titers were increased in 5/6 Bts at presentation compared to controls. After nine months there was clinical and serological improvement in all Bts with CECS strictly adherent to a gluten-free diet. One dog had persistently increased antibody titers, but was found to have scavenged horse manure. On the strict introduction of a gluten-free diet this dog also had an improved clinical and serological response. The diet-associated improvement was reversible in two dogs on completion of the study, both of which suffered a clinical relapse of CECS on the re-introduction of gluten.

CECS is best described as a gluten sensitive movement disorder triggered and perpetuated by gluten and thus responsive to a gluten-free diet.

**NEUROSTIMULATION IN EPILEPSY – VAGUS NERVE STIMULATION –**

**Sofie F. M. Bhatti**, Ghent University, Faculty of Veterinary Medicine, Dept. of Small Animal Medicine and Clinical Biology, Neurology and neurosurgery unit

Neurostimulation is a treatment modality in which electrical pulses are administered to nerve tissue in order to manipulate a pathological substrate and to achieve a symptomatic or even curative therapeutic effect. In human epilepsy, a substantial number of patients either lack good seizure control with pharmacological or surgical treatment, or they experience major adverse effects (or both). Therefore, neurostimulation-based treatments have gained considerable interest the last 10-15 years. Electrical stimulation of the tenth cranial nerve or vagus nerve stimulation (VNS) is an extracranial form of neurostimulation that was developed in the 1980s and is currently routinely and worldwide available for the management of human epilepsy. VNS is indicated in patients with refractory epilepsy who are unsuitable candidates for epilepsy surgery or who have had insufficient benefit from such a treatment. VNS influences crucial brainstem and intracranial structures; including the nucleus of the solitary tract, the locus coeruleus, the thalamus, and limbic structures. The precise mechanism of action (MOA) by which VNS exerts its antiepileptic effect is still unknown. The current consensus on efficacy is that one third of patients have a considerable improvement in seizure control with a reduction in seizure frequency of at least 50%, one third of patients experience a worthwhile reduction in seizure frequency between 30% and 50%, and the remaining one third of patients experience little or no effect. Increased insight into the MOA may help to identify responders and increase clinical efficacy. Also, from a veterinary point of view, research towards neurostimulatory treatments for refractory canine epilepsy is needed. Only one study evaluated the efficacy and safety of VNS in dogs with idiopathic epilepsy and the results seemed promising. We examined the MOA of VNS in healthy Beagle dogs and demonstrated changes in cerebral perfusion using micro SPECT and increases in CSF norepinephrine concentrations induced by VNS.


**THE EFFECT OF IMEPITOIN, A RECENTLY DEVELOPED ANTI-EPILEPTIC DRUG, ON THYROID FUNCTION TEST PARAMETERS AND FAT METABOLISM IN HEALTHY BEAGLE DOGS**

**K. Bossens**1, S. Daminet1, M. Ricki1, L. Duchateau1, L. Van Ham1, S. Bhatti1
1Department of Small Animal Medicine and Clinical Biology, Ghent University, Belgium. 2Department of Pathobiology and Diagnostic Investigation, Michigan State University, United States. 3Department of Physiology and Biometrics, Faculty of Veterinary Medicine, Ghent University, Belgium.

Since early 2013, imepitoin has been introduced in most European countries for the management of recurrent single generalized epileptic seizures in dogs with idiopathic epilepsy. It has been demonstrated that imepitoin is similarly effective as phenobarbital in controlling seizures in dogs with newly diagnosed idiopathic epilepsy. Furthermore, a clinically significant superior safety profile was shown compared to phenobarbital administration. As the use of imepitoin gains popularity, its effect on serum thyroid function test parameters warrants further investigation. It is well known that long-term phenobarbital administration influences thyroid function test parameters in dogs. This alteration in serum thyroid hormone concentrations can lead to misinterpretation of results and incorrect diagnosis of thyroidal illness. A prospective study was conducted to compare the effect of phenobarbital and imepitoin on serum concentrations of total thyroxine (TT4), triiodothyronine, free thyroxine, thyroglobulin auto-antibodies, thyroid-stimulating hormone, cholesterol and triglycerides in healthy Beagle dogs. These parameters were determined prior to initiation of antiepileptic drug administration, and after 6, 12 and 18 weeks of antiepileptic drug administration. The oral starting dose of phenobarbital was 5 mg/kg PO q12h and was monitored and adjusted to obtain optimal therapeutic serum concentrations (30-35 µg/mL). Imeptoin was given at 30 mg/kg PO q12h.

The results of our study showed that, firstly, imepitoin administration did not affect mean serum TT4 concentrations over an 18 week period. In contrast, mean serum TT4 concentrations decreased significantly over time in dogs where phenobarbital was administered. Secondly, mean serum cholesterol concentrations increased significantly over time in the dogs on imepitoin but not to the same extent as commonly seen in dogs with primary hypothyroidism.
Efficacy of Imepitoin as First Choice Drug in the Treatment of 53 Naïve Dogs Affected by Idiopathic Epilepsy

A. Gallucci1, T. Gagliardo1, M. Menchetti1, A. Ruffini1, E. Bianchi1, A. Micili1, P. Tosolini1, A. Caudo1, G. Gandin1
1Department of Veterinary Medical Sciences, University of Bologna, Ozzano Emilia, Italy. 2Department of Veterinary Medical Sciences, University of Parma, Parma, Italy. 3Veterinary Clinic “Città di Saronno”, Saronno, Italy. 4Veterinary Clinic “Schiavi”, Udine, Italy, 5Veterinary Clinic “Ass. Prof. Neurovet”, Legnano, Italy.

The efficacy and safety of Imepitoin, a novel antiepileptic drug, were retrospectively evaluated in 53 naïve idiopathic epileptic dogs that met the inclusion criteria (diagnosis of Idiopathic Epilepsy and Imepitoin monotherapy).

Follow-up information was obtained through a questionnaire sent to the owners: 53 (100%), 35 (66%) and 13 (25%) dogs had follow-up information at 3, 6 and 9 months, respectively. Imepitoin starting dosage (expressed as mg/kg Bid) was 8-10 in 11 dogs (21%), 11-20 in 40 dogs (75%) and 21-30 in 2 dogs (4%). During time, dosage was increased in 20 dogs (34%), becoming 11-20mg/kg in 39 dogs (74%) and 21-30mg/kg in 10 dogs (19%). Successful treatment, defined as >50% reduction in seizures was noted in 25 (47%), in 20 (57%) and 9 (69%) dogs at 3, 6 and 9-months follow-up, respectively. Complete remission was achieved in 15 (28%), 9 (26%) and 3 (23%) dogs at 3, 6 and 9-months follow-up. Cluster seizures (before treatment: n=16, 30%) turned to single seizures in 8 (15%), 4 (11%) and 1 (8%) at 3, 6 and 9 months follow-up. Side effects were observed in 19 dogs (36%) and consisted in transient sedation/somnolence, hyperexcitability, aggressiveness, tremors, gastrointestinal problems. Two dogs still showed mild aggressiveness in long term. Lack of efficacy and consequent change/association of drug was the reason for 9 and 11 dogs that missed the follow-up at 6 and 9 months.

In this study Imepitoin showed its major efficacy at a dosage >15mg/kg Bid. Side effects were mild and, mostly, transient.

VALUE OF CEREBROSPINAL FLUID ANALYSIS IN EPILEPTIC DOGS THAT LACK INTERICTAL NEUROLOGICAL ABNORMALITIES AND HAVE UNREMARKABLE MAGNETIC RESONANCE IMAGING OF THE BRAIN

A M Coelho1, T Madoo2, D Sanchez-Masian, R Goncalves. Small Animal Teaching Hospital, University of Liverpool, Neston, CH64 7TE, UK.

To diagnose idiopathic epilepsy (IE), CSF analysis is essential to exclude other causes of seizures. Dogs with interictal neurological deficits are likely to have a underlying brain disease but the value of performing CSF analysis in dogs that lack interictal changes and have normal advanced imaging of the brain has not yet been assessed.

The database of the University of Liverpool was searched for dogs presenting for recurrent seizures with no interictal abnormalities according to the owners. Inclusion criteria for the study comprised an unremarkable neurological examination, exclusion of possible causes of reactive seizures (including a normal haematology and biochemistry profiles) and normal magnetic resonance (1-Tesla) of the brain. Results of the CSF analysis in these patients were evaluated and considerate abnormal if the protein concentration was >30mg/dl and/or the total nucleated cell count was >5cells/μl. Dogs were excluded if the CSF RBC count was >5,000μl.

A total of 120 dogs met the inclusion criteria. An abnormal CSF was only found in 5.8% (7/120) of dogs. The prevalence found for diagnosis other than IE was 1/120 dogs (0.8%). The site of CSF sampling, the interval between last seizure and CSF collection and the seizure type had no correlation with having an abnormal CSF. The number of red blood cells in the sample was significantly different between dogs with normal and abnormal CSF (P<0.01).

These results suggest that CSF analysis has a poor incremental diagnostic value for other than idiopathic epilepsy in patients with the same inclusion criteria of this study.

PHENOTYPIC CHARACTERIZATION OF SPINNING AND TAIL-CHASING IN GERMAN SHEPHERD AND JACK RUSSELL TERRIER

Catherine Escriche1, Julie Meynet1, Caroline Dufaure de Citres2, Anne Thomas2
1Neurology, VeDagro Sup, Lyon Veterinary Campus, France; 2Antigène, France

A predisposition to develop “spinning” (S) and “tail chasing” (TC) behavior has been observed in Bull Terriers (BT). These stereotypic behaviors are related to Obsessive Compulsive Disorder (OCD) and genetic basis have been demonstrated. Signs of hallucination and psychotic like behavior (freezing, staring, unprovoked aggression, unexplained growing, fly catching) are commonly associated and a complex neurological and evolutive syndrome related to autism is suspected were OCD coexists with focal/partial seizures.

In order to determine if others breeds could be affected by this disease, we recruited other dogs than BT affected by spinning or tail-chasing using a web questionnaire. We recruited 20 German Shepherd (GS) and 14 Jack Russell Terrier (JRT). A precise phenotypic characterization could have been done and compared to BT’s one. As in BT, we describe an association between spinning/tail chasing and psychotic like behavior with alteration of dog’s personality. Trance like behavior were observed in a majority of dogs with disconnection from the environment during episodes. Same stages of disorders reflecting the severity and evolutive nature of the disease were applied to GS or JRT as for BT. Some minor breed specificities seems to exists as high spinning velocity in JRT, spinning with tail in the mouth in JRT and GS (but not in BT) for example. Spinning or tail chasing in GH and JRT must be considered as the same complex neurobehavioral disease as in BT although its prevalence in this breeds seems to be lower than in BT.

VLDLR-ASSOCIATED CEREBELLAR HYPOPLASIA IN EURASIER DOGS

A. Fischer1, K. Rentmeister2, S. Lindstedt1, F. Bernardino1, E. Manz1
1Centre for Clinical Veterinary Medicine, LMU University of Munich, Munich, Germany; 2Tierärztliche Praxis für Neurologie, Dettelbach, Germany; 3Kynologische Zuchtgemeinschaft Eurasier e. V., Germany; 4Generatio Sol GmbH, Heidelberg, Germany

The very low density lipoprotein receptor (VLDLR) is part of the reelin signaling pathway, which directs neuroblast migration. VLDLR-associated cerebellar hypoplasia in Eurasier dogs is an autosomal recessive inherited cerebellar malformation due to a one base pair deletion in the very low density lipoprotein receptor (Gerber et al., 2015). It is characterized by absence of the caudal portions of the cerebellar vermis and the
cerebellar hemispheres, large retrocerebellar fluid accumulations, enlarged fourth ventricle and variable caudal fossa size (Bernardino et al., 2015). Non-progressive ataxia is the main neurologic sign. Phenotypic variation in the severity of the ataxia may occur, ranging from severe cerebellar ataxia in puppies, to improved ataxia in adults, and an almost normal gait in one affected dog despite the presence of a severely hypoplastic cerebellum.

Therefore the aim of the present investigation was to estimate the prevalence of the VLDLR:c.1713delC mutation in a population of Eurasier dogs. EDTA blood samples from 397 Eurasier dogs were collected in association with the respective breed club and stored frozen at -20°C until further analysis. Genetic testing for the VLDLR-mutation was done as described previously (Gerber et al., 2015). Results identified 46 heterozygous carriers of the mutation and indicated an estimated prevalence of 11.59% (CI95% 8.44%–14.74%). In conclusion, the VLDLR:c.1713delC mutation has a high prevalence in this population. Genetic testing is recommended prior to breeding in any Eurasier dog to avoid breeding carriers to each other, and also as a simple diagnostic tool in any Eurasier dog with non-progressive ataxia.

Magnetic resonance imaging (MRI) is a pivotal diagnostic test for epilepsy and movement disorders (MD) however the sensitivity is limited because diagnosis of idiopathic epilepsy is one of exclusion. Reliability is limited by available technology and variations in protocol which may not be optimised for detection of subtle epileptogenic lesions. There are 3 main aims: 1) rule out diseases which may be treatable with means other than anticonvulsant therapy (e.g, inflammatory or infectious brain disease) 2) identify lesions which are caused by seizures but not necessarily the source of seizures, for example hippocampal sclerosis 3) to provide data to further advance the field of research into the pathogenesis and/or treatment of epilepsy and MD. There is a need for a further standardized veterinary epilepsy and MD MRI protocol which will: facilitate more detailed examination of areas susceptible to generating and perpetuating seizures / MD; complement pathological studies; is economical; simple to perform; and can be adapted for both low and high field scanners. Standardisation of imaging will improve clinical communication and uniformity of case definition between research studies. The International Veterinary Epilepsy Task force proposes a 6-7 sequence epilepsy-specific MRI protocol including sequences orientated parallel and perpendicular to the hippocampus.

A finite element model was constructed, using geometry extracted from MRI scans of a Cavalier King Charles spaniel with syringomyelia, to explore possible mechanisms of syrinx formation. The model included the spinal cord, subarachnoid space (SAS), dura mater, and the epidural space. It has been shown in patients with restricted CSF flow, that there is exaggerated movement of the spinal cord during the cardiac cycle. This motion was applied to the cranial end of the spinal cord of the model. The peak longitudinal and radial pressure differences in the SAS oscillated between -22.3 to +90.0 Pa, and -100 to +100 Pa, respectively. Low-amplitude cyclic shear stresses were present in the cervical spinal cord (C2 – C6), where the cavities typically originate.

In conclusion it was proposed that the CSF pressure gradients are unlikely to cause fluid movement into the cord, sufficient to generate syrinxes. On the other hand, although the shear stress in the cord is low, its location and cyclic nature indicates the possibility that this may be the factor that generates the initial tissue damage, which eventually leads to the formation of syrinxes.

A PRELIMINARY STUDY OF THE PENLIGHT-COVER TEST IN DIFFERENTIATING BETWEEN PERIPHERAL AND CENTRAL VESTIBULAR DISEASE IN DOGS AND CATS


Differentiating between peripheral and central vestibular disease is important for clinical decision-making and prognostication. Current diagnostic methods used for differentiation are infrequently available in general practice, require general anaesthesia and carry a high cost. The penlight-cover test (PCT) may provide a simple, safe and cost-effective clinical test to overcome these limitations. Masking visual input using a bright pencil tip should reduce suppression of spontaneous nystagmus in cases of peripheral but not central vestibular disease.

The utility of the PCT to differentiate between peripheral and central vestibular disease was evaluated by measuring changes in slow phase velocity (SPV) and beat frequency (BF) of nystagmus whilst masking visual input. Data were collected from 5 peripheral and 8 central cases in a referral population of dogs and cats. SPV increased when visual input was removed in 60% of peripheral cases but 0% of central cases (Fisher’s exact test, p=0.035). BF increased when visual input was removed in 80% of peripheral cases but 12.5% of central cases (Fisher’s exact test, p=0.039). The BF increased by a median of 11 beats in 7 seconds in peripheral cases and 0 beats in 7 seconds in central cases (Mann-Whitney U test, p=0.0128).

In conclusion, increased SPV or BF when visual input is removed may increase suspicion of peripheral vestibular disease, whereas unchanged SPV, or unchanged or decreased BF, may increase suspicion of central vestibular disease. Further studies are required to calculate the sensitivity, specificity and predictive values of the PCT in a larger population of dogs and cats.

COMPUTER SIMULATION OF THE CANINE SPINE: THE EFFECTS OF INCREASED SPINAL CORD MOTION ON THE DEVELOPMENT OF SYRINGOMYELIA

R. Lloyd1, S. Cirovic2, J. Jovanovik3, H. Voli3, C. Rusbridge1,4. 1Dept. of Mechanical Engineering Sciences, University of Surrey Guildford, Surrey, UK; 2Fitzpatrick Referrals, Halfway Lane, Eashing, Godalming, Surrey, UK; 3Royal Veterinary College, University of London, London, UK; 4School of Veterinary Medicine, University of Surrey, Guildford, Surrey, UK.

Syringomyelia is a disorder characterised with the presence of fluid-filled cavities in the spinal cord. The condition occurs in both humans and animals. The mechanism(s) of cavity formation are not clear, leading to limited treatment. Current theories are based on the assumption that abnormalities in the circulation of the cerebrospinal fluid (CSF) generates pressures that drive the fluid into the cord.
INTERLEUKIN-17 AND CD40 LIGAND IN CANINE STEROID-RESPONSIVE MENINGITIS-ARTERITIS

J. Freundl Revilla1, R. Carlson1, A. Maolini1, A. Tipold2.1Dept. of Small Animal Medicine and Surgery, University of Veterinary Medicine, Hannover, Germany.

Steroid-Responsive Meningitis-Arteritis (SRMA) is an immune mediated disorder characterized by a neutrophilic pleocytosis in the cerebrospinal fluid (CSF). Previous studies of the disease have shown increased levels of IL-6 and TGF-β, in CSF indicating the presence of Th17 lymphocytes. IL-17 induces and mediates inflammatory responses and plays an important role in recruitment of neutrophils. The hypothesis of a Th17 skewed immune response in SRMA should be confirmed by evaluating IL-17 and CD40L, inducing B-T cell interaction.

Enzyme-linked immunosorbent assays (ELISA) were performed to measure IL-17 and CD40L in serum and CSF of patients suffering from SRMA in the acute stage (SRMA A), under treatment with glucocorticosteroids (SRMA T), other neurological disorders and in healthy dogs (animal experiment05-14A453). Significant higher levels of IL-17 were found in CSF of dogs with SRMA A compared with SRMA T, other neurological disorders and healthy dogs (p<0.0001). In addition, levels of CD40L in CSF in dogs with SRMA A and SRMA R were significantly higher than in healthy controls (p<0.05). Furthermore, CSF concentrations of IL-17 and CD40L showed a strong positive correlation among each other (rspear= 0.4215; p<0.0003) and with the degree of pleocytosis (rSpear= 0.8924; p<0.0001 and rSpear= 0.3817; p<0.0034).

These results imply that Th17 cells are inducing the autoimmune response in SRMA and are involved in the development of the severe neutrophilic pleocytosis. The investigation of the role of IL-17 and CD40L in SRMA adds to the knowledge of pathophysiological mechanisms in SRMA and opens the discussion about new therapeutic strategies.

MORPHOLOGICAL RECLASSIFICATION OF IMMUNE-MEDIATED NEUROPATHIES (IMNP) IN DOGS AND CATS: BEYOND THE CONCEPT OF AXONAL AND DEMYELINATING DISEASE


1Section of Clinical & Comparative Neuropathology, Centre for Clinical Veterinary Medicine, Ludwig-Maximilians University, Munich, Germany; 2Neurology Referral Service, Tierklinik Haar, Haar, Germany; 3Clinica Veterinaria Roma Sud, Rome, Italy; 4North Downs Specialist Referrals, Bletchingley, Surrey, UK; 5Langford Veterinary Services, University of Bristol, Bristol, UK; 6Section of Neurology, Centre for Clinical Veterinary Medicine, Ludwig-Maximilians University, Munich, Germany; 7Neurology Service, Small Animal Clinic, Luenen, Germany; 8Department of Veterinary Medical Science, University of Bologna, Italy; 9Avetia Clinic for Small Animal Medicine, Paris, France; 10Ecole Nationale Vétérinaire d’Alfort, Maisons Alfort, France; 11Clinica Veterinaria Valdanevole, Monsummano, Italy; 12Neurology Service, Clinic of Small Animal Medicine, University of Berlin, Berlin, Germany; 13Section of Neurology, Clinic of Small Animal Medicine, University of Leipzig, Leipzig, Germany; 14Clinica Veterinaria Malpensa, Samarate, Italy

Recent developments in human Guillain-Barre research challenge the classification of IMPN according to axonal and demyelinating features even though unsupported by morphological investigations. To contribute to this discussion, this study screened for an association of inflammatory features and fibre pathologies amongst IMPN affected nerves from dogs and cats. Archived nerves from dogs and cats exhibiting fibre-invasive inflammatory cells underwent duo dichromatic teasing preparation (DTP) and were reevaluated for affection of functional fibre subunits. The features were correlated to clinical records and electrophysiological data. Altogether 19 IMPN affected dogs and 15 cats were included. In each species, the mode of inflammation and fibre degeneration gave rise to four IMPN subtypes, with affection of Schmidt-Lantermann clefts (SLC) (9/34), nodes-panarodes (NPN) (10/34) or both (14/34). Amongst dogs displaying NPN (9/19) two different stages were distinguished suggesting humoral immune mechanisms to precede cellular infiltrates (4/19). Feline biopsies mostly featured involvement of both subunits (12/15) at advanced stages. Electrophysiology was rarely predictive of the primarily affected subunit in pure SLC and NPN types. Evaluation of fibre subunits provide a better insight into the immunobiology of IMPN by unravelling the primary targets. Like in humans, nodo-panarodopathies and internodopathies can be distinguished in animals. In a strict sense, the former relates to acute motor axonal neuropathy (AMAN) while the latter corresponds to acute inflammatory demyelinating polyneuropathy (AIDP) in people. Correct IMPN subtyping by DTP is recommended in order to achieve a correct diagnosis and to base prospective clinical studies on a scientific ground.

IMUNOHISTOCHEMICAL CHARACTERIZATION OF THE ANTI-INFLAMMATORY EFFECT OF TWO TREATMENT PROTOCOLS IN DOGS WITH GRANULOMATOUS MENINGENCEPHALOMYELITIS OR NECROTIZING (MENINGO)-ENCEPHALITIS

F. Salger1,2, A. Oevermann1, L. Kreipe1, T. Flegel1, M. Vandevelde1,2, B. Vldondo Curras1, D. Henke1,2

1Division of Neurological Sciences, 2Division of Clinical Neurology, Department of Clinical Veterinary Medicine, 3Department of Small Animal Medicine, Faculty of Veterinary Medicine, University of Leipzig, Germany, 4Department of Veterinary Public Health, Vetsuisse Faculty, University of Bern, Switzerland.

There is little objective evidence about the effect of immunosuppressive treatment of canine granulomatous meningoencephalomyelitis (GME) and necrotizing encephalitides (NME/NLE summarized as NE). To assess the effect of two different treatment protocols for GME and NE, the inflammatory reaction in these conditions was evaluated quantitatively and qualitatively in 18 untreated dogs (5 GME, 13 NE), in 10 dogs treated with prednisolone (4 GME, 6 NE), and in 7 dogs treated with prednisolone and lomustine (CCNU-P) (4 GME, 3 NE) which were all euthanized because of their CNS disease or other reasons. Randomly selected areas of representative lesions were examined for the total inflammatory cell count (TCC), and the number and relative distribution of T-lymphocytes (CD3), B-lymphocytes (CD20) and macrophages (CD163, F4/80) using immunohistochemistry.

In all untreated dogs, macrophages were the most common cell population, followed by T-lymphocytes and much less B-lymphocytes. The TCC was decreased following both treatment protocols as compared to untreated dogs; however, only after CCNU-P this difference reached significance. In dogs treated with CCNU-P for GME, all cell types, and in dogs with NME/NE specifically macrophages and T-lymphocytes were significantly decreased.

Our results regarding the qualitative distribution of inflammatory cells were consistent with previous reports in dog with GME; however, we did not find a predominance of T-lymphocytes in in dog with NE as reported before. The immunosuppressive effect of both treatment protocols was evident, but the CCNU-P...
A meningoencephalocele is a protrusion of cerebral tissue and meninges through a cranial defect. There are few case reports describing this uncommon condition in dogs. The aim of this retrospective case series is to describe the clinical presentation, magnetic resonance imaging (MRI) or necropsy features, treatment and outcome of dogs with meningoencephalocele.

Four young dogs (aged between 8 weeks and 10 months) were presented with a history of seizures. In one dog, neurological examination was unremarkable. In three dogs, neurological deficits indicated a forebrain neurolocalisation. Intranasal meningoencephalocele was diagnosed with MRI in all four cases. MRI revealed signs of inflammation of herniated meninges and brain parenchyma. One dog was euthanased after MRI. Necropsy confirmed the imaging diagnosis. The three other dogs were treated with phenobarbitone. In the three cases, seizures were well controlled under treatment for four months to four years after diagnosis.

In these four cases, intranasal meningoencephalocele was diagnosed with MRI in young dogs presenting with seizures. It has been hypothesised that a meningoencephalocele could be a possible seizure focus. In humans, surgical excision of the herniated brain tissue is the treatment of choice. In veterinary medicine, two cases (one dog and one cat) were described in which the neurological signs disappeared after surgical treatment. In three of the dogs presented here, adequate seizure control and quality of life were achieved with medical treatment only. Antiepileptic treatment is a valid non-invasive treatment option when there is no cerebrospinal fluid leakage and the neurological deficits are mild.

Seventeen dogs were treated by multiple TPLCs for ventral spinal cord compression caused by Hansen type I or II IVDD. The presurgical spinal cord compression and postsurgical decompression, as well as slot dimensions were measured based on computed tomography-myelography images. The neurological outcome was assessed based on repetitive examinations and owner questionnaires. Fourteen dogs had two TPLCs, two dogs had three TPLCs and one dog had four TPLCs performed. The mean slot depth, height and length were 63%, 29% and 25% of the vertebral body, respectively. The mean residual vertebral interslot length between two adjacent TPLCs was 65% of the vertebral body length. At re-evaluation four weeks after surgery, seven dogs (35.3%) had the same modified Frankel score compared to the presurgical examination, whereas eleven dogs (64.7%) had neurologically improved. According to the owners, 78.6% of the dogs showed a normal gait within six months after surgery. In conclusion, multiple spinal cord compressions caused by IVDD can be eliminated by multiple TPLCs. The benefit of complete spinal cord decompression at all relevant disc locations seems to outweigh the risk of potential spinal instability.

MULTIPLE THORACOLUMBAR PARTIAL LATERAL CORPECTOMIES IN 17 DOGS

S. Hanemann1, M. Münch1, K. Held1, F. Salger1, L. Ziegler1, P. Böttcher1, T. Flegel1
1Department of Small Animal Medicine, University of Leipzig, Germany; 2Division of Clinical Neurology, Vetsuisse Faculty, University of Bern, Switzerland; 3Clinic for Birds, Reptiles, Amphibians and Fish, Faculty of Veterinary Medicine, Justus Liebig University Giessen, Germany

Thoracolumbar partial lateral corpectomies (TPLC) facilitates access to the ventral spinal canal and allows spinal cord decompression by minimizing spinal cord manipulation. In veterinary literature multiple TPLCs have not been recommended because of potential spinal instability. The aim of this retrospective study was to report the feasibility of multiple thoracolumbar partial lateral corpectomies in dogs with intervertebral disc disease (IVDD) and to describe the clinical outcome.

In conclusion, multiple spinal cord compressions caused by IVDD can be eliminated by multiple TPLCs. The benefit of complete spinal cord decompression at all relevant disc locations seems to outweigh the risk of potential spinal instability.

COMPARISON OF CONVENTIONAL AND HIGH DEFINITION VIDEO TELESCOPE ASSISTED VENTRAL SLOT DECOMPRESSION FOR CERVICAL INTERVERTEBRAL DISC HERNIATION IN 51 DOGS

D. Rossetti, C. Ricco, G. Ragetly, C. Poncet, Centre Hospitalier Vétérinaire Frégis, Arcueil France

The ventral slot surgery is the treatment of choice for cervical intervertebral disc disease (IVDD) in dogs. Little is known about the use of magnification in veterinary neurosurgery. The objective of this prospective study was to compare the use of a Video Telescope Operating Monitor (VITOM) with the conventional approach.

Fifty-one dogs that presented with cervical intervertebral disc disease between June 2013 and September 2014. The 40 dogs were assigned to a VITOM group (n = 30) or a conventional group (n = 21). Signalement, pre-operative neurological status, preoperative spinal cord dimension at the compression level obtained with CT myelography, operative time, surgical complications, ventral slot size, postoperative spinal height and diameter at the compression level obtained with CT myelography, hospitalization time and the postoperative outcome were compared between the two groups.

No significant differences in the surgical time were noted (62.4 ± 14.2 min, p = 0.6). The VITOM group was associated with a greater post-operative spinal diameter (p = 0.01) and spinal height (p = 0.002) as well as a smaller ventral slot (p = 0.007) in comparison with the conventional group. The VITOM group was associated with a better post-operative outcome (p < 0.01) and a shorter post-operative hospitalization time (p = 0.006).

The VITOM installation was not time consuming and the learning curve was considered to be fast. The results of this study support the view that the VITOM technique could be advantageous compared with conventional ventral slot surgery.
Spinal walking (SW) is described as the acquisition of an involuntary motor function in paraplegic dogs and cats without deep pain perception (DPP) affected by a thoracolumbar (TL) lesion. Aim of this retrospective study, was to evaluate the number of TL paraplegic dogs without DPP that developed an autonomous spinal walking after intensive physical rehabilitation, consisting in passive and active assisted exercises. Cornerstone of the treatment were the use of underwater treadmill, and cage restriction to avoid the dogs to drag on their hind limbs. The medical records of 81 acute paraplegic TL dogs were selected according to the inclusion criteria (intensive rehabilitation treatment in paraplegic dogs with absence of DPP on admission and during the whole treatment). Nonparametric and parametric statistics were used to analyze possible correlation between variables and acquisition of SW. Autonomous SW was achieved in 48 dogs (59%). 34 had intervertebral disk disease, 14 had traumatic injury. Out of these, 31 underwent surgery. Age and weight were significantly lower (P=0.0064 and P=0.0269) in SW than in No-SW group. Type and site of the lesion and hospitalization were not significantly correlated to development of SW. Early start of physiotherapy and its duration, were positively associated with becoming SW (P=0.0426 and P=0.0001). Time of physiotherapy had a median of 86 days in SW and 64 days in No-SW dogs. Our data suggested that dogs with irreversible TL lesion had significant possibilities to develop SW after intensive physiotherapy treatment, especially when started soon and in lightweight dogs.

Role of Therapy with Growth Factors in the Management of Pain Perception Negative Dogs Caused by Thoracolumbar Disk Extrusions

D. Faisstler, E. Rozanski, M. Kowaleski, Cummings School of Veterinary Medicine at Tufts University, North Grafton 01532, MA, USA

The purpose of this prospective pilot study was to examine the hypothesis that dogs with an acute onset of paraplegia and absent pain perception treated with either subdural platelet rich plasma injections or the intravenous application of erythropoietin at the time of decompressive surgery will have a higher likelihood of a functional recovery than dogs treated with surgery alone. Inclusion criteria included chondrodystrophic dogs presenting with acute thoracolumbar disk extrusion, paraplegia, and absent pain perception. All dogs underwent decompression within 24 hours after admission and were randomly assigned to: 1) saline subdural, 2) autologous platelet rich plasma subdural or 3) erythropoietin (EPO) IV. Initial and follow-up examinations were performed at the time of admission, and 1, 3, 7, 14, 42, and 84 days post-surgery, with a focus on ambulation and fecal/urinary continence. Statistical analysis was performed with SPSS for Windows 20 software. The level of significance was defined as P < 0.05.
The prognosis for canine nasal tumors with intracranial extension is poor with an expected survival of 1 month with palliation and 6.7 months with irradiation. However, studies regarding stage IV nasal tumors treated with brain irradiation technique are limited. The aim of this prospective study was to evaluate feasibility and efficacy of definitive intent stereotactic radiotherapy in dogs with nasal tumors with massive intracranial extension.

Seven dogs with stage IV nasal tumors were treated with high-dose hypo-fractionated stereotactic radiotherapy with volumetric modulated arc therapy technique. Of these dogs, 32 were prescribed 36-36 Gy in four consecutive-day fractions to the gross tumor and 30 Gy to the hypoxic region. Adjuvant treatment included carboplatin. Serial clinical and Computed Tomography/Magnetic Resonance Imaging examination were performed. Disease control and toxicity effects were evaluated according to response evaluation criteria in solid tumors and veterinary radiotherapy oncology group criteria. Median survival time (MST) was evaluated using Kaplan-Meier curves. Six carcinoma and 1 sarcoma were treated. Prescriptions were accepted to limit maximum brain punctual dose<27 Gy. Two partial response and 5 complete responses were observed. Relapse pathways involve diffuse meningeal and sphenoid invasion. The initial experiences with the radiation therapy regimen adopted indicate a feasibility and effectiveness in modified stage IV nasal tumors. The relapse pathways observed suggested to evaluate alternative adjuvant treatment in dogs treated with stereotactic radiotherapy.

Surgical stabilization of canine lumbosacral spine can be challenging. The aim of this research was to evaluate two surgical techniques to treat lumbosacral spinal disease in dogs either with normal or transitional vertebrae. Lumbosacral instability and degenerative stenosis were evaluated by dynamic Computed Tomography (CT) and Magnetic Resonance Imaging (MRI). In dogs with normal vertebrae, 4.5 mm screws were bicortically inserted in S1 with the heads behind the caudal articular process of L7 to prevent the extension of the lumbosacral joint; if vertebral lysis was evident, the heads of the screws were augmented by methyl methacrylate. In dogs with transitional vertebrae, 4.5 mm screws were inserted in the iliac wings, two 3.5 mm screws were inserted in the spinous process of L6 and end plates were also embedded in methyl methacrylate after flexion of the lumbosacral spine. In cases of residual radicular compression, dorsal laminectomy and partial disectomy were accomplished. Serial clinical and imaging follow-up examinations were performed. Twenty-two large breed dogs were enrolled. In 14 dogs stop-screws (in 4 augmented) and in 8 dogs iliac wings technique are effective surgical stabilization of canine lumbosacral spine.

EVALUATION OF MAGNETIC RESONANCE IMAGING GUIDELINES TO DIFFERENTIATE BETWEEN NUCLEUS PULPOUS EXTRUSIONS AND ANULUS FIBROSUS PROTRUSIONS IN LARGE BREED DOGS


Four MRI variables have recently been identified, which are independently associated with a diagnosis of thoracolumbar nucleus pulposus extrusion (NPE) or anulus fibrosus protrusion (AFP), midline intervertebral disk (IVD) herniation, and partial IVD degeneration were as associated with AFP, while a presence of a single IVD herniation and disk material dispersed beyond the boundaries of the IVD space were associated with NPE. The aim of this study was to evaluate if using these MRI variables improves differentiation between NPE and AFP. No ethical approval was required for this study. Eighty large breed dogs with surgically confirmed thoracolumbar NPE or AFP that underwent MRI were included. Studies were randomized and presented on 2 occasions to 6 blinded observers, which were divided into 3 experience categories. During the first assessment, observers made a presumptive diagnosis of thoracolumbar NPE or AFP without guidelines. During the second assessment they were asked to make a presumptive diagnosis with the aid of the guidelines. Additionally, they were asked to record the presence or absence of each MRI variable. Agreement was evaluated by Kappa-statistics. Diagnostic accuracy improved from 70.8% to 79.6% and inter-observer agreement for making a diagnosis of NPE or AFP improved from (k = 0.27) to moderate (k = 0.41) after using the proposed guidelines. Diagnostic accuracy was influenced by degree of observer experience. Intra-observer agreement for the assessed variables was variable, while inter-observer agreement ranged from fair to moderate.

The results of this study suggest that the proposed imaging guidelines can aid in differentiating thoracolumbar NPE from AFP.
COMPUTATIONAL DEVELOPMENT AND EVALUATION OF A CERVICAL INTERVERTEbral DISC PROTRUSION MODEL OF THE CERVICAL SPINE: THE MEANS OF THE FINAL ELEMENT METHOD

P.V. Marinho¹, A.P. Macedo¹, P.C. Sampayo¹, A.C. Shimano², C.C. Zani², M.V.B. Arias³, Dept. of Surgery, University of São Paulo, Brazil; 1Dept. of Medicine and Prolopathology, Ribeirão Preto Dental School, University of São Paulo, Brazil; 2Dept. of Design, State University of Londrina, Brazil; 3Dept. of Medicine and Locomotive Apparatus Rehabilitation, Ribeirão Preto Medical School, University of São Paulo, Brazil; 4Dept. of Veterinary Medicine, State University of Londrina, Brazil.

Currently the most effective treatment of disc-associated cervical spondylomyelopathy is distraction—merger, however this technique can lead to biomechanical changes in the adjacent segments increasing the risk of “domino” type injury. The objective of this study was to develop a cervical intervertebral disc prosthesi and evaluate main stress points in the prosthesi system - vertebral body using the Finite Element Method (FEM). The prosthesi sizing was based on width, height and measuring of the vertebral body according to the BML. a three dimensional vertebral spine of mature canine cadavers weighing between 25-35 kg. The prosthesi was developed on Rhinoceros® software and 3D prototyping to refine its design. the analysis was done using the FEM using the Aways® Workbench sofware after applying extension, lateral and ventral bending forces, assessing the average equivalent von-Mises stresses on the periphery of the vertebral body. The vertebral body received much lower stress than the prosthesis for all applied forces. the average stresses on the vertebral body were superior on the lateral and ventral surfaces when compared to the cranial surface of C5 and the caudal of C6. Finally, the average stress on the prosthesi was intensely more focused on the bearing surface contact and less intensely on the interface between the prosthesis and the vertebral body, the bigger stress happened on the cranial surface.

The developed prosthesis had an adequate design and good fitness on the intervertebral space between C5 and C6, allowing a homogeneous stress on 6×36° surfaces on the vertebral body, which lowered stress on vertebral endplates.

CHANGES IN INTESTINAL VS. POSTICAL DIFFUSION AND PERFUSION MR PARAMETERS IN FAMILIAL SPONTANEOUS EPIDEMIC CATS D. Hasegawa¹, T. Iwabuchi¹, T. Y. A. Fujikura¹, M. Fujita¹, Dept. Clinical Veterinary Medicine, Nippon Veterinary and Life Science University, Tokyo, Japan.

Familial spontaneous epileptic cats (FSECs) are the only genetic model of epilepsy in cats. The epileptogenic zone of FSECs is thought to exist in the amygdala and/or hip pocampus, as determined by seizure semiology (spontaneous focal seizures, status epilepticus and clinical signs). the objective of this study was to compare the clinical presentation and outcome of dogs with a presumptive diagnosis of ischemic myelopathy and acute non-compressive neural pulse pressure extrusion (IM). Medical records and MRI studies were reviewed, yielding 93 suitable dogs with an antemortem presumptive diagnosis of IM or ANPNE. Concurrent clinical presentation and short-term outcome were retrospectively retrieved from clinical records. Long-term follow-up was achieved by telephone questionnaire with veterinarians and owners.

Compared to the hospital population of the study institution, English Staffordshire Bull Terriers were overrepresented with IM, and Border Collies with ANPNE. Dogs with ANPNE were significantly older (mean 7.0 y ±2.2) than those with IM (mean 5.9 y ±2.8). Dogs diagnosed with ANPNE were significantly more likely to present with a history of onset vocalization (50%), spinocerebellar atrophy (47.6%) and a CL-C5 myelopathy (16.7%) compared to those with IM. Dogs with IM were more likely to present with a L4-S3 myelopathy (11.8%), compared to dogs with ANPNE (0%). Dogs with ANPNE were more likely to be ambulatory at discharge (69.0%) than those with IM (43.1%). Although long-term follow-up did not reveal a difference in quality of life or outcome success, dogs with IM were “Department of Small Animal Medicine and Surgery, University of Veterinary Medicine Hannover, Germany”. Veterinary Practice A. Batten-Nöthen, Cologne, Germany.

The aim of this study was to prove the reliability of the neuroanatomical examination in localizing a vestibular syndrome in dogs using magnetic resonance imaging (MRI) as gold standard of correct lesion localization. Neuroanatomical examination results and MRI findings of 91 dogs with vestibular signs were retrospectively enounced in a central vestibular syndrome (CVS) and peripheral vestibular syndrome not responding to treatment (PVS). The majority of dogs (78/91; 85.7%) showed a head tilt as cardinal sign indicating vestibular disease. Additional signs comprised ataxia, strabismus, nystagmus, proprioceptive deficits, other cranial nerve deficits, and tetraparesis. Based on the neuroanatomical examination, 31 dogs had PVS, 57 had CVS and 3 remained unclear. After MRI examination 20 dogs had PVS, 70 dogs had CVS, and the diagnosis remained unclear in one dog. The neuroana...
EVALUATION OF POSTOPERATIVE SURVIVAL AND COMPLICATIONS AFTER INTRACRANIAL SURGERY IN DOGS

A.K. Forward, H.A. Volk, S. De Decker. Royal Veterinary College, University of London, Hatfield, United Kingdom.

Although intracranial surgery is increasingly performed in veterinary medicine, little is known about the nature and prevalence of post-operative complications and survival of dogs undergoing intracranial surgery. This study aimed to describe survival and early postoperative outcome following intracranial surgery in dogs.

Records were searched and analysed for dogs that had intracranial surgery between 2005-2015. Signalment, clinical presentation, neurological deficits, concurrent medical conditions, perioperative laboratory data, mode of perioperative analgesia and sedation, and outcome data were collected. Dogs were categorised by surgical indication and study was performed by logistic regression of survival postoperatively.

Of the 48 dogs included in this study, various complications were diagnosed. The most common complications included seizures, brain oedema, haemorrhage, and chronic meningitis. Overall, the survival rate for dogs undergoing intracranial surgery was 60.8% (30 dogs) at 1 month postoperatively. The mortality rate was highest in dogs with brain oedema and haemorrhage.

MAGNETIC RESONANCE FEATURES OF SUSPECTED LEUKOARAIOSIS IN ELDERLY DOGS


Age-related changes have been identified on brain MRI (magnetic resonance imaging) of dogs over 9 years of age. Changes identified in the white matter of the brain include areas of hypointensity on T1-weighted images and hyperintensity on T2-weighted images. The pathophysiology of leukoaraiosis remains incompletely understood, but an ischaemic origin of these lesions has been favoured. Endothelial damage, leading to narrowing of the vessels lumen, and ultimately reduced blood supply to the brain structures affected by leukoaraiosis is postulated.

PREVALENCE AND BREED PREDISPOSITION OF THORACOLUMBAR INTERVERTEBRAL DISC DISEASE IN CATS

S. De Decker, A.S. Warner, H.A. Volk. The Royal Veterinary College, University of London, Hatfield, United Kingdom.

Although several studies have evaluated the prevalence and breed predisposition of intervertebral disk disease (IVDD) in dogs, such information is not yet available for cats. In this study, the prevalence and possible breed predispositions for thoracolumbar IVDD in cats were examined. The study included a total of 465 cats that were presented for physical and neurological examinations. The prevalence of thoracolumbar IVDD in cats was 7.2% (33/465). The most common clinical signs associated with IVDD included neurological deficits, such as hindlimb weakness, incontinence, and decreased pain sensation. The results of this study suggest that the prevalence of thoracolumbar IVDD in cats is lower than that reported in dogs.

COMPARISON OF CERVELICAL CANAL OCCUPANCY IN GIANT, LARGE, AND SMALL BRED DOGS: AN MRI MORPHOMETRIC STUDY

M. Gattone, G. da Costa, H.C. Pernambuco, Recife-PE, Brazil, 2The Ohio State University, Columbus-OH, USA.

The pathophysiologic of cervical spondylomyelopathy (CSM) appears to involve a major static component, namely relative and absolute stenosis of the vertebral canal, with consequent compressive effect on the spinal cord. Our objective was to compare the cervical vertebral canal occupany ratio of the spinal cord (C3-C7) in giant, large, and small breeds. The C3-C7 canal area was calculated using MRI. Giant breeds were more affected compared to small breeds. The study suggests that the prevalence of CSM in giant dogs is higher than in small breeds. The results of this study highlight the need for further research to understand the pathophysiology of CSM across different breeds.

RETROSPECTIVE STUDY OF SPINAL ARACHNOID DIVERTICULUM IN 15 DOGS DIAGNOSED WITH INJURY

A. Tauro, J. Jovorovic, V. Crvje, V. Rudignje. Fitzgerald referrals, Easing, UK. University of Surrey, Guildford, UK.

Spinal arachnoid diverticula (SAD) are considered rare conditions in veterinary medicine, although the number of diagnosed cases is currently increasing. The purpose of this study was to describe the clinical presentation, diagnosis, and outcomes of dogs with SAD. The study included 15 dogs diagnosed with SAD between 2008 and 2014. The most common clinical signs associated with SAD were hindlimb weakness, incontinence, and decreased pain sensation. The results of this study suggest that SAD is a rare but important condition in veterinary medicine, with a broad range of clinical presentations and possible etiologies.
steady state (Ciss), which has a combination of high signal levels and extremely high spatial resolution and offers excellent contrast. Csf and pathological structures in providing anatomical information to identify SAD with minimal signal loss due to Csf pulsations. The presence of compatible clinical signs and advanced imaging using the 3D-CISS sequences confirming the presence of SAD. Male Pugs were over-represented, while caudal terriers (sBts) appeared to be predisposed to multiple SAD located dorsally at C2-C3 and thoracic spine. 3D-CISS sequences were beneficial in identifying SAD secondary to scarring and adhesion following surgical correction. Findings from this study support the superiority of 3D-CISS imaging in all cases particularly when spinal cord injury is suspected, as gait atrophy, spasticity, or syringomyelia may obscure the true spinal cord lesion.

CLINICAL FEATURES AND DISEASE PROGRESSION OF L-2-HYDROXYGLUTARIC ACIDURIA IN 27 STAFFORDSHIRE BULL TERRIERS

L. A. Riso1, H. Caruncho1, E. Beltran1. 1Centre for Small Animal Studies, The Animal Health Trust, Newmarket, United Kingdom; 2Tay Valley Veterinary Centre, Perth, Scotland; 3Queen Mother Hospital for Animals, Royal Veterinary College, Putney’s Bar, United Kingdom

L-2-hydroxyglutaric aciduria (L2HGA) results from autosomal recessive mutations within the L-2-hydroxyglutarate dehydrogenase (L2HGDH) gene. Accumulation of L-2-hydroxyglutaric acid in brain tissue causes oxidative stress and interferes with cerebellar creative kinase activity. There is no published data on clinical outcomes or prognostic factors. Our aim was to describe the onset, pattern and nature of clinical signs (CS) in a cohort of L2-HGA-affected Staffordshire Bull Terriers (SBTs).

Owners of 119 SBTs positive for the L2HGDH genetic mutation were reviewed retrospectively. Inclusion criteria were the availability of data, with stiffness of all four limbs the most common (24/26) and earliest recognized abnormality. The mean age of onset of CS was 12 months (range 2.5-60). Gait dysfunction was reported in all 26 dogs with available data, with stiffness of all four limbs the most common (24/26) and earliest recognized abnormality.

The mean age of onset of CS was 12 months (range 2.5-60). Gait dysfunction was reported in all 26 dogs with available data, with stiffness of all four limbs the most common (24/26) and earliest recognized abnormality. The mean age of onset of CS was 12 months (range 2.5-60). Gait dysfunction was reported in all 26 dogs with available data, with stiffness of all four limbs the most common (24/26) and earliest recognized abnormality.

L2-HGA is considered a progressive neurological disease, however symptoms can be successfully managed to allow dogs a good quality of life long term.

IDENTIFICATION OF THE CAUSAL GFAP MUTATION IN A LABRADOR RETRIEVER WITH ALEXANDER’S DISEASE

V. Martle, M. Van Pouckel, L. Van Brantegem2, L. Van Ham3, S. Bhatti3, R. Ducatel3, L. Peelman1. 1Department of Small Animal Medicine and Clinical Biology, Ghent University, Merelbeke, Belgium; 2Department of Pathology, Bacteriology and Animal Diseases, Faculty of Veterinary Medicine, Ghent University, Belgium

Alexander’s disease is a neurodegenerative disorder of astrocyte dysfunction in humans, for which already a number of causal mutations have been identified. A 3-month-old Labrador retriever pup was euthanized at the age of 4.5 months and on histopathological examination of the brain tissue was diagnosed with Alexander’s disease (GFP). The pup was euthanized at the age of 4.5 months and on histopathological examination of the brain tissue was diagnosed with Alexander’s disease (GFP). During this study, we performed a comprehensive genotyping of the canine GFAP gene and compared our results to the canine reference sequence. Genetic analysis of the GFAP gene identified a heterozygous G9A nucleotide substitution resulting in an arginine to histidine amino acid substitution at position 240.

We detected a consistent point mutation in a Labrador retriever with obvious improvement in 1 dog. Moreover, we identified a heterozygous G9A nucleotide substitution resulting in an arginine to histidine amino acid substitution at position 240.

APPLICATION OF MAGNETIC RESONANCE SPECTROSCOPY IN CANINE EPILEPSY OF UNKNOWN ORIGIN - THE PRELIMINARY RESULTS

Agnieszka Olzewska1, Marta Płonek1, Józef Nicpoń2, Małgorzata Wilk1, Józef Waliszewski1, 1Department of Internal Medicine and Clinic of Diseases of Horses, Dogs and Cats, Center of Experimental Diagnostics and Innovative Biomedical Technology, The Faculty of Veterinary Medicine, Wroclaw University of Environmental and Life Sciences

Canine epilepsy posts a diagnostic challenge for veterinary professionals. Magnetic resonance spectroscopy (MRS) is a noninvasive method that determines the chemical composition of brain tissue and is displayed as a spectrum of peaks fit along the x-axis, labeled in parts per million (ppm). The three major peaks observed in the MRS spectrum are: phosphocreatine (P), choline (Cho) and Cr. Data from 21 canine epileptic patients (9 epileptic and 12 non-epileptic controls) were analyzed.

The study was carried out on 10 dogs of different breeds with generalized epilepsy of unknown origin. We aimed at identifying factors influencing the occurrence of epileptic crises. The aim was to describe the phenotypical description of a dog with a possible paroxysmal dyskinesia with an adult onset is suspected in the Maltese dog, although additional cases and expanding pedigree and genetic analysis is necessary.

A MUTATION IN MFSD8 CAUSES NEURONAL CERIOD LIPOFUSCINOSIS IN CHIHUAHUA DOGS

F. J. E. Faller1, J. Bras2, S. Sharpe3, L. Darwent1, C. Kun-Rodrigues1, J. Aroyi1, J. Perdener1, S. E. Meier1, R. Gutierrez-Quintana1, R. J. Guerreiro2. 1Dept. of Small Animal Medicine, University of Leipzig, Leipzig, Germany; 2Dept. of Small Animal Medicine and Surgery, University of Veterinary Medicine Hannover, Hannover, Germany

The purpose of this study was to describe the clinical outcomes or prognostic factors. Our aim was to describe the onset, pattern and nature of clinical signs (CS) in a cohort of L2-HGA-affected Staffordshire Bull Terriers (SBTs).

Owners of 119 SBTs positive for the L2HGDH genetic mutation were reviewed retrospectively. Inclusion criteria were the availability of data, with stiffness of all four limbs the most common (24/26) and earliest recognized abnormality. The mean age of onset of CS was 12 months (range 2.5-60). Gait dysfunction was reported in all 26 dogs with available data, with stiffness of all four limbs the most common (24/26) and earliest recognized abnormality. The mean age of onset of CS was 12 months (range 2.5-60). Gait dysfunction was reported in all 26 dogs with available data, with stiffness of all four limbs the most common (24/26) and earliest recognized abnormality. The mean age of onset of CS was 12 months (range 2.5-60). Gait dysfunction was reported in all 26 dogs with available data, with stiffness of all four limbs the most common (24/26) and earliest recognized abnormality. The mean age of onset of CS was 12 months (range 2.5-60). Gait dysfunction was reported in all 26 dogs with available data, with stiffness of all four limbs the most common (24/26) and earliest recognized abnormality.

The mean age of onset of CS was 12 months (range 2.5-60). Gait dysfunction was reported in all 26 dogs with available data, with stiffness of all four limbs the most common (24/26) and earliest recognized abnormality. The mean age of onset of CS was 12 months (range 2.5-60). Gait dysfunction was reported in all 26 dogs with available data, with stiffness of all four limbs the most common (24/26) and earliest recognized abnormality. The mean age of onset of CS was 12 months (range 2.5-60). Gait dysfunction was reported in all 26 dogs with available data, with stiffness of all four limbs the most common (24/26) and earliest recognized abnormality. The mean age of onset of CS was 12 months (range 2.5-60). Gait dysfunction was reported in all 26 dogs with available data, with stiffness of all four limbs the most common (24/26) and earliest recognized abnormality.
Two littermates Chiuhua dogs (one male, one female) were investigated for progressive blindness, ataxia and cognitive impairment with an onset from around one year of age. Magnets were used to elicit anaesthesia and marked generalised brain atrophy suggestive of a neurodegenerative disorder. Due to worsening of the signs, both dogs were euthanized before the age of two years. Brain histopathological examination demonstrated abundant neuronal accumulation of autofluorescent intracytoplasmic storage material characteristic of Neuronal Ceroid Lipofuscinosis (NCL), with a lamellar ultrastructure. Following ethical approval, DNA was collected from both affected dogs and five related dogs including the sire and dam. Whole genome sequencing was performed on the affected dogs. Sequence alignment and variant calling was done against the canine reference genome. Homozygous variants were found in the coding or splicing regions of four genes previously identified as causing NCL (ARSG, CLN2-TPP1, CLN6, CLN7-MSDOB). Segregation analysis by Sanger sequencing characterised MSDOB:c.843delT - predicted to cause a truncated protein - as the causal mutation.

Although NCL has previously been reported in Chiuhua dogs on three different occasions, the causal mutation had remained unknown. In a recent report, the identical mutation uncovered in this study was strongly suggested to be the causative variant in a single case in the Chinese Crested dog, suggesting a genetic relationship between both breeds. Considering the strong similarity in the clinical presentation and histological changes between humans with CLN7 disease and the two dogs presented here, Chiuhua could potentially be used as a large animal model of the human disease.

LAFORA DISEASE IN A BEAGLE
I. Hajek, V. Simenová, P. Wang, S.F.M. Bhati', B.A. Moharani, V. Palus'. Centre of Veterinary Medicine, Bratislava, Slovakia, 2Small Animal Clinic, Faculty of Veterinary Medicine, University College London, Gower Street, London, WC1E 6BT, UK.

This case report describes the mutation in a clinically diagnosed Lafora disease, also called progressive myoclonic epilepsy in a beagle dog breed. This case report suggests that the similar mutations can be present also in other breeds suffering of progressive myoclonic epilepsy. Preferred format: Poster presentation. Submitting author has finished his postgraduate study in neurology and currently is in neurology training under Dr. Viktor Palus, DVM, Dipl. ECVN, MRCVS.

| EXPRESSION OF EPIDERMAL GROWTH FACTOR RECEPTOR AND Ki-67 IN CANINE GLIOMAS |
| A. Fraser, B. Baccar, M. Ie Chevalier, S. Long. 'Translation Research and Animal Clinical Trials Study Group, 'Department of Anatomic Pathology, Faculty of Veterinary and Agricultural Sciences, The University of Melbourne, Werribee, Victoria, Australia. |

The histologic classification of gliomas is challenging. In people, accurate diagnosis is essential as the treatment and prognosis varies between tumour type and grade. Numerous biomarkers have been investigated in human gliomas to aid in their diagnosis and to act as potential therapeutic targets. To our knowledge, for evaluation in veterinary medicine investigation of biomarkers in canine gliomas is required. Epidermal growth factor receptor (EGFR) is associated with glioma grade and is a therapeutic target in human gliomas. The Ki-67 labelling index (LI) is a marker of proliferation which is a prognostic indicator in human gliomas. The objectives of the current study were to evaluate EGFR and Ki-67 expression immunohistochemically in canine gliomas and to determine if immunofluorescence is associated with histologic tumour type and grade. Formalin-fixed paraffin-embedded canine gliomas were assessed for EGFR, Ki-67 expression and histopathologic abnormalities. EGFR expression was evaluated using a semi-quantitative score and the Ki-67 LI was calculated. Thirty-one canine gliomas were evaluated. EGFR expression was identified in 16/31 (51.6%) tumours; expression was significantly greater in high grade tumours when compared to low grade tumours (P=0.042). EGFR expression was also significantly greater in glioblastomas. Ki-67 was expressed in 28/31 (90.3%) gliomas and the Ki-67 LI was significantly greater in the high grade tumours (P=0.024). A significant moderate correlation was identified between EGFR immunopositivity and Ki-67 LI (r=0.472, P=0.007).

While EGFR is expressed in approximately 50% of canine gliomas, investigation in to other therapeutic targets is required. EGFR is a suitable therapeutic target for glioblastomas cerebri.

IDENTIFICATION OF MOTOR PATTERNS AND ASSESSMENT OF PROGRESSIVE REFLEXES IN CANINE NEONATES
C. Morales, J. Fatjo, V. Aigües, P. Montoliu'. Neurorcat Veterinary, Barcelona, Spain, 'Chair Affinity Foundation Animals and Health, Department of Psychiatry and Forensic Medicine, Universitat Autònoma de Barcelona, Bellaterra, Barcelona, Spain, 'Department of Sanitat i Anatomia Animals, Universitat Autònoma de Barcelona, Bellaterra, Barcelona, Spain.

Progressive reflexes are brainstem-mediated, complex, automatic and stereotyped movement patterns that appear during development. They are usually present at birth and become more difficult to elicit with central nervous system maturation or are replaced by more complex postural responses. The early motor behaviour of the newborn is related to primitive reflexes. Descriptions of primitive reflexes in dogs are sparse, and their influence on neurological examination is poorly documented. The purpose of this study was to describe in detail the main primitive reflexes that can be assessed in canine neonates and how these motor patterns influence neurological examination. This is part of a larger study with the aim to develop a neurological examination procedure for neonatal dogs. This study obtained ethical approval.

Serial neurologic examinations were performed in 110 puppies during the first 3 months of life. Among all these反射s, detailed descriptions and preliminary analyses can be present also in other breeds suffering of comparable motor characteristics. A total of 13 primitive reflexes were evaluated. It was possible to elicit righting, withdrawal and crossed extension reflexes, walking reflex, hopping, and pelvic limb straightening-supporting reflex from birth in most dogs, and uniform and reproducible responses were observed. For all these reflexes, detailed descriptions of initial posture, stimulus, supporting manoeuvres, and a gradation of normal responses were established. Influences of instinctive motor patterns and optimal behavioural states for each item were identified. This work established a basis for standardization of canine neonatal neurological examination.

PROGNOSTIC FACTORS FOR ONE WEEK SURVIVAL IN DOGS DIAGNOSED WITH MENINGOCOELITIS OF UNKNOWNT AETIOLOGY
I. Cornelis', R. Ducatelle'. 1Dept. of Pathology, Bacteriology and Avian Medicine and 'Dept. of Medicine and Clinical Biology of Veterinary Animals, Faculty of Veterinary Medicine, Ghent University, Belgium. 'Clinical Science and Services, The Royal Veterinary College, Hatfield, United Kingdom.

Medical records were reviewed for dogs diagnosed with MUA between 2004 and 2015. Previously described inclusion criteria were used, and for all dogs 7-day survival was required. A poor outcome was defined as death.

One hundred and sixteen dogs met the inclusion criteria. Thirty-two (38%) dogs died within 7 days after making a presumptive diagnosis of MUA. Dogs were typically treated with steroids and/or corticosteroids. Both the presence of seizures and the presence of cluster seizures were significantly associated with a poor outcome. Age, sex, neuter status and location(s), total protein concentration, central venous blood and lactate cell count in cerebrospinal fluid and lactate levels on venous blood analysis at time of diagnosis were significantly associated with poor outcome. Age, sex, neuter status and location(s), total protein concentration, cerebrospinal fluid and lactate cell count were not associated with 7-day survival.

Every neuron needs to be able to detect and respond to electrical stimuli in order to help the nervous system to function properly. Anticonvulsant hypersensitivity syndrome in dogs is a condition that occurs when a dog is sensitised to an antibiotic and develops an allergic reaction to it. This can happen with a variety of antibiotics, including penicillin, tetracycline, and ciprofloxacin. Anticonvulsant hypersensitivity syndrome is a condition that can cause the dog to experience seizures, tremors, and other neurological symptoms.

Animal disease and veterinary medicine experts have been working on developing effective treatments for anticonvulsant hypersensitivity syndrome in dogs. One promising approach is to use immunotherapy with the antigen from the antibiotic that caused the hypersensitivity reaction. This can help the dog to desensitise to the antibiotic and prevent future reactions.

However, the development of effective treatments for anticonvulsant hypersensitivity syndrome in dogs is still ongoing. More research and clinical trials are needed to better understand the underlying mechanisms of the condition and to develop safer and more effective treatments. In the meantime, it is important to be aware of the potential for anticonvulsant hypersensitivity syndrome in dogs and to take steps to prevent it from occurring, such as using appropriate antibiotic dosing and monitoring the dog for signs of a reaction.

The prevalence of anticonvulsant hypersensitivity syndrome in dogs varies depending on the region and the breed. In some areas, it is more common in certain breeds, such as the Beagle. However, any dog can develop anticonvulsant hypersensitivity syndrome, regardless of breed or age. It is important to be aware of the potential for anticonvulsant hypersensitivity syndrome in all dogs on antibiotic treatment.

In conclusion, the first report of an EPM2B mutation causing ncl is a large animal model of the human disease.
This is the first case report of suspected aHSS after phe- notobarbitial treatment in a dog and the first description of the pathological findings. Clinicians should be aware of this serious and possibly fatal complication. With aggres- sive and early treatment, chances of survival are good in human patients.

POST-OPERATIVE SYMPTOMATIC PNEUMORRACHIS IN A DOG WITH A THORACOLUMBAR INTERVERTEBRAL DISC EXTRUSION

I. Cornelis1, P. Monticelli2, S. De Decker1. Clinical Science and Services, The Royal Veterinary College, Hatfield, United Kingdom.

Although pneumorrhachis, the presence of air in the vertebral canal, is extensively described in human medi- cine, it has been described only twice in veterinary lit- erature.

A 6-year-old crossbreed presented with acute, progres- sive ambulatory paraparesis localized to the T3-L3 spinal cord segments. MRI revealed an L1-L2 intervertebral disc extrusion, which was removed by a right-sided T3-L2 hemilaminectomy. Surgery and recovery from anesth- esia were uneventful. One day after surgery, the dog demonstrated slight deterioration, being non-ambula- tory paraparetic. The dog however became paraplegic with intact nociception and marked thoracolumbar hypalgesia 9 hours after surgery. A CT scan of the thoracolumbar vertebral column revealed the presence of a sphenical, gas filled structure at the level of T3. The structure had a maximum diameter of 4.8mm, filling up approximately 50% of the vertebral canal and was as- sociated with marked spinal cord compression. A hyper- dense lesion possibly surrounding the gas bubble was concurrently identified, so the dog was presumptively diagnosed with an extradural gas bubble and hema- toma, causing marked spinal cord compression. Revision surgery confirmed the presence of a hematoma, which was removed. Biopsies of spinal and parietal muscle were examined histologically at the epicenter and remote from the epicenter. Endothelin-1 (ET-1) expression was examined immunohistochemically and by in situ hybridisation. Using statistical analysis, we searched for associations between the expression of ET-1 and the severity of intradural haemorrhage or the extension of myelomalacia. Endothelin-1 was mainly expressed by astrocytes, mac- rophages and neurons and only rarely by endothelial cells in both control and affected dogs. In astrocytes at the epicenter, ET-1 expression was significantly higher in affected dogs than in control dogs irrespective of the severity of haemorrhage or myelomalacia (P = 0.001, P = 0.001, respectively). ET-1 expression in neurons at the epicenter was lower than in control dogs (P = 0.004, P = 0.008, respectively), in both astrocytes and neurons, there was a higher ET-1 expression, which was particularly evident remote from the epicenter than in the epicenter itself. Our observation of enhanced ET-1 expression over mul- tiple spinal cord segments could suggest a spinal microangiopathy, with a pathological finding of ADMIv. However, because more quantitative tech- niques and larger case numbers are required to investigate this further.

EVALUATION OF CEREBROSPINAL FLUID BIOMARKERS IN PARALPYGIC DOGS WITH INTERVERTEBRAL DISC HERNIATION

S.J. Wicha1, R. Carlson; A. Tipold; V.M. Stein, Dept. of Small Animal Medicine and Surgery, University of Veterinary Medicine Hannover, Hannover, Germany.

The definition of a reliable prognosis is challenging in paralpygic dogs with intervertebral disc herniation (IVDH). Early postoperative neurological deterioration in dogs undergoing decom- pressive spinal surgery. Surgical revision might result in a good outcome. This is the first case report of post- operative management for pneumorrhachis after thoracolumbar hemi- laminectomy in a dog.

ENDOTHELIN-1 EXPRESSION IN A CANINE MODEL OF SPINAL CORD INJURY

D. Mayer1, A. Devermann1, T. Seuberlich1, M. Van de Velde1, A. Casanova-Hakoyama1, S. Selimovic-Hanzes1, D. Henke,2 Division of Neurological Sciences, Department of Clinical Veterinary Medicine, “Department of Clinical Research and Veterinary Public Health, “Centre for Fish and Wildlife Health, Institute of Animal Pathology, Vetsuisse Faculty, University of Bern, Switzerland.

The pathophysiology of ascending / descending my- elomalacia (ADMM) following canine intervertebral disc (IVD) extrusion remains poorly understood. Vasoactive molecules may contribute to this pathophysiology. The aim of the study was to investigate the expression of endothelin-1 (ET-1) in the uninjured and injured canine spinal cord and its potential association with intramed- ullary haemorrhage and extension of myelomalacia. Spinal cord tissue of 11 normal control dogs and 34 dogs with IVD extrusion was examined histologically at the level of the extrusion (epicenter) and in segments remote from the epicenter. Endothelin-1 expression in most paralpygic dogs significantly exceeded that in control dogs. The ratio of ET-1 to ET-2 expression was significantly higher in dogs with grade V compared to the control group. CSF concentra- tions of tau protein, MIP3b and GFAP could not discrimi- nate between grade IV and V. Cisternal tau protein concentrations were significantly increased in dogs with IVDH and only cisternal tau protein concentrations seem to be correlated with the outcome of paralpygic dogs. The measurement of multiple biomarkers did not enhance outcome prediction. Paralpygic dogs had significantly (p<0.05) higher tau protein and MIP3b levels than the control group of which lumbar levels were also significantly higher compared to cisternal CSF samples. GFAP only showed significantly higher values in cisternal CSF in dogs with grade V compared to the control group. CSF concentra- tions of tau protein, MIP3b and GFAP could not discrimi- nate between grade IV and V. Cisternal tau protein values were significantly lower in dogs showing neurological improvement at least one grade within four weeks. In dogs not showing improvement, ET-1 and MIP3b values were significantly increased in dogs with IVDH and only cisternal tau protein concentrations seem to be correlated with the outcome of paralpygic dogs. The measurement of multiple biomarkers did not enhance outcome prediction.

BONE REMODELING AFTER CONSERVATIVE MANAGEMENT OF HYPOVITAMINOSIS A IN A AFRICAN LION

J. Siedenburg1, S. Wicha2, V. Mohar, P. Dziallas1, M. Shami2, V.M. Stein1, A. Tipold1, Department of Small Animal Medicine and Surgery, University of Veterinary Medicine Hannover, Hannover, Germany, Zoo Hannover, Hannover, Germany, Koret School of Veterinary Medicine, Hebrew University of Jerusalem, Jerusalem, Israel.

A four-month-old, intact, captive male African lion (Panthero leo) was presented with a history of mild ves- tibular signs. The lion was 20 months old when it was examined. To confirm the diagnosis of suspected hypovitaminosis A an associated occult bone malformation, computed tomography (CT) and magnetic resonance imaging (MRI) of the skull were performed. To obtain compa- rable ratios, CT and MRI based measurements were normalized with skull width and diameter of vertebral height. Tentorium cerebelli and occipital bone were significantly thicker and cerebellar herniation was evident on MRI. T2w midsagittal planes. The tentorium cerebelli/skull width ratio (TCSR) was 0.839, basiphenoid/skull width ratio (BBr) 0.067, occipital bone/vertebral height ratio (OBr) 0.5. A cervical intrameditullary T2w hyperintensity extending for at least two cervical vertebrae was visible. Conservative treatment consisted of intramuscular vitamin-A supplementation (2000 IU/kg/week for four weeks, 2000 IU/kg/14 days for five months, than 1000 IU/kg/14 days) and feeding of whole carcasses. After three months post initiation of the treatment only and very a slight ataxia was visible. In control MRI examinations the T2w declined to 0.0388, the BBr to 0.0462, and the OBr to 0.46. Though the tentorium cerebelli was minimally thickened, the cerebellum remained mildly herniated, cervical hyperintensities were no longer visible. Another five months later MRI ratios and pathologies in CT scans changed again (TCSR 0.346, BBr 0.0425, OBr 0.51). A subtle cerebellar herniation was still evident. In conclu- sion, vitamin A supplementation seems to ameliorate clinical signs and positively influence bone remodeling in young lions with hypovitaminosis A.

DISTAL POLYNEUROPATHY IN A BIRMAN CAT WITH TOXOPLASMOSIS

M. Rosati1, M. Leipig, K. Matiasek. Section of Clinical & Comparative Neuropathology, Ludwig-Maximilians University, Munich, Germany.

Lymphocytic invasion of predegenerate myofibers re- serves the hallmark of histopathological findings. However, a recent distribution and preceding treatment may preclude detection of specific infiltrates on microscopy. Thus im- munohistochemical markers have been used to increase sensitivity and specificity of biopsy studies. Among those MHC-II was evaluated in immune cells. Since my- ocytes also may act as antigen-presenting cells, their immunohistochemical markers have been used to increase sensitivity and specificity of biopsy studies. Among those MHC-II was evaluated in immune cells. Since my- ocytes also may act as antigen-presenting cells, their diagnostic value of myocytic MHC-II expression in the diagnosis of canine immune-mediated myositis (CIMM) has been evaluated.

CIMM diagnosis.

Diagnosis of Myocytic MHC-II Expression in the Diagnosis of Canine Immune-Mediated Myositis (CIMM)

M. Rosati, M. Leipig, K. Matiasek. Section of Clinical & Comparative Neuropathology, Ludwig-Maximilians University, Munich, Germany.

Lymphocytic invasion of predegenerate myofibers re- serves the hallmark of histopathological findings. However, a recent distribution and preceding treatment may preclude detection of specific infiltrates on microscopy. Thus im- munohistochemical markers have been used to increase sensitivity and specificity of biopsy studies. Among those MHC-II was evaluated in immune cells. Since my- ocytes also may act as antigen-presenting cells, their immunohistochemical markers have been used to increase sensitivity and specificity of biopsy studies. Among those MHC-II was evaluated in immune cells. Since my- ocytes also may act as antigen-presenting cells, their
evaluated immunohistologically for expression of CD3, CD8, CD20, IBA-1 and MHC-II in immune cells and myocytes. The intensity was scored (all marks) and sorted for subcellular distribution (MHC-II) after which group specific data were obtained according to standard algorithms. Myocytic MHC-II expression was significantly increased in CiMM if compared to the other diseases (p≤0.02), which only stained weakly positive in 35% of cases. There was a moderate correlation (0.52; p=0.01) between CD3 and MHC-II expression. With a cut off between scores 1 and 2, MHC-II reached 100% specificity and 75% sensitivity with 23/34 CiMM (67.6%) staining positive. The overall calculated accuracy was 85%. MHC-II expression was a valid diagnostic marker for CiMM that extends beyond inflammatory foci. Subthreshold MHC-II expression requires further investigation since normal treated Beagles or other dogs might have not reached cut-off threshold.

**MRI FINDINGS IN A CARBON MONOXIDE INTOXICATION IN 2 DOGS**

K. von Pückler1, MrI FINDINGs IN A CARBON MoNoxIDE evaluated immunohistologically for expression of Cd3, MHC-ii expression requires further investigation since that extends beyond inflammatory foci. subthreshold calculated accuracy was 83%.

A six years old female intact Jack russel Terrier was found in a burning house without consciousness. After emergence from the coma, the dog showed ataxia, circling and vocalisation. Magnetic resonance imaging was performed 13 days after admission. T2w, FLAIR and diffusion weighted imaging revealed symmetrical diffusion and homogenous hyperintensity of the caudate nu-
cleii. ADC mapping showed a hyperintensity of the cau-
date nuclei. Four weeks after admission the dog showed normal neurological deficits and was discharged. A second dog (4 years old neutered male crossbreed) was presented 2 weeks after smoke exposure. Initially the dog showed no abnormalities. At the presentation the dog was obtunded and disorientated, showing signs of cerebellar ataxia and proprioceptive deficits. T2w and FLAIR images revealed a heterogeneous hyperintensity of both caudate nuclei. Their signal was hypointense in T1. Generalised hyperintensity of the grey matter surrounding the cerebellar folia was visible in T2w and FLAIR sequences. Due to progressive deterioration of clinical signs the dog was euthanized 5 months later.

The MRI findings presented in this report are partially compatible with acute and chronic findings reported in human patients after carbon monoxide intoxication. Hypoperfusion in cerebellar pathways, hypoponsus and cerebellum in T2w and FLAIR images with restricted diffusion were major findings in the acute phase. In the chronic phase changes involve predominantly the white matter. Lesions were observed as bi-
llateral, diffuse hyperintense signal changes in T2w and FLAIR sequences with a restricted diffusion. Cerebellar dysfunc-
tion was observed in the acute and chronic phase.

**RESIDUAL DISC VOLUMES IN CHONDRODYS-
TROPHIC VERSUS NON-CHONDRODYS-
TROPIC DOGS WITH AND WITHOUT SPINAL DISEASE TREATED BY ASSISTED MINI-HENILAMINECTOMY**

F. Innerkofler1, K. Matiasek1, S. Medi1. Neurology Referral Service, Tierklinik Babenhausen, Germany. Clinical & Comparative Neuropathology, Ludwig-Maximilians University of Munich, Germany.

For years, hemilaminectomy (HM) has been the most ad-
vanced decortication technique for surgical therapy of intervertebral disc disease (IVD) in chondrodys-
trophic (CD) and non-chondrodystrophic dogs (NCD). However, a mini-hemilaminectomy (MH) very early has been shown to preserve better the stability of the vertebral column and to yield considerably more promising results than HM, in terms of both postoperative status and recovery time.

This monocentre trial evaluated the efficacy of volume reduction by MH in combination with power-fenestration or small cærectomy in CD and NCD suffering from thoracolumbar IVD. Medical charts of dogs with thora-
columbar IVD from 2010-2015 were reviewed. Inclusion criteria were (1) IVD documented by native-phase CT imaging pre and post surgery and (2) treatment via MH with small cærectomy or power fenestration. A total of 74 patients were included (CD: 59, NCD:15).

The mean FP was 27.9±11.2, mean MFP 10.3±5.1, mean MFP 48.5±19.8, mean MFP 18.9±9.1, and mean RDP was 31.2±18.5. The mean FP was 30.0±17.5, NCD: 31.8±22.7. Assisted MH comprises a tissue sparing decortica-
tion technique that proved successful in both CD and NCD. Likewise, Volume reduction assessed by RDP appears superior to previously published.

**CORRELATION OF TRANSCRANIAL MAGNETIC MOTOR EVOKED POTENTIALS AND MRI MORPHOMETRY IN DOGS WITH FUNCTIONAL LIMB PAIN ASSOCIATED TO PHOTON LIMB PAIN IN A CLIENT-OWNED DOG POPULA-
TION AFTER LIMB AMPUTATION**

M. Menchetti1, A. Gallucci2, G. della Rocca3, L. Matiasek3, K. Matiasek4, G. Gandini4, G. Gandini5, M. Rosati6. Section of Clinical & Comparative Neuropathology, Ludwig-Maximilians University, Munich, Germany; Department of Veterinary Medical Science, University of Bologna, Italy; Department of Veterinary Medicine, University of Perugia, Italy. 2Neurology Referral Service, Tierklinik Babenhausen, Germany; 3Clinical & Comparative Neuropathology, Ludwig-Maximilians University, Munich, Germany; 4Neurology Referral Service, Tierklinik Haar, Haar, Germany.

Phantom Limb Pain (PLP) refers to pain perceived in the area of an amputated limb. Despite the comparatively high rate of limb amputation in dogs, occurrence of PLP has not been studied in this species. We have assessed a client-owned population of dogs with limb amputation through an online survey aimed to document PLP preva-
ce, risk factors and owner’s perception of their pets’ quality of life (QOL) after amputation. The 75 questions survey evaluated reasons of amputation, pain before and after amputation, pain frequency (expressed as daily, weekly, monthly or yearly episodes) and QOL after amputation. Data were analysed with a Chi-squared test. 165 dog owners completed the survey. The main rea-
sons for amputation were cancer (59%) and trauma (30%). According to the baseline owners perception, pain ap-
ppeared similar before (81%) and after (86%) amputa-
tion (p=0.44). Over time, 66% of dogs experienced pain between 24 hours to 1 week, 20% between the second and fourth week, 9% between one and three months and 5% between three months and one year. Duration of preamputation pain correlated positively to the frequency of painful episodes after amputation (p=0.001). Despite 22% of owners were not satisfied with pain control, 86% of them did not regret the decision of amputation.

Post-amputation pain is a common problem in dogs af-
flicting 86% of patients and requiring appropriate treat-
ment. dog amputation was performed in 20 dogs. 20% of dogs showed pain at a later moment resembling PLP. Duration of preamputation pain is a risk factor associated with occurrence of PLP.

**VASULAR AND METABOLIC COMPROMISE OF THE LG DOUG'G DOGS WITH PAINFUL NERVE ROOT COMPRESSI0N**

M. Menchetti1, U. Foltz2, M. Rosati2, T. Gödde1, A. Blutke1, F. Steffen1, H. Volk1, T. Fleget1, R. Cappello1, M. Lowy4, G. Gandini1, K. Matiasek1, F. Gentilini2, G. Gandini2, M. Rosati1. 1Section of Clinical & Comparative Neuropathology, Ludwig-Maximilians University, Munich, Germany; 2Department of Veterinary Medical Science, University of Bologna, Italy; 3Neurology Referral Service, Tierklinik Babenhausen, Germany; 4Neurology Unit, Tierespital, Wetsuise Faculty, University of Zurich, Switzerland; 5Clinical & Science Services, Royal Veterinary College, UK; 6Section of Neurology, Department of Small Animal Medicine, University of Veterinary Medicine, Austria; 7North Downs Specialist Referrals, Bletchingley, UK; 8Davies Veterinary Specialists, Higham Gobion, Herts, UK.

Neuroformal Stenosis (NFS) and consequent nerve root compression has been shown to cause morpho-
logical changes of entrapped blood vessels and increase neuronal VEGF expression in entrapped dorsal root gan-
glia (DRG). These findings indicate adaptive changes to hyoxia and circulatory impairment. Reduced oxygen supply could furthermore stimulate a shift in neuronal metabolism, a valid ultimately influences neuronal function. To challenge this hypothesis we evaluated the expres-
sion of neuroglia (NGC), neurocanic anhydride IX (CA-IX), monocarboxyate transporter 1 (MCT-1) and -4 (MCT-4) in compressed canine DRG. Altogether, 15 L7-DRG were evaluated from nine dogs by lumbosacral NFS. Results were compared to age/breed-
matched, healthy control group.

All entrapped DRG showed significant neuronal positivi-
ty for NGb (p<0.0001), CA-IX (p<0.0001), MCT-1 (p<0.0001) and MCT-4 (p<0.0001). Substantial increase of endothe-
*logic changes of entrapped blood vessels and increase neuronal VEGF expression in entrapped dorsal root gan-
glia (DRG). These findings indicate adaptive changes to hyoxia and circulatory impairment. Reduced oxygen supply could furthermore stimulate a shift in neuronal metabolism, a valid ultimately influences neuronal function. To challenge this hypothesis we evaluated the expres-
sion of neuroglia (NGC), neurocanic anhydride IX (CA-IX), monocarboxyate transporter 1 (MCT-1) and -4 (MCT-4) in compressed canine DRG. Altogether, 15 L7-DRG were evaluated from nine dogs by lumbosacral NFS. Results were compared to age/breed-
matched, healthy control group.

All entrapped DRG showed significant neuronal positivi-
ty for NGb (p<0.0001), CA-IX (p<0.0001), MCT-1 (p<0.0001) and MCT-4 (p<0.0001). Substantial increase of endothe-
ly changes of entrapped blood vessels and increase neuronal VEGF expression in entrapped dorsal root gan-
glia (DRG). These findings indicate adaptive changes to hyoxia and circulatory impairment. Reduced oxygen supply could furthermore stimulate a shift in neuronal metabolism, a valid ultimately influences neuronal function. To challenge this hypothesis we evaluated the expres-
sion of neuroglia (NGC), neurocanic anhydride IX (CA-IX), monocarboxyate transporter 1 (MCT-1) and -4 (MCT-4) in compressed canine DRG. Altogether, 15 L7-DRG were evaluated from nine dogs by lumbosacral NFS. Results were compared to age/breed-
matched, healthy control group.
of satellite cells further indicates activation of scavenging mechanisms. These findings highlight significant neuropathological alterations in canine cervical idiopathic disc degeneration.}

**EVALUATION OF THE RISK FACTORS AND ABC1B GENOTYPE IN DOGS AFFECTED BY REFRACTORY IDIOPTIC EPILEPSY**

T. Gagliardo, E. Della Nave, F. Gentiletti, A. Galuzzi, M. Mernichietti, M. Turba, E. Bianchi, A. Cauduro, C. Di Cori, S. Giannì, M. Baroni, M. Bernardini, G. Gandini. 1Department of Veterinary Medical Sciences, University of Bologna, Italy; 2Department of Veterinary Medical Sciences, University of Parma, Italy; 3Neurovet Professional Association, Milan, Italy; 4Roma Sud Veterinary Clinic, Rome, Italy; 5Gran Sasso Veterinary Clinic, Teramo, Italy; 6Valdinevol Veterinary Hospital, Pistoia, Italy; 7Portoni Rossi Veterinary Hospital, Bologna, Italy.

The aim of this study was to evaluate the risk factors in a population of dogs affected by refractory idiopathic epilepsy (RIE) and assess the frequency of the ABC1B gene mutation (c.6-1807G>C) previously associated to phenobarbital-resistant idiopathic epilepsy in Border Collies (BCs). The multicentric study had a cross-sectional design. Among a population of idiopathic epileptic dogs, 52 dogs affected by RIE (defined as a condition in which two anti-epileptic drugs at adequate serum concentration did not achieve a decrease in seizure frequency ≥50%) were selected. Breed, sex, body weight, age, onset of seizures (<12 months; 12-24 months; >24 months), age at the onset of seizures (<12 months; 12-24 months; >24 months), and seizure type (cluster/single and generalized) were analyzed. The most represented breeds were mongrel (40%) and Beagle (7%). The aim of the study was to evaluate the impact of seizure-related factors on the occurrence of severe clinical signs. A significant reduction of sp expression was observed in the hippocampus sub-regions and its subcellular distribution. All hippocampal areas showed synaptic immunoreactivity in Hs compared to controls only in CA1. A significant reduction of sp expression was observed after SE in CA3 (p<0.05) and PHG (p<0.004). Decrease of SP expression in the hippocampus of epileptic cats in course of HS could be attributed to loss of synapses in course of HS. SE and prolonged excitatory activity might as well decrease SP expression through its synaptic depletion. Assessment of candidate neuropeptides for neuronal modulation is a preliminary step towards development of adjuvant therapies to assist conventional antiepileptic treatments.

**SEIZURES DO NOT MASK THE PATTERN OF PERVERSIVE CHANGES IN CATS WITH HYPERENCEPHALOPATHY**

S. Benaromin, E. Castei, E. Wagner, H.A. Volk, A. Fischer, K. Matiasek, S. Bertram, H.A. Flegel, K. Matiasek. 1Sections of Clinical & Comparative Neurorpathology and Neurology, Centre for Clinical Veterinary Medicine, Ludwig-Maximilians University, Munich, Germany; 2Neurology Referral Service, Tierklinik Haar, Haar, Germany; 3Clinical Science & Services, Royal Veterinary College, Hatfield, UK.

Systemic arterial hypertension impacts on the brain by maladaptive and pervasive effects that show a characteristic topography and therefore may allow for an imaging diagnosis of feine hypertensive encephalopathy (FHE). Neurological complications of FHE, on the other hand, include seizures in up to 30% of affected animals. It was the aim of this study to evaluate the impact of seizures on the spatial pattern of pervasive changes in order to differentiate target areas of hypertensive damage from those prone to seizure-related blood brain barrier breakdown (BBB) breakdown. This study enrolled brains of 31 cats, including 9 epileptics, with FHE identified through blood pressure increase, compatible primary lesions and target organ damage of brain vessels. Brain tissue was screened for actual and post-registration stages of oedema, perivascular microgliosis, parenchymal and vascular lesions. Apart from a significantly increased involvement of hippocampus, cerebral and cerebellar nuclei, e.g. by actual oedema and aquaporin-4 expression, there were no differences in between lesion maps or damage scores of epileptic and non-epileptic FHE cases (p>0.05). Brain oedema and perivascular gloss in FHE resemble blood pressure-induced vascular dysfunction rather than consequences of local excitotoxicity. The lesion pattern persists throughout epileptic and non-epileptic FHE courses and therefore an accurate imaging diagnosis of systemic hypertensive.

**ALTERED EXPRESSON OF AN EXCITATORY TACHYKININ NEUROPEPTIDE IN SEIZURE-ASSOCIATED HIPPOCAMPUS**

G. Turba2, E. Bianchi3, A. Cauduro4, D. Corlazzoli5, S. Gianni6, S. Bertram1,2, L. Matiasek3, M. Rosati1, E. Wagner7, A. Fischer8, L. Matiasek9, T. Fiegl9, K. Matiasek1. 1Section of Clinical & Comparative Neurorpathology, Ludwig-Maximilians University, Munich, Germany; 2Neurology Referral Service, Tierklinik Haar, Haar, Germany; 3Section of Neurorpathology, Ludwig-Maximilians University, Munich, Germany; 4Neurology Referral Service, Tierklinik Haar, Haar, Germany; 5Section of Neurorpathology, Ludwig-Maximilians University, Munich, Germany; 6Gran Sasso Veterinary Clinic, Teramo, Italy; 7Portoni Rossi Veterinary Hospital, Bologna, Italy; 8Tosco Vétérinaire Frégis, Arcueil France. AHT Landwades Park Kentford Newmarket Suffolk CB8 7TU, United Kingdom.

An association between the number of calcified intervertebral discs (IVDs) on survey radiographs and recurrence of clinical signs following surgery for thoracolumbar intervertebral disc extrusion (IVDE) in dogs has been previously described. MRI is now the imaging modality of choice in canine IVDE. The aim of this study is to examine the relationship between MRI-assessed IVD degeneration and recurrence of clinical signs in surgically treated dogs. Medical records and stenographic notes of 46 dogs, with IVDE laminectomy without fenestration for thoracolumbar IVDE at two centres (2010-2014) were reviewed. Recurrence was assessed by telephone follow-up to either referring vets or owners. Recurrence was recorded if thoracolumbar pain or neurological deficits occurred after a period of at least 6 months during which recovery was seen. Fifty-four dogs were included and MRI studies from T1 to L3 were assessed. The most common outcome in addition to the affected one in mid-sagittal T2WI images. In common with previous studies, most animals were Dachshund (43%) and mean age was 5.7 years (2.6-11.96% of extruded discs were between T11 and L2). Recurrence of clinical signs was seen in 13/34 (24%) of cases, and 100% of these cases had at least one other degenerative change seen on MRI. Twenty three dogs (24%) constituted the group 23/41 (56%) showed no other degenerate discs. There was a significant correlation between number of degenerate discs and recurrence. These results might be of value in predicting recurrence in dogs, assisting surgical decision making with regard to prophylactic laminectomy.

**OUTCOME AND COMPLICATION RATE IN CANINE CERVICAL DISK EXTRUSIONS TREATED EITHER WITH A VENTRAL SLOT OR A CERVICAL HEMILAMINECTOMY PROCEDURE**

D. Faissler, S. Samuels. Cummings School of Veterinary Medicine at Tufts University, Grafton, MA 01532, USA.

Cervical disk extrusions account for 15-25% of canine IVDD. The purpose of this study was to assess post-surgical complications, short and long-term outcome canine cervical IVDD treated with either a ventral slot (VSL) or cervical hemilaminectomy (CHL) procedure. Our hypothesis is that dogs undergoing VSL or CHL have a similar incidence of post-surgical complications and outcome, but a significantly higher incidence of recurrence at an adjacent disk space after a VLS procedure. Inclusion criteria were complete medical history, advanced diagnostic imaging and postsurgical follow-up of ≥2 years. Eighty seven dogs were enrolled in a VSL surgery and 26 dogs had a CHL procedure. There was no difference in breed, weight, age, sex, onset, and clinical presentation, and degree of spinal cord compression between the two groups. Dogs undergoing CHL had a lateral or foraminal extrusion more frequently. Surgery time, days on a fentanyl CRi and hospitalization time were significantly shorter in group 2. The rate of adverse events assessed with sa Ves grades was similar in both groups (8.4% versus 7.7%). Both procedures had good return to normal function (80% versus 88%). Dogs in the VSL group had a higher incidence of postoperative pain than in the CHL group (21.7% versus 7.7%) with the ad- jacent disk space affected (13.3% versus 3.8%). The VSL procedure is faster, requires less post-operative pain control and hospitalization time. However, with the VSL procedure patients are at a higher risk of recurrence, especially at the adjacent disk.
Patients were enrolled from the population referred to the Neurology and Neurosurgery service at the Queen Mother Hospital for Animals, Royal Veterinary College, between 2005 and 2014. Inclusion was based upon previously published criteria. Cytosine arabinoside administration was typically by subcutaneous injection at 50mg/m² body surface area every 12 hours over a 48 hour period, initially, at three weekly intervals. Prednisolone was started at 1mg/kg every 12 hours and tapered based on clinical response. Outcome data were collected by regular re-examination (1x) at our dedicated cytosine arabinoside clinic and a standardised telephone questionnaire. 135 dogs were included in this study. Survival times ranged from 0 to 3630 days (mean 710 days, median 377.5 days, lower quartile 85 days, upper quartile 1104 days). None of the evaluated variables were significantly associated with prognosis. 9% of dogs demonstrated side effects associated with cytosine arabinoside, which included vomiting or diarrhea (5.2%), calcinosis cutis (1.5%), myelosupression (2.2%), and injection site infection (0.7%). Clients reported 76% of dogs showed one or more side effects associated with glucocorticoid use, these were reduced or alleviated through dose reduction in 98% of cases. Cytosine arabinoside can be safely used for the long-term management of MUA, and in dogs with a history of non-compliance, can significantly reduce the side effects associated with concomitant glucocorticoid use through dose reduction.

Epilepsy in the cat is a serious medical condition as in all species. To date there are no licensed treatments for feline epilepsy and no well-controlled clinical studies on the efficacy or safety of antiepileptic drugs in cats. The new antiepileptic drug imepitoin has demonstrated clinical efficacy in the treatment of human epilepsy and to reduce the stricture of generalised seizures due to idiopathic epilepsy. The aim of this randomised, controlled, blinded GLP study was to investigate the tolerance of imepitoin in cats with idiopathic epilepsy, and to determine the mean measurements for mixed and pure-breed cats at risk. In conclusion, reductions in cortisol levels and decrease of pain were observed between the t0 and t2.

In summary, in this laboratory study imepitoin was well tolerated in cats, even at very high doses. For definitive evidence on safety and efficacy, clinical trials in feline epilepsy patients are warranted. The studies described here were approved by the relevant authorities for protection of animals.
GENETIC EPILEPSY IN CANE CORSO AND DOGUE DE BORDEAUX

Catherine Escrou1, Pascale Quignon1, Emilie Menzer1, Solennine Corrard1, Catherine André2, Neurology, VetAgro Sup, Lyon Veterinary Campus, France. 1CNRs, UMR 6290, Genetic and development institute, Rennes, France.

Genetic factors are increasingly being identified as the underlying mechanism of breed specific type of epilepsy in dogs. Prevalence and phenotypic characteristics of epilepsy are well described in popular breeds like Belgian Shepherd, Australian Shepherd, Border Collie or Labrador. To date, no descriptions have been reported in molossoid breeds like Cane Corso and Dogue de Bordeaux.

In order to determine breed specific epilepsy presentation we asked breeders or owners to complete a questionnaire about epilepsy. We collected 25 epileptic Cane Corso and 5 epileptic Dogue de Bordeaux.

Cane Corso displayed severe epilepsy with very homogeneous presentation appearing during teen age (median 17.5 months) with no sex predisposition. All dogs presented generalized seizures and 91% have systematic clusters. 30% of dogs are euthanized or deceased from status epilepticus before 3 years. In 54% of dogs, seizure frequency didn’t decreased with anticonvulsants. Epilepsy in Dogue de Bordeaux was similar (generalized seizures and clusters for all dogs) but a juvenile form is observed in 4 dogs (first seizure before 4 months).

In Cane Corso, 17 dogs were related and pedigree analysis is in favor of recessive autosomal transmission. Whether this severe epilepsy is linked to the size of the dogs as previously described or to the specific underlying genetic factors remains questionable.

SEIZURES IN A COATI (NASUA NASUA) WITH CRANIOPHARYNGIOMA- A CASE REPORT

D. Farke1, M. Kolecka1, S. Kirsten2, L. Rydewski2, A. Schänzer3, M. J. Schmidt1. 1Clinic for Small Animals-Surgery, Justus Liebig University, Giessen Germany. 2Institute of Veterinary Pathology, Justus Liebig University, Giessen, Germany. 3Institute of Neuropathology, Justus Liebig University, Giessen, Germany.

A ten years old coati was presented with a 3 week history of generalized seizures, altered consciousness, circling and blindness. Abdominal and chest radiographs, complete blood cell count and biochemistry panel from the referring veterinarian revealed no abnormalities. Neurological examination showed a reduced menace response on both eyes. Magnetic-resonance-imaging showed an extra-axial, well demarcated mass lying in the middle cranial fossa, which had a mass effect on the brainstem, cerebellum, thalamus and corpus callosum. The mass was heterogeneous with a central hyperintensity in T2, T2FSE and FLAIR. In T1- weighted images a central hypointensity and a moderate contrast enhancement was visible. Differential diagnosis included makroadenoma/carcinoma of the pituitary gland, germinoma meningioma, lymphoma and craniopharyngioma. The animal was euthanized and a pathological examination was performed. Macroscopic examination revealed a pressure induced atrophy of the brainstem. Histological findings included satellitosis, mild mononuclear cell infiltration within the meninges and a mild vacuolization of the white matter adjacent to the mass. The mass itself was organized in nests of round to polygonal cells with eosinophilic cytoplasm. Few mitoses were visible and within the stroma areas of necrosis and calcification were visible around one big central necrosis. Immunohistochemistry showed that tumour cells strongly expressed epithelial marker, and ß-catenin (NSE) and showed a low mitotic activity (MiB-1) <5%. The histology is consistent with a low grade tumor of epithelial differentiation and in combination with the radiological findings a craniopharyngioma of papillary type was diagnosed.

Immunohistological stains were positive for vimentin, cytokeratine, NSE within the stroma and the mass and synaptophysine. They were negative for GFAP, S100 and NF. So the histopathological examination considered a pituitary gland neoplasia most likely.