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Editorial note

Reviewers: the backbone of an excellent peer-reviewed journal

It is traditional at the end of each calendar year, in the final issue of the journal for the year, to acknowledge the support of the many colleagues who have acted as honorary reviewers of manuscripts during the past 12 months. They bring a commitment and enthusiasm to what is a time-consuming and sometimes challenging role, but nevertheless one that is vital for the success of any peer-reviewed publication. Their insightful comments help authors, especially those embarking on research or writing for the first time, to hone their skills and to craft a more readable and scientifically sound manuscript. Occasionally the process results in a paper being rejected for publication, often with a recommendation to the authors to rework the data, rewrite the manuscript, or undertake further research, before resubmitting it for peer-review once more. The care and dedication of our reviewers in these regards are greatly appreciated. They make the task of the editor much more straight-forward!

An editor’s special thanks also to Monika Cole, Liliane Cabral and Anne Jackson in the office, and to the folk at Southern Design, especially Jessica Bloor, for their helpful advice and assistance, as I have settled into the new role.

Professor Emeritus Bruce Parry, AVP Editor, December 2014

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- Gary Wilson, University of Queensland
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- Guy Yates, Centre for Animal Referral and Emergency
Outcomes of mechanical ventilation in 302 dogs and cats in Australia (2005-2013)
- pages 698-703
Trigg et al look at the ‘whys and wherefores’ of positive-pressure ventilation in canine and feline patients. They review the medical records of more than 250 dogs and 40 cats. Their study shows higher survival rates than previously reported. Cases of snake envenomation and tick paralysis were those most commonly treated in this way, with good survival rates. The take-home message is to use mechanical ventilation early, when the patient begins to show evidence of unsustainable respiratory effort for the best chance of survival. An interesting study, contrasting the situation in Australia with that overseas.

Clinical features of Mainland tiger and Eastern brown snake envenomation in dogs and cats in Melbourne
- pages 704-712
Indrawirawan, Sheridan and McAlees have conducted a retrospective study of snakebite in 104 dogs and 45 cats seen at an emergency centre in the outer suburbs. They document the common clinical signs, treatment and outcomes of these cases. Although an in vitro coagulopathy was often noted, it did not usually result in clinical signs of haemorrhage. With appropriate treatment, survival rates were remarkably good. It’s a study that is applicable well beyond the outskirts of Melbourne!

Concurrent Sertoli-Leydig cell ovarian tumour and Gartner’s duct cyst in an 8-year-old bitch
- pages 713-718
It begins as another case of generalised hyperpigmentation, hyperkeratoses and non-pruritic alopecia in a Staffy-cross. Doesn’t sound all that remarkable? The diagnostic trail that Wasik, Bacci and Witham get to follow is an unusual one, resulting in another ‘first’ for the AVP! Don’t remember your embryology? Not sure about a Gartner’s duct cyst? Never fear, the authors will lead you gently through the case.

Water-clear cell hyperplasia of the parathyroid gland in a dog
- pages 719-722
Hyperparathyroidism is not a common diagnosis, and Range et al document a hitherto unreported cause of this disorder in dogs. If you know about parathyroid “water-clear cells” you might not be surprised that they can become hyperplastic and hyperfunctional, but if you did not know of them, this article will be of interest to you (and likely a nostalgic journey down the path of endocrine-histopathology).

Angular limb deformity associated with injury to the metacarpal physes in a dog
- pages 723-727
This paper is one for the surgeon in you. Wilson and Smith describe the diagnosis and correction of an angular deformity of the distal left forelimb in a puppy. Surgery on the metacarpal bones involved wedge osteotomies, a mid-diaphyseal ostectomy on metacarpal II, stabilisation using a veterinary cuttable plate and bone screws and external coaptation. All for an elegant outcome.

Pneumorrhachis associated with hemivertebrae in a French Bulldog
- pages 728-732
Hall, Deruddere and Snelling describe an unusual cause of hind limb paresis in a dog. It was associated with spinal cord compression which resulted, not so much from the dog’s congenital hemivertebrae, but more from spontaneous pneumorrhachis. What’s that? How do you diagnose it? What can be done to treat it? Have a read of the article to find out!

What is your diagnosis?
- pages 735-736
Moller and Mills challenge your microscopic memory, with a feline blood smear that contains some unusual cells. What are they and what do they mean?
Outcomes of mechanical ventilation in 302 dogs and cats in Australia (2005-2013)

Trigg NL,a Leister E,b Whitney J,c McAlees TP
a Animal Accident and Emergency, 72 Hargrave Ave, Essendon Fields, Victoria 3041, Australia
b Veterinary Specialist Services, PO Box 1145, Springwood, Queensland 4127, Australia
c University Veterinary Teaching Hospital, The University of Sydney, NSW 2006, Australia
* Corresponding author: nicole.trigg@animalemergency.com.au

ABSTRACT
Aim: To determine the indications for, duration of, complications, duration of hospitalisation and outcomes in patients treated with positive-pressure ventilation (PPV) in Australia. Materials and Methods: Medical records of 302 patients (262 dogs and 40 cats) treated with PPV were reviewed for the indication for ventilation, duration of PPV, complications, duration of hospitalisation and outcome. Patients were assigned into one of four groups based on the indication for ventilation. Results: The present study showed higher survival rates for patients treated with PPV than previously reported. Patients ventilated for hypoventilation and unsustainable respiratory effort had the highest survival rates. Snake envenomation and tick paralysis were the most common underlying diseases in patients treated with PPV and had good survival rates. Conclusion: The indication for ventilation is associated with outcome. PPV should be instigated early when unsustainable respiratory effort is evident to give patients the best chance of survival. Aust Vet Pract 2014;44(4):698-703.

INTRODUCTION
Positive-pressure ventilation (PPV) is used to support respiratory function in veterinary critical care patients with a variety of underlying diseases including pulmonary parenchymal diseases, neuromuscular diseases, cervical spinal disease and intracranial disease.1-3 Reported survival rates are 20-90%, but frequently < 50%.1-4,9 Respiratory failure can be broadly divided into oxygenation failure, due to pulmonary parenchymal disease, or ventilatory failure due to neurological or muscular disease.2,4,9 In the largest veterinary PPV study to date, survival-to-discharge in patients ventilated for oxygenation failure was 22%, significantly lower than in patients ventilated for ventilatory failure where survival-to-discharge was 39%.2

Indications for mechanical ventilation are hypoxaemia which is unresponsive to oxygen therapy, hypoventilation and unsustainable respiratory effort where respiratory arrest is imminent.2,4,10,11 Hypoxaemia is defined as a partial pressure of arterial oxygen (PaO2) < 80 mmHg or a haemoglobin saturation (SPO2) of < 95%.10 Severe hypoxaemia is defined as a PaO2 < 60 mmHg or a SPO2 < 90%.10 Hypoventilation is defined as a reduction in alveolar ventilation leading to an increase in the partial pressure of carbon dioxide (PaCO2) > 50 mmHg.10,11 Severe hypoventilation is defined as a PaCO2 > 60 mmHg.10,11

With the exception of one recent study in tick paralysis patients,4 all studies on PPV in dogs and cats are from North America. These patients often have different underlying diseases to the patients commonly treated in Australia, particularly for patients ventilated for hyperventilation. Survival in these studies has generally been < 50% and these results have reinforced the perception that PPV is an expensive and invasive procedure that is unlikely to be beneficial in veterinary patients. Our hypothesis was that, due to the different underlying diseases in patients treated in Australia compared to those in North America, there would be higher survival and shorter duration of PPV in Australian patients.

MATERIAL AND METHODS
Patient records from Animal Accident and Emergency (46 patients, Melbourne, Victoria), Veterinary Specialist Services/Animal Emergency Service (239 patients, Brisbane/Gold Coast, Queensland) and the University Veterinary Teaching Hospital (17 patients, Sydney, New South Wales) were examined. The records of 322 patients that underwent mechanical ventilation from 2005-2013 were reviewed. The records were examined for indication for ventilation, duration of ventilation, complications of ventilation, duration of hospitalisation and outcome. Twenty patients were excluded from the study, due to incomplete medical records that resulted in an inability to determine the primary indication for ventilation (17 patients) or ventilation only being performed in the post-surgical period (3 patients). Patients that required ventilation only in the post-surgical period were excluded as they were ventilated to control their ventilation rather than for respiratory failure. Of the 302 cases included in the study, 262 were dogs and 40 were cats. Patients were divided into 4 categories based on the primary indication for ventilation:

- Group 1 (69 cases): Hypoxaemia, defined as SPO2 < 92%, PaO2 < 60 mmHg on room air, or PaO2 < 5 x Fraction of inspired oxygen (FiO2) when on supplemental oxygen.
- Group 2 (171 cases): Hypoventilation, defined as a PaCO2 > 60 mmHg, PaCO2 > 55 mmHg, or clinical examination findings consistent with hypoventilation (e.g. shallow respiration, minimal chest movement), respiratory fatigue or apnoea.
- Group 3 (47 cases): Hypoxaemia and hypoventilation (each defined as above).
- Group 4 (15 cases): Unsustainable respiratory effort, based on clinical assessment that respiratory effort was excessive and unsustainable or that respiratory arrest was imminent.

Patient outcome was classified as discharged, died, euthanasia due to poor prognosis, or euthanasia due to cost of treatment (‘cost-based euthanasia’). For some patients it was not possible to determine whether euthanasia was due to the cost of treatment or to poor prognosis, or to a combination of the two factors. For the purposes of analysis, such
patients were classified as euthanasia due to poor prognosis. For each group of patients, ‘cost-based’ euthanasia cases were excluded in the analysis of duration of PPV and duration of hospitalisation. Any complications noted in the patient’s medical history were recorded and the prevalence was compared to similar veterinary PPV studies. Hypothermia was defined as a temperature < 37.5°C and hypotension was defined as a systolic blood pressure < 180 mmHg and/or a mean arterial blood pressure < 120 mmHg. However, not all patients had a specific temperature or blood pressure value recorded in their medical history, therefore, patients with a notation of hypothermia or hypotension in their medical history were also included.

Survival-to-discharge was compared between the 4 different indications for ventilation using two-tailed, Fisher’s exact tests to determine statistical significance (GraphPad InStat 3, www.graphpad.com). Hypoxaemia was used as the reference indication to compare between the 4 groups to allow comparison with other veterinary ventilation studies that have also used hypoxaemia as the reference indication. Duration of PPV and hospitalisation of survivors and non-survivors in each patient group was compared using two-tailed, Mann-Whitney U tests to determine statistical significance (Statistics Open For All – SOFA, www.sofastatistics.com). A two-tailed, Fisher’s exact test was also used to compare survival between dogs and cats to determine statistical significance (GraphPad InStat3). For all comparisons, \( P \leq 0.05 \) was considered significant.

RESULTS

The underlying disease processes for each ventilation group are detailed in Table 1.

All indications for ventilation

A total of 302 patients, including 262 dogs and 40 cats, were included, with 160 (53%) of animals surviving. When cost-based euthanasia cases (n=21) were removed from the study, the survival rate rose to 57% (160/281). Overall, 52% (135/262) of dogs survived and when cost-based euthanasia cases (n=18) were excluded, survival increased to 55% (135/244). For cats, overall 63% (25/40) survived and when cost-based euthanasia cases (n=3) were excluded, survival increased to 68% (25/37). There was no significant difference in survival between dogs and cats (\( P = 0.21, \text{OR} = 1.68, 95\% \text{CI: 0.81 - 3.50} \)).

<table>
<thead>
<tr>
<th>Disease</th>
<th>All Groups</th>
<th>Group 1: Hypoxaemia</th>
<th>Group 2: Hypoventilation</th>
<th>Group 3: Hypoxaemia and hypoventilation</th>
<th>Group 4: Unsustainable respiratory effort</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute respiratory distress syndrome</td>
<td>7</td>
<td>6</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Aspiration pneumonia</td>
<td>33</td>
<td>8</td>
<td>17</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Central neurological disease</td>
<td>14</td>
<td>0</td>
<td>14</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Cardiac arrest</td>
<td>7</td>
<td>1</td>
<td>6</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>7</td>
<td>2</td>
<td>0</td>
<td>5</td>
<td>0</td>
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<tr>
<td>Diaphragmatic hernia</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Drowning</td>
<td>4</td>
<td>3</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Generalised paresis/collapse</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Heat stroke</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Immune-mediated haemolytic anaemia</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Neoplasia</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Neurological disease (no definitive diagnosis)</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Non-cardiogenic pulmonary oedema</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>4</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Pulmonary contusions</td>
<td>7</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Pulmonary disease (no definitive diagnosis)</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Pulmonary haemorrhage</td>
<td>2</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Respiratory arrest/fatigue</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Seizures</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Septic peritonitis</td>
<td>2</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Snake bite</td>
<td>42</td>
<td>2</td>
<td>39</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Cervical spinal disease</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Tick paralysis (with and without aspiration pneumonia)</td>
<td>164</td>
<td>35</td>
<td>89</td>
<td>26</td>
<td>14</td>
</tr>
<tr>
<td>Toxicities</td>
<td>11</td>
<td>1</td>
<td>10</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Trauma</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 1. Underlying disease processes for each indication for ventilation
The association between the indication for ventilation and the proportion surviving to discharge (excluding cost-based euthanasia cases) is shown in Table 2.

Group 1: Hypoxaemia

Sixty-nine patients were ventilated for hypoxaemia (66 dogs and 3 cats). When cost-based euthanasia cases (n = 4) were excluded overall survival was 42% (27/65 patients), while survival for dogs was 42% (26/62). One of the 3 cats in this group survived. Of the two cats that did not survive, one died during ventilation and the other was euthanased due to poor prognosis for recovery.

Group 2: Hypoventilation

A total of 141 dogs and 30 cats were ventilated because of hypoventilation. This included 10 dogs and 3 cats in the cost-based euthanasia category. When those cases were excluded, 69% of all animals survived (109/158), with a survival rate of 67% (88/131) for dogs and 78% (21/27) for cats. Survival in animals ventilated for hypoventilation was significantly improved compared to hypoxaemic patients (P = 0.0003).

Group 3: Patients with hypoxaemia and hypoventilation

This group contained 47 patients (42 dogs and 5 cats), with only one dog in the cost-based euthanasia category. When the latter animal was excluded, 30% (14/46) of these cases survived, which comprised 32% (13/41) of dogs and 20% (1/5) of cats. There was no difference in survival-to-discharge between hypoxaemic patients and those that were both hypoxaemic and hypercapnic (P = 0.32, OR = 1.62, 95% CI 0.73-3.61).

Group 4: Unsustainable respiratory effort

This group included 15 patients (13 dogs and 2 cats), with an overall survival rate of 67% (10/15). Of the dogs, 69% (9/13) survived, three were cost-based euthanasia cases and one died after it was weaned from PPV. When cost-based euthanasia cases were excluded, 90% (9/10) of dogs survived. Of the 2 cats in this group, one survived and one was euthanased due to poor prognosis, after the development of ventilator-associated pneumonia and lung consolidation. Patients ventilated for unsustainable respiratory effort had significantly improved survival when compared to hypoxaemic patients (P = 0.011).

Median duration of PPV and hospitalisation (Table 3)

Hypoxaemic patients were mechanically ventilated for a median of 18 hours (range 1-136 hours). Duration of ventilation was not recorded in one patient. The median duration of hospitalisation was 75.5 hours (range 3-262 hours). Duration of hospitalisation was not recorded in one patient. Survivors were ventilated for a significantly longer duration (median of 27 hours) than non-survivors (median of 14 hours) (P < 0.001), and had a significantly longer duration of hospitalisation (P < 0.001).

Patients ventilated for hypoventilation had a median duration of ventilation of 20 hours (range 1-168 hours) and median duration of hospitalisation of 112 hours (range 3-600 hours). Duration of ventilation was not recorded in one patient. Duration of hospitalisation was not recorded in one patient. Survivors were ventilated for a significantly longer duration (median of 22 hours) than non-survivors

### Table 2. Association between indication for ventilation and proportion surviving to discharge (excluding cost-based euthanasia cases)

<table>
<thead>
<tr>
<th>Indication for ventilation (Group)</th>
<th>Number of patients in group</th>
<th>Number of patients that survived</th>
<th>P value</th>
<th>Number of patients in group</th>
<th>Number of patients that survived</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxaemia</td>
<td>65</td>
<td>27 (42%)</td>
<td>Reference</td>
<td>158</td>
<td>109 (69%)</td>
<td>0.0003</td>
</tr>
<tr>
<td>Hypoventilation</td>
<td>158</td>
<td>109 (69%)</td>
<td></td>
<td>46</td>
<td>14 (30%)</td>
<td>0.32</td>
</tr>
<tr>
<td>Hypoxaemia and hypoventilation</td>
<td>46</td>
<td>14 (30%)</td>
<td></td>
<td>12</td>
<td>10 (83%)</td>
<td>0.011</td>
</tr>
<tr>
<td>Unsustainable respiratory effort</td>
<td>281</td>
<td>160 (57%)</td>
<td>NA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total number of patients</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 3. Duration of positive-pressure ventilation (PPV) and hospitalisation in survivors and non-survivors, after cost-based euthanasia cases were excluded

<table>
<thead>
<tr>
<th>Indication for ventilation</th>
<th>Duration of PPV</th>
<th>Duration of hospitalisation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Median (range)</td>
<td>Median (range)</td>
</tr>
<tr>
<td></td>
<td>in hours</td>
<td>in hours</td>
</tr>
<tr>
<td></td>
<td>All</td>
<td>Survivors</td>
</tr>
<tr>
<td>Hypoxaemia</td>
<td>18 (1-136)</td>
<td>27 (1-136)</td>
</tr>
<tr>
<td></td>
<td>n = 27</td>
<td>n = 10</td>
</tr>
<tr>
<td>Hypoventilation</td>
<td>20 (1-168)</td>
<td>22 (1-152)</td>
</tr>
<tr>
<td></td>
<td>n = 109</td>
<td>n = 48</td>
</tr>
<tr>
<td>Hypoxaemia and hypoventilation</td>
<td>11.5 (1-132)</td>
<td>22.5 (1-132)</td>
</tr>
<tr>
<td></td>
<td>n = 14</td>
<td>n = 32</td>
</tr>
<tr>
<td>Unsustainable respiratory effort</td>
<td>21 (3-108)</td>
<td>21 (3-108)</td>
</tr>
<tr>
<td></td>
<td>n = 10</td>
<td>n = 2</td>
</tr>
</tbody>
</table>

NA# Not applicable, two patients in this group with hospitalisation time known for one (28 hours)

n = number of patients

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(median of 16 hours) (P = 0.016), and had a significantly longer duration of hospitalisation (P < 0.0010).

Patients with both hypoxaemia and hypercapnia had a median duration of ventilation of 11.5 hours (range 1-132 hours) and median duration of hospitalisation of 49 hours (range 3-408 hours). Survivors had a median duration of PPV of 22.5 hours and non-survivors had a median duration of PPV of 6.5 hours. Survivors were ventilated for a significantly longer duration than non-survivors (P = 0.016) and had a significantly longer duration of hospitalisation (P < 0.001).

Patients with unsustainable respiratory effort had a median duration of ventilation of 21 hours (range 3-108 hours) and median duration of hospitalisation of 96 hours (range 14-237 hours). Duration of hospitalisation was not recorded in one patient. There was no difference in duration of PPV between survivors and non-survivors (P = 0.75).

**Underlying Disease**

Tick paralysis (Ixodes holocyclus) and elapid snake envenomation were the two most common reasons for PPV. Of the 164 patients ventilated for lower motor neurone disease secondary to tick paralysis, 104 (63%) survived, which increased to 71% (104/147 patients) when cost-based euthanasia cases (n = 17) were excluded.

Of the 103 patients that were ventilated for hyperventilation or unsustainable respiratory effort, 73 (71%) survived. This increased to 80% (73/91) when cost-based euthanasia cases (n = 12) were excluded. By comparison, tick paralysis patients ventilated for hypoxaemia or both hypoxaemia and hypercapnia, had survival rates of 61% (19/31) and 48% (12/25) respectively when cost-based euthanasia cases were excluded.

When cost-based euthanasia cases were excluded, duration of PPV for patients with tick paralysis ranged from 1-152 hours (median 24 hours) and duration of hospitalisation ranged from 3-408 hours (median 96 hours), in the 163 patients for which hospitalisation time was known. There was no significant difference (P = 0.082) in median duration of PPV between survivors (median 24 hours, range 3-152 hours) and non-survivors (median 24 hours, range 1-96 hours). Duration of hospitalisation was significantly longer (P < 0.001) in survivors (median 120 hours, range 24-408 hours) than non-survivors (median 36.5 hours, range 3-120 hours). Forty-two patients were ventilated after snake envenomation. The type of snake was identified in 27 cases: 24 being Pseudonaja textilis (Eastern Brown Snake), one Notechis scutatus (Tiger Snake), one Acanthophis antarcticus (Death Adder) and one Pseudechis porphyriacus (Red-bellied Black Snake). Thirty-two of 42 (76%) patients ventilated after snake envenomation survived. Survival increased to 82% (32/39), once cost-based euthanasia cases (n = 3) were excluded.

Forty patients were ventilated for hyperventilation or unsustainable effort, of which 31 (78%) survived. Survival increased to 84% (31/37), once cost-based euthanasia cases (n = 3) were excluded. The remaining 2 snake envenomation cases were ventilated for hypoxaemia, of which one patient survived and one was euthanased due to worsening lung function. Two cases of possible snake envenomation were excluded from this analysis because a definite diagnosis was not determined. One patient that was diagnosed with snake envenomation due to Red-bellied Black Snake envenomation and tick paralysis during the same period was also excluded from both the snake envenomation and tick paralysis analyses, as it was impossible to determine which was the primary underlying disease necessitating ventilation.

When cost-based euthanasia cases were excluded, the duration of PPV for snake envenomation patients ranged from 1-96 hours (median 20 hours) and the duration of hospitalisation ranged from 3-378 hours (median 120 hours). The median duration of PPV of survivors (22.5 hours) was significantly longer (P = 0.027) than non-survivors (12 hours). Survivors were hospitalised for a significantly longer duration than non-survivors (P < 0.001).

Complications during PPV included, aspiration pneumonia/ventilator associated pneumonia (38/302, 13%), corneal ulceration (20/302, 7%), hypotension (14/302, 5%), cardiac arrhythmias (12/302, 4%), hyperthermia (12/302, 4%), pneumothorax (10/302, 3%), pulmonary oedema (10/302, 3%), acute respiratory distress syndrome (9/302, 3%), acute kidney injury (7/302, 2%) and sepsis/SIRS (5/302, 2%).

**DISCUSSION**

The present study reports higher survival-to-discharge rates for patients treated with PPV than previous studies.1-4 This is likely due to the large proportion of patients ventilated for hyperventilation to lower motor neurone disease associated with snake or tick envenomation, but with normal lung function. In comparison, patients requiring PPV for hyperventilation in North America are more commonly patients with myasthenia gravis, polyradiculoneuritis, cervical spinal disease or central neurological disease. The latter diseases require prolonged PPV for days to weeks in many cases. Snake envenomation and tick paralysis patients usually show rapid improvement once the toxin has been neutralised, requiring shorter ventilatory support than patients with other lower motor neurone conditions.4

An attempt was made to assess the effect of cost-based euthanasia on survival rates of PPV patients. Many patients in the present study that were euthanased due to financial reasons were ventilated because of hyperventilation or unsustainable respiratory effort. When the latter patients were excluded from the analysis, cases with hyperventilation or unsustainable effort had a survival of 69% and 83% respectively. On this basis, it is reasonable to assume that a substantial proportion of the patients that were euthanased due to cost of treatment may have survived if there were no financial constraints. The latter patients were excluded from the analyses to give a more accurate medical prognosis for survival in patients treated with PPV, rather than the results being skewed by the negative effects of cost-based euthanasia.

Hypoxaemia and hypercapnia have wide-ranging deleterious effects on the body. Hypoxaemia results in generalised tissue hypoxia with the central nervous system and the myocardium being at greatest risk.12 In addition, hypoxaemia can also result in impaired renal function and pulmonary hypertension.12 Hypercapnia results in an increase in cerebral blood flow and increased intracranial pressure, potentially resulting in cerebral injury, a decrease in myocardial contractility and systemic vascular resistance, activation of the sympathetic nervous system and release of catecholamines.12,13 To prevent injury to the tissues, PPV should ideally be instigated prior to the development of hypoxaemia and hypercapnia, at which point unsustainable respiratory effort becomes the primary indication for ventilation.

Hypoxaemia results from pulmonary parenchymal disease, which interferes with respiratory gas exchange at the alveolar level.10 To achieve effective oxygenation and ventilation, patients with pulmonary parenchymal disease require more aggressive ventilator settings, including increased respiratory rates and higher mean airway pressures. These patients are thus at higher risk of ventilator-associated complications, such as ventilator-induced lung injury or pneumothorax due to repeated shearing injury, and subsequent pulmonary parenchymal inflammatory response associated with recruitment of collapsed alveoli. Hypercapnic patients have normal lung function but are unable to ventilate adequately. They usually
require less aggressive ventilator settings to maintain normocapnia and normoxaemia and so are at reduced risk of ventilator-associated complications.2,12 The goal of PPV is to achieve adequate oxygenation and ventilation with the least aggressive ventilator settings possible.

Primary hypoventilation is due to central neurological disease, lower motor neurone disease or respiratory muscle paralysis. In the present study, 171 patients with hypoventilation were treated, including 39 with snake envenomation and 89 with tick paralysis. The remaining 43 patients included 16 patients with neurological diseases such as meningitis, progressive paralysis and seizures. Thirteen of these patients died or were euthanased due to poor prognosis. The overall survival rate for this group was 69%, when cost-based euthanasia cases were excluded. In previous studies, patients ventilated for primary hypoventilation had survival-to-discharge rates of 0%,3 21.4%,4 39.0%,7 71.4%,7 and 90.5%.4 The study with a 90.5% survival rate involved patients ventilated due to lower motor neurone disease resulting from tick paralysis.4

Patients ventilated for hypercapnia have been consistently shown in human and veterinary studies to have a higher survival than patients ventilated for hypoxaemia.2-4,14 The survival rate of hypercapnic patients was significantly higher than hypoxaemic patients (P = 0.0003) in the present study. Secondary hypoventilation occurs as a consequence of respiratory fatigue and was shown to have a poor survival prognosis in the present study, with these patients included in the hypoxaemic and hypoventilation group of patients. Hypoxaemic and hypercapnic patients either had a clinically significant pulmonary disease and an important neuromuscular disease, or they were patients with hypoxaemia that had developed respiratory fatigue. This group contained patients with a high severity of illness and had the lowest survival-to-discharge (30%) of the 4 indications for ventilation, however, there was no significant difference in survival between patients with hypoxaemia and patients with both hypoxaemia and hypoventilation. These results are consistent with another large veterinary study that found similar results in this category of patients.2

This group had a shorter median duration of PPV and hospitalisation than the other groups in the present study, due to the high mortality rate. The majority of patients that did not survive either died or were euthanased in the first 12 hours of PPV, which skewed the overall median duration of PPV and hospitalisation to be shorter than for the remaining 3 indications for ventilation. Survivors in this group had a similar duration of PPV (median 22.5 hours) and hospitalisation (median 132 hours) as the other 3 groups, which had a median duration of PPV between 21 and 27 hours, and median hospitalisation between 101 and 128 hours. Non-survivors had a significantly shorter duration of PPV (median 6.5 hours) and hospitalisation (median 24.5 hours) than survivors in this group.

Fifteen patients were ventilated due to unsustainable respiratory effort, 14 patients with tick paralysis and one patient with snake envenomation. These patients were ventilated based on clinical assessment that their respiratory effort was excessive and unsustainable. These patients had the highest survival of all patient groups (83%) and a significantly higher survival rate than patients with hypoxaemia (P = 0.011). The clinical assessment of unsustainable respiratory effort can be a much earlier indicator than hypoxaemia or hypercapnia of the patient's deteriorating ventilatory function.3,15

Cats have been reported to have survival rates of 15-21% when treated with PPV for any indication,2,12 and were recently reported to have a survival rate of 83% in tick paralysis patients when cost-based euthanasia cases were excluded.17 In the present study, cats had an overall survival rate of 68% (25/37) when cost-based euthanasia cases were excluded. Of the 25 cats that survived, 13 were diagnosed with tick paralysis and all of these cats survived to discharge. This is consistent with the high survival in cats treated with PPV secondary to tick paralysis reported in a previous study. The remaining cats in the present study had various indications for ventilation and underlying diseases, but still had a survival rate of 50%. Furthermore, there was no significant difference in survival between cats and dogs (P = 0.21) indicating that cats can also be treated successfully with PPV.

Dogs have been reported to have a higher survival rate (21-71%) than cats when treated with PPV for any indication,2,3,5,9 and a survival rate of 74% in tick paralysis patients, when cost-based euthanasia cases were excluded.4 In the present study, dogs had an overall survival rate of 55% (135/244) when cost-based euthanasias were excluded.

The present study compared the duration of PPV in the 4 different indications for ventilation and also in snake envenomation and tick paralysis patients. There were significant differences in duration of PPV between survivors and non-survivors that were ventilated for hypoxaemia, hypoventilation, both hypoxaemia and hypercapnia and patients treated for snake envenomation. The non-survivors in these groups had a significantly shorter duration of PPV than survivors. There was no difference in duration of PPV between survivors and non-survivors that were ventilated for unsustainable respiratory effort, or treated for tick paralysis. As death or euthanasia generally occurred early in the course of treatment, surviving patients had a significantly longer duration of hospitalisation than non-survivors.

The majority of snake envenomation and tick paralysis patients seen in the present study required mechanical ventilation for hypoventilation or unsustainable respiratory effort, but had no clinical evidence of pulmonary pathology (Groups 2 and 4). Snake envenomation patients had an 84% survival rate, and tick paralysis patients had an 80% survival rate. These are encouraging statistics for the success of treatment of such patients with PPV. There were insufficient numbers of snake envenomation patients ventilated for hypoxaemia or both hypoxaemia and hypercapnia to be able to compare their survival to patients with hypoventilation or unsustainable respiratory effort. Tick paralysis patients ventilated for hypoxaemia or for both hypoxaemia and hypercapnia, had a diagnosis of aspiration pneumonia noted in their medical records, and for this reason showed a significantly lower survival rate of 61% and 48% respectively, when cost-based euthanasia cases were excluded. This is an important distinction and enables veterinarians to give the owners of tick paralysis patients, with and without aspiration pneumonia, a more accurate prognosis.

In the present study, there were no cases of aspiration pneumonia secondary to lower motor neurone disease in snake envenomation patients, however, aspiration pneumonia prior to PPV was a significant complication seen in tick paralysis patients. Bronchopneumonia, suspected to be secondary to aspiration, was reported in 15 of 25 tick paralysis patients that died or were euthanased in a recent study.16 Tick paralysis patients can develop respiratory failure from pulmonary dysfunction due to aspiration pneumonia or pulmonary oedema, resulting in hypoxaemia, or due to lower motor neurone signs resulting in hypoventilation. Both pulmonary disease and lower motor neurone signs contribute to the patient developing unsustainable respiratory effort.4,17 The high prevalence of aspiration pneumonia in tick paralysis patients in the present study resulted in a reduced survival rate compared to snake envenomation patients.

Although pneumonia was the most prevalent complication noted in the present study, most likely due to the large number of tick
paralysis patients included, where aspiration pneumonia is a common complication, the prevalence of pneumonia was lower than in previous studies. However, it is possible that the actual prevalence of pneumonia was under-diagnosed in the present study, as bacterial airway cultures and thoracic radiographs were not performed in many cases.

Corneal ulceration occurred in 7% of cases in the present study, which is consistent with a previous study. Tear production in critically ill dogs has been shown to be reduced, resulting in a higher risk of corneal ulceration. This highlights the importance of diligent nursing care to prevent corneal ulceration in both PPV and other critically ill veterinary patients.

The prevalence of pneumothorax in the present study was lower than in previous studies where pneumothorax was reported in 7-28% of patients. This may have been due to a larger number of patients with healthy lungs in the present study, as pneumothorax has been reported to occur more often in patients with pulmonary parenchymal disease. PPV is often perceived in veterinary practice to be a technically difficult, expensive and futile procedure, with little chance of survival. For this reason, it is often instigated only when the patient develops respiratory arrest due to fatigue and the prognosis at this point is grave. The latter patients (included in the hypoxaemic and hypercapneic group in the present study) had a much poorer survival than other patients in the present study and in another veterinary study. Given the higher survival in patients that are ventilated for unsustainable respiratory effort alone, prior to the development of hypercapnia and/or hypoxaemia, PPV should be instigated early in patients that are at risk of respiratory fatigue, rather than waiting for the patient to progress to respiratory arrest, when the prognosis for survival is poor. A proactive approach to early PPV is likely to result in higher ventilation success rates for all categories of patients. In addition, the results of the present study demonstrate that non-survivors tended to die in the early stages of PPV.

Often, patients that require PPV are considered to have a poor prognosis if they cannot be weaned off the ventilator in only a few hours. The present study, however, indicates that a patient that is stable at greater than 20 hours after the commencement of PPV is likely to survive and is not the ‘hopeless case’ that they are commonly considered.

CONCLUSION
The results of the present study show that PPV can be used very successfully in veterinary medicine to treat respiratory failure. Patients with unsustainable respiratory effort had the highest survival-to-discharge (83%) in the present study. As hypercapnia and hypoxaemia are later manifestations of respiratory failure, it is recommended to initiate PPV before their development, to give the patient the best chance of survival. Patients with snake envenomation and tick paralysis were the most common cases treated with PPV in the present study (in Australia) and these patients have a good prognosis for survival with such treatment.

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REFERENCES
Clinical features of Mainland tiger and Eastern brown snake envenomation in dogs and cats in Melbourne

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ABSTRACT
A retrospective study was conducted in cats and dogs with Mainland tiger snake (Notechis scutatus) and Eastern brown snake (Pseudonaja textillis) envenomation in Melbourne, Australia, to compare clinical signs and treatment and to determine if a coagulopathy is seen as frequently as in human envenomation cases.

Neurotoxicity was common in both tiger and brown snake envenomation. Myotoxicity was seen in 83% of dogs and 96% of cats with tiger snake envenomation. Prolonged coagulation tests were common in animals with tiger snake envenomation, but not in those with brown snake envenomation. Clinical bleeding was less common than laboratory evidence of a coagulopathy. Dogs were treated with a median of 2 vials and cats with a median of 1 vial of tiger/brown polyvalent antivenom. The overall proportion of cases presenting for snake envenomation that were euthanased for financial reasons was 12% for dogs and 13% for cats. Of the remaining cases, 95% of dogs and 97% of cats survived.

Due to the similarity of clinical signs in tiger and brown snake envenomation, it is not recommended that diagnosis and choice of antivenom be based on clinical signs alone. Aust Vet Pract 2014;44(4):704-712.

INTRODUCTION
There are 4 venomous snakes found in Victoria: Eastern tiger snake (Notechis scutatus), Eastern brown snake (Pseudonaja textillis), Red-bellied black snake (Pseudechis porphyraicus) and Copperhead snake (Austrelaps sp.). Of these, only tiger snakes and brown snakes are known to exist in the area involved in the present study, namely the northern and western suburbs of Melbourne.

Tiger snake venom is composed of various toxic compounds. The most important of these is notexin, a potent myotoxin and pre-synaptic phospholipase 2 (PLA2). Notexin causes marked local and generalised skeletal muscle myodegeneration in a dose-dependent manner, with dogs particularly sensitive to its effects. Notexin also acts presynaptically to reduce the amount of acetylcholine (ACh) available intracellularly, through mobilisation of vesicular ACh, resulting in significant neuromuscular paralytic effects.

Numerous other less potent pre-synaptic neurotoxic PLA2 enzymes such as scutatoxin and notechis are found in tiger snake venom. They enhance the paralytic effects by blocking ACh release in a more direct manner. Several reversible post-synaptic neurotoxins are also present in small amounts. Tiger snake venom also contains group D prothrombin activators, which are similar to activated coagulation Factor X (FXa) and with activated coagulation Factor V (FVa), become effective procoagulants. The FXa-FVa (prothrombinase) complex leads to the formation of thrombin from prothrombin in the presence of calcium and phospholipids. Thrombin provides positive feedback in the coagulation cascade, resulting in activation of other coagulation factors (the thrombin burst), subsequent fibrin formation and almost complete consumption of Factor V, Factor VIII and fibrinogen. The latter consumption of coagulation factors results in the situation of venom-induced consumptive coagulopathy (VICC) that is seen in tiger snake envenomation in people.

Neurological and myolytic effects are the most commonly reported findings in dogs after tiger snake envenomation. Clinical signs of weakness, ataxia, dilated pupils, paralysis, salivation, tremors and vomiting are common. The procoagulant effect of tiger snake venom has been confirmed in previous studies by the histological presence of thrombi in the heart, lungs and kidney. The activation of the clotting cascade may lead to the coagulopathy which has been observed in clinical settings and in experimental studies with a large dose of venom. Marked prolongation in activated partial thromboplastin time (APTT) and prothrombin time (PT) have been reported in dogs, with some also showing increased fibrin degradation products (FDP) and hypofibrinogenaemia. Clinical coagulopathy is manifested as haematoma formation post-venepuncture, gingival petechiae and haemoptysis.

Common clinical signs of tiger snake envenomation in cats include depression or generalised weakness, hind limb ataxia, decreased to absent pupillary light reflex (PLR), mydriasis and vomiting. In contrast to dogs, cats show minimal to no prolongation in PT and APTT values and have not been documented to have increased FDP.

In a recent human study, tiger snake envenomation was characterised by VICC in almost all cases, systemic symptoms in 80%, neurotoxicity in 30.3% and myotoxicity in 19.6% of cases seen. VICC can be complete or partial. In complete VICC, there is undetectable fibrinogen and/or increased D-dimer concentration in citrated plasma and an International Normalised Ratio (INR) of > 3.0 and APTT that exceeds the upper limit of detection. In partial VICC, there is a low but detectable fibrinogen concentration and/or increased D-dimer and a maximum INR < 3, and prolonged APTT. (The INR is the ratio of the patient’s PT to mean normal PT and allows valid comparison of results regardless of which reagents are used at different laboratories.) Clinical signs of VICC in people include bleeding at the bite site or intravenous cannula site, bleeding of the gums and haematemesis. Major haemorrhage is not
noted in human patients with VICC.59 Thrombotic microangiopathy also occurs in patients with VICC.14,15 Myotoxicity is defined as an increased creatine kinase (CK) value > 1000 U/L.17 Clinical signs of myotoxicity include local muscle pain and tenderness, generalised myalgia and trismus.16

Eastern brown snake venom contains textilotoxin, pseudonaja toxin and group C prothrombin-like activators.3,10 Textilotoxin is a potent pre-synaptic PLA2 neuromyotoxin that blocks presynaptic neuromuscular transmission by disrupting ACh release.3 Pseudonaja toxin is a potent post-synaptic neurotoxin, which acts by binding to the ACh receptors.21 Group C prothrombin activators are similar to the FXa-FVa prothrombinase complex and act as effective procoagulants.7 In people, brown snake envenomation causes a VICC that is faster in onset than tiger snake envenomation,4 while no myotoxic effect is noted from brown snake envenomation.22,23

The clinical signs of brown snake envenomation in dogs and cats are not as well documented as those of tiger snake envenomation. Signs are thought to be similar in all species and include a lower motor neuron paralysis syndrome and a hypocoagulable state.24 Initial haemodynamic collapse, severe systemic hypotension, prolonged coagulation time and thrombocytopenia have also been documented in canine experimental studies.25-28 Prolonged coagulation time is noted in all dogs with experimental brown snake envenomation with all doses of venom studied.29 In dogs, signs most noted in clinical cases include weakness, ataxia, mydriasis, paralysis, salivation and tremors.30 Symptoms of brown snake envenomation in experimental studies in cats include weakness, depression, ataxia, tachypnoea, dyspnoea, flaccid paralysis, mydriasis and prolonged clotting time.31 Reported clinical signs in cats with suspected brown snake envenomation include mydriasis, hypothermia, flaccid paralysis and ataxia.32 Vomiting, diarrhoea and prolonged clotting times were not common in the latter case series.29

A recent survey of veterinary clinics in Australia noted that tiger and brown snake envenomation produces similar clinical syndromes with weakness, ataxia and paralysis as the most common signs reported.8 Pigmenturia (not distinguished as specifically haemoglobinuria or myoglobinuria) occurred significantly more often in tiger snake envenomations than in brown snake envenomations.30 The latter survey also noted that the snake venom detection kit (SVDK, CSL Limited, Victoria, Australia) was used infrequently, with clinical signs being used to determine which snake species might be responsible for the envenomation.9

A recent human study found that all patients with brown snake envenomation developed VICC, with 45% developing systemic symptoms and 2% developing a mild neurotoxicity.31 No myotoxicity was noted.31 VICC manifested as bleeding at an intravenous cannula site (32%), at the bite site (24%) and from the gums (14%).31 Major haemorrhage was observed in 4% of cases, gastrointestinal haemorrhage in 2% and intracranial haemorrhage in 1% of cases. Severe envenomation was characterized by collapse/hypotension, seizures, cardiac arrest, major haemorrhage, thrombosis, microangiopathy and death.31 Systemic symptoms included nausea, vomiting, headache, abdominal pain, diaphoresis and diarrhoea.31 Signs of neurotoxicity included ptosis, lateral gaze diplopia, bulbar weakness, dysphonia and facial weakness.31

Previous veterinary clinical studies have not reported the prevalence of coagulopathy in dogs or cats.2,9,11,13,32-34 The present study aimed to investigate the common clinical signs associated with tiger and brown snake envenomation in dogs and cats in Melbourne and to determine if coagulopathy is seen as frequently as in human cases. Treatment and mortality rate were also noted.

MATERIALS AND METHODS

Records from Animal Accident and Emergency (AAE), Melbourne, Victoria, were searched for cases of snake envenomation from April 2005 until March 2014. As the clinical software was unable to do a keyword search, invoice codes were instead used. The following codes were searched: tiger-brown antivenom, SVDK, activated coagulation time (ACT), APTT and CK. The clinical records were then examined and cases of snake envenomation were selected.

An animal was diagnosed with snake envenomation by the attending veterinarian based on one or more of the following criteria:

1. Clinical signs consistent with snake envenomation, which include acute collapse, vomiting, ptyalism, dyspnoea, hind limb paresis or ataxia, generalised weakness or recumbency, mydriasis, reduced PLR, reduced gag reflex and pigmenturia.
2. It was seen to interact with a snake
3. An increased CK concentration and/or prolonged APTT or ACT
4. A positive SVDK.

If an animal is presented with clinical signs consistent with snake envenomation and has increased CK and/or APTT values, it is clinic policy to not perform a snake venom detection test (SVDT) because of the likelihood that the hook effect would interfere with the test.33 (The hook effect occurs when there is a high concentration of venom causing the binding antibodies in the SVDK to be saturated to the point where a sandwich cannot be formed and the labeled conjugate is removed in the wash step, leading to a false negative result.) In such cases, tiger/brown polyvalent antivenom (TBAV) is administered and the bladder is emptied after administration of antivenom. A freshly formed urine sample is subsequently collected and the SVDT performed on the fresh urine. Further TBAV is administered only if the urine SVDT is then positive.

The following information was collected from each case: presenting clinical signs, diagnostics performed, whether a SVDT was performed and the result, the dose of antivenom administered, treatment given, clinical outcome and days of hospitalisation. The following blood test results were recorded if available: ACT (Becton Dickson ACT tube, USA), APTT (Coag Dx Analyzer, IDEXX Laboratories, Australia) and CK activity. Most CK tests were performed in-house (Catalyst Dx Chemistry Analyser, IDEXX Laboratories, Australia), while some were sent to a referral laboratory (IDEXX Laboratories, Australia). For those CK tests run in-house, when the result was above the maximum readable CK activity of 2036 U/L, the sample was diluted 1:10, increasing the maximum value to 22,396 U/L. Many CK results > 22,396 U/L were then sent to the referral laboratory for further dilution to obtain a result, while a few were simply recorded as > 22,396 U/L in the patient file. The latter were ascribed a value of 22,396 U/L for calculation of the median result for an envenomation group.

If more than one result was available for any blood test in the patient’s record, the first result was used. All blood results for data analysis were obtained within 24 hours of admission.

If the animal was referred to AAE, the presenting clinical signs and any diagnostic tests performed at the referring practice were used in the present study. If no diagnostics were performed at the referring clinic, the first ACT or APTT, CK and SVDK result after admission to AAE were used.

Animals were diagnosed with either tiger or brown snake envenomation on the basis of a SVDT, definitive snake identification by a professional snake handler, or examination of the snake’s scales as noted on the case record. Animals with a CK activity > 1000 U/L were categorised as suspected tiger snake envenomation. This value was chosen based
on the human definition of snake venom myotoxicity,\textsuperscript{17} as there is no veterinary definition of snake venom myotoxicity. Otherwise, the animal was categorised as an unknown snake envenomation.

The animal was considered to have a coagulopathy if the APTT or ACT was prolonged. Reference values for APTT in cats were < 119 s and in dogs < 102 s. Reference values for the ACT were < 110 s in cats and < 160 s in dogs. Both APTT and ACT results longer than 300 s were recorded as > 300 s in the patient file, but were ascribed a value of 300 s for calculation of the mean result for an envenomation group. Fibrinogen and D-dimer concentrations were not performed, so a diagnosis of VICC could not be made. However, based on previous animal and human studies,\textsuperscript{8,10,36} it was assumed that the coagulopathy was seen in 36 hours of snake envenomation was due to VICC and not to disseminated intravascular coagulation (DIC).

Treatment for snake envenomation in the present study included intravenous fluid therapy, antivenom administration, oxygen therapy, mechanical ventilation, opioid pain relief, packed red blood cells and fresh frozen plasma. Patients were hospitalised and treated until their CK value was < 5000 U/L.

In cases treated at AAE prior to 2008, adrenaline, chlorpheniramine, with or without dexamethasone were used as premedication before administration of antivenom. After 2008, chlorpheniramine was the only medication given before antivenom administration in cases treated at AAE.

The antivenom used in the present study was CSL polyvalent tiger/brown antivenom (Commonwealth Serum Laboratories, Australia) or Summerland tiger/multi-brown snake antivenin (Summerland Serums, Australia). The total number of vials of antivenom administered to the patients was recorded, including any administered by the referring veterinarian. The number of vials of antivenom that were administered was determined by the attending clinician, following clinic policy. At AAE, one TBA V is usually administered to animals with a diagnosis of snake envenomation, although two vials may be administered in animals presenting with severe neurological changes, dyspnoea or in respiratory arrest, or having laboratory evidence of coagulopathy. Subsequent TBA V are administered only if a freshly formed urine sample is positive on testing with a SVDK, or if the animal is deteriorating.

Animals that were euthanased due to financial reasons were excluded from the determination of mortality rate. Animals that died during treatment, or were euthanased due to poor prognosis, were included. If the reason for euthanasia was not able to be determined from the records, the animal was placed into the non-survivor group.

**RESULTS**

Based on the search parameters, over 500 records were reviewed and 149 animals comprising 104 dogs and 45 cats were included in the study. Thirty dogs (29%) and 10 cats (22%) were seen to interact with a snake. The latter included playing with, or being in proximity to a dead or live snake, or being struck by a snake. The bite site was found only in one dog (1%) and 3 cats (7%) and all were located on the face. Two of the 3 cats had facial swelling, associated with the bite site.

Clinical findings, laboratory results, treatments and outcomes (including survival and euthanasia for financial reasons) are tabulated for dogs (Table 1) and cats (Table 2).

Most animals with a coagulopathy on the basis of laboratory results did not show clinical signs of bleeding. Of the 72 dogs with prolonged coagulation times, only 3 (4%) showed signs of clinical bleeding. In total, 5 dogs showed clinical signs of bleeding, 2 of which had ACT and/or APTT within reference limits. The signs of bleeding in dogs included bleeding from the gums (2 dogs), epistaxis (one dog), haemorrhagic diarrhoea (one dog) and haematemesis (one dog). All dogs with clinical bleeding survived. No cats showed clinical signs of bleeding.

Hypothermia (temperature < 35°C) was noted on presentation in 2 of 41 dogs (3%) with suspected tiger snake envenomation and in 4 of 41 dogs (10%) with unclassified snake envenomation. Of these 6 dogs, one was euthanased due to suspected intracranial haemorrhage or thrombosis causing obtundation that did not improve over 6 hours. The remaining 5 dogs survived. Hypothermia (temperature < 35°C) with generalised weakness was noted in one of 32 cats (3%) with suspected tiger snake envenomation and hypothermia only was noted in one of 32 cats (3%) with suspected tiger snake envenomation. Both cats survived.

There were 2 dogs in the tiger snake and 4 dogs in the suspected tiger snake envenomation groups with values recorded as > 22,396 U/L, while there was one cat in the tiger snake and 4 cats in the suspected tiger snake envenomation groups with values recorded as > 22,396 U/L. The CK values in the latter groups are therefore likely an underestimate of the median values. All other cases where the in-house result was originally > 22,396 U/L were sent to the referral laboratory, to obtain a final CK value. The median and range of CK values for each envenomation group for dogs and cats are given in Tables 1 and 2 respectively.

Dogs were given a median of 2 vials of TBA V for tiger snake envenomation. Of the 21 dogs in the tiger snake envenomation group, one dog was transferred to another facility for further treatment and one dog was euthanased without treatment. The remaining 19 dogs were treated with TBA V. Five dogs (26%) received one vial, 7 dogs (37%) received 2 vials and 7 dogs (37%) received 3 vials of TBA V. Cats were given a median of 1 vial of TBA V for tiger snake envenomation. Of the 4 cats in the tiger snake envenomation group, one cat was euthanased without treatment. Of the remaining 3 cats, 2 (67%) received one vial and one (33%) received 2 vials of TBA V.

The 3 dogs treated for brown snake envenomation were given a median of 2 vials of TBA V. Two dogs received 2 vials and one dog received 3 vials of TBA V. Both cats with brown snake envenomation received 1 vial of TBA V.

Of 104 dogs with snake envenomation, 7 were euthanased without treatment and one was transferred to another facility for further treatment. The remaining 96 dogs were treated with TBA V. Thirty dogs (31%) received 1 vial, 58 dogs (60%) received 2 vials and 8 dogs (8%) received 3 vials of TBA V; a median of 2 vials.

Of the 45 cats with snake envenomation, one cat was transferred to another facility and 3 cats were euthanased without treatment. Forty-one cats were treated for snake envenomation, with 5 (15%) receiving no antivenom, and 34 (83%) receiving 1 vial and one (2%) receiving 2 vials of TBA V; a median of 1 vial.

The average length of hospitalisation for cases of tiger snake envenomation was 3.6 days (range 1 to 11 days) in dogs and 3.0 days (range 1 to 7 days) in cats. The average length of hospitalisation for dogs with brown snake envenomation was 2.5 days (range 2 to 3 days). The cat with brown snake envenomation was hospitalised for one day.

Average length of hospitalisation for all cases of snake envenomation was 3.1 days (range 1 to 11 days) in dogs and 3.3 days (range 1 to 12 days) in cats.

The overall survival rate for cases that were treated was 95% in dogs and 97% in cats. The overall rate of euthanasia for financial reasons was 12% in dogs and 13% in cats.
### Table 1. Findings in dogs with snake envenomation

<table>
<thead>
<tr>
<th>Envenomation Group</th>
<th>Tiger snake</th>
<th>Suspected tiger snake</th>
<th>Brown snake</th>
<th>Unknown snake</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of dogs</td>
<td>21</td>
<td>41</td>
<td>3</td>
<td>39</td>
<td>104</td>
</tr>
<tr>
<td>Seen to interact with the snake</td>
<td>4 (19%)</td>
<td>12 (29%)</td>
<td>0</td>
<td>14 (36%)</td>
<td>30 (29%)</td>
</tr>
</tbody>
</table>

#### Clinical signs

**Neurotoxicity**

- **Mydriasis:**
  - Tiger snake: 6 (29%)
  - Suspected tiger snake: 20 (49%)
  - Brown snake: 0
  - Unknown snake: 20 (51%)
  - Total: 46 (44%)

- **Reduced pupillary light reflex:**
  - Tiger snake: 5 (24%)
  - Suspected tiger snake: 11 (27%)
  - Brown snake: 0
  - Unknown snake: 10 (26%)
  - Total: 26 (25%)

- **Other ocular neuropathy:**
  - Tiger snake: 1 (5%)
  - Suspected tiger snake: 1 (2%)
  - Brown snake: 1 (33%)
  - Unknown snake: 1 (3%)
  - Total: 4 (4%)

- **Extraocular ophthalmoplegia:**
  - Tiger snake: 1 (5%)
  - Suspected tiger snake: 2 (5%)
  - Brown snake: 0
  - Unknown snake: 1 (3%)
  - Total: 4 (4%)

- **Ataxia/hindlimb paresis:**
  - Tiger snake: 2 (10%)
  - Suspected tiger snake: 14 (34%)
  - Brown snake: 1 (33%)
  - Unknown snake: 7 (18%)
  - Total: 24 (23%)

- **Dyspnoea/respiratory arrest:**
  - Tiger snake: 9 (43%)
  - Suspected tiger snake: 12 (29%)
  - Brown snake: 1 (33%)
  - Unknown snake: 13 (33%)
  - Total: 35 (34%)

- **Generalised tremors:**
  - Tiger snake: 1 (5%)
  - Suspected tiger snake: 1 (2%)
  - Brown snake: 1 (33%)
  - Unknown snake: 1 (3%)
  - Total: 4 (4%)

- **Generalised weakness/recumbency:**
  - Tiger snake: 5 (24%)
  - Suspected tiger snake: 6 (15%)
  - Brown snake: 0
  - Unknown snake: 15 (39%)
  - Total: 26 (25%)

- **Reduced gag reflex:**
  - Tiger snake: 4 (19%)
  - Suspected tiger snake: 8 (20%)
  - Brown snake: 0
  - Unknown snake: 9 (23%)
  - Total: 21 (20%)

**Alimentary symptoms**

- **Ptyalism:**
  - Tiger snake: 2 (10%)
  - Suspected tiger snake: 10 (24%)
  - Brown snake: 1 (33%)
  - Unknown snake: 7 (18%)
  - Total: 20 (19%)

- **Vomiting:**
  - Tiger snake: 8 (38%)
  - Suspected tiger snake: 14 (34%)
  - Brown snake: 1 (33%)
  - Unknown snake: 9 (23%)
  - Total: 32 (31%)

- **Abdominal pain:**
  - Tiger snake: 0
  - Suspected tiger snake: 1 (2%)
  - Brown snake: 0
  - Unknown snake: 1 (3%)
  - Total: 2 (2%)

- **Diarrhoea:**
  - Tiger snake: 0
  - Suspected tiger snake: 1 (2%)
  - Brown snake: 0
  - Unknown snake: 3 (8%)
  - Total: 4 (4%)

**Other effects**

- **Collapse:**
  - Tiger snake: 6 (29%)
  - Suspected tiger snake: 17 (42%)
  - Brown snake: 0
  - Unknown snake: 15 (39%)
  - Total: 38 (37%)

- **Depressed mentation/lethargy:**
  - Tiger snake: 3 (14%)
  - Suspected tiger snake: 8 (20%)
  - Brown snake: 0
  - Unknown snake: 15 (39%)
  - Total: 26 (25%)

- **Hyperthermia:**
  - Tiger snake: 0
  - Suspected tiger snake: 2 (5%)
  - Brown snake: 0
  - Unknown snake: 2 (5%)
  - Total: 4 (4%)

- **Hypothermia (<35°C):**
  - Tiger snake: 0
  - Suspected tiger snake: 2 (5%)
  - Brown snake: 0
  - Unknown snake: 4 (10%)
  - Total: 6 (6%)

- **Pigmenturia:**
  - Tiger snake: 4 (19%)
  - Suspected tiger snake: 9 (22%)
  - Brown snake: 1 (33%)
  - Unknown snake: 1 (3%)
  - Total: 15 (14%)

- **Cardiovascular shock:**
  - Tiger snake: 2 (10%)
  - Suspected tiger snake: 2 (5%)
  - Brown snake: 0
  - Unknown snake: 2 (5%)
  - Total: 6 (6%)

- **Clinical bleeding:**
  - Tiger snake: 1 (5%)
  - Suspected tiger snake: 2 (5%)
  - Brown snake: 0
  - Unknown snake: 2 (5%)
  - Total: 5 (5%)

#### Laboratory tests

- **Coagulopathy present:**
  - Tiger snake: 12 (57%)
  - Suspected tiger snake: 34 (83%)
  - Brown snake: 1 (33%)
  - Unknown snake: 25 (64%)
  - Total: 72 (69%)

- **Mean (Range) APTT (s):**
  - Tiger snake: 213 (66 - >300)
  - Suspected tiger snake: 237 (70 - >300)
  - Brown snake: 193 (85 - >300)
  - Unknown snake: 216 (51 - >300)
  - Total: 223 (51 - >300)

- **Number of APTT results > 300 s:**
  - Tiger snake: 12
  - Suspected tiger snake: 31
  - Brown snake: 1
  - Unknown snake: 21
  - Total: 65

- **Mean (Range) ACT (s):**
  - Tiger snake: 120 (120)
  - Suspected tiger snake: 226 (94 - >300)
  - Brown snake: -
  - Unknown snake: 168 (90 - 240)
  - Total: 235 (90 - >300)

- **Number of ACT results > 300 s:**
  - Tiger snake: 0
  - Suspected tiger snake: 2
  - Brown snake: -
  - Unknown snake: 0
  - Total: 2

- **Median (Range) CK (U/L):**
  - Tiger snake: 1,878 (100 - 62,586)
  - Suspected tiger snake: 3,151 (1,010 - 202,880)
  - Brown snake: 539 (115 - 963)
  - Unknown snake: 248 (28 - 878)
  - Total: 1,506 (28 - 202,880)

#### Treatment

- **Median (Range) number of vials of antivenom administered:**
  - Tiger snake: 2 (1-3)
  - Suspected tiger snake: 2 (1-2)
  - Brown snake: 2 (2-3)
  - Unknown snake: 2 (1-2)
  - Total: 2 (1-3)

- **Number of dogs mechanically ventilated:**
  - Tiger snake: 2 (10%)
  - Suspected tiger snake: 1 (2%)
  - Brown snake: 0
  - Unknown snake: 5 (13%)
  - Total: 8 (8%)

- **Average (Range) length of hospitalisation (days):**
  - Tiger snake: 3.6 (1-11)
  - Suspected tiger snake: 3.4 (1-7)
  - Brown snake: 2.5 (2-3)
  - Unknown snake: 2.6 (1-5)
  - Total: 3.1 (1-11)

- **Number of dogs that survived:**
  - Tiger snake: 17 (90%)
  - Suspected tiger snake: 38 (100%)
  - Brown snake: 2 (100%)
  - Unknown snake: 30 (91%)
  - Total: 87 (95%)

- **Number of dogs euthanased (for financial reasons):**
  - Tiger snake: 2 (10%)
  - Suspected tiger snake: 3 (7%)
  - Brown snake: 1 (33%)
  - Unknown snake: 6 (15%)
  - Total: 12 (12%)

---

* Miosis, bilateral third eyelid protrusion, anicosoria, * Reduced palpebral reflex, reduced menace reflex, * Based on laboratory tests: activated partial thromboplastin time (APTT) or activated coagulation time (ACT), * The upper limits of measurement for the APTT and ACT were 300 s; results > 300 s were ascribed a value of 300 s for calculation of the mean
### Table 2. Findings in cats with snake envenomation

<table>
<thead>
<tr>
<th>Envenomation Group</th>
<th>Tiger snake</th>
<th>Suspected tiger snake</th>
<th>Brown snake</th>
<th>Unknown snake</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of cats</td>
<td>4</td>
<td>32</td>
<td>2</td>
<td>7</td>
<td>45</td>
</tr>
<tr>
<td>Seen to interact with snake</td>
<td>1 (25%)</td>
<td>6 (19%)</td>
<td>1 (50%)</td>
<td>2 (29%)</td>
<td>10 (22%)</td>
</tr>
<tr>
<td>Bite site found&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0</td>
<td>2 (6%)</td>
<td>0</td>
<td>1 (14%)</td>
<td>3 (7%)</td>
</tr>
<tr>
<td>Clinical signs</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Neurotoxicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mydriasis</td>
<td>0</td>
<td>17 (53%)</td>
<td>1 (50%)</td>
<td>6 (86%)</td>
<td>24 (53%)</td>
</tr>
<tr>
<td>Reduced pupillary light reflex&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0</td>
<td>10 (31%)</td>
<td>1 (50%)</td>
<td>2 (29%)</td>
<td>13 (29%)</td>
</tr>
<tr>
<td>Other ocular neuropathy&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0</td>
<td>2 (6%)</td>
<td>1 (50%)</td>
<td>1 (14%)</td>
<td>4 (9%)</td>
</tr>
<tr>
<td>Extraocular ophthalmoplegia&lt;sup&gt;c&lt;/sup&gt;</td>
<td>0</td>
<td>2 (6%)</td>
<td>0</td>
<td>2 (29%)</td>
<td>4 (9%)</td>
</tr>
<tr>
<td>Ataxia/hindlimb paresis</td>
<td>3 (75%)</td>
<td>9 (28%)</td>
<td>1 (50%)</td>
<td>2 (29%)</td>
<td>15 (33%)</td>
</tr>
<tr>
<td>Dyspnoea/respiratory arrest</td>
<td>2 (50%)</td>
<td>6 (19%)</td>
<td>1 (50%)</td>
<td>3 (43%)</td>
<td>12 (27%)</td>
</tr>
<tr>
<td>Generalised tremors</td>
<td>1 (25%)</td>
<td>1 (3%)</td>
<td>1 (50%)</td>
<td>1 (14%)</td>
<td>4 (9%)</td>
</tr>
<tr>
<td>Generalised weakness/recumbency</td>
<td>1 (25%)</td>
<td>0</td>
<td>10 (31%)</td>
<td>1 (14%)</td>
<td>11 (24%)</td>
</tr>
<tr>
<td>Reduced gag reflex</td>
<td>1 (25%)</td>
<td>7 (22%)</td>
<td>1 (50%)</td>
<td>1 (14%)</td>
<td>10 (22%)</td>
</tr>
<tr>
<td>Alimentary symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pylialism</td>
<td>1 (25%)</td>
<td>4 (13%)</td>
<td>1 (50%)</td>
<td>1 (14%)</td>
<td>7 (16%)</td>
</tr>
<tr>
<td>Vomiting</td>
<td>1 (25%)</td>
<td>11 (34%)</td>
<td>1 (50%)</td>
<td>1 (14%)</td>
<td>14 (31%)</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>0</td>
<td>1 (3%)</td>
<td>0</td>
<td>1 (14%)</td>
<td>2 (4%)</td>
</tr>
<tr>
<td>Other effects</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Collapse</td>
<td>0</td>
<td>13 (41%)</td>
<td>1 (50%)</td>
<td>4 (57%)</td>
<td>18 (40%)</td>
</tr>
<tr>
<td>Depressed mentation/lethargy</td>
<td>1 (25%)</td>
<td>13 (41%)</td>
<td>0</td>
<td>2 (29%)</td>
<td>16 (36%)</td>
</tr>
<tr>
<td>Hyperthermia</td>
<td>0</td>
<td>2 (6%)</td>
<td>0</td>
<td>0</td>
<td>2 (4%)</td>
</tr>
<tr>
<td>Hypothermia (&lt;35°C)</td>
<td>0</td>
<td>2 (6%)</td>
<td>0</td>
<td>0</td>
<td>2 (4%)</td>
</tr>
<tr>
<td>Pigmenturia</td>
<td>1 (25%)</td>
<td>3 (9%)</td>
<td>0</td>
<td>0</td>
<td>4 (9%)</td>
</tr>
<tr>
<td>Cardiovascular shock</td>
<td>1 (25%)</td>
<td>3 (9%)</td>
<td>1 (50%)</td>
<td>0</td>
<td>5 (11%)</td>
</tr>
<tr>
<td>Clinical bleeding</td>
<td>0</td>
<td>0</td>
<td>10 (31%)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Laboratory tests</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coagulopathy present&lt;sup&gt;d&lt;/sup&gt;</td>
<td>2 (50%)</td>
<td>11 (34%)</td>
<td>0</td>
<td>3 (43%)</td>
<td>16 (36%)</td>
</tr>
<tr>
<td>Mean&lt;sup&gt;e&lt;/sup&gt; (Range) APTT (s)</td>
<td>107 (93-124)</td>
<td>144 (81 -&gt; 300)</td>
<td>85 (81-89)</td>
<td>185 (82-300)</td>
<td>144 (81-300)</td>
</tr>
<tr>
<td>Number of APTT results &gt; 300 s</td>
<td>0</td>
<td>3</td>
<td>0</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Mean&lt;sup&gt;e&lt;/sup&gt; (Range) ACT (s)</td>
<td>300 (&gt;300)</td>
<td>213 (125-300)</td>
<td>-</td>
<td>-</td>
<td>222</td>
</tr>
<tr>
<td>Number of ACT results &gt; 300 s</td>
<td>1</td>
<td>1</td>
<td>-</td>
<td>-</td>
<td>2</td>
</tr>
<tr>
<td>Median (Range) CK (U/L)</td>
<td>22,396 (1,058-216,147)</td>
<td>7,925 (1,194-385,120)</td>
<td>340 (340)</td>
<td>422 (144-946)</td>
<td>2036 (144-385,120)</td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median (Range) number of vials of antivenom administered</td>
<td>1 (0-2)</td>
<td>1 (0-1)</td>
<td>1 (1)</td>
<td>1 (1)</td>
<td>1 (0-2)</td>
</tr>
<tr>
<td>Number of cats mechanically ventilated</td>
<td>0</td>
<td>1 (3%)</td>
<td>0</td>
<td>0</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Average (Range) length of hospitalisation (days)</td>
<td>3.0 (1-7)</td>
<td>3.4 (1-12)</td>
<td>1.0 (1)</td>
<td>3.6 (1-5)</td>
<td>3.3 (1-12)</td>
</tr>
<tr>
<td>Number of cats that survived</td>
<td>3 (100%)</td>
<td>29 (97%)</td>
<td>1 (100%)</td>
<td>5 (100%)</td>
<td>38 (97%)</td>
</tr>
<tr>
<td>Number of cats euthanased (for financial reasons)</td>
<td>1 (25%)</td>
<td>2 (6%)</td>
<td>1 (50%)</td>
<td>2 (29%)</td>
<td>6 (13%)</td>
</tr>
</tbody>
</table>

<sup>a</sup>The only bite sites that were found were on the faces of the cats. <sup>b</sup>Miosis, bilateral third eyelid protrusion, anicosoria. <sup>c</sup>Reduced palpebral reflex, reduced menace reflex. <sup>d</sup>Based on laboratory tests; activated partial thromboplastin time (APTT) or activated coagulation time (ACT). <sup>e</sup>The upper limits of measurement for the APTT and ACT were 300 s; results > 300 s were ascribed a value of 300 s for calculation of the mean.
DISCUSSION

In the present study the majority of envenomations were due to tiger snakes. Previous studies conducted in the western suburbs of Melbourne also found that tiger snake envenomations were predominant.2,4,15,32

Bite sites were rarely found, and when found they were located on the face, as noted in previous studies.13,15 This may be due to the predatory nature of dogs and cats causing them to attack the snake or due to the fact that the face has less hair than other parts of the body, allowing easier identification of the wound. Two cats with suspected tiger snake envenomation in the present study had facial swelling, associated with the bite site, likely because of subcutaneous haemorrhage and oedema secondary to puncture wounds.17 Swelling and bruising, with associated pain, has been noted in human patients.16 With more thorough examination of veterinary patients, localised swelling at a bite site might be found more frequently.

In people, a hypersensitivity reaction to snake venom may also be the cause of facial swelling.27 The reaction is mediated by IgE and related to prior exposure to either bites or inhalation of dried venom. It is possible that the cats may have had previous unknown snake envenomation.

Signs of neurotoxicity were common in both tiger and brown snake envenomation in the present study, similar to previously reported clinical, experimental and survey studies in dogs2,9,12,32 and cats.2,14,15 The common clinical signs of neurotoxicity in dogs with brown snake envenomation were ataxia, due to hind limb paresis, dyspnoea, ocular neuropathy and generalised tremors. The clinical signs of neurotoxicity in the one cat with brown snake envenomation were mydriasis, ataxia due to hind limb paresis, dyspnoea, ocular neuropathy, reduced gag reflex and reduced pupillary light reflex. These are consistent with previous clinical and experimental findings.12,29 Clinical signs of neurotoxicity are not common in humans following brown snake envenomation.14,29 Thus there is likely to be species variation in the susceptibility to brown snake neurotoxins.

Myotoxicity was seen in 59% of dogs with tiger envenomation, but not in any dogs with brown snake envenomation. The median CK activity in dogs with tiger snake envenomation was 1878 U/L; the highest CK value was 62,586 U/L. Previous studies have noted mean CK values of 974 U/L to 1271 U/L,22 and as high as 220,000 U/L in dogs. In recent human studies, myotoxicity was defined by CK values > 1000 U/L and was noted in 29% of cases of tiger snake envenomation,16 but not in brown snake envenomation.11 The higher percentage of dogs with increased CK values when compared to humans is possibly due to an increased susceptibility to notoxin in this species,6,46 or to delayed presentation. An experimental study in dogs found ongoing muscle necrosis spreading from the notoxin injection site, with associated increase in serum CK activity, lasting for up to 48 hours when higher notoxin doses were used.6

Myotoxicity was seen in all cats with tiger snake envenomation. The median CK activity in cats with tiger snake envenomation was 22,396 U/L, with the highest CK value of 216,147 U/L. The median CK value in cats with tiger snake envenomation is greater in the present study than the previously reported mean value of 428 U/L.15 This is likely to be a result of delayed presentation in some cats,2 allowing CK values to increase further.2 The 6 cats in the present study with CK values between 10,000 U/L and 192,730 U/L that did not require antivenom reflect such delayed presentation.

Systemic signs were seen commonly in snake envenomations in both dogs and cats in the present study (Tables 1 and 2 respectively) and were consistent with those reported in previous clinical, experimental and survey studies.2,9,12,14,15,29,32

Hypothermia was noted in 6 dogs and 2 cats in the present study. Some of the dogs also presented with generalised weakness. No dogs or cats presented with dyspnoea. Of the animals with hypothermia, one dog was euthanased due to neurological signs, but the remaining dogs and both cats survived. Previous studies found that hypothermia, flaccid paralysis and dyspnoea were negative prognostic indicators in dogs12 and cats.15

The results of the present study show that laboratory evidence of coagulopathy was more common in dogs (69%) than cats (36%), consistent with a previous studies.11 Possible explanations for this include an increased resistance in cats to snake venom compared to dogs, or that cats tend to be presented to veterinarians longer after envenomation, allowing time for the coagulation profile to return to normal.11 Prothrombin activator toxin is rapidly deactivated independent of antivenom administration,6,46 and recovery of clotting factor occurs by synthesis of new clotting factors.4

Laboratory evidence of a coagulopathy was detected in 12 of 21 dogs (57%) with tiger snake envenomation and in 34 of 41 dogs (83%) with suspected tiger snake envenomation in the present study; in contrast to the presence of VICC in 95% of human tiger snake victims.48 An experimental study noted a coagulopathy only in dogs that were administered a larger dose of tiger snake venom subcutaneously than that required to cause neurotoxicity signs.12 This may indicate that dogs are less sensitive to the procoagulant effects of tiger snake venom than humans.

Another possible explanation for the lower percentage of coagulopathies noted in the present study when compared to studies in people is that some of the dogs had ACT measured, instead of APTT. The ACT is prolonged when there is a 95% or more decrease of a single clotting factor,69 compared to the APTT, which is prolonged when there is a 70% or more decrease in activity of a single factor.66 Thus, some of the dogs classed as having normal coagulation, based on an ACT result, may have been classed as coagulopathic if the APTT was performed. The present study did not measure fibrinogen or D-dimer concentrations, therefore, some partial VICC could not be detected. Partial VICC has been diagnosed in human patients with INR within reference limits, but with a high D-dimer concentration or decreased fibrinogen concentration.17

Laboratory evidence of a coagulopathy was present in 2 of 4 cats (50%) with tiger snake envenomation and in 11 of 32 cats (34%) with suspected tiger snake envenomation. Similarly, a previous study noted laboratory evidence of coagulopathy in 57% (4 of 7) cats.11

One of the 3 dogs and none of the 2 cats with brown snake envenomation had a clinically detectable coagulopathy. This is in contrast to a recent human study where VICC was found in all patients with brown snake envenomation.13 This maybe for the reasons elucidated above. An in vitro study found that the plasmas of cats and dogs are less susceptible to the procoagulant action of brown snake venom than are those of horses and wallabies.46 Hence species variation between dogs, cats and humans is also possible. Another possible explanation is that venom from a less venomous subtype of the brown snake is cross-reacting with the brown snake venom well in the SVDK.49 However, given the presence of the other clinical signs of envenomation in the present cases, this explanation is less plausible.

Most animals with a coagulopathy, as diagnosed by laboratory tests in the present study, did not show clinical signs of bleeding. This is consistent
with a previous study, where profound abnormalities of laboratory tests of coagulation were noted, but did not result in clinically significant bleeding, unless trauma or another inciting cause was present.\textsuperscript{44} Sites of bleeding in the dogs in the present study included gums, nostrils and the gastrointestinal tract. The areas involved are similar to the sites found in human studies.\textsuperscript{16,31} All of the animals with clinical bleeding in the present study survived, which is consistent with human studies.\textsuperscript{44} One dog with unknown snake envenomation might have had intracranial haemorrhage or thrombosis (based on clinical signs) and this dog was euthanased. Intracranial haemorrhage has been reported as a cause of death in brown snake envenomation in humans.\textsuperscript{51}

Overall, in the present study, dogs received a median of 2 vials of TBAV, with the majority (61\%) being given 2 vials and 3 vials being the maximum administered. Cats received a median of 1 vial of TBAV, with 78\% being administered one vial and only one cat receiving 2 vials, the maximum dose of TBAV given to a cat. Six cats (14\%) had a history of progressive generalised weakness or ataxia, snake interaction a few days prior to presentation, markedly increased CK values, but were negative on SVDT. It is likely that delayed presentation meant there was no longer any unbound venom circulating, although the hook effect cannot be ruled out fully in these cases. These cats were not treated with antivenom.

Previously published doses of antivenom have varied from a maximum of 2 vials of tiger snake antivenom and 1 vial of brown snake antivenom in dogs and cats,\textsuperscript{2,15,32} up to 7 vials of tiger/multibrown antivenom in a cat.\textsuperscript{45} In an early report, up to 10 vials of antivenom were used to treat brown snake envenomation in people.\textsuperscript{4} Recent human studies state that 1 vial of antivenom is adequate to neutralise the venom in all cases of tiger snake\textsuperscript{14} and brown snake\textsuperscript{32} envenomation. The clinical outcome for people who received one vial of antivenom was similar to those who received higher doses.\textsuperscript{51} A few patients still had a very small amount of unbound venom detectable by venom-specific enzyme immunoassay after administration of up to 2 vials of antivenom.\textsuperscript{11} Nevertheless, despite the presence of residual venom, the coagulopathy and clinical signs in the latter patients were improving, and it was concluded that one vial of antivenom was adequate to treat brown snake envenomation in people.\textsuperscript{51} Further study is required to determine the optimum dose of antivenom needed in dogs and cats.

With the exception of mechanical ventilation, which was used in some cases in the present study, other treatment modalities employed were similar to those used in previously reported studies.\textsuperscript{2,9,15,29,32} Prior to 2008, adrenaline was commonly used as part of the premedication prior to administration of antivenom. After 2008, chlorpheniramine was the only premedicant given before administration of antivenom. This change is in line with recommendations in human medicine. Previously adrenaline was recommended to prevent acute adverse reactions to the snake antivenom;\textsuperscript{45} however, in more recent human studies, it has been shown that the use of any premedication was not associated with a reduction in hypersensitivity reactions.\textsuperscript{44} Currently, premedication with adrenaline, antihistamine or corticosteroids prior to administration of antivenom is not recommended in human medicine in Australia.\textsuperscript{13}

In the present study, the average length of hospitalisation was 3.1 days for dogs and 3.3 days for cats. Previous studies have reported hospitalisation times of 1.6 days following tiger snake envenomation\textsuperscript{2} and 1.5 days for all snake envenomations\textsuperscript{12} in dogs. Both of the latter studies reported a lower mean CK activity (974 U/L\textsuperscript{2} and 1271 U/L\textsuperscript{12}) than seen in the dogs in the present study. Previously reported hospitalisation times in cats include 4.5 days for tiger snake envenomation;\textsuperscript{7} 3.3 days for brown snake envenomation\textsuperscript{9} and 4.2 days for all snake envenomations.\textsuperscript{35} In the present study, patients were hospitalised and treated until their CK value was < 5000 U/L. The risk of acute kidney injury increases with CK values > 5000 U/L, because the iron in myoglobin can cause direct oxidative injury to the renal tubule cells.\textsuperscript{10} The protocol of continuing treatment of animals with CK > 5000 U/L, contributed to the longer average hospitalisation times for dogs in the present study.

Eighteen dogs and cats were euthanased for financial reasons, with 11 of these because of the need for mechanical ventilation. Of the 8 dogs that were mechanically ventilated, 5 survived and 3 were euthanased for financial reasons. One cat was mechanically ventilated, but was subsequently euthanased for financial reasons. The survival percentage for the ventilated animals in the present study is similar to that of animals ventilated because of hypoventilation related to tick paralysis (91\%).\textsuperscript{46} The overall proportion of cases in the present study that were euthanised for financial reasons was 12\% (12 of 104) for dogs and 13\% (6 of 45) for cats. Of the remaining cases, 95\% (87 of 92) of dogs and 97\% (38 of 39) of cats survived. The survival percentages in dogs that were treated in the tiger snake envenomation group was 90\% (17 of 19 dogs), while for those suspected as cases of tiger snake envenomation it was 100\% (all 38 dogs) and for cases of brown snake envenomation it was 100\% (2 of 2 dogs). The survival percentages in cats that were treated in the tiger snake envenomation group was 100\% (all 3 cats), while for suspected tiger snake envenomation it was 97\% (29 of 30 cats), with the only case of brown snake envenomation also surviving. Previous studies have reported survival percentages of 75\% to 87\% for canine snake envenomation cases and 66\% to 91\% for feline snake envenomation cases that were treated with antivenom.\textsuperscript{2,15,29,30,32,33} The greater survival percentages in the present study may be in part attributed to the use of mechanical ventilation of patients with ventilatory failure (5 of 92 dogs survived with mechanical ventilation, increasing the survival percentage by 5.4\%). Unfortunately the cost of mechanical ventilation may also contribute to an increase in financial euthanasia rates.

A limitation of the present study is the small number of confirmed brown snake envenomations. Further study is currently underway in this regard. Another limitation in the present study was our inability to perform a keyword search in the database. The invoice code search excluded any animal with suspected or definite snake envenomation that was euthanised or died soon after presentation, without the performance of any confirmatory tests or treatment. If such animals were included, it would likely increase the percentage of animals showing more severe clinical signs, such as dyspnoea, and might have reduced the survival and euthanasia rates in the present study.

CONCLUSION

Laboratory evidence of a coagulopathy is common in dogs and cats with tiger snake envenomation and with more sensitive testing may approach the rate seen in human cases. Such a coagulopathy was less common in brown snake envenomation in dogs and was not noted in cats, however, this is possibly due to the very small numbers of animals with confirmed brown snake envenomation in the present study. Due to the similarity of clinical signs in tiger and brown snake envenomation, it is not recommended that diagnosis of the species of snake involved is based on clinical signs alone. The use of a SVDK to confirm the monovalent antivenom required or the use polyvalent antivenom is recommended.
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Concurrent Sertoli-Leydig cell ovarian tumour and Gartner’s duct cyst in an 8-year-old bitch

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ABSTRACT
An 8-year-old female Staffordshire bull terrier-cross dog, reportedly spayed, was presented for evaluation of generalised hyperpigmentation, hyperkeratosis and non-pruritic alopecia, in addition to intermittent haematuria. Abdominal palpation revealed an abnormal tubular structure within the caudal abdomen. Oestradiol and progesterone assays revealed hyperoestrogenism with normal progesterone concentration. Abdominal ultrasonography identified a large, fluid-filled, tubular structure located within the mid- to caudal abdomen, and a polycystic right ovary. Exploratory laparotomy was performed to remove the right ovary and the fluid-filled tubular structure, which was adherent to the vaginal wall. Evaluation of these structures by histopathology and immunohistochemistry confirmed the diagnoses of a Gartner’s duct cyst and a Sertoli-Leydig cell tumour of the right ovary. Within weeks of surgery, improvement of the dog’s endocrine-related skin disease occurred. To the authors’ knowledge, this is the first report of a Gartner’s duct cyst and Sertoli-Leydig cell tumour in the same patient within the veterinary literature. Aust Vet Pract 2014;44(4):713-718.

INTRODUCTION
Ovarian neoplasms are uncommon in the dog, accounting for approximately 1% of all canine neoplasms.¹ The overall incidence is low (4%) in cats and dogs, presumably due to the prevalence of elective ovariohysterectomy in most parts of the world. The incidence in intact female dogs is slightly higher (6%), with middle-aged to older dogs (mean 10 years of age) usually affected.²,³ Tumours of the ovary are almost exclusively primary, and may originate from germ, epithelial or sex cord stromal cells.²-⁴ Mesenchymal tumours and metastatic lesions affecting the ovary are rare.¹ Ovarian tumours are generally unilateral, although some tumours, such as dysgerminomas, papillary adenomas and Sertoli-Leydig cell tumours (SLCT) may present bilaterally.²,⁵-⁶ The vast majority of canine ovarian tumours (80-90%) are of epithelial or sex cord stromal cell origin. Of the sex cord stromal cell tumours, granulosa cell tumours account for approximately 50% of cases.²,⁴ To the authors’ knowledge, the incidence of SLCT in dogs has not been reported; in women these represent 0.2% of all ovarian neoplasms.³

During embryological development, the urinary and genital systems share a common mesodermal origin. When sexual development progresses normally in the female fetus, ovarian differentiation occurs in the absence of testis determining factor and testosterone. Similarly, normal regression of mesonephric (Wolffian) ducts occurs in the absence of Müllerian inhibiting substance, which is not secreted in the immature female fetus.² Disturbances in the regression of the mesonephric duct system can be the result of hormonal imbalances in utero. This can lead to urogenital abnormalities such as the development of Gartner’s duct cysts (GDC), which may also be associated with ipsilateral renal and ureteral anomalies.⁴-⁵ The Wolffian duct system also impacts the early development of the Müllerian ducts; therefore, uterine anomalies can also occur.⁶ GDC represent the vaginal and uterine remnants of the Wolffian duct system, and may form anywhere along its course, most commonly adjacent to the lateral wall of the vagina.⁷ Occasionally seen in women between the ages of 20-40 years,⁸-¹² GDC are rare in dogs, with less than 10 cases reported.⁹,¹⁰-¹²

In the present report, an unusual case of a SLCT and a GDC occurring concurrently in a dog is described. To the authors’ knowledge, there is no other report within the veterinary literature describing these unusual pathologies in the same patient.

CLINICAL FEATURES
An 8-year-old, nulliparous 21-kg female Staffordshire bull terrier-cross dog was referred for evaluation of generalised hyperpigmentation, hyperkeratosis and non-pruritic alopecia, in addition to intermittent haematuria of approximately 10 months’ duration. The dog was obtained by the present owner as a “rescue”, adopted from a local welfare organisation as a young adult that was reportedly spayed prior to surrender. The primary clinical signs first noticed by the owner were lack of hair regrowth, subsequent to surgery for correction of a ruptured cranial cruciate ligament of the right hind limb, and a persistently swollen, thickened vulva, with intermittent blood-tinged discharge. Haematuria, with pollakiuria and occasional inappropriate urination, had also been observed. The patient was treated medically at the referring veterinary hospital with several courses of amoxicillin clavulanate (12 mg/kg PO bid for 10 days), which resulted in short-term resolution of the vulval discharge and haematuria. However, upon completion of each course of antibiotics, these clinical signs always recurred. The last course of antibiotic therapy was completed 3 weeks prior to referral.

Physical examination findings at the time of the initial referral consultation revealed vital parameters within reference ranges. No external lymphadenomegaly was detected. Non-pruritic alopecia was associated with the entire right hind limb, lateral aspect of the left hind limb and the tail and tail base. Slow hair regrowth and partial alopecia was observed at previously clipped sites. Generalised hyperpigmentation and hyperkeratosis of the skin (Figure 1) and vulva were also present. Abdominal palpation yielded signs of repeatable discomfort, and an abnormal tubular structure could be palpated within the caudal abdomen. The vulva and perineal regions were swollen and engorged, and a prominent vaginal fold was noted via digital vaginal examination. Digital examination of the vaginal vault revealed thickening/oedema of the ventral floor, but no evidence of a distinct mass. A surgical...
scar was observed over the lateral aspect of the right stifle. No ventral midline or flank abdominal scars could be appreciated and no ear tattoo, commonly placed at the time of ovariohysterectomy, was present. Attempts were made to recover the previous medical records of the patient to verify that she had been spayed; however, these were not available.

A complete haematology and serum biochemistry blood profile was obtained, with all parameters within canine reference ranges. Blood was also collected for oestradiol and progesterone assays; the results of which were not available until approximately one week later. In the interim, abdominal ultrasound identified a large, fluid-filled tubular structure (suspected to be uterine) located within the mid-to caudal abdomen. A heterogenous pattern of echogenicity was noted within the fluid-filled components of the structure, suggesting compartmentalisation or torsion of the contents. A hypoechoic, heterogenous mass-like lesion (dimensions 54.1 x 28.2 x 44.1 mm) was observed within the cranial left margin of the structure. The right ovary was identified (17 mm diameter) and appeared to be polycystic; the left ovary could not be visualised. Mild hydronephrosis of the right kidney was present (renal pelvis 4 mm diameter); otherwise both kidneys and the remainder of the abdominal contents were sonographically unremarkable. The mild hydronephrosis was considered an incidental finding and was not investigated further. Surgical intervention, with ovariohysterectomy and complete exploration of the abdominal cavity, was recommended.

A ventral midline laparotomy was performed and a large, convoluted, fluid-filled structure, similar in appearance to the left uterine horn was encountered. A number of adhesions associated with this structure were identified; these were broken down to enable the structure to be exteriorised from the abdominal cavity. The cranial pole of this structure was carefully evaluated for the presence of an ovary; no distinct ovarian tissue was identified, although a focal, discrete thickening was identified at the cranial aspect. The “horn” could be traced caudally to an apparent uterine “body”, and appeared to be attached to the vaginal wall adjacent to the cervix; however, no right uterine horn was present. The right ovary was located at the normal anatomic location, adjacent to the caudal pole of the right kidney. It was approximately 2-3 cm in diameter with multiple cystic structures present on its surface. The right ovarian pedicle, the cranial attachment of the left uterine “horn” and the uterine “body” were triple-clamped and double ligated. The uterine “body” was also secured with a Parker-Kerr oversew. A sample of fluid from the lumen of the “uterus” and a small section of tissue from the wall was collected for bacterial analysis. The excised tissue was then placed in 10% neutral buffered formalin and submitted for histopathological examination. An intra-operative urine sample was obtained via cystocentesis and sent for urinalysis and culture/sensitivity. The remainder of the abdominal organs were explored and no abnormalities were detected. The abdominal wall was closed routinely in 3 layers, with staples placed in the skin. The patient was recovered from general anaesthesia and maintained on ticarcillin (50 mg/kg IV q 8h) and methadone (0.2 mg/kg IV q 4h), which was subsequently transitioned to buprenorphine (0.01 mg/kg IV q 8h). The patient was ultimately discharged from hospital 2 days after surgery with a 5-day course of tramadol (2.4 mg/kg PO tid) and four weeks of amoxicillin-clavulanate (12 mg/kg PO bid).

The results of the urinalysis obtained intra-operatively were unremarkable, and the urine culture was negative. Culture/sensitivity of the “uterine” fluid and of the tissue collected from the wall yielded pure growths of E. coli and E. avium, both sensitive to amoxicillin-clavulanate. Gross pathologic examination of the submitted “uterine” tissue revealed a large, compartmentalised cystic structure that was attached to the cervix but not directly communicating with it (Figure 2). The lumen of this structure was filled with green-tinged, turbid fluid. The right ovary was 20 x 20 x 25 mm in dimensions and haemorrhagic on the cut section (Figure 3).
Histopathologic assessment of the cystic structure revealed a thin wall, composed of connective tissue stroma lined by a non-ciliated monostratified epithelium (Figure 4); consistent with a remnant mesonephric duct cyst (GDC). In some areas of the wall, thin bundles of smooth muscle cells were observed. Sections inclusive of the cervix revealed normal mucosa. No ovarian tissue was identified at the cranial aspect of the structure.

The right ovary was well-demarcated and encapsulated by a thin connective tissue capsule in which few remnants of sex-cord cells were visible. Within the centre of the ovary, there was cellular proliferation amongst areas of haemorrhage and necrosis. Normal ovarian architecture was completely effaced by a highly cellular neoplasm composed of two distinct cellular populations. The predominant population was represented by polygonal to oval cells, arranged in sheets and cords, separated by thin fibrovascular stroma. Cells had abundant clear to foamy cytoplasm (consistent with Leydig cells). The second population was composed of columnar cells, in well-formed tubules arranged perpendicularly to the basement membrane, consistent with Sertoli cells (Figure 5). Both populations of neoplastic cells demonstrated mild anisocytosis and anisokaryosis. The observed mitotic index was low (<1 per 10 high power fields). This finding was consistent with a benign SLCT.

Immunohistochemistry was performed on representative sections of the ovarian tumour; by the acidin-biotin-peroxidase complex (ABC) method. The following antibodies were used: vimentin (mouse monoclonal antibody, Dako, Denmark; diluted at 1:100), cytokeratin AE1/AE3 (mouse monoclonal antibody, Dako; diluted at 1:100), Melan-A (mouse monoclonal antibody, Dako; diluted at 1:400), Wilms Tumor-1 (WT-1; rabbit polyclonal antibody, Santa Cruz Biotechnology, Inc., Dallas, TX, USA; diluted at 1:100),
inhibin-α (mouse monoclonal antibody, AbD Serotec, Kidlington, Oxfordshire, UK; diluted at 1:50). This revealed that neoplastic cells in the Leydig cell portion were strongly positive for vimentin and Melan-A. The sex cord component (Sertoli cell tumor) was strongly positive for vimentin and inhibin-α and moderately positive for Melan-A (Figure 6). Both components were negative for cytokeratin AE1/AE3 and WT-1. The pre-operative progesterone concentration was 2.7 nmol/L (reference range < 9 nmol/L), consistent with pre-ovulation. The pre-operative oestradiol concentration was 80 pmol/L (reference range > 73 pmol/L), suggestive of follicular activity. A post-operative oestradiol/progesterone assay has not been performed to date.

The patient was returned for a follow-up appointment 2 weeks after surgery. The laparotomy incision had completely healed and the skin staples were removed. At this time, the alopecia of the tail and hind limbs was beginning to resolve. Digital examination of the vaginal vault revealed resolution of the thickening/oedema of the vaginal floor, and the vulval and perineal swelling was no longer present. The owner reported the onset of mild urinary incontinence, particularly when the patient was sleeping or at rest. Aside from incontinence, no other lower urinary tract signs had been observed. Urinalysis performed at that time was unremarkable, with no evidence of persisting urinary tract infection. Therefore, it was suspected that the patient was suffering from post-ovariohysterectomy urethral sphincter mechanism incontinence (USMI), and treatment with phenylpropanolamine (1.6 mg/kg PO bid) was commenced.

The patient was re-examined 2 months post-operatively, at which time the hyperpigmentation, hyperkeratosis and non-pruritic alopecia had significantly improved (Figure 7). However, the owner advised that the urinary incontinence was persistent, despite phenylpropanolamine therapy. Therefore, stilboestrol (1 mg PO sid for 5 days; subsequently tapered to 1 mg PO once weekly) was added to the treatment regime. Blood was obtained for karyotyping, however this was unsuccessful as the sample failed to yield complete metaphases suitable for analysis of genotypic sex. This failure was likely due to damage to the mononuclear cells during sample acquisition, storage or transport.

**DISCUSSION**

SLCT are predominantly composed of cells with male morphologic features interspersed with the normal ovarian stromal architecture. In humans, SLCT are divided into 3 separate histological classifications: well differentiated tubular adenoma, intermediate type composed of testicular tubules in all stages of gonadogenesis, and an undifferentiated sarcomatoid type. A fourth classification is the retiform variant, with an appearance similar to the rete testis. The latter variant is characterised by the presence of irregular anastomosing channels lined by cuboidal or columnar cells in addition to the usual SLCT architecture. This classification scheme has not been directly followed in dogs; rather, SLCT are frequently classified within the granulosa cell tumour group. However, some authors describe SLCT as a separate entity with distinct histological characteristics. As they originate from the specialised gonadal stromal cells of the ovary, SLCT may be functional, secreting one or multiple hormones, or none at all. It has been reported that up to 50% of canine SLCT are functional. In humans, most SLCT are virilising, with testosterone secretion noted in approximately 80% of cases, except the retiform variant which is rarely androgenic. The secretion of oestrogen, with associated signs of hyperoestrogenism, occurs in approximately 10% of cases. In contrast, in the dog, the endocrinological behaviour of SLCT specifically, as opposed to the overall behaviour of sex cord stromal tumours, has not been well documented. Cystic endometrial hyperplasia, and endometritis with or without squamous metaplasia of the uterine mucosa, have been reported, implicating increased progesterone secretion, but only in a few dogs.

We have been unable to locate any reliable information relating to the oestrogen secreting behaviour of SLCT in dogs. In the current case, the dog was clearly exhibiting signs of excessive oestrogen secretion at the time of presentation. Such clinical signs included: vulval enlargement, bloody vulval discharge and bilaterally symmetrical, non-pruritic alopecia with hyperpigmentation and hyperkeratosis. However, no evidence of bone marrow suppression or pancytopenia was observed, based on the dog's pre-operative blood screen. Objective evidence of hyperoestrogenism, without concurrent hyperprogesteronism, was confirmed by pre-operative oestradiol and progesterone assays. Based on these observations, and the rapid regression of the clinical signs associated with hyperoestrogenism post-operatively, we deduce that the dog was affected by an estrogen secreting SLCT; a rare finding within the veterinary literature.

The results of the immunohistochemical analysis of neoplastic ovarian tissue in the present case were in accordance with those expected, based on the available information within the literature. The Sertoli cell components of the tumour were strongly positive for vimentin and inhibin-α and moderately positive for Melan-A. The Leydig cell components were strongly positive for vimentin and Melan-A. Both components were negative for cytokeratin AE1/AE3 and WT-1. Positivity to inhibin-α is a consistent feature of sex cord stromal tumours in both humans and dogs. Inhibin-α is a gonadal glycopeptide which is known to be a feedback inhibitor of pituitary secretion of follicle stimulating hormone (FSH). It is not expressed by epithelial or germ cell tumours of the ovary, but can be expressed by other sex-cord stromal ovarian tumours, such as granulosa-theca cell tumours. SLCT are also consistently positive to vimentin, although the intensity of the reaction can be variable.

SLCT are used in the diagnosis of melanocytic lesions, however, it has also been shown to label normal steroid-producing cells, including adrenocortical cells and gonadal sex cord and stromal cells. Melan-A expression has also been described in adrenocortical tumours, testicular Leydig cell tumours, and ovarian SLCT. This is considered an immunological cross-reaction, as mRNA for Melan-A has not been

![Figure 7. Appearance of the dog's right hind limb and tail 2 months post-operatively, demonstrating resolution of alopecia.](image-url)
detected in non-melanocytic sites. Therefore, variation in the degree of immunoreactivity to Melan-A across the range of adrenocortical and gonadal tumours exists, such that a positive staining reaction of an ovarian tumour to Melan-A is suggestive of sex cord stromal origin, but a negative reaction is not clinically significant. SLCT also co-express vimentin and cytokeratin A1/AE3, but expression can be variable and these markers can also be expressed by other ovarian tumours. In dogs with sex cord stromal tumours, reaction to cytokeratin A1/AE3 can either be positive or negative. In the present case, both Sertoli and Leydig cell components yielded negative reactions. Similarly, reactivity to WT-1 is variable, with either positive or negative reactions recorded in both humans and dogs, with negative reactions more commonly observed in the dog. A marker that was not assessed in the present case was epithelial membrane antigen (EMA); which always yields a negative reaction in the case of SLCT in both humans and dogs.

These results confirm, in agreement with the published literature, that immunohistochemistry can be a valuable tool for differentiating SLCT from more common epithelial tumours of the canine ovary. The latter can be difficult to achieve with conventional histopathology alone, due to the complex histological structure of the canine ovary. Where neoplastic proliferation occurs, a variety of histologies may emerge, due to the proliferation of neoplastic cells within the ovary, proliferation of the tubular or cord cells of the ovary, or both. The antibodies that are most sensitive and specific for SLCT are inhibin-α and EMA, always yielding positive and negative results respectively. Of these two antibodies, inhibin-α is probably the most useful as it is only expressed by sex-cord stromal tumours.

In women, GDC are usually small (< 2 cm diameter) and therefore asymptomatic, although some may eventually grow to extremely large sizes (> 10 cm), when they may cause pelvic pain, dyspareunia or complications during parturition. Affected dogs are usually middle-aged to older, and in most reports, at the time of diagnosis, the GDC are very large. Clinical signs in the dog are related to the space-occupying effect of the GDC within the caudal abdomen and include dysuria, dyschezia and dystocia. Sonographically, GDC characteristically appear as large, often multilobulated structures filled with anechoic to hypoechoic fluid, originating from the lateral vaginal wall, but distinct to the uterus. Histologically, GDC are lined by a pseudostatified, layer of high cuboidal to columnar epithelium. The wall of the cyst may contain a thin layer of smooth muscle, although the latter finding is variable. The lumen of the GDC is usually filled with proteinaceous fluid, secreted by the mucosal lining. As observed in the present case, infection of this structure is also possible, but infrequently documented. The sonographic and histopathologic evaluations of the lesion in the present case were consistent with those found in the literature.

Surgical management of GDC may involve simple drainage of the cyst via episiotomy (if extending caudally into the pelvic canal) or laparotomy. Excision of the GDC from its point of origin on the vaginal wall has also been described, with or without concurrent ovariohysterectomy in the intact female. This was the approach undertaken in the present case. It has been mentioned on one occasion in the literature to avoid excision of GDC at its point of origin, due to the risk of damage to the urethral innervation and blood supply. During our dissection, extreme care was taken to identify and preserve the important structures within close proximity to the bladder neck, such as the ureters and the urethral blood and nervous supply. Despite this approach, the patient developed urinary incontinence post-operatively. It is unclear whether this was caused by disruption to the urethral innervation during surgery, or related to USMI, as the condition appeared to partially respond to medical management. Alternatively, marsupialisation of the cyst into the vagina via episiotomy has also been advocated as a surgical management strategy.

To the authors’ knowledge, GDC with a concurrent ovarian tumour has not been described in humans. A review of the veterinary literature has identified only one case of a dog suffering from these concurrent conditions. In the latter case, multiple papillary adenomas and papillary cystadenomas, common ovarian tumours in dogs, were confirmed histologically in both ovaries. In the present case, a concurrent SLCT, a much rarer neoplasm, was confirmed histologically and with immunohistochemical staining.

Due to the unavailability of the previous medical records of the patient, we are unable to conclusively determine whether she had been previously spayed (given the absence of a uterus and the left ovary), or whether she was exhibiting concurrent congenital anomalies of the uterus and ovaries. However, the absence of an abdominal scar and ear tattoo suggest that such surgery had not been undertaken. Similarly, it was unknown whether the presence of the right ovary at the time of surgery was indicative of the presence of genuine ectopic ovarian tissue, ovarian remnant syndrome or another embryological defect.

**CONCLUSION**

The present report describes the unusual occurrence of 2 rare forms of genitourinary tract pathology, namely SLCT and GDC within the same patient. Although the dog’s endocrine-related skin disease has markedly improved following ovariohysterectomy, urinary incontinence has occurred post-operatively. It is unknown whether this is due to USMI, or potential disruption to the innervation of the urethral sphincter during resection of the GDC, or a combination of these factors.

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Water-clear cell hyperplasia of the parathyroid gland in a dog


ABSTRACT

A 10-year-old male neutered cross-bred dog was presented with a 2-month history of polyuria and polydipsia. Biochemistry revealed hypercalcaemia. Parathyroid hormone was increased, consistent with primary hyperparathyroidism. Ultrasonographic imaging was consistent with a parathyroid mass. A bilateral caudal parathyroidectomy was performed. The left caudal parathyroid gland was firm and enlarged at 7 mm diameter. The right caudal parathyroid gland was also prominent at 4 mm diameter, but had normal consistency. Histopathology revealed a water-clear cell hyperplasia of the left caudal parathyroid gland. The right caudal parathyroid gland had normal morphology. Water-clear cells are derived from chief cells, differentiated by their abundant cytoplasmic glycogen. Water-clear cell hyperplasia has been described as a cause of primary hyperparathyroidism in humans. To the authors’ knowledge, this is the first reported case of water-clear cell hyperplasia of the parathyroid gland in a dog.

INTRODUCTION

The present case describes a dog with initial signs of polyuria and polydipsia due to hypercalcaemia, which was ultimately found to have a previously unreported form of parathyroid hyperplasia. Primary hyperparathyroidism should always be considered in cases of hypercalcaemia with normo- to hypophosphataemia, but needs to be differentiated from other diagnoses, such as humoral hypercalcaemia of malignancy, on the basis of a parathyroid hormone (PTH) assay. Primary hyperparathyroidism is a well-recognised cause of hypercalcaemia in dogs, due to increased PTH activity caused by excessive production by parathyroid chief cells, most often in parathyroid gland adenomas and less commonly in hyperplasia and carcinomas. The present case is unusual in relation to the type of cells found to be hyperplastic in the affected parathyroid gland. Water-clear cells are derived from chief cells of the parathyroid gland, but are not recognised as part of the normal architecture of the canine parathyroid gland. While water-clear cell hyperplasia (WCCH) of the parathyroid gland has been reported as a histopathological finding and a cause of hypercalcaemia in the human medical literature and once in a bovine case series, to the authors’ knowledge this is the first reported case in a dog.

CLINICAL FEATURES

A 10-year-old male neutered 9.5-kg cross-breed dog was presented initially with a 2-month history of polyuria and polydipsia. On physical examination, including rectal palpation, no abnormalities were detected. Biochemistry performed at the referring veterinary hospital revealed hypercalcaemia (total serum calcium of 3.66 mmol/L; reference range 2.60-3.10 mmol/L), normophosphataemia (1.16 mmol/L; reference range 0.81-2.19 mmol/L) and normoalbuminaemia (36 g/L; reference range 22-39 g/L). A subsequent sample was tested simultaneously for total serum calcium and serum PTH concentrations at an external laboratory (Vetnostics Specialist Diagnostic Services Pty Ltd). In tact PTH was measured via a polyclonal antibody chemiluminescent assay (LIAISON N-tact PTH Assay, DiaSorin Inc, Stillwater, MN, USA). Both were increased (total serum calcium 3.59 mmol/L, reference range 2.00-2.80 mmol/L; PTH 277 pg/mL, reference range 12-145 pg/mL). These findings were consistent with primary hyperparathyroidism. A mild increase in total bilirubin (21 umol/L; reference range 0-15 umol/L) was also noted at that time. Ultrasonography of the urinary bladder revealed no evidence of cystic calculi. In the following 2 months (between diagnosis and referral), the dog was treated with frusemide (Urex-M; Aspen Pharma, St Leonards, NSW) at 5 mg/kg, to increase calciuresis.

On referral to the Small Animal Specialist Hospital, no significant abnormalities were noted on physical examination and rectal palpation. The plasma calcium and ionized calcium measurements were repeated and found to be 3.04 mmol/L (reference range 2.80-3.20 mmol/L) and 1.32 mmol/L (reference range 1.25-1.55 mmol/L) respectively. Other biochemical parameters tested included albumin, urea, creatinine and phosphate, which were all within reference limits. Packed cell volume was also within reference limits. Ultrasonographic imaging of the parathyroid glands identified enlargement of the left caudal parathyroid gland, measuring 5.43 x 6.11 mm.

A bilateral caudal parathyroidectomy was performed. Grossly, both the caudal intracapsular parathyroid glands were white in appearance and enlarged. The left caudal parathyroid gland was firm and measured 7 mm in diameter. The right caudal parathyroid gland was also prominent at 4 mm diameter but had normal consistency. The cranial extracapsular parathyroid glands were bilaterally small and grey, and subjectively normal. The thyroid glands were bilaterally normal in appearance, texture and size. Both of the enlarged caudal parathyroid glands were excised utilising a guillotine procedure: the caudal poles of the thyroid glands (containing parathyroid glands) were isolated from surrounding tissue using bipolar electrocautery. Monoplus 3-0 (B Braun, Bella Vista, NSW) was used to guillotine the thyroid half way along its length. Care was taken to preserve the cranial (extracapsular) parathyroid glands and their blood supply (the cranial thyroid artery). All specimens were fixed in neutral-buffered 10% formalin and routinely processed for microscopic examination.
The dog became hypocalcaemic (ionized calcium of 1.08 mmol/L) 6 hours post-operatively, then developed clinical evidence of hypocalcaemia (tremors) 9.5 hours post-operatively. Both biochemical and clinical evidence of hypocalcaemia resolved within 6 hours of initiating intravenous calcium supplementation. An initial dose of a 10% solution of calcium gluconate (Phebra, Lane Cove West, NSW), equivalent to 88.35 mg of elemental calcium, was administered intravenously, slowly over 40 minutes. A continuous rate intravenous infusion of calcium gluconate was then administered at a rate of 1 mg/kg/hour for 15 hours, then at 0.5 mg/kg/hour for a further 3 hours and was discontinued thereafter. Oral supplementation of calcitriol (Rocaltrol; Roche Products Pty Ltd, Dee Why, NSW) and calcium carbonate (Caltrate; Wyeth, Australia) at 0.027 µg/kg SID and 48 mg/kg BID respectively, was initiated 22 hours after surgery. The dog was discharged 4 days after surgery. Calcitriol dose was tapered in three-weekly increments from once every 24 hours to once every 48 hours, then to once every 72 hours, then once per week for the final 3 weeks. The calcium carbonate was continued for a total of 20 days.

Ionized calcium concentration was measured at 8 weeks post-surgery (1.28 mmol/L), 15 weeks post-surgery (1.06 mmol/L) and 19 weeks post-surgery (1.27 mmol/L), with only the 15-week sample being mildly decreased.

On histopathology, the right caudal parathyroid and thyroid glands had normal morphology. The left caudal parathyroid gland was expanded by a circumscribed, non-encapsulated nodule composed of nests of large polyhedral to rounded cells (water-clear cells) that had multiple, variably sized clear cytoplasmic vacuoles with well-defined light eosinophilic borders (Figure 1). Nuclei were round to oval, eccentric, with dense chromatin and no distinct nucleolus. No mitotic figures were seen. Some distinct chief cells were scattered around the periphery. Adjacent was some normal thyroid with dilated follicles filled with colloid. The histopathological diagnosis was WCCH of the left caudal parathyroid gland.

DISCUSSION

The normal canine parathyroid gland is comprised of chief (or ‘principal’) cells, oxyphil cells and transitional cells, which have structural characteristics intermediate between those of chief and oxyphil cells. Water-clear cells are not among the typical cells found in the canine parathyroid gland, whereas in the human parathyroid gland, water-clear cells are observed as part of the normal solid parenchyma along with chief cells, transitional-oxyphil, and oxyphil cells.

Water-clear cells are derived from chief cells and similarly produce PTH. They are differentiated by their abundant cytoplasmic vacuoles surrounded by an eosinophilic cytoplasm. When compared with chief cells, water-clear cells are larger and the glycogen is more dispersed, the plasma membrane is often much more tortuous. However, many cells may display histological features intermediate

Figure 1. Histologic sections of the hyperplastic left parathyroid gland.

(A) The hyperplastic parathyroid gland with an abundance of water-clear cells (red arrow heads) can be seen, with some chief cells scattered around the periphery. The component of parathyroid gland tissue comprised of water-clear cells is not encapsulated and is not compressing the chief cells or any adjacent structures, consistent with water-clear cell hyperplasia. Some normal thyroid tissue (black arrow heads) can also be seen in this section. Haematoxylin & Eosin stain (H&E), bar = 300 µm.

(B) Normal parathyroid chief cells (black arrow heads) are easily distinguished from the hyperplastic water-clear cells (red arrow heads). H&E, bar = 50 µm.

(C) Water-clear cells are characterised by their large clear vacuoles within well-defined light eosinophilic borders (red arrow heads). H&E, bar = 30 µm.
between those of active chief cells and large clear cells. In WCCCH, the individual cells are ultrastructurally composed of numerous membrane-bound vacuoles, 0.2 to 2 µm in diameter, thought to be derived from the Golgi apparatus.12

WCCCH has been described as a cause of primary hyperparathyroidism in humans.5,7,11 To the authors’ knowledge, this is the first reported case of WCCCH of the parathyroid gland of a dog and only the second in the veterinary literature, the other in a bovine case series of nutritional hypercalcitonism.4

In the present case, PTH concentration was increased in association with clinical findings consistent with primary hyperparathyroidism. This suggests that the hyperplastic water-clear cells were functional and maintained a similar function to chief cells in the production of PTH.

In the human literature, histological differentiation between parathyroid gland hyperplasia and adenoma has been described as difficult, especially if only one specimen is submitted.1 In human cases of parathyroid hyperplasia, the glands may not all be of equal size.9 Pseudo-adenomatous chief cell hyperplasia has also been described in humans, wherein one gland is enlarged and the others are of a normal size but all glands exhibit active hyperplastic cells.9 Therefore, it is essential to submit not only the enlarged parathyroid gland but also at least one of the other glands when making a diagnosis. In cases of adenoma, the other glands should appear normal or suppressed on histopathological examination, whereas diagnosis of hyperplasia can be made if the cells of the second normal-sized gland appear to also be excessively active.9 Resting chief cells can be detected in an apparently inactive phase when the rough endoplasmic reticulum and Golgi apparatus are not prominent and with a less tortuous plasma membrane than that seen in active chief cells, which display a more prominent rough endoplasmic reticulum and large Golgi apparatus.9 Staining of glycogen by Periodic-acid-Schiff-diastase has been utilised in human cases of parathyroid adenoma to further distinguish between chief cells, which stain positive for glycogen, and water-clear cells, which are negative for glycogen.13 Since non-parathyroid forms of clear cell proliferation have been reported in the human literature, such as renal cell carcinomas, paragangliomas and salivary gland tumours, immunohistochemistry for PTH and chromogranin A can also be utilised to confirm that the submitted sample is of parathyroid gland origin.13 While the latter techniques were not performed in the present case, they could be considered for use if further confirmation of the diagnosis was required. In canine hyperparathyroidism due to hyperplasia, it has been shown that hyperplastic parathyroid nodules can also show intrinsic autonomy, similar to parathyroid adenomas, causing suppression of the remaining parathyroid endocrine tissue, and it has been suggested that primary multi-nodular hyperplasia may in fact be a form of parathyroid adenoma.2 It has been suggested that there is a path of sequential transformation of parathyroid cells in a neoplastic state, since clear morphological characteristics to distinguish between hyperplasia, adenomas and adencarcinomas reportedly do not exist.14 The parathyroid proliferation in the present case was difficult to classify, but was ultimately designated as hyperplasia based on the absence of a capsule and the lack of compression of adjacent tissue, both of which would be expected with an adenoma.3

In dogs, parathyroid gland hyperplasia, adenoma and carcinoma have been reported as causes of primary hyperparathyroidism.2,15,16 Parathyroid neoplasia is uncommon in dogs.14 In a case series of 29 dogs in which 18 parathyroid nodules were submitted, a histopathological diagnosis of neoplasia was made 5.3 times more often than hyperplasia.2 In contrast to the human literature, while canine parathyroid hyperplasia can involve any number of glands, it most often only affects one gland and almost always appears in a nodular pseudoadenomatous pattern, with the remaining parathyroid tissue appearing atrophic.3,10 Even in cases of canine primary parathyroid hyperplasia, where more than one gland is affected, the hyperplastic tissue almost always appears in discrete nodules within the other glands, while the diffuse form is reported exceedingly rarely in dogs.7 The most common presenting signs of primary hyperparathyroidism in dogs are polyuria and polydipsia.7,13,14 This is due to a progressive increase in serum calcium concentration, secondary to inappropriately high PTH values. Primary hyperparathyroidism occurs most commonly in middle aged to older dogs, except for those cases that have a probable familial basis, such as in Keeshonds and German shepherd dogs.2,14

In humans, primary hyperparathyroidism has been reasonably well documented in adults, with opinions divided on whether hyperplasia or adenomas are the more common diagnosis.7 Primary hyperparathyroidism is rare in children with both hyperplasia and adenomas having been documented.17 WCCCH has been documented in all age categories, from a reported case in a neonate, large case series of adults, as well as in the elderly.18-21 WCCCH has been reported as an O-allele associated condition in humans, as patients with primary hyperparathyroidism due to WCCCH frequently belong to blood group O.16 This may suggest a degree of inheritability in humans. In a large case series, multiple endocrine syndrome was documented in patients with chief cell hyperplasia, but this was not observed in any of the WCCCH cases.20 While chief cell hyperplasia has a female to male ratio of almost 2:1, WCCCH affects more men than women.20-22 WCCCH is rare but well-documented, water clear cell adenomas are exceptionally rare.20,21 In one case series where hyperplasia represented 15% of all primary hyperparathyroid cases, 22% of hyperplasia cases were of water-clear cell type with the remainder being chief cell hyperplasia.21 Therefore, in the latter case series, 3.3% of primary hyperparathyroidism cases were caused by WCCCH.

Reported presenting signs and clinical signs in cases of WCCCH in humans include recurrent renal calculi, impaired renal function and failure, gastrointestinal signs including nausea, vomiting, gastric or duodenal peptic ulcer disease, neuromuscular signs including fatigue, muscular weakness, mental symptoms including dementia (organic brain syndrome), and bone disease including rickets, cyst formation and subperiosteal resorption.1,10,11,21 These clinical signs are similar to those reported in cases of chief cell hyperplasia in humans, but occur at different ratios, for example nephrolithiasis is far more common in WCCCH than in chief cell hyperplasia.21

In the reported human cases of WCCCH, patients who presented with a primary hyperparathyroidism and associated hypercalcemia, sometimes also had hypophosphatemia.6,10 Normocalcaemic primary hyperparathyroidism due to WCCCH has also been documented, with total serum calcium concentration within the reference range, despite increased serum ionized calcium and PTH.24 One case report of a patient with parathyroid WCCCH presented with chronic hypocalcemia.25 Other reported concurrent laboratory abnormalities include increased alkaline phosphatase activity.2 Finally, WCCCH with concurrent parathyroid carcinoma has been noted to cause more severe hypercalcemia than other kinds of primary hyperparathyroidism.26 In one case series of people with primary hyperparathyroidism, the parathyroid glands of most patients with WCCCH weighed > 5 g, while those with chief cell hyperplasia generally weighed < 5 g.2 Thus, while water-clear cells produce less PTH/g of tissue, the typically larger overall mass in WCCCH results in a more pronounced hypercalcaemia.
than that seen in cases of chief cell hyperplasia.\(^2\)

The magnitude of the hypercalcaemia in the present case was comparable to that reported previously in dogs with primary hyperparathyroidism, with total serum calcium concentration slightly higher than the median value in a reported case series.\(^2\)

In the present case, an increased ionized calcium concentration was not documented after initiation of calciuresis by frusamide therapy. While it would have been ideal to confirm an ionized hypercalcaemia alongside an inappropriately high PTH value via radioimmunoassay, it was unnecessary for ultimate diagnosis of hyperparathyroidism. Discontinuation of frusamide-induced calciuresis to attempt to detect ionized hypercalcaemia was contraindicated, due to the risk of increasing morbidity prior to surgery\(^2\) and therefore was not attempted. It is also noteworthy that the accuracy of ionized calcium measurement can be affected by a multitude of factors, including sample storage and handling methods, pH adjustment and instrumentation utilised, all of which may contribute to spurious results for this test.\(^2\)

The post-operative hypocalcaemia reported in the present case is a well-recognised complication of surgical treatment of primary hyperparathyroidism, and typically occurs within 7 days of surgery.\(^1\)

The present case does not differ on a clinical scale from previously reported aetiologies of primary hyperparathyroidism in dogs. While not reported before, WCCH must be considered as a differential diagnosis for hypercalcaemia due to primary hyperparathyroidism.

**REFERENCES**


Angular limb deformity associated with injury to the metacarpal physis in a dog

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ABSTRACT
A 14-week-old female entire English bull terrier was presented for surgical evaluation of an angular deformity of the manus of the left thoracic limb. Radiographic assessment revealed injury to the physis of metacarpals III and IV with a resultant ALD and the dog was referred to the veterinary literature. This case report documents the successful surgical management of such an injury with corrective osteotomies, internal fixation and adjunctive external coaptation.

INTRODUCTION
Angular limb deformity (ALD) is a not-infrequent presentation in small animal surgical referral practice.1,2 ALD can result from a number of mechanisms, but in immature dogs it is often the result of premature closure of a long bone physis.3 When closure occurs in the physis of just one bone in a multiple bone model, or across only part of a physis, the consequences of continued growth from the remaining open physeal regions can include limb angulation, torsion and length discrepancy. Most frequently encountered in the antebrachium, ALD have also been demonstrated in other long bones, including the humerus, femur and tibia.1,3

To the authors’ knowledge, ALD secondary to premature closure of metacarpal (MCP) physis has not previously been described in the veterinary literature. This case report documents the successful surgical management of such an injury with corrective osteotomies, internal fixation and adjunctive external coaptation.

CLINICAL FEATURES
A 12-week-old female entire English bull terrier was initially presented to the referring veterinarian for evaluation of a left thoracic limb, non-weight bearing lameness, sustained after a fall from the owner’s arms. Physical examination on the day of injury revealed moderate soft tissue swelling of the manus and pain on direct palpation and manipulation of the MCP region. Non-steroidal anti-inflammatory medication (Meloxicam 0.1 mg/kg PO once daily; Metacam, Boehringer Ingelheim, North Ryde, NSW) and strict rest resulted in a marked improvement in the lameness within 48 hours. Seven days after the injury, the owner noticed that the dog’s paw was beginning to deviate laterally and the dog was re-presented to the referring veterinarian. Radiographs demonstrated injury to the physis of MCP III and IV with a resultant ALD and the dog was referred to the authors’ institution for ongoing care.

On physical examination at referral presentation, 14 days after the injury, the dog was ambulatory on the left thoracic limb, with a mild to moderate weight-bearing lameness. Moderate valgus deformity of the left manus was noted and the dog was primarily weight-bearing on the medial aspect of digital pads II and III. Firm swelling and pain was elicited with direct palpation over the distal MCP region, however gross instability was not present. Repeat radiographs under sedation with acepromazine (0.02 mg/kg IV; ACP, Delvet Pty Ltd, Seven Hills, NSW) and butorphanol (0.1 mg/kg IV; Butorgesic, Ilum, Troy Laboratories (Australia) Pty Ltd, Glendenning, NSW) were obtained and were similar in appearance to the referring veterinarian’s radiographs and indicated damage to the physis of MCP III and IV (Figure 1). The physis of MCP III and IV were widened, with adjacent radiolucency in the metaphyseal bone, a surrounding region of reactive, sclerotic bone and valgus angulation of the epiphyses and distal digits. Mild lateral displacement of the epiphyses of MCP III and IV was appreciated.

Joint orientation lines, anatomic axes and resultant joint orientation angles were determined by comparison with radiographs of the right manus to quantify the length discrepancy and angular deformity of each MCP, consistent with centre of rotation of angulation (CORA) methodology.4 MCP II-V measured 44, 49, 47 and 41 mm and 44, 52, 50 and 44 mm for the left (affected) and right (unaffected) thoracic limbs respectively, confirming a 3 mm length discrepancy in MCP III-V. The anatomical lateral distal MCP angle (aLDMA) measured 90° for each of MCP II-V of the right manus; in comparison the aLDMA for MCP II and V of the left manus were also 90°, while the aLDMA for MCP III was 72° and the aLDMA for MCP IV was 78°, demonstrating an 18° and 12° valgus deformity for each respective bone. The CORA for MCP III and IV was determined, with a location 13 mm proximal to the metacarpophalangeal joint in MCP III and 20 mm proximal to the metacarpophalangeal joint in MCP IV. Given the degree of angular deformity and lameness, coupled with the possibility of deformity progression due to remaining residual growth potential of the unaffected MCP II, corrective osteotomy surgery was advised.

Twenty-two days after the injury, the dog underwent surgery. The dog was sedated with acepromazine (0.04 mg/kg SC) and methadone (0.5 mg/kg SC; Methone, Parnell, Mascot, NSW) and anaesthetised with intravenous alfaxalone (1.3mg/kg IV; Alfaxan, Jurox Pty Ltd, Rutherford, NSW). Intubation was achieved with a cuffed 7-mm endotracheal tube and general anaesthesia maintained on isofluorane (Isoflo, Abbott Animal Health, Abbott Park, IL, USA) in 100% oxygen. A brachial plexus nerve block (bupivacaine HCl 2 mg/kg; Bupivicaine, Pfizer Animal Health, Zoetis, Rhodes, NSW) was administered and...
peri-operative antibiotic prophylaxis consisting of cephazolin (22 mg/kg IV; Kefzol, Aspen Pharmacare, St Leonards, NSW) every 90 minutes was initiated at induction and continued for 6 hours after surgery. The left thoracic limb was clipped from the shoulder, distally, to include the digits. Aseptic surgical skin preparation was performed and the dog was positioned in dorsal recumbency in a hanging limb position. After four-quadrant draping, a tourniquet, comprising multiple tight layers of sterile self-adhesive conforming bandage (Vet-wrap, 3M Australia, North Ryde, NSW), was applied from the toes proximally to the mid antebrachium. Application time was noted to ensure the tourniquet duration did not exceed 90 minutes. The tourniquet was removed from the limb distal to the level of the carpus prior to skin incision. A previously described dorsal approach was employed to allow access to the diaphyses and heads of MCP bones II-V.1

Direct palpation and manipulation of the epiphyses of MCP III and IV failed to demonstrate instability, and a fracture line was not discernible. The digital extensor tendon was elevated from the dorsal surface of MCP III and retracted with paired Senn-Miller retractors. The osteotomy site, 20 mm proximal to the metacarpophalangeal joint, was marked with an electrocautery pencil (Valleylab electrosurgery pencil, Covidien Ltd, Dublin, Ireland). The periosteum was sharply incised with a scalpel and bluntly reflected with a Freer periosteal elevator. The periosteum and interosseous muscles were retracted with, and protected by, paired mini Hohman retractors, placed medially and laterally at the osteotomy site. An oscillating bone saw (E Pen, DePuy Synthes, North Ryde, NSW) was employed, under sterile saline irrigation, to perform a transverse osteotomy. Osteotomies were created at a similar level in MCP IV and MCP V using the same surgical technique.

An opening wedge osteotomy of 15°, as measured with an angle template (TPLO wedge templates, Veterinary Instrumentation Limited, Sheffield, UK), was created in MCP III and stabilised with small bone holding forceps. Visual assessment of the phalanges and reference to MCP II was employed to confirm alignment. An 8-hole, 1.5-mm veterinary cuttable plate (Veterinary Instrumentation Limited) was contoured and placed on the dorsal surface of MCP III, and secured with cortical self-tapping screws (Veterinary Instrumentation Limited). A screw hole was left unfilled over the osteotomy site. Metacarpal IV was stabilised in an identical manner, while the osteotomy performed in MCP V was not stabilised. A mid-diaphyseal ostectomy, entailing removal of approximately 6 mm of bone and surrounding periosteum, was performed in MCP II, and an autogenous free fat graft, harvested from the lateral brachial region, was placed in the ostectomy gap.

Figure 1. Dorso-palmar (a) and medio-lateral (b) radiographs of the left manus at referral presentation. The physis of MCP III and IV appear damaged, with subsequent angulation and shortening of each respective bone. The right manus is shown for comparison (c and d).

Figure 2. Dorso-palmar (a) and medio-lateral (b) post-operative radiographs. The MCP are well aligned. A mild medial translational deformity is present in MCP III due to location of the osteotomy proximal to the CORA and insufficient lateralisation of the distal fragment.
The subcutaneous layer was apposed with 4-0 poliglecaprone 25 (Monocryl, Ethicon, Johnson & Johnson Medical Pty Ltd, North Ryde, NSW) in a simple continuous pattern. A continuous intradermal suture of the same material was utilised to appose the skin. Post-operative radiographs showed acceptable limb re-alignment and implant placement (Figure 2). External coaptation, incorporating a palmar splint constructed from 6 layers of a thermoplastic resin impregnated mesh cotton splint material (Vet-Lite 5 cm, Runlite SA, Belgium) was placed to augment the surgical repair.

The dog was discharged from the hospital the following day, with instructions to continue daily oral administration of meloxicam at a rate of 0.1 mg/kg once daily for 7 days and to maintain strict cage confinement for 4 weeks. Weekly scheduled rechecks and bandage changes were performed with unremarkable findings.

Four weeks after the surgery, the manus was noted to be well aligned. Mild flexor tendon laxity of the carpus and manus was documented, in conjunction with a mild to moderate carpal valgus and torsional deformity, seemingly centred over the distal antebrachium. Radiographs detailed osseous union of the MCP III-V osteotomy sites and stable implants, while the MCP II ostectomy remained unhealed (Figure 3). The physes of MCP II-V appeared radiographically closed, while the length of MCP III-V was unchanged from the initial radiographs obtained at presentation. It was noted that the distal ulnar physis appeared to be closing prematurely, with a large region of lysis in the adjacent metaphyseal bone. This was deemed to be the cause of the new ALD.

Surgical intervention was recommended to address the consequences of altered physeal growth at the distal ulnar physis and was performed 5 days later. Anaesthesia and surgical site preparation were performed as previously described. After placement of a self-adhesive bandage tourniquet (Vet-wrap) from the toes to the proximal antebrachial region, a lateral approach to the distal ulna diaphysis was employed to allow a distal ulna ostectomy. Approximately 15 mm of distal ulna with its surrounding periosteum was excised with an oscillating bone saw. An autogenous free fat graft was harvested from the lateral brachial region and placed in the osteotomy defect. A soft padded bandage was placed post-operatively and the dog discharged from hospital that afternoon. The bandage was removed 3 days after surgery.

At revisit examination 4 weeks after ulnar ostectomy, the dog had no evidence of lameness in the left thoracic limb, with no evidence of ALD or flexor tendon laxity. Moderate soft tissue swelling was noted over the MCP implants with mild discomfort elicited on palpation. At final recheck, performed approximately 22 weeks after the initial injury, the dog had appropriate limb alignment with no evidence of lameness. Mild soft tissue swelling was present over the MCP implants, with no evidence of pain on direct palpation. Radiographs revealed resolution of the ALD and persistence of the distal ulnar ostectomy gap (Figure 4). The implants were unchanged in their radiographic appearance, while synostoses were noted between MCP II and III and between MCP III and IV. Length of MCP II-V of the left limb was measured at 42, 48, 46 and 41 mm respectively, compared with 47, 55, 53 and 46 mm for the right limb, while the left radius measured 137 mm in length compared to 129 mm for the right.

**DISCUSSION**

The present report describes an unusual ALD presentation that appeared to develop as a consequence of injury to the physes of MCP III and IV in an immature dog. Due to the cartilaginous composition and hence fragile nature of growth plates, they remain a point of weakness in juvenile patients.7 Salter and Harris described a classification system for fractures of the juvenile physis in humans,8 which remains widely used in the veterinary field. Unfortunately, the lack of radiographs at the initial presentation, coupled with the unusual radiographic appearance of the fractures 2 weeks after the injury, made it difficult to accurately classify the fracture configuration in the present case. For the same reason it cannot be...
stated with certainty that underlying MCP metaphyseal lesions were not present before the trauma, which subsequently either contributed to the physeal fractures, or were merely revealed after presentation for the fall. Nevertheless, it appears likely that the initial traumatic event resulted in premature closure of the physes of MCP III and IV, which, with or without initial angular displacement of the epiphyses, culminated in shortening and angulation of the MCP III and IV and lateral deviation of the manus. While an injury to the physe of MCP V was not evident radiographically, serial radiographs demonstrated a progressive length discrepancy in the bone, when compared with the right MCP V, indicating that some interference with physeal growth may have occurred.

The single physe located in the head of MCP II-V has been demonstrated to close between 5 and 7 months of age in medium-sized dogs. Surgical correction was recommended in the present case, due to the degree of mechanical dysfunction and the potential for progressive deformity.

The CORA was calculated pre-operatively at a position 13 mm and 20 mm proximal to the distal end of MCP III and IV respectively. Paley’s first rule of osteotomies states that creation of the correction osteotomy at the CORA enables appropriate realignment of the bone with angulation only. In the present case, positioning of the osteotomy in MCP III at the site of CORA would have resulted in sub-optimal implant purchase and implant-related complications, such as interference with the metacarpophalangeal joint, the digital extensor tendons and the proximal palmar sesamoid bones. The osteotomy was therefore created 7 mm proximal to the measured CORA, at a level adjacent to the CORA of MCP IV. In an attempt to ensure co-linearity of the axes, in accordance with Paley’s second rule of osteotomies, the distal fragment was translated slightly laterally at the level of the osteotomy. Unfortunately, optimal implant positioning limited the degree of distal fragment lateralisation and a mild translational deformity remained post-operatively. This did not appear to have any clinical manifestation.

The radiographic appearance of the MCP III and IV physes indicated generalised disruption to the physes, with a likely resultant complete closure. The lack of longitudinal growth in MCP III-V in the follow-up radiographs confirmed this supposition. For this reason, it was not felt necessary to span the physes with the implants and obtain purchase in the epiphyseal bone. However, if an asymmetrical closure of the physes had been present, continued growth from the undamaged regions could have occurred and recrudescence of the deformity may have resulted.

Corrective osteotomy of MCP bones have not been described previously in the veterinary literature, however, several retrospective studies have been published that detail the management of MCP fractures in the dog, with therapy consisting of either surgical stabilisation or non-surgical management with external coaptation. Surgical stabilisation of MCP fractures is recommended if more than two MCP bones are involved, if the two central MCP are fractured, if the MCP fractures are articular, if the fracture fragments are displaced more than 50%, if the fracture involves the base of MCP II or V, or if the dog is a large-breed, athletic or show dog. In the present case, accuracy correction of the angular deformity necessitated precise osteotomy placement and surgical stabilisation.

Surgical stabilisation techniques for diaphyseal MCP fractures include bone plate and screws, intramedullary pins, dowel pins, external skeletal fixation and lag screws. In the present case, 1.5-mm veterinary cuttable plates with 1.5-mm cortical self-tapping screws were selected because the high screw density enabled adequate implant purchase in the relatively short section of justa-articular bone.

Internal fixation of MCP V was not performed, as it was anticipated that the fixation of MCP III and IV alone, in conjunction with external coaptation, would provide sufficient stability to engender uncomplicated clinical union, as per the recommendations for minimally displaced diaphyseal fractures of MCP II and/or V. A partial ostectomy of MCP II was performed to allow manus realignment and to prevent further longitudinal growth in the bone via its undamaged physe. It was therefore considered important to prevent premature healing of this osteotomy, prior to normal cessation of physeal growth. Documented methods of preventing bony union include removal of a critical amount of bone, typically greater than 1.5 times the diaphyseal diameter, excision of all periosteum and placement of a physical barrier such as an autogenous fat graft. With the partial ostectomy of MCP II, the periosteum was circumferentially excised and a fat graft placed, but a critical defect was not created. The justification for this decision was the desire for MCP II to eventually heal, albeit at a slower than normal rate. This aim was realised on sequential radiographs, with fracture gap persistence and physeal closure seen at 4 weeks post-operatively, and healing of the osteotomy demonstrated at 5-month follow-up. This is in comparison to the partial ostectomy of the distal ulna, whereby a critical defect was created, in addition to periosteal resection and fat graft placement. As non-union of an ulnar diaphyseal deficit does not appear to result in clinically adverse sequelae, prevention of ulnar union prior to distal radial physeal closure was considered the prime objective and the fracture gap was noted to have persisted at the final radiographic evaluation.

The dorsal position of the bone plates on the compression side of the MCP bones place them in a suboptimal position biomechanically, with the subsequent potential for implant failure. It is for this reason that the authors elected to supplement the repair with external coaptation, in line with previously documented guidelines. Synostosis between healed MCP bones is frequently seen after MCP fracture, with a recent retrospective study that evaluated the long-term prognosis of MCP and metatarsal fractures finding that 62% of dogs with 3 or 4 fractured bones that were treated with surgery had demonstrable synostoses at radiographic follow-up. In the present case, synostoses were noted between MCP II and III and between MCP III and IV at final radiographic evaluation, 22 weeks after the injury (20 weeks after surgery). However, these synostoses did not result in any clinical signs of pain or lameness.

At final radiographic evaluation, the lengths of the left MCP III, IV and V were 6.5, 6.5 and 4 mm less than the corresponding MCP bones of the right limb. At the same time, interestingly, the left radius measured 8 mm longer than the right. Termed compensatory growth, this increased growth rate in adjacent bones has been described previously in dogs that experienced premature physeal closure and subsequent comparative bone shortening.

The aetiopathogenesis of premature closure of the distal ulnar physeis, the ulnar metaphyseal lesion, and the subsequent antebrachial ALD remains unknown. While early physeal closure was documented in the present case, a radiographic reason for the closure was not seen, and the appearance of the metaphyseal lesion is unusual. Previously documented reasons for premature closure of the distal ulnar physeis include trauma, hypertrophic osteodystrophy, retained cartilaginous cores and heritable idiopathic closure. No episode of significant trauma was noted in the 4 weeks following the index surgery and the limb was protected by a bandage and palmar splint. It remains possible...
that the splint in some way contributed to the closure via direct compressive injury to the physis, although no discomfort or soft tissue injury from the bandage was seen at any stage. Traumatic injury to the distal ulnar physis sustained at the time of the original MCP physeal fractures is also unlikely, given the lack of clinical and radiological evidence over the 2-week period after the incident. However, a mild or focal injury that created minimal initial radiographic pathology and resulted in only slowing, but not cessation, of physeal growth remains a possibility. A congenital cause is possible, however this also seems unlikely given that the contralateral limb remained unaffected and that the condition has, to the authors’ knowledge, not been previously reported in the English bull terrier.

A previous study on ALD secondary to premature closure of the distal ulnar physis demonstrated that, in mildly affected cases, distal ulnar osteotomy alone can result in resolution of the ALD. A congenital cause is possible, however this also seems unlikely given that the contralateral limb remained unaffected and that the condition has, to the authors’ knowledge, not been previously reported in the English bull terrier.

Similarly, in the present case, timely intervention via release of the constraining effect brought about by retarded ulnar growth allowed radial development to continue unimpeded and limb re-alignment to occur.

**CONCLUSION**

The present case report describes the successful surgical management of an unusual post-traumatic MCP ALD by opening wedge osteotomy, internal bone plate fixation and external coaptation. Re-alignment of the manus was achieved and bony union proceeded without complication. Premature closure of the distal ulnar physis complicated recovery, however, subsequent distal ulnar osteotomy allowed resolution of the developing antebrachial ALD and restoration of normal limb function.

**REFERENCES**

The present report describes a case of pneumorrhachis, associated with hemivertebrae, that spontaneously resolved. A 4-year-old, female, neutered, French Bulldog was presented with hind limb paresis of one week’s duration. A lesion was localised to the T3-L3 spinal cord segments by neurological examination. Plain radiographs revealed hemivertebrae at T7, T8, T10 and T11. Computed tomography (CT) myelogram demonstrated mild osseous spinal cord compression at T7-8 and T10-11. A hypoattenuated spherical lesion in the epidural space associated with the hemivertebrae of T10-11 was identified by Hounsfield values as gas (pneumorrhachis) and was suspected to be contributing to clinical signs of spinal cord compression. The dog was treated conservatively and follow-up imaging demonstrated the pneumorrhachis had resolved.

INTRODUCTION
Pneumorrhachis is the presence of intraspinal gas, which can be categorised as primary or secondary, and aetiolologically divided into iatrogenic, traumatic or atraumatic.1,2 Pneumorrhachis has been variously described as spinal pneumocele, spinal pneumatosis, spinal emphysema, aerorrhachia, pneumosaccus and pneumomyelogram.1,2 Pneumorrhachis is most frequently associated with trauma in humans and is reported concurrently with head trauma, skull fractures, vertebral fracture/luxations, pneumothorax, chest and pelvic injuries.3-9 Iatrogenic pneumorrhachis has been associated with epidural injections, thoracostomy tube placement, lung tumour resection and craniotomy.10-14 Respiratory conditions such as asthma, which create significant pressure differentials, have been reported to induce pneumomediastinum and subsequent communicating pneumorrhachis.15 The latter pathophysiologic process has been proposed to explain spontaneous pneumorrhachis following the lifting of heavy objects.16 Epidural empyema has been reported to lead to epidural gas formation.17 The vacuum phenomenon described in degenerate intervertebral discs is also associated with pneumorrhachis.18-22 There are many reports of vacuum phenomenon in the veterinary literature, as well as iatrogenic cases, but the only report of non-iatrogenic pneumorrhachis is a case of spontaneous pneumorrhachis in a Rottweiler.23 It was treated by hemilaminectomy.

The current case report describes a case of a spontaneous pneumorrhachis, secondary to hemivertebrae and vacuum phenomenon.

CASE HISTORY
A 4-year-old, 10.6-kg, female, neutered French Bulldog was presented with a 7-day history of acute onset pelvic limb weakness. Treatment had been initiated by the referring veterinarian at the onset of these signs with oral carprofen (Rimadyl 20 mg, Pfizer Australia, West Ryde, NSW), at a dose of 20 mg q 12 hours for 4 days then 20 mg q 24 hours for 3 days, and a recommendation of rest. Pelvic limb function slowly improved over the 7-day period prior to presentation at our hospital. Routine examination of the chest and abdomen was unremarkable. The dog’s vital signs were within reference limits. Orthopaedic examination revealed a grade 2 left medial patella luxation. Neurologic examination revealed delayed bilateral pelvic limb proprioceptive paw positioning with exaggerated patellar, sciatic and cranial tibialis reflexes. Cutaneous trunci reflex was intact and the thoracic limb function was normal. Voluntary motor function was present in the hind limbs, however, although the dog could stand, it could not ambulate without assistance. No spinal pain was noted. Based on these findings a lesion was localised to the T3-L3 spinal cord segments. The owner elected not to proceed with imaging at that stage. The dog was treated with ongoing medical management, which consisted of cage rest and oral carprofen (1 mg/kg q 12 hours for 3 days, then 1 mg/kg q 24 hours for 4 days).

The dog was re-presented 10 days later with acute onset worsening of the pelvic limb paresis. Voluntary motor function was still present but had deteriorated. The dog still exhibited upper motor neuron signs to the hind limbs. The cutaneous trunci reflex was intact to the level of T13-L1 and mild discomfort was now noted on palpation of the spine in the thoracolumbar region.

Radiography (Fujiﬁlm FDR D-EVO (DR-ID 600), Fujifilm Corporation, Tokyo, Japan) of the thoracolumbar vertebral column revealed hemivertebrae and spinous process malformations at T8, T10 and T11, as well as multiple spinous process malformations throughout the remainder of the thoracic vertebral column. Computed tomography (CT; Siemens Somatom Emotion, Siemens AG, Munich, Germany) was performed (130 kV, 25 mA, 1.0 mm slice thickness, 16-slice helical acquisition) in dorsal recumbency and demonstrated mild vertebral canal narrowing at T7-8 and T10-11 adjacent to the congenital vertebral deformities. A hypoattenuated region was noted in the right lateral spinal canal at the level of the T10-11 intervertebral disc space, with a second, smaller hypoattenuated region 6 mm caudal to the former region but on the left (Figure 1A-B).

A myelogram was performed using iohexol contrast agent (Omnipaque, 300 mg/ml iiodine, GE Healthcare Australia, Rydalmere, NSW) introduced via the L5-6 intervertebral space (Figure 2). CT myelography identified minor extradural ventral spinal cord compression from the hemivertebrae malformations at the T7-8 location. Further minor extradural ventral compression of the spinal cord was identified from the hemivertebrae malformations at the T10-11 site. A hypoattenuated (-990 HU; Hounsfield units) spherical lesion in the right epidural space, that prevented contrast from filling the subarachnoid space between the lesion and spinal cord, was noted at this site (Figure 3A).
Figure 1. Transverse (1A) and sagittal (1B) CT images of the vertebral column at the level of the T10-11 intervertebral disc space, taken in dorsal recumbency, showing a hypoattenuated region in the spinal canal (black arrows).

Figure 2. Lateral myelogram of the thoracolumbar spine. Note the vertebral malformations at T7-8 and T10-11, as well as the malformations of the dorsal spinous processes.

The lesion occupied nearly 25% of the cross-sectional area of the vertebral canal and extended into the right intervertebral foramen. A second smaller lesion was noted on the left about 6 mm caudal to the larger one, but was not causing similar compression and collapse of the subarachnoid space adjacent to the spinal cord (Figure 3B). A hypoattenuated region was also noted in the ventral aspect of the T10-11 intervertebral disc space (Figure 4A-B). The Hounsfield values for these lesions were consistent with gas.

A diagnosis of extradural pneumorrhachis and vacuum phenomenon was made.

Figure 3. Transverse CT myelogram images of the vertebral column at the level of the T10-11 intervertebral disc space, taken in dorsal recumbency, showing an extradural hypoattenuated region in the spinal canal (black arrow), compressing the right subarachnoid space (3A). A similar, smaller lesion (black arrow) appears 6 mm caudal and on the left of the compressive lesion (3B).

Figure 4. Transverse (4A) and sagittal (4B) CT myelogram images of the vertebral column, taken in dorsal recumbency, showing a hypoattenuated region within the T10-11 intervertebral disc space (black arrows).
at the T10-11 intervertebral disc space was made. It was suspected the pneumorrhachis was contributing to the clinical signs, because intervertebral disc-associated compression was not evident and the osseous changes did not appear to significantly compress the spinal cord. Gabapentin (10 mg/kg q 24 hours; Neurontin 100 mg, Pfizer, West Ryde, NSW) was prescribed for mild spinal pain. Treatment with omeprazole (1 mg/kg q 24 hours; Losec 10 mg, AstraZeneca, North Ryde, NSW) was initiated to prevent gastric ulceration when changing from carprofen to prednisolone. The carprofen course was discontinued and 48 hours later prednisolone (Pred-X 5 mg, Apex Laboratories, Somersby, NSW) was prescribed (tapered dose starting at 1mg/kg q 12 hours for 5 days), due to clinician preference over carprofen for presumed inflammation in the affected region of the spinal cord. Cage rest was continued as previously advised.

Three days later the dog was ambulatory. CT was repeated under sedation with medetomidine (0.02 mg/kg IV; Domitor, Pfizer Animal Health, West Ryde, NSW) in each of sternal recumbency, left lateral recumbency and right lateral recumbency, to determine if the improved clinical signs could be attributed to decompression of the spinal cord due to the gas being resorbed or moved. A myelogram was not performed at this visit, so as not to expose the dog to unnecessary risk of myelogram-related complications and the requirement for general anaesthesia for myelography. The imaging demonstrated that the larger lesion on the right had resolved, but the smaller lesion on the left remained (Figure 5A-B). With the dog positioned in each of left lateral recumbency and right lateral recumbency it was noted that the T10-11 intervertebral disc space had a marked increase in the amount of gas compared to the previous CT, however, in sternal recumbency the gas was not visible (Figure 6A-B). The major pneumorrhachis lesion was not appreciable with the dog in any of the positions tested. It was decided not to proceed with decompressive or stabilizing surgery at this stage.

Figure 5. Transverse CT myelogram images of the vertebral column at the level of the T10-11 intervertebral disc space, taken in dorsal recumbency, showing resolution of the pneumorrhachis which had previously compressed the subarachnoid space (5A). The smaller lesion (black arrow) has reduced in size compared to the previous images but is still present (5B).

Figure 6. Paramedian (6A) and median (6B) CT views of the vertebral column showing that gas within the intervertebral disc space is visible when the image was acquired with the dog in right lateral recumbency (black arrow; 6A), but not when in sternal recumbency (6B).

One week following the diagnosis of pneumorrhachis the dog was freely ambulatory without support. There was slightly reduced conscious proprioception in the pelvic limbs. There was no pain response noted on palpation of the thoracolumbar region. The dog was re-presented 6 weeks later for review. The prednisolone dose had been tapered to 1 mg/kg q 48 hours. Ataxia was noted but assessed as minimal. There were slightly increased patellar, sciatic and cranial tibial reflexes in the pelvic limbs, and a normal cutaneous trunci reflex. CT was performed and the pneumorrhachis was no longer evident. Prednisolone therapy was discontinued. The dog was re-presented 6 months later when neurological examination revealed no deficits.

DISCUSSION
The most likely cause of the pneumorrhachis in the present case was thought to be gas escaping from the degenerate T10-11 intervertebral disc. By definition, Hounsfield values for water and air are arbitrarily set at 1 HU and -1000 HU respectively. In the present case, the Hounsfield
value for the hypoattenuated compressive lesion was -990 HU, confirming the lesion had a density similar to air (gas). The Hounsfield value for the intervertebral disc space when in sternal recumbency on the second CT was -400 HU, which is comparable to lung and likely indicates CT volume averaging of gas and other adjacent tissues. Vacuum phenomenon results from negative pressure in a potential cavity which fills with nitrogen from the surrounding extracellular fluid and is known to occur in degenerate intervertebral discs. Gas retrieved from a disc by a closed system was demonstrated to be over 90% nitrogen in one study, which is higher than the atmospheric concentration, suggesting the origin is not gas. Intervertebral disc degeneration was noted at multiple sites in the present case, however, narrowing of the vertebral canal at these locations was minor. Vertebral malformations are common among brachiocephalic breeds and were recently reported to be the cause of neurological signs in over 40% of cases in one study. Conversely, it should be noted that the finding of vertebral malformation on plain radiographic films does not confer a diagnosis of spinal cord compression at that site. Intervertebral disc degeneration as a result of hemivertebrae is well documented and vacuum phenomenon is frequently associated with disc disease, however, pneumorrhachis specifically in association with intervertebral disc degeneration at the site of a hemivertebra in the dog has not been reported. In the present case, gas within the intervertebral disc space was not noted with the dog imaged in ventral recumbency, but it was noted in left lateral recumbency, right lateral recumbency and dorsal recumbency. Spinal extension, which increases negative pressure within a disc, has been reported to give rise to intervertebral disc vacuum phenomenon in humans. We hypothesise that instability related to the malformation (hemivertebrae) may lead to a variable pressure within the intervertebral disc space. It is possible that the malformed vertebrae exhibited similar variable pressure changes when in different positions. The positioning of the dog between CT examinations did not cause the pneumorrhachis to reform. Other possible causes of pneumorrhachis were not evident in the present case. The dog had no history of trauma, and neither pneumothorax nor pneumomediastinum were evident on CT. The lesion was visible prior to performing lumbar puncture and no surgery had been performed. The spherical shape of the lesion would indicate that it was encapsulated rather than dispersed within the epidural space. Although the dog was a brachiocephalic breed, the dog’s history and clinical examination did not suggest a respiratory cause. Discospondylitis was excluded due to clinical signs and absence of end-plate lysis, and there were no clinical or CT findings suggestive of spinal empyema. The CT myelogram indicated a spinal cord gas lesion at a site consistent with the neuroanatomic localisation. Although most cases of epidual pneumorrhachis in humans are asymptomatic, several reports exist which demonstrate neurologic signs attributable to gas in the spinal canal. The only reported veterinary case of non-iatrogenic pneumorrhachis was attributed to vacuum phenomenon associated with degenerative disc disease. That report described treatment with a hemilaminectomy because of worsening neurological signs. The decision not to operate in the current case was made because of the improvement in clinical signs and the unpredictable outcomes following hemilaminectomy in dogs with hemivertebrae. Destabilisation of the vertebral column at the site of the hemivertebrae was considered a significant risk following hemilaminectomy. Concurrent decompression and stabilisation of the affected vertebrae using pins and polymethylmethacrylate was planned, had medical therapy failed. It was thought that stabilisation of the vertebrae may also prevent worsening of the vacuum phenomenon by preventing sudden pressure changes within the intervertebral disc space. Most cases of pneumorrhachis in humans resolve spontaneously by absorption of the gas and treatment is usually directed towards the inciting cause, if evident. Oxygen therapy and hyperbaric treatments have been used to help speed up absorption in specific cases. Aspiration of the extradural gas was reported to temporarily resolve signs in one report, but clinical signs and gas returned and surgery was eventually performed. Percutaneous CT-guided laminar trephination has also been reported to treat cauda equina syndrome resulting from epidural pneumorrhachis.

Other possible causes of the clinical signs in the current case would include intervertebral disc protrusion or extrusion, spinal cord fibrocartilagenous embolism, various forms of meningitis, arachnoid diverticulae, syringomyelia and spinal cord neoplasia. CT and CT myelogram were able to rule out significant spinal cord compressive lesions, including disc compression, and neoplastic and granulomatous masses. Magnetic resonance imaging (MRI) may have been useful in this case, to better image the spinal cord parenchyma and thereby help rule out differential diagnoses such as fibrocartilagenous embolism, syringomyelia and intramedullary spinal cord neoplasia. The medical management used in the present case may have initially improved the clinical signs for many neurological diseases affecting the spinal cord, so a diagnosis of spinal cord compression as a result of pneumorrhachis is not definitive. However, due to a lack of other diagnostic findings, it is believed that the clinical signs may have been a result of eruption of gas into the epidural space causing spinal cord compression injury. The anti-inflammatory effect of carprofen and prednisolone may have improved clinical signs in the present case, by reducing spinal cord inflammation and oedema secondary to compression; however, it is unlikely to have contributed to the regression of the pneumorrhachis and is not reported to reduce vacuum phenomenon in humans. The long-term prognosis for the present case is unknown, despite the dog being clinically normal at its last examination. Disc extrusion and osseous spinal cord compression at the site of vertebral malformations carry a higher risk than for normal intervertebral disc spaces, but the recurrence rate of pneumorrhachis in the dog is not reported.

**CONCLUSION**

The present report details a case of suspected spinal cord compression as a result of pneumorrhachis, which may have been secondary to spinal malformation and vacuum phenomenon. The clinical signs resolved with medical management alone. Pneumorrhachis should be considered as a possible cause of acute neurologic signs in dogs with vertebral malformations and associated intervertebral disc degeneration, which, in the face of improving clinical signs, may not require surgical decompression.

**REFERENCES**


Marbofloxacin is a synthetic, broad spectrum bactericidal agent. The bactericidal activity of marbofloxacin is concentration-dependent, with susceptible bacteria cell death occurring within 20–30 minutes of exposure.

Like other fluoroquinolones, marbofloxacin has demonstrated a significant post-antibiotic effect and is active in both stationary and growth phases of bacterial replication. High sensitivity for the difficult otitis externa causing gram negative bacteria *P. aeruginosa*, *Proteus*, *Klebsiella*, *E.coli.*

Clotrimazole is an antifungal of the ‘azole’ group. The primary mechanism is against dividing and growing fungi.

Dexamethasone acetate is a synthetic anti-inflammatory corticosteroid. The comparative anti-inflammatory activity of corticosteroids is reported as:

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<th>Corticosteroid</th>
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<td>Hydrocortisone</td>
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Managing inflammation and allergic reaction in the ear is important for the resolution of otitis externa.

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2. Data on file
3. Data on file
4. APVMA – External Review
6. www.pharmacoevidence.com
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*Cegedim: Veterinarians Comfortis Survey December 2012 (Australia) commissioned by Elanco. Survey data on file. Statistically significant difference in one-tailed test versus the number two veterinary recommended flea product for dogs.

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What is your diagnosis?
Blood smear from a cat

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SIGNALMENT AND HISTORY
A 10-year-old female spayed ragdoll cat was presented to Murdoch University Veterinary Hospital following episodes of acute collapse over several
days and a 6- to 8-week history of facial swelling. Dexamethasone and chlorpheniramine injections had been administered the previous day. There
was a grade 1/6 left parasternal heart murmur and a 10 mm diameter, firm swelling on the left side of the nose. An unclassified cardiomyopathy
was diagnosed by echocardiogram. A complete blood count showed a relative mild mature neutrophilia, moderate lymphopenia and absolute
eosinopenia, indicative of a stress/corticosteroid response. A mild thrombocytopenia was also present. A blood smear was examined, with 4% of the
leukocytes having the appearance of those in Figure 1.

Figure 1: Cells from peripheral blood smear. Wright-Giemsa stain, 1000x magnification.

DIAGNOSTIC CHALLENGES:
What are your differential diagnoses for the cells of concern on the blood smear and what further diagnostic steps would you take?

Diagnostic findings and interpretation
The cells are neutrophils. The cytoplasm of approximately 5% of the
segmented neutrophils contained 1-10 basophilic to amphophilic
to slightly eosinophilic, round to smudged granules of 0.5-1 µm
in diameter (as seen in Figure 1). These granules were not seen
within any other cells. Occasional large platelets were also observed. Differential diagnoses included May-Hegglin anomaly, congenital
mucopolysaccharidosis, or a Birman cat-like granulation anomaly.1-3
Chédiak-Higashi disease was considered unlikely given this was not
a Persian cat, and the granules were only within neutrophils and no
other leukocytes or platelets.1-3 The small granules were of somewhat
variable size, shape and colour, thus phagocytosed bacteria were
unlikely. The cat did not have any prior blood smears with which
to compare.
Fine needle aspirates of the cutaneous nasal mass indicated a
well-differentiated mast cell tumour. Due to the findings of neutrophil granulation with mild
thrombocytopenia and large platelets, May-Hegglin anomaly was
suspected. To investigate, Dr Shinji Kunishima of the Department
of Advanced Diagnosis, Nagoya Medical Center, Japan, performed
myosin IIA immunofluorescence on blood smears from this cat and
a control cat. The granules were negative for myosin IIA, ruling out
May-Hegglin anomaly. A congenital mucopolysaccharidosis was
considered unlikely due to lack of concurrent clinical signs such as
facial deformity, and that granules were not detected within other
leukocytes. A Birman cat-like granulation anomaly was considered,
however, such granules stain red on Wright-Giemsa smears, and are
smaller than those seen in the present case.
Cytochemistry was performed on air-dried blood smears to further
characterise the chemical content of the granules. A toluidine blue
stain produced metachromatic staining of the granules (Figure
2). A single mast cell was also detected (Figure 2). Mast cells were
not detected following examination of two buffy coat smears.
Metachromasia is the result of polymerisation of large anionic
molecules within the granules of mast cells and basophils,
follow the binding of large amounts of toluidine blue.4
The granules in mucopolysaccharidoses also stain positively with toluidine blue, so staining characteristics need to be considered in light of the clinical presentation and concurrent findings.\textsuperscript{2}

Based on the cutaneous mast cell tumour, the very rare mast cells in the peripheral blood, and the positive toluidine blue staining of the granules within the neutrophils, a diagnosis of phagocytosis of mast cell granules by peripheral blood neutrophils was made. The mast cell granules may have originated from circulating mast cells, or from degranulated mast cells within the cutaneous tumour following examination and manipulation. The owner opted for no further investigation or treatment, and the cat was subsequently euthanased. A post mortem was not permitted.

COMMENTS

Mastocythaemia, or the presence of circulating mast cells in the peripheral blood, is an uncommon finding in cats and is most often associated with systemic mastocytosis (mast cell tumours) or, rarely, other neoplasms.\textsuperscript{3} This is in contrast to dogs, where mastocythaemia may be seen in a variety of inflammatory as well as neoplastic diseases.\textsuperscript{4} Evaluation of a smear made from the leukocyte-rich buffy coat of a spun microhaematocrit tube is typically a more sensitive method of detecting mastocythaemia than by blood smear examination alone.\textsuperscript{5} Toluidine blue staining may be a helpful adjunct.

In the present case, only a single cutaneous mast cell tumour was identified. In the cat, solitary dermal mast cell tumours are usually behaviourally benign, although they may recur if excision is incomplete.\textsuperscript{6} When there is concern for more extensive mast cell neoplasia, assessment of a buffy coat smear, and cytology and/or histopathology of regional lymph nodes and abdominal viscera, especially the spleen, is required to rule out disseminated disease (mastocytosis), which is important for prognostication.\textsuperscript{7} Further staging was not performed in the present case and, as a post mortem was not permitted, the extent of mast cell disease remained unknown.

Although the cause of the episodes of collapse could not be confirmed, mast cell degranulation, resulting in histamine-induced hypotension, or arrhythmia due to cardiomyopathy, were considered most likely.

Phagocytosis of mast cell granules by circulating neutrophils has been previously reported in the cat, and should be included on the list of differential diagnoses when basophilic or amphophilic granules are seen within the cytoplasm of peripheral blood neutrophils.\textsuperscript{10}

REFERENCES

Abstracts

Abstracts have been arranged under the headings of Review Papers, and Research Papers and Reports; within the latter they are arranged by Species (in this issue: Cat, Dog, Dog and Cat, Greyhound, Ferret and Rabbit), while within each species, articles relating to the a major body system tend to be grouped together.

Abstracts in this issue are from the Journal of the American Veterinary Medical Association (JAVMA), the Journal of Veterinary Internal Medicine (JVIM), Veterinary Surgery (Vet Surg) and the Journal of Veterinary Emergency and Critical Care (JVECC).

REVIEW PAPERS

A systematic review of the literature describing the efficacy of surgical treatments for canine hip dysplasia (1948-2012)

Bergh MS, Budsberg SC
Vet Surg 2014;43:501-506; doi 10.1111/j.1532-950X.2014.12208.x

Objective - To systematically evaluate the literature reporting outcome of surgical treatments for canine hip dysplasia (CHD) and to evaluate whether adequate evidence exists to support a procedure that will allow a consistent return to normal function. Study Design - Systematic literature review. Animals - Dogs with naturally occurring CHD. Methods - An a priori question was defined and a computer-based bibliographic search was performed on PubMed, Medline, CAB Abstracts, and Veterinary Information Network through November 2012. Studies were compared and evaluated with regard to surgical technique, study design, outcome measurements, evidence classification, and evidence quality. Unilateral surgeries with >6 months postoperative follow-up were included. Results - Manuscripts (n = 477) were identified and reviewed; 17 met the inclusion criteria. One study provided level I evidence, 2 provided level II evidence, 3 provided level III evidence, and 11 provided level IV evidence relative to the study question. The most common outcome measurements were orthopedic examination (70.6%), owner interview (70.6%), and visual gait observation (64.7%). Three studies used objective kinetic gait assessment. Two studies with level III evidence (total hip replacement) and 1 study with level IV evidence (juvenile pubic symphysiodesis) documented a consistent return to normal function after surgery. Conclusions - Despite a large number of publications describing clinical outcome after surgical treatments for CHD, few provided strong evidence to allow an adequate assessment of therapeutic efficacy.

Multiple organ dysfunction syndrome in humans and animals

Osterbur K, Mann FA, Kuroki K, DeClue A
JVIM 2014; 28:1141-1151; doi.org/10.1111/jvim.12364

Multiple organ dysfunction syndrome (MODS) defined as the presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention, is a cause of high morbidity and mortality in humans and animals. Many advances have been made in understanding the pathophysiology and treatment of this syndrome in human medicine, but much still is unknown. This comparative review will provide information regarding the history and pathophysiology of MODS in humans and discuss how MODS affects each major organ system in animals.

Cystatin C: A New Renal Marker and Its Potential Use in Small Animal Medicine

Ghys L, Paepe D, Smets P, et al
JVIM 2014; 28:1152-1164; doi.org/10.1111/jvim.12366

The occurrence of chronic kidney disease is underestimated in both human and veterinary medicine. Glomerular filtration rate (GFR) is considered the gold standard for evaluating kidney function. However, GFR assessment is time-consuming and labor-intensive and therefore not routinely used in practice. The commonly used indirect GFR markers, serum creatinine (sCr) and urea, are not sufficiently sensitive or specific to detect early renal dysfunction. Serum cystatin C (sCysC), a proteinase inhibitor, has most of the properties required for an endogenous GFR marker. In human medicine, numerous studies have evaluated its potential use as a GFR marker in several populations. In veterinary medicine, this marker is gaining interest. The measurement is easy, which makes it an interesting parameter for clinical use. This review summarizes current knowledge about cystatin C (CysC) in humans, dogs, and cats, including its history, assays, relationship with GFR, and biological and clinical variations in both human and veterinary medicine.

Extradural spinal cysts in dogs

Lowrie ML, Platt SR, Garosi LS

Objective - To (1) synthesize the terminology used to classify extradural spinal cysts in dogs to clarify some of the commonly reported misconceptions, and (2) propose a classification scheme to limit confusion with terminology. Study design - Literature review. Methods - An online bibliographic search was performed in January 2013 for articles relating to extradural spinal cysts in dogs using PubMed (http://www.pubmed.gov/) and Google Scholar (http://scholar.google.com/) databases. Only peer-reviewed clinical literature describing cystic lesions pertaining to the spinal cord and associated structures was included. Results - From 1962 to 2013, 42 articles were identified; 25 (95 dogs) reported meningeal cysts, 10 (24 dogs) described 60 extradural cysts, 3 reports (18 dogs) described discal cysts or acute compressive hydrated nucleus pulposus extrusions (HNPE). Spinal cysts were categorized by location based on cross-sectional imaging as meningeal or extradural non-meningeal. Sub-classification was then performed based on surgical findings and pathology. Meningeal cysts included arachnoid diverticulae and Tarlov (perineural) cysts. Extradural non-meningeal cysts included intrasinal cysts of the vertebral joints, ligaments and discs. Discal cysts also fit this category and have been reported extensively in humans but appear rare in dogs. Conclusions - Extradural spinal cysts should be first classified according to location with a sub-classification according to pathologic and surgical findings. Previous canine cases of discal cysts appear to represent a different disease entity and the term acute compressive HNPE is therefore preferred.

Advances in diagnostic and treatment modalities for intracranial tumors

Dickinson PJ
JVIM 2014; 28:1165-1185; doi.org/10.1111/jvim.12370

Intracranial neoplasia is a common clinical condition in domestic companion animals, particularly in dogs. Application of advances in standard diagnostic and therapeutic modalities together with a broad interest in the development of novel translational therapeutic strategies in dogs has resulted in clinically relevant improvements in outcome.
DNA testing is available for a growing number of hereditary diseases in neurology and other specialties. In addition to guiding breeding decisions, DNA tests are important tools in the diagnosis of diseases, particularly in conditions for which clinical signs are relatively nonspecific. DNA testing also can provide valuable insight into the risk of hereditary disease when decisions about treating comorbidities are being made. Advances in technology and bioinformatics will make broad screening for potential disease-causing mutations available soon. As DNA tests come into more common use, it is critical that clinicians understand the proper application and interpretation of these test results.

RESEARCH PAPERS AND REPORTS
CATS
Use of a morphometric method and body fat index system for estimation of body composition in overweight and obese cats
Witzel AL, Kirk CA, Henry GA, et al
JAVMA 2014;244:1285-1290; doi: 10.2460/javma.244.11.1285

Objective - To develop morphometric equations for prediction of body composition and create a body fat index (BFI) system to estimate body fat percentage in overweight and obese cats. Design - Prospective evaluation study. Animals - 76 overweight or obese cats ≥ 1 year of age. Procedures - Body condition score (BCS) was determined with a 5-point scale, morphometric measurements were made, and dual-energy x-ray absorptiometry (DEXA) was performed. Visual and palpation-based evaluation of various body regions was conducted, and results were used for development of the BFI system. Best-fit multiple regression models were used to develop equations for predicting lean body mass and fat mass from morphometric measurements. Predicted values for body composition components were compared with DEXA results. Results - For the study population, prediction equations accounted for 85% of the variation in lean body mass and 98% of the variation in fat mass. Values derived from morphometric equations for fat mass and lean mass were within 10% of DEXA values for 55 of 76 (72%) and 66 of 76 (87%) cats, respectively. Body fat as a percentage of total body weight (ie, body fat percentage) predicted with the BCS and BFI was within 10% of the DEXA value for 5 of 39 (13%) and 22 of 39 (56%) cats, respectively. Conclusions and Clinical Relevance - The BFI system and morphometric equations were considered accurate for estimation of body composition components in overweight and obese cats of the study population and appeared to be more useful than BCS for evaluation of these patients. Further research is needed to validate the use of these methods in other feline populations.

Editor’s note: There was a similar study reported by Witzel AL, et al in the same issue of JAVMA on overweight dogs.

The effect of Chinese rhubarb, Rheum officinale, with and without benazepril on the progression of naturally occurring chronic kidney disease in cats
Hanzlicek AS, Roof CJ, Sanderson MW, Grauer GF
JVIM 2014;28:1221-1228; doi.org/10.1111/jvim.12365

Background - Renal fibrosis is common in progressive kidney disease. Transforming growth factors β (TGF-β) are important mediators of all types of fibrosis, including renal fibrosis. Chinese rhubarb has been shown to have antifibrotic properties in part because of inhibition of TGF-β and has slowed the progression of kidney disease in rodent models. Hypothesis - That administration of a Chinese rhubarb supplement will slow the progression of chronic kidney disease (CKD) in cats and the concurrent administration of Chinese rhubarb and benazepril will be more effective than either alone. Animals - Twenty-nine client-owned cats with naturally occurring IRIS Stage 2 or early Stage 3 CKD and without comorbidity such as cancer, urinary tract obstruction, urinary tract infection, poorly controlled hyperthyroidism, or systemic hypertension were enrolled in the study. Methods - A randomized, positive-controlled, prospective study was performed. Cats received Chinese rhubarb, benazepril, or both in addition to standard treatment for CKD. Repeated measures ANOVA was used to assess changes in serum creatinine concentration, body weight, hematocrit, urine protein: urine creatinine ratio (UPC), and systemic arterial blood pressure over time between and within treatment groups over an average of 22 months. Results - No significant differences were detected in serum creatinine concentration, body weight, hematocrit, urine protein: urine creatinine ratio (UPC), and systemic arterial pressure over time between or within treatment groups. Conclusions and Clinical Importance - This study failed to detect a significant difference in the progression of CKD in cats treated with Chinese rhubarb, benazepril, or both. Further study in specific subsets of cats with CKD is warranted.

Characterization of kidney injury molecule-1 in cats
Bland SK, Côte O, Clark ME, et al
JVIM 2014;28:1454-1464; doi.org/10.1111/jvim.12428

Background - Kidney disease (KD) is common in older cats and presumed to arise from subclinical kidney injuries throughout life. Sensitive markers for detecting kidney injury are lacking. Kidney injury molecule 1 (KIM-1) is a useful biomarker of kidney injury in humans and rodents. Hypothesis/Objectives - Feline KIM-1 is conserved across species, expressed in kidney, and shed into urine of cats with acute kidney injury (AKI). The objectives were to characterize the feline KIM-1 gene and protein, assess available immunoassays for detecting KIM-1 in urine of cats, and identify KIM-1 expression in kidney sections. Animals - Samples from 36 hospitalized and 7 clinically healthy cats were evaluated. Hospitalized cats were divided into 2 groups based on absence (n = 20) or presence (n = 16) of historical KD. Methods - Feline KIM-1 genomic and complementary DNA sequences were amplified, sequenced and analyzed to determine the presence of isoforms, exon-intron organization and similarity with orthologous sequences. Presence in urine was evaluated by immunoassay and expression in kidney by immunohistochemistry. Results - Three expressed feline KIM-1 transcript variants comprising 894, 810, and 705 bp were identified in renal tissue. KIM-1 immunoassays yielded positive results in urine of cats with conditions associated with AKI, but not chronic KD. Immunohistochemistry of kidney sections identified KIM-1 in proximal tubular cells of cats with positive urine immunoassay results. Conclusions and Clinical Importance - Kidney injury molecule 1 was expressed in specific segments of the nephron and detected in urine of cats at risk of AKI. Urine KIM-1 immunoassay may be a useful indicator of tubular injury.
Association between oral health status and retrovirus test results in cats
Kornya MR, Little SE, Scherk MA, et al
JAVMA 2014;916-922; doi: 10.2460/javma.245.8.916

Objective - To determine associations between oral health status and seropositivity for FIV or FeLV in cats. Design - Cross-sectional survey. Animals - 5,179 cats. Procedures - Veterinarians at veterinary clinics and animal shelters completed online training on oral conditions in cats and then scored oral health status of cats with no known history of vaccination against FIV. Age, sex, and results of an ELISA for retroviruses were recorded. Results were analyzed by means of standard logistic regression with binary outcome. Results - Of 5,179 cats, 237 (4.6%) and 186 (3.6%) were seropositive for FIV and FeLV, respectively, and of these, 12 (0.2%) were seropositive for FIV and FeLV. Of all 5,179 cats, 1,073 (20.7%) had gingivitis, 576 (11.1%) had periodontitis, 203 (3.9%) had stomatitis, and 252 (4.9%) had other oral conditions (overall cardiac hypertrophy. Results and Clinical Relevance - Inflammatory oral disease was associated with an increased risk of seropositivity for retroviruses in naturally infected cats. Therefore, retiroval status of cats with oral inflammatory disease should be determined and appropriate management initiated.

Cardiac troponin I and T as prognostic markers in cats with hypertrophic cardiomyopathy
JVIM 2014;28:1485-1491; doi.org/10.1111/jvim.12407

Background - Myocardial injury detected by cardiac troponin I and T (cTnI and cTnT) in cardiac disease is associated with increased risk of death in humans and dogs. Hypothesis - Presence of myocardial injury predicts long-term death in cats with hypertrophic cardiomyopathy (HCM), and ongoing myocardial injury reflects change in left ventricular wall thickness over time. Animals - Thirty-six cats with primary HCM. Methods - Prospective cohort study. Cats with HCM were included consecutively and examined every 6 months. Echocardiography, ECG, blood pressure, and serum cTnI and cTnT were evaluated at each visit. Cox proportional hazards regression analysis was performed to evaluate prognostic potential of serum troponin concentrations at admission and subsequent examinations. Correlations were used to examine associations between troponin concentrations and cardiac hypertrophy. Results - Troponin concentrations at admission were median [range] 0.14 [0.004-1.02] ng/mL for cTnI, and 13 [13-79.5] ng/L for cTnT. Both were prognostic for death (p = 0.032 and 0.026) as were the last available concentrations of each (p = 0.016 and 0.003). The final cTnT concentration was a significant predictor of death even when adjusting for the admission concentration (p = 0.043). In a model containing both markers, only cTnT remained significant (p = 0.043). Left ventricular free wall thickness at end-diastole (LVFWd) at admission was correlated with cTnI at admission (r = 0.35, p = 0.035), however no significant correlations (r = 0.2-0.31, p = 0.007-0.26) were found between changes in troponin concentrations and left ventricular thickness over time. Conclusions and Clinical Importance - Myocardial injury is part of the pathophysiology leading to disease progression and death. Low sensitivities and specificities prevent outcome prediction in individual cats.

Case-control study of the effects of pimobendan on survival time in cats with hypertrophic cardiomyopathy and congestive heart failure
Reina-Doreste Y, Stern JA, Keene BW, et al
JAVMA 2014;534-539; doi: 10.2460/javma.245.5.534

Objective - To assess survival time and adverse events related to the administration of pimobendan to cats with congestive heart failure (CHF) secondary to hypertrophic cardiomyopathy (HCM) or hypertrophic obstructive cardiomyopathy (HOCM). Design - Retrospective case-control study. Animals - 27 cats receiving treatment with pimobendan and 27 cats receiving treatment without pimobendan. Procedures - Medical records between 2003 and 2013 were reviewed. All cats with HCM or HOCM treated with a regimen that included pimobendan (case cats) were identified. Control cats (cats with CHF treated during the same period with a regimen that did not include pimobendan) were selected by matching to case cats on the basis of age, sex, body weight, type of cardiomyopathy, and manifestation of CHF. Data collected included signalment, physical examination findings, echocardiographic data, serum biochemical values, and survival time from initial diagnosis of CHF. Kaplan-Meier survival curves were constructed and compared by means of a log rank test. Results - Cats receiving pimobendan had a significant benefit in survival time. Median survival time of case cats receiving pimobendan was 626 days, whereas median survival time for control cats not receiving pimobendan was 103 days. No significant differences were detected for any other variable. Conclusions and Clinical Relevance - The addition of pimobendan to traditional treatment for CHF may provide a substantial clinical benefit in survival time for HCM-affected cats with CHF and possibly HOCM-affected cats with CHF.

Evaluation of the perioperative analgesic efficacy of buprenorphine, compared with butorphanol, in cats
Warrn LN, Betts T, Holm M, et al
JAVMA 2014;195-202; doi: 10.2460/javma.245.2.195

Objective - To compare the analgesic effects of buprenorphine and butorphanol in domestic cats. Design - 2-phase positive-controlled randomized masked clinical trial. Animals - 39 healthy female cats (10 in phase 1 and 29 in phase 2). Procedures - Cats admitted for ovariohysterectomy received buprenorphine (4 in phase 1; 14 in phase 2) or butorphanol (6 in phase 1; 15 in phase 2). In phase 1, cats were premedicated with buprenorphine (0.02 mg/kg [0.009 mg/lb], IM) or butorphanol (0.4 mg/kg [0.18 mg/lb], IM), in combination with medetomidine. Anesthesia was induced with propofol (IV) and maintained with isoflurane in oxygen. After extubation, medetomidine was antagonized with atipamezole. A validated multidimensional composite scale was used to assess signs of pain after surgery starting 20 minutes after extubation and continuing for up to 360 minutes, and pain score comparisons were made between the 2 groups. Phase 2 proceeded similar to phase 1 with the following addition: during wound closure, cats from the butorphanol and buprenorphine groups received buprenorphine (0.4 mg/kg, IM) or buprenorphine (0.02 mg/kg, IM), respectively. Results - Phase 1 of the study was stopped after 10 cats were ovariohysterectomized because 9 of 10 cats required rescue analgesia at the first evaluation. In phase 2, at the first pain evaluation, pain scores from the buprenorphine group were lower, and all cats from the butorphanol group required rescue analgesia. None of the cats from the buprenorphine group required rescue analgesia at any time. Conclusions and Clinical Relevance -Buprenorphine (0.02 mg/kg, IM) given before surgery and during wound closure provided adequate analgesia for 6 hours following ovariohysterectomy in cats, whereas butorphanol did not.
Effect on renal function of restoration of euthyroidism in hyperthyroid cats with iatrogenic hypothyroidism

Williams TL, Elliott J, Syme HM
JVIM 2014;28:1251-1255; doi.org/10.1111/jvim.12359

Background - Iatrogenic hypothyroidism is associated with an increased incidence of azotemia after treatment of hyperthyroidism, and decreased survival time in azotemic hyperthyroid cats. Hypothesis - Restoration of euthyroidism will decrease plasma creatinine concentrations. Animals - Nineteen client-owned, methimazole- or carbimazole-treated, hyperthyroid cats with documented iatrogenic hypothyroidism (based on subnormal plasma total thyroxine concentrations [TT4] and increased plasma thyroid-stimulating hormone concentrations). Methods - Prospective interventional study. Doses of antithyroid medication were reduced until euthyroidism was restored (TT4 10–40 nmol/L). Plasma creatinine concentration and selected other clinicopathologic variables were evaluated before and after restoration of euthyroidism and compared by nonparametric statistics. Data are presented as median [25th, 75th percentile]. Results - Restoration of euthyroidism was associated with a significant decrease in plasma creatinine concentrations (2.61 [1.90, 3.26] mg/dL versus 2.07 [1.42, 2.82] mg/dL; P < .001) and body weight (4.03 [3.59, 4.53] kg versus 3.89 [3.34, 4.18] kg; p = 0.019), and a significant increase in packed cell volume (30 [28, 39]% versus 34 [29, 39%]; p = 0.038), heart rate (174 [163, 201] bpm versus 190 [164, 202] bpm; p = 0.009), and plasma alkaline phosphatase activity (26.6 [17.0, 33.0] IU/L versus 38.0 [23.5, 46.5] IU/L; p < 0.001). Conclusions and Clinical Importance - Restoration of euthyroidism in medically treated hyperthyroid cats with iatrogenic hypothyroidism causes a reduction in plasma creatinine concentrations, and thus might improve renal function; however, this could be influenced by concurrent changes in body weight.


Holly D. Burr HD, Keating JH, Clifford CA, Burgess KE
JAVMA 2014;244:1429-1434; doi: 10.2460/javma.244.12.1429

Objective - To determine features of lymphoma of the tarsus in cats. Design - Multi-institutional retrospective study. Animals - 23 cats with cutaneous lymphoma of the tarsus. Procedures - Veterinary oncologists were requested to submit cases fitting the following criteria: histologically or cytologically confirmed lymphoma with a location at or near the tarsus and described as subcutaneous or mass-like. Data regarding breed, sex, age, FeLV and FIV status, and reason for evaluation were collected. Results of staging tests, location of the tumor, immunophenotype, and histopathologic description were recorded. Type of treatments, Interventions - Blood collection by jugular venipuncture. Transfusion with labeled, autologous, fresh RBCs. Measurements and Main Results - Antiocoagulated whole blood (35 mL/cat) was collected in 2 equal aliquots. RBCs were washed and labeled at 2 different biotin densities, before suspension in autologous plasma. Labeled RBCs were then transfused using 2 methods, gravity flow and pump delivery using a 20 mL syringe and 18µm microaggregate filter. Whole blood samples were collected from each cat at 2-hour intervals for 12 hours following completion of the transfusions. Additional samples were collected at weekly intervals up to 6 weeks to assess circulating half-life of the transfused cells. Cell survival was assessed via flow cytometry. The proportion of transfused cells remaining in each of the 2 populations was measured. Biotinylated RBCs were readily detected in all cats over the 6-week sampling period. There was a significant decrease in both populations of labeled cells over the 6-week period (P < 0.01), as expected. There was no difference in probability that the RBCs would survive up to 12 hours immediately following transfusion, and no significant difference in survival between the 2 groups over 6 weeks. The average half-life of all labeled cells was approximately 23 days. Conclusions - We conclude that, in contrast to findings from dogs, transfusion of autologous feline RBCs using a syringe + aggregate filter method does not significantly impact short- or long-term survival of the transfused cells.

Feline secondary spontaneous pneumothorax: A retrospective study of 16 cases (2000-2012)

Liu DT, Silverstein DC
JVECC 2014; 24:316-325; doi.10.1111/jvec.12150

Objective - To describe the demographics, clinical characteristics, diagnostic findings, underlying etiologies, treatment, and outcome associated with secondary spontaneous pneumothorax (SSP) in cats; and to identify clinical feature differences among cats with asthma associated secondary spontaneous pneumothorax (AASSP) versus non-asthma-associated secondary spontaneous pneumothorax (NAASSP). Design - Retrospective case series. Setting - University teaching hospital. Animals - Sixteen client-owned cats with secondary spontaneous pneumothorax. Interventions - None. Measurements and Main Results - Domestic shorthair was the predominant breed in this study (n = 15). The median age was 8 years old (range: 7 weeks to 17 years) with no sex predilection. Fourteen cats were affected by multi-lobar pulmonary pathology of infectious, inflammatory, or neoplastic causes. Asthma was the most common cause of spontaneous pneumothorax (25%). Ten of 12 treated cats survived the initial episode of spontaneous pneumothorax to discharge with medical management, including all 4 cats with AASSP. Reoccurrence was documented in 4 cats. Pulmonary lobectomy was curative for 1 cat with congenital accessory lung lobe...
emphysema. No difference in clinical presentation was identified between cats with AASSP and cats with NAASSP. Conclusions - Feline SSP is frequently associated with extensive pulmonary pathology. Supportive medical management is most appropriate, except in rare cases with focal congenital abnormalities that may benefit from surgical intervention. AASSP appears to carry a good prognosis for short-term outcome (survival to discharge). Clinical assessment, imaging, and invasive diagnostics were required to differentiate between AASSP and NAASSP.

**DOGS**

**Natural history of arrhythmogenic right ventricular cardiomyopathy in the Boxer dog: A prospective study**

*Meurs KM, Stern JA, Reina-Doreste Y, Spier AW, Koplitz SL, Baumwart RD*

**JVIM 2014;28:1214-1220; doi.org/10.1111/jvim.12385**

**Background** - Boxer arrhythmogenic right ventricular cardiomyopathy (ARVC) is a disease that may result in sudden death or heart failure.

**Hypothesis/objectives** - To prospectively study the natural history of Boxer ARVC.

**Animals** - 72 dogs (49 ARVC, 23 controls).

**Methods** - Boxers >1 year of age were recruited for annual reevaluation. Controls were defined as being ≥6 years of age and having <50 ventricular premature complex (VPCs)/24 h. ARVC was defined as ≥300 VPCs/24 h in the absence of other disease. Dogs were genotyped for the striatin deletion when possible. Descriptive statistics were determined for age; VPC number; annual change in VPC number; and left ventricular (LV) echocardiographic dimensions. Survival time was calculated.

**Results** - Controls: median age of 7 years (range, 6-10); number of VPCs 12 (range, 4-32). Median time in study of 6 years (range, 2-9). Seventeen of 23 were genotyped (5 positive, 12 negative). ARVC: median age of diagnosis of 6 (range, 1-11). Median time in study 5 years (range, 3-8). A total of 33% were syncopal and 43/49 were genotyped (36 positive, 7 negative). Yearly change in VPCs was 46 (range, -7,699 to 33,524). Annual percentage change in LV dimensions was 0, and change in fractional shortening (FS%) was 2%. Two dogs had F S% <20%. Although ARVC dogs died suddenly, there was no difference in survival time between groups. ARVC median age of survival was 11 years, and for controls was 10 years.

**Conclusions/Clinical Importance** - Arrhythmogenic right ventricular cardiomyopathy is a disease of middle age and frequently is associated with the striatin deletion. Syncope occurs in approximately 1/3 of affected dogs; systolic dysfunction is uncommon. The prognosis in many affected dogs is good.

**Prediction of long-term outcome by measurement of serum concentration of cardiac troponins in critically ill dogs with systemic inflammation**

*Langhorn R, Thawley V, Oyama MA, et al*

**JVIM 2014;28:1492-1497; doi.org/10.1111/jvim.12402**

**Background** - Myocardial injury, detected by cardiac troponin I and T (cTnI and cTnT), has been associated with long-term death in the noncardiac human intensive care unit (ICU). **Hypothesis** - Presence of myocardial injury predicts 1-year case fatality in critically ill dogs with systemic inflammation. **Animals** - Thirty-eight dogs with evidence of systemic inflammation and no primary cardiac disease. **Methods** - Prospective cohort study. In dogs admitted to the ICU with evidence of systemic inflammation, blood samples were obtained at ICU admission for measurement of cTnI and cTnT, and cTnI was measured once daily during ICU hospitalization. Receiver operating characteristic (ROC) curves were used to examine prognostic capacity of admission cTnI, admission cTnT, and peak cTnI concentrations. **Results** - One-year case fatality rate was 47% (18/38 dogs). Admission cTnI concentrations were (median [range]) 0.48 [0.004-141.50] ng/mL, and peak cTnI concentrations were 1.21 [0.021-141.50] ng/mL. Admission cTnT concentrations were 15 [<13-3744] ng/L. For each marker, non-survivors had significantly higher concentrations than survivors (p = 0.0082-0.038). ROC analyses revealed areas under curves [95% CI] of 0.707 [0.537-0.843] for peak cTnI and 0.739 [0.571-0.867] for admission cTnT, respectively. At the optimal cut-off, concentrations were 1.17 ng/mL (peak cTnI) and 23 ng/L (admission cTnT), sensitivities were 72% and 72%, and specificities were 70% and 80%, respectively. **Conclusions and Clinical Importance** - While peak cTnI and admission cTnT are significantly related to 1-year case fatality in critically ill dogs with systemic inflammation, low sensitivities and specificities prevent their prediction of long-term outcome in individual patients. Troponins might play a role in identification of dogs at long-term risk of death.

**Comparison of manual and suction pump aspiration techniques for performing bronchoalveolar lavage in 18 Dogs with respiratory tract disease**

*Woods KS, Defarges AMN, Abrams-Ogg ACG, et al*

**JVIM 2014;28:1398-1404; doi.org/10.1111/jvim.12403**

**Background** - Different aspiration techniques to retrieve bronchoalveolar lavage fluid (BALF) affect sample quality in healthy dogs. Studies evaluating these techniques in dogs with respiratory disease are lacking. **Objectives** - To compare sample quality of BALF acquired by manual aspiration (MA) and suction pump aspiration (SPA). **Animals** - Eighteen client-owned dogs with respiratory disease.

**Methods** - Randomized, blinded prospective clinical trial. Manual aspiration was performed with a 35-mL syringe attached directly to the bronchoscope biopsy channel and SPA was performed with a maximum of 50 mmHg negative pressure applied to the bronchoscope suction valve using the suction trap connection. Both aspiration techniques were performed in each dog on contralateral lung lobes, utilizing 2 mL/kg lavage volumes per site. Samples of BALF were analyzed by percentage of retrieved infusate, total nucleated cell count (TNCC), differential cell count, semiquantitative assessment of slide quality, and diagnosis score.

Data were compared by paired Student’s t-test, Wilcoxon signed-rank test, chi-squared test, and ANOVA. Cohen’s kappa coefficient was used to assess agreement.

**Results** - The percentage of retrieved BALF (P = .001) was significantly higher for SPA than MA. Substantial agreement was found between cytologic classification of BALF obtained with MA and SPA (kappa = 0.615). There was no significant difference in rate of definitive diagnosis achieved with cytologic assessment between techniques (p = 0.78).

**Conclusions and Clinical Importance** - Suction pump aspiration, compared to MA, improved BALF retrieval, but did not significantly affect the rate of diagnostic success of bronchoalveolar lavage (BAL) in dogs with pulmonary disease.

**Hypomagnesemia in brachycephalic dogs**

*Mellema MS, Hoareau GL*

**JVIM 2014;28:1418-1423; doi.org/10.1111/jvim.12393**

**Background** - Brachycephalic dogs are at risk for arterial hypertension and obstructive sleep apnea, which are both associated with chronic magnesium (Mg) depletion. **Hypothesis/Objectives** - To compare the period prevalence of hypomagnesemia between Boxers and Bulldogs presented to a referral teaching hospital. To screen a group of Bulldogs for evidence of hypomagnesemia, and to obtain pilot data regarding the utility of parenteral Mg tolerance testing (PMgTT) in the diagnosis
of whole-body Mg deficiency. Animals - Chemistry laboratory submissions were retrospectively analyzed for serum total Mg (tMg) in Boxers and Bulldogs. Prospectively, 16 healthy client-owned Bulldogs were enrolled. Methods - Retrospective case study. tMg concentrations were compared between Boxers and Bulldogs. Dogs with low serum albumin or high serum creatinine concentrations were excluded. Prospectively, ionized Mg (iMg), tMg, and arterial blood pressure were measured and iMg-to-tMg ratio (iMgtMg) was calculated. Parenteral Mg tolerance testing (PMgTT) was performed in 3/16 dogs. Results - In the retrospective study, period prevalence of hypomagnesemia was 4.7% in Boxers and 15% in Bulldogs (p = 0.02). The ratio for hypomagnesemia in Bulldogs was 1.8 when compared to Boxers (CI: 1.3-2.7). In the prospective study, iMg was [median (interquartile)] 0.43 (0.42-0.46) mmol/L (reference range 0.4-0.52), tMg was 1.9 (1.8-1.9) mg/dL (reference range 1.9-2.5), iMgtMg was [mean (±SD)] 0.59±0.04. Percentage retention after PMgTT were 55%, 95%, and 67%, respectively. Conclusions and Clinical Importance - Mg deficiency is common in Bulldogs and could contribute to comorbidities often observed in this breed. iMgtMg and PMgTT might prove helpful in detecting chronic subclinical Mg deficiency. 

Comparison of adrenocorticotropic hormone stimulation test results started 2 versus 4 hours after trilostane administration in dogs with naturally occurring hyperadrenocorticism

Bonadio CM, Feldman EC, Cohen TA, Kass PH
JVIM 2014;28:1239-1243; doi.org/10.1111/jvim.12357

Background - Trilostane medical treatment of naturally occurring hyperadrenocorticism (NOH) in dogs is common, as is use of the adrenocorticotropic hormone (ACTH) stimulation test (ACTHst) in monitoring response to treatment. There is uncertainty regarding when the ACTHst should be started relative to time of trilostane administration. Objective - To compare ACTHst results in dogs being treated for NOH with trilostane when the test is begun 2 versus 4 hours after trilostane administration. Animals - Twenty-one privately owned dogs with NOH, each treated with trilostane for at least 30 days. Methods - Each dog had 2 ACTHst completed, 1 started 2 hours and the other 4 hours after trilostane administration. The second test was started no sooner than 46 hours and no later than 74 hours after the first. Results - For all 21 dogs, the mean post-ACTH serum cortisol concentration from tests started 2 hours after trilostane administration (5.4±3.7 µg/dL) was significantly lower (p = 0.03) as compared with results from the tests started 4 hours after administration (6.5±4.5 µg/dL). Conclusions - Results of ACTHst started at different times yield significantly different results. Dogs with NOH, treated with trilostane, and monitored with ACTHst results should have all of their subsequent ACTHst tests begun at or about the same time after trilostane administration. 

Canine pancreatic-specific lipase concentrations in clinically healthy dogs and dogs with naturally occurring hyperadrenocorticism

Mawby DI, Whittemore JC, Fecteau KA
JVIM 2014;28:1244-1250; doi.org/10.1111/jvim.12376

Background - Specificity of canine pancreatic lipase immunoreactivity (cPLI) assays in dogs with hyperadrenocorticism (HAC) is unknown. Hypothesis - Results of cPLI assays differ for clinically healthy dogs and dogs with HAC. Animals - Seventeen healthy dogs and 20 dogs with HAC diagnosed by ACTH stimulation test results without evidence of clinical pancreatitis. Methods - Dogs were enrolled between December 2009 and November 2010. Serum cPLI concentrations were determined by quantitative (Spec cPLI test, SPEC) and semiquantitative (SNAP cPLI test, SNAP) assays. Results were categorized as normal, equivocal, or abnormal (SPEC) or negative or positive (SNAP). Associations between group and cPLI were assessed using Fisher’s exact test or the Mann–Whitney U-test. Spearman rank correlation coefficients (ρ) were determined for SNAP and SPEC results. Significance was set at P < .05. Results - Spec cPLI test concentrations were significantly (p < 0.001) higher in dogs with HAC (491.1±74.5 µg/L) than in healthy dogs (75.2±39.7 µg/L), with more abnormal SPEC results in HAC dogs (P < .001). There were more (p = 0.002) positive SNAP results in dogs with HAC (55%) than in healthy dogs (6%), SNAP and SPEC results were highly correlated (ρ = 0.85; p < 0.001). Conclusions and Clinical Importance - Dogs with HAC had higher SPEC concentrations and more positive SNAP results than clinically healthy dogs with normal ACTH stimulation test results. Specificity of SPEC and SNAP assays in HAC dogs without clinical pancreatitis was 65 and 45%, respectively. Pending further study, SNAP and SPEC results should be interpreted cautiously in dogs with HAC to avoid false diagnosis of concurrent pancreatitis. 

Comparison of perioperative morbidity and mortality rates in dogs with noninvasive adrenocortical masses undergoing laparoscopic versus open adrenalectomy

Mayhew PD, Culp WTN, Hunt GB, et al
JAVMA 2014;1028-1035; doi: 10.2460/javma.245.9.1028

Objective - To describe the clinicopathologic features of a cohort of dogs with adrenocortical masses that underwent laparoscopic adrenalectomy and to compare perioperative morbidity and mortality rates in these dogs with rates for dogs that underwent open adrenalectomy for resection of similarly sized (maximal diameter, ≤ 5 cm) adrenocortical masses. Design - Retrospective case series. Animals - 48 client-owned dogs that underwent laparoscopic (n = 23) or open (25) adrenalectomy for noninvasive tumors. Procedures - Medical records were reviewed. History, clinical signs, physical examination findings, clinicopathologic findings, imaging results, and surgical variables were recorded. A 3- or 4-port approach was used for laparoscopic adrenalectomy. Surgical time, perioperative complications, postoperative and overall hospitalization times, and perioperative deaths were recorded and compared between groups. Results - The surgical method for 1 dog was converted from a laparoscopic to an open approach. Perioperative death occurred in no dogs in the laparoscopic group and 2 dogs in the open adrenalectomy group. Surgical time was shorter for laparoscopic (median, 90 minutes; range, 40 to 150 minutes) than for open (median, 120 minutes; range, 75 to 195 minutes) adrenalectomy. Laparoscopic adrenalectomy was associated with shorter hospitalization time and more rapid discharge from the hospital after surgery, compared with the open procedure. Conclusions and Clinical Relevance - With careful patient selection, laparoscopic adrenalectomy was associated with a low complication rate and low conversion rate for resection of adrenocortical masses as well as shorter surgical and hospitalization times, compared with open adrenalectomy. 

Basal serum cortisol concentration as a screening test for hypoadrenocorticism in dogs

Bovens C, Tennant K, Reeve J, Murphy KF
JVIM 2014;28:1541-1545; doi.org/10.1111/jvim.12415

Background - Measurement of basal serum or plasma cortisol concentration is used as a screening test for hypoadrenocorticism in dogs, but is not well characterized. Objectives - To evaluate the sensitivity
and specificity of basal serum cortisol to detect hypoadrenocorticism in a population of dogs with a clinical suspicion of hypoadrenocorticism. Animals - Four hundred and fifty dogs with nonadrenal gland illness and 14 dogs with naturally occurring hypoadrenocorticism were included. Methods - Retrospective case-control study. The records of all dogs having had an ACTH stimulation test performed between January 2005 and September 2011 at the University of Bristol were reviewed. Dogs were included if the test was performed as a screening for hypoadrenocorticism. The sensitivity and specificity of basal serum cortisol concentration to detect dogs with hypoadrenocorticism were calculated using 2 cut-offs and compared to the gold standard ACTH stimulation test. Results - Using a cut-off of ≤ 2 µg/dL (≤ 55 nmol/L), the sensitivity and specificity of basal cortisol to detect hypoadrenocorticism were 100% and 63.3%, respectively, whereas for a cut-off of ≥ 1 µg/dL (≥ 28 nmol/L), the sensitivity and specificity were 85.7% and 91.8%, respectively. Conclusions and Clinical Importance - Measurement of basal serum cortisol is useful as a screening test for hypoadrenocorticism in dogs using a cut-off of ≤ 2 µg/dL (≤ 55 nmol/L), and the disease is unlikely with a basal serum cortisol >2 µg/dL (>55 nmol/L). A basal serum cortisol ≥ 2 µg/dL (≥ 55 nmol/L) cannot be used to diagnose hypoadrenocorticism, and an ACTH stimulation test should be performed in these cases.

Use of the cortisol-to-ACTH ratio for diagnosis of primary hypoadrenocorticism in dogs

Lathan P, Scott-Moncrieff JC, Wills RW
JVIM 2014;28:1546-1550; doi.org/10.1111/jvim.12392

Background - The ACTH stimulation test is currently required for definitive diagnosis of hypoadrenocorticism. Increased cost of synthetic ACTH (cosyntrin®) has prompted a search for alternative diagnostic methods. Objective - The purpose of this study was to determine whether a cortisol-to-ACTH ratio (CAR) can be used to differentiate dogs with hypoadrenocorticism from normal dogs and those with nonadrenal illness. Animals - Eight healthy dogs (H), 19 dogs with nonadrenal illness (NAI), and 15 dogs with hypoadrenocorticism (HAD). Methods - Dogs in the HAD group were retrospectively identified from PUVTH medical records. The NAI group consisted of hospitalized dogs with clinical signs, clinicopathologic findings, or both, consistent with a diagnosis of hypoadrenocorticism, but in which hypoadrenocorticism was ruled out based on ACTH stimulation test results. Healthy dogs were recruited from hospital staff and students. Endogenous ACTH concentrations and cortisol concentrations before and after ACTH stimulation were measured in all dogs. Results - Baseline cortisol concentration was significantly lower, and ACTH concentration was significantly higher, in the HAD group versus the H and NAI group (P < .001). However, there was overlap among groups. Cortisol-to-ACTH ratio was significantly lower in the HAD group versus the H and NAI groups (P < .001), and there was no overlap between the HAD group and the other 2 groups. Conclusions and Clinical Importance - CAR can be used for definitive diagnosis of primary hypoadrenocorticism.

Use of plasma renin activity to monitor mineralocorticoid treatment in dogs with primary hypoadrenocorticism: desoxycorticosterone versus fludrocortisone

Baumstark ME, Nussberger J, Boretti FS, et al
JVIM 2014;28:1471-1478; doi.org/10.1111/jvim.12426

Background - Measurement of plasma renin activity (PRA) is the gold standard for monitoring mineralocorticoid treatment in humans with primary hypoadrenocorticism (PH). Objectives - To compare PRA in dogs with newly diagnosed PH, dogs with diseases mimicking PH, and healthy dogs, and evaluate measurement of PRA to monitor therapeutic effects in dogs with PH treated with different mineralocorticoids. Animals - Eleven dogs with newly diagnosed PH (group 1), 10 dogs with diseases mimicking PH (group 2), 21 healthy dogs (group 3), 17 dogs with treated PH (group 4). Methods - In group 1, PRA was measured before treatment and at different times after initiating treatment. In groups 2 and 3, PRA was measured at initial presentation only. In group 4, no baseline PRA was obtained but PRA was measured once or every 1–6 months during treatment. Mineralocorticoid treatment consisted of fludrocortisone acetate (FC) or desoxycorticosterone pivalate (DOCP). Results - Plasma renin activity before treatment was increased in dogs with PH compared to normal dogs and dogs with diseases mimicking PH with median activity of 27.0, 8.0, and 1.0 ng/mL/h, respectively. In dogs with PH, PRA decreased and normalized with mineralocorticoid treatment using DOCP but not with FC. In dogs treated with DOCP, PRA was lower than in dogs treated with FC. Plasma sodium concentrations were higher and potassium concentrations were lower with DOCP treatment compared to FC treatment. Conclusion and Clinical Importance - Plasma renin activity is a reliable tool for monitoring mineralocorticoid treatment. DOCP treatment more effectively suppresses PRA compared to FC in dogs with PH.

Pharmacokinetics of total thyroxine after repeated oral administration of levothyroxine solution and its clinical efficacy in hypothyroid dogs

van Dijl IC, Le Traon G, van de Meulengraaf BDAM, Burgaud S, Horspool LJ, Kooistra HS
JVIM 2014;28:1229-1234; doi.org/10.1111/jvim.12363

Background - Oral levothyroxine (l-T4) supplementation is commonly used to treat hypothyroid dogs. Objectives - Investigate the plasma profile and pharmacokinetics of total thyroxine (tT4) after PO administration of a l-T4 solution and its clinical efficacy in hypothyroid dogs. Animals - Ten dogs with naturally occurring hypothyroidism. Methods - After hypothyroidism diagnosis and supplementation with l-T4 solution PO q24h at 20 µg/kg BW for minimum 4 weeks, the plasma profile and pharmacokinetics of tT4 were determined over 34 hours and the clinical condition of the dogs was evaluated. Results - Before dosing for pharmacokinetic evaluation, mean tT4 concentration was 23 ± 9 nmol/L. l-T4 was absorbed rapidly (tmax, 5 hours), reaching a mean maximal tT4 concentration of 56 ± 11 nmol/L. The apparent terminal half-life was 11.8 hours. Clinical signs of hypothyroidism improved or resolved in all dogs after 4 weeks of treatment. The dosage of 20 µg/kg PO q24h was judged appropriate in 5 dogs, and 4 dogs required slight increases (9–16%). Twice daily treatment, with a 30% increase in dosage, was necessary for 1 dog. Conclusions and Clinical Importance - The pharmacokinetics of l-T4 in hypothyroid dogs was similar to that reported in healthy euthyroid dogs. Clinical and hormonal responses to l-T4 solution were rapid in all dogs. The starting dosage of 20 µg/kg PO q24h was suitable for maintenance supplementation in 50% of the dogs, minor dosage modification was required in 4 other dogs, and treatment q12h was required in 1 dog.

Clinical features and treatment outcomes of 41 dogs with sublingual ectopic thyroid neoplasia

Broome MR, Peterson ME, Walker JR
JVIM 2014;28:1560-1568; doi.org/10.1111/jvim.12406

Background - Thyroid neoplasia is common in dogs, but there are few reports of dogs with ectopic, sublingual thyroid tumors. Objectives - To describe clinical features and outcomes of dogs with ectopic, sublingual
Evaluation of urethral stent placement for benign urethral obstructions in dogs
Hill TL, Berent AC, Weisse CW
JVIM 2014;28:1384-1390; doi.org/10.1111/jvim.12412

Background - Benign urethral obstructions (BUO) in dogs result in substantial morbidity because of challenges with conventional therapies. Treatment of malignant urethral obstructions with intraluminal urethral stents is reported to successfully relieve obstructions. Hypothesis/ Objectives - To evaluate the efficacy and outcome of urethral stent placement for treatment of BUO in dogs. Animals - Eleven client-owned animals with urethral stents placed for treatment of BUO. Methods - Retrospective study in which medical records were reviewed in dogs diagnosed with BUO and treated with a metallic urethral stent. Data collected included signalment, cause of benign obstruction, procedure time, size and type of stent, complications, and short- and long-term outcome. Results - Eleven dogs with 15 urethral stents were included. Intraluminal urethral stent(s) relieved the obstructions in all dogs. Four dogs had 2 stents placed in separate procedures because of incomplete patency after treatment (n = 1), inadvertent compression of the stent (n = 1), or tissue ingrowth through the stent (n = 2). The median continence score after stent placement was 10 of 10 (range 3-10) with 6 dogs being continent, 3 mildly incontinent, and 1 each moderately and severely incontinent. All owners considered their dog to have an excellent long-term clinical outcome with long-term urethral patency. The median follow-up time was 24 months (range 4-48). Conclusions and Clinical Importance - Urethral stents appear to be an effective treatment for benign urinary obstructions. Moderate to severe incontinence developed in a minority (12.5%) of dogs. Stents relieved obstructions in all dogs with an excellent long-term outcome.

Urethral prolapse in dogs: A retrospective study
Carr JG, Tobias KM, Smith L
Vet Surg 2014;43:574-580; doi.10.1111/j.1532-950X.2014.12190.x

Objective - To evaluate the signalment, clinical signs, treatment, and outcome of dogs with urethral prolapse and identify risk factors associated with prolapse or treatment. Study Design - Retrospective case series. Animals - Dogs (n = 48) with urethral prolapse. Methods - Medical records (May 1995–June 2010) from 2 referral centers were reviewed. Retrieved data included signalment, clinical signs, laboratory findings, treatment, complications, results of long-term follow-up. Records from Veterinary Medical Data Base (VMDB) were evaluated to determine odds ratios. Results - Odds ratio for urethral prolapse in English bulldogs compared to all breeds was 366.99 (95% CI: 265.83, 506.65). Of 48 affected dogs, 46 had either resection and anastomosis (43 dogs) or urethropexy (3 dogs). The most common early postoperative complication was hemorrhage (39%); postoperative hemorrhage was less common when a simple continuous pattern was used for resection and anastomosis. Prolapse occurred in 57% of dogs available for long-term follow-up; recurrence was less common in dogs that were administered postoperative butorphanol or acepromazine. Gender was not associated with urethral prolapse or postoperative complications. Conclusions - Urethral prolapse occurs most commonly in English bulldogs. Postoperative hemorrhage and prolapse recurrence may be reduced with use of a simple continuous pattern for urethral anastomosis and by administration of postoperative sedation, respectively. Castration status did not appear to affect prolapse development or outcome.

Prevalence and clinical outcome of subclinical bacteriuria in female dogs
Wan SY, Hartmann FA, Jooss MK, Viviano, KR
JAVMA 2014;106-112; doi: 10.2460/javma.245.1.106

Objective - To determine the prevalence of subclinical bacteriuria and its natural clinical course over a 3-month period in healthy female dogs. Design - Observational, prospective, cross-sectional study. Animals - 101 healthy client-owned female dogs. Procedures - In all dogs, screening clinicopathologic tests and bacteriologic culture of urine were performed. In culture-positive dogs, subclinical bacteriuria was confirmed by 2 positive culture results within 2 weeks and dogs were reevaluated at 3 months. Results - The prevalence of subclinical bacteriuria in healthy female dogs was 9 of 101 (8.9%). Three-month follow-up data were available for 8 of 9 dogs with subclinical bacteriuria. Four dogs had persistent bacteriuria, and 4 had transient bacteriuria. No dogs with subclinical bacteriuria developed clinical signs during the 3-month observation period. Subclinical bacteriuria was diagnosed in 6 of 51 (12%) young and middle-aged dogs and 3 of 50 (6.0%) senior and geriatric dogs. No significant difference was found in the prevalence of subclinical bacteriuria with age. Conclusions and Clinical Relevance - Results suggested that subclinical bacteriuria is a nonprogressive condition in healthy female dogs and can be persistent or transient. No significant difference in the prevalence of subclinical bacteriuria in young and middle-aged dogs versus senior and geriatric dogs was detected. No dogs with subclinical bacteriuria developed clinical signs requiring antimicrobial treatment during the 3-month observation period. Healthy female dogs with subclinical bacteriuria may be a population of dogs in which antimicrobial treatment is unnecessary.

Lippi I, Guidi G, Marchetti V, et al
JAVMA 2014;1135-1140; doi: 10.2460/javma.245.10.1135

Objective - To investigate serum calcium-phosphorus concentration product (sCaPP) as a predictor of mortality rate in dogs with chronic kidney disease (CKD). Design - Retrospective case-control study.
Animals - 31 dogs with definitive CKD and 35 apparently healthy dogs. Procedures - All dogs had been referred for nephrological consultation between December 2008 and December 2010. Dogs with CKD had stable disease for ≥ 3 months. On the basis of glomerular filtration rate < 60 mL/min/m², 13 of the 35 apparently healthy dogs were subsequently classified as having early CKD. Disease stage among dogs was determined on the basis of plasma creatinine concentration as follows: stage 1, < 123.7 μmol/L (n = 13), stage 2, 123.7 to 176.8 μmol/L (7); stage 3, 185.6 to 442 μmol/L (13); or stage 4, > 442 μmol/L (11). For each dog, serum concentrations of ionized and total calcium and phosphorus were evaluated once; the latter 2 variables were used to determine sCaPP. Results - The sCaPP differed significantly between the 22 healthy dogs and dogs with stage 3 or stage 4 CKD. The proportion of dogs with sCaPP > 70 mg/dL increased with stage of disease. Mortality rate among the 24 dogs with sCaPP > 70 mg/dL was higher than that among the 42 dogs with sCaPP ≤ 70 mg/dL. Dogs with sCaPP > 70 mg/dL had a comparatively lower survival rate, and risk of death was 4.2 times as high for dogs with sCaPP > 70 mg/dL. Conclusions and Clinical Relevance - For dogs with CKD, sCaPP > 70 mg/dL appeared to be a negative prognostic indicator, which was not influenced by the concomitant serum concentrations of phosphorus and total or ionized calcium.

Prognostic value of early magnetic resonance imaging in dogs after traumatic brain injury: 50 cases

Background - The prognostic value of early magnetic resonance imaging (MRI) in dogs after traumatic brain injury (TBI) remains unclear. Objectives - Determine whether MRI findings are associated with prognosis after TBI in dogs. Animals - Fifty client-owned dogs. Methods - Retrospective study of dogs with TBI that underwent 1.5T MRI within 14 days after head trauma. MRI evaluators were blinded to the clinical presentation, and all images were scored based on an MRI grading system (Grade I [normal brain parenchyma] to Grade VI [bilateral lesions affecting the brainstem with or without any lesions of lesser grade]). Skull fractures, percentage of intraparenchymal lesions, degree of midline shift, and type of brain herniation were evaluated. MGCS was assessed at presentation. The presence of seizures was recorded. Outcome was assessed at 48 h (alive or dead) and at 3, 6, 12, and 24 months after TBI. Results - Sixty-six percent of the dogs had abnormal MRI findings. MRI grade was negatively correlated (P < 0.001) with MGCS. A significant negative correlation of MRI grade, degree of midline shift, and percentage of intraparenchymal lesions with follow-up scores was identified. The MGCS was lower in dogs with brain herniation (P = 0.0191). Follow-up scores were significantly lower in dogs that had brain herniation or skull fractures. The possibility of having seizures was associated with higher percentage of intraparenchymal lesions (P = 0.0054) and 10% developed PTE. Conclusions and Clinical Importance - Significant associations exist between MRI findings and prognosis in dogs with TBI. MRI can help to predict prognosis in dogs with TBI.

Chiari-like malformation and syringomyelia in American Brussels Griffon dogs

Background - Although Chiari-like malformation (CM) and syringomyelia (SM) have been described in many small breed dogs, the prevalence and clinical manifestations of this complex have not been documented in a large cohort of American Brussels Griffon (ABG) dogs. Objectives - To characterize the clinical and magnetic resonance imaging (MRI) features of CM and SM in the ABG breed. Animals - Eighty-four American Kennel Club registered ABG dogs were recruited. Methods - Prospective study. Complete histories and neurologic examinations were obtained before MRI. Images were blindly reviewed and calculations were made by using OsiriX. All analyses were performed by Student’s t-test, Spearman’s correlation, ANOVA, and chi-square test where appropriate. Results - Chiari-like malformation and SM were present in 65% and 52% of dogs, respectively. Twenty-eight percent of dogs had neurologic deficits and 20% had neck pain. Mean central canal (CC) transverse height was 2.5 mm with a mean length of 3.6 cervical vertebrae. Neurologic deficits were significantly associated with a larger syrinx (p = 0.04, p = 0.08) and syrinx size increased with age (p = 0.027). SM was associated with a smaller craniovertebral junction (CCJ) height (p = 0.04) and larger ventricles (p = 0.0001; p < 0.001). Conclusions and Clinical Importance - Syringomyelia and CM are prevalent in American Brussels Griffon dogs. Syrinx size is associated with neurologic deficits, CM, larger ventricles, a smaller craniovertebral junction height, neurologic deficits, and cerebellar herniation. Fifty-two percent of dogs with a SM were clinically normal.

Osseous-associated cervical spondylomyelopathy in dogs: 27 cases (2000-2012)

Objective - To evaluate the signalment, neurologic examination and imaging findings, and outcome in dogs treated medically or surgically for osseous-associated cervical spondylomyelopathy (OACSM). Design - Retrospective case series. Animals - 27 client-owned dogs. Procedures - Medical records for dogs with OACSM (diagnosis made in 2000 through 2012) were reviewed. Collected data included signalment, neurologic examination findings (graded from 0 [normal] to 5 [tetraplegia]), imaging findings, treatment, and outcome. From MRI and CT images, measurements were obtained for subjective grading of spinal cord compression. Results - Among the 27 dogs, the median age was 2 years; there were 15 Great Danes, 3 Mastiffs, 3 Newfoundlands, and 6 other large-breed dogs. For medically treated dogs (n = 7), the median initial neurologic grade was 2; for surgically treated dogs (20), the median initial neurologic grade was 3. Magnetic resonance imaging revealed dorsolateral spinal cord compression in 22 dogs and lateral spinal cord compression in 5 dogs. Dogs with more severe compressions were slightly more likely to undergo surgical than medical treatment. Median survival time of medically treated dogs was 43 months, and that of surgically treated dogs was 60 months. Fifteen of 19 dogs treated surgically had improved neurologic grades at 4 to 8 weeks after surgery and had a good to excellent long-term outcome. Conclusions and Clinical Relevance - Surgical treatment of dogs with OACSM resulted in neurologic improvement and was associated with a good long-term outcome. For dogs that received medical treatment, neurologic deterioration continued but some patients did well for several years.

Neurologic outcome after thoracolumbar partial lateral corpectomy for intervertebral disc disease in 72 dogs
Objective - To determine neurologic outcome and factors influencing outcome after thoracolumbar partial lateral cortectomy (PLC) in dogs with intervertebral disc disease (IVDD) causing ventral spinal cord compression. Study Design - Retrospective case series. Animals - Dogs with IVDD (n = 72; 87 PLC). Methods - Dogs with IVDD between T9 and L5 were included if treated by at least 1 PLC. Exclusion criteria were: previous spinal surgery, combination of PLC with another surgical procedure. Neurologic outcome was assessed by: (1) modified Frankel score (MFS) based on neurologic examinations at 4 time points (before surgery, immediately after PLC, at discharge and 4 weeks after PLC); and (2) owner questionnaire. The association of the following factors with neurologic outcome was analyzed: age, body weight, duration of current neurologic dysfunction (acute, chronic), IVDD localization, breed (chondrodystrophic, nonchondrodystrophic), number of PLCs, degree of presurgical spinal cord compression and postsurgical decompression, slot depth, presurgical MFS. Presurgical spinal cord compression was determined by CT myelography (71 dogs) or MRI (1 dog), whereas postsurgical decompression and slot depth were determined on CT myelography (69 dogs). Results - MFS was improved in 18.7%, 31.7%, and 64.2% of dogs at the 3 postsurgical assessments, whereas it was unchanged in 62.6%, 52.8%, and 32.0% at corresponding time points. Based on owner questionnaire, 91.4% of dogs were ambulatory 6 months postsurgically with 74.5% having a normal gait. Most improvement in neurologic function developed within 6 months after surgery. Presurgical MFS was the only variable significantly associated with several neurologic outcome measurements (p < 0.01). Conclusions - PLC is an option for decompression in ventrally compressing thoracolumbar IVDD. Prognosis is associated with presurgical neurologic condition.

Clinical signs and outcome of dogs treated medically for degenerative lumbosacral stenosis: 98 cases (2004–2012)
De Decker S, Wawrzenski LA, Volk HA
JAVMA 2014;408-413; doi: 10.2460/javma.245.4.408
Objective - To compare clinical signs of dogs treated medically or surgically for degenerative lumbosacral stenosis (DLS) and assess outcome after medical treatment. Design - Retrospective case series. Animals - Client-owned dogs treated medically (n = 49) or surgically (49) for DLSS. Procedures - Medical records from 2004 to 2012 were reviewed. Dogs were included if they had clinical signs, clinical examination findings, and MRI abnormalities consistent with DLS. Several variables were compared between surgically and medically treated dogs: age, sex, duration of clinical signs, presence or absence of neurologic deficits, urinary and fecal incontinence, concurrent medical conditions, and medical treatment before referral. Medical treatment after obtaining a final diagnosis of DLSS consisted of restricted exercise in combination with anti-inflammatory and analgesic drugs. Surgical treatment consisted of dorsal lumbosacral laminectomy. Outcome for medically treated dogs was obtained via a standardized questionnaire. Results - Neurologic deficits were observed significantly more often in surgically treated dogs. Surgically treated dogs had unsuccessful medical treatment before referral significantly more often than did medically treated dogs. Thirty-one of 49 (63.3%) medically treated dogs were available for follow-up evaluation. Of these 31 dogs, 17 (55%) were managed successfully, 10 (32.3%) were managed unsuccessfully and underwent surgical treatment, 3 (9.7%) were euthanized because of progression of clinical signs, and 1 (3.2%) was alive but had an increase in severity of clinical signs after medical management. Conclusions and Clinical Relevance - Clinical signs differed in dogs treated medically or surgically for DLSS. Medical treatment for dogs with DLSS was associated with a fair prognosis.

Arthroscopically assisted treatment of injury to the lateral glenohumeral ligament in dogs
Ridge PA, Cook JL, Cook CR
Vet Surg 2014;43:958-962; doi 10.1111/j.1532-950X.2014.12205.x
Objective - To report short and medium term outcomes, and complications, in dogs treated for rupture of the lateral glenohumeral ligament (LGHL) with a novel, arthroscopically assisted technique. Study Design - Retrospective case series. Animals - Dogs (n = 10) with LGHL injury. Methods - Dogs were included after arthroscopic confirmation of rupture of the LGHL and treatment using an arthroscopically assisted suture anchor technique with informed owner consent. Outcomes were assessed by the authors and owners and complications arising from treatment recorded. Results - After 6 months, 7 dogs were assessed as having full function, 2 as having acceptable function, and 1 had poor function. There was 1 minor intra-operative and 1 minor post-operative complication. There were no major complications encountered 6 months after treatment. Conclusions - Dogs with LGHL injury can be treated using this novel technique with low complication rates and good outcomes after 6 months.

Short- and Long-term outcomes after arthroscopic treatment of young large breed dogs with medial compartment disease of the elbow
Barthélémy NP, Griffon DJ, Ragetly GR, et al
Vet Surg 2014;43:935-943; doi 10.1111/j.1532-950X.2014.12255.x
Objectives - To report short- and long-term outcomes after arthroscopic treatment in young large breed dogs affected by medial coronal process disease (MCPD) and identify variables affecting outcome. Study Design - Prospective observational case series. Animals - Large breed dogs <3 years old (n = 15; 23 elbows). Methods - MCPD was confirmed by radiography, computed tomography, and arthroscopy. Dogs were treated by arthroscopy. Variables recorded at time of treatment included radioulnar incongruity (RUI) and degree of cartilage erosion. Variables recorded before, 6 weeks, and ±23 months after surgery included radiographic score for osteoarthritis, trochlear notch sclerosis, muscle circumference, range of motion (ROM), and the load distribution of ground reaction forces between thoracic and pelvic limbs. Results - A greater load distribution to the pelvic limbs was identified preoperatively in dogs with RUI than in dogs with congruent elbows. Load distribution was not significantly improved at 6 weeks compared with preoperatively. Muscular circumference and vertical impulse distributions were improved at long-term evaluation despite an increased osteoarthritis score. This improvement was more obvious in dogs with RUI or a high degree of cartilage erosion at initial presentation. Conclusion - Some evidence of improvement in long-term function was found in dogs with MCPD after arthroscopic treatment. RUI and cartilage erosion at the time of diagnosis were associated with more lameness preoperatively but did not affect the final gait assessment or osteoarthritis score in this small cohort.

Incidence and type of meniscal injury and associated long-term clinical outcomes in dogs treated surgically for cranial cruciate ligament disease
Ritzo ME, Ritzo BA, Siddens AD, et al
Objective - To evaluate factors related to meniscal pathology and their effect on clinical outcome in dogs treated for cranial cruciate ligament (CCL) disease. Study Design - Prospective cross-sectional study,
Animals - Dogs (n=163) with CCL disease (n=223 stifles). Methods - CCL disease was treated by (1) arthroscopy and TightRope (TR) stabilization; (2) arthroscopy and tibial plateau leveling osteotomy (TPLO); or (3) open arthroscopy and TPLO. Incidences of concurrent and subsequent meniscal tears, meniscal treatments, mid-(6 months) and long-(>1 year) term outcomes by owner assessment were compared among surgical treatment groups. Results - Concurrent meniscal tears were diagnosed in 83% of stifles assessed by arthroscopy and 44% of stifles assessed by arthroscopy, with concurrent tear diagnosis being 1.9 times more likely by arthroscopy than arthrotomy (p<0.001). Incidence of diagnosis of subsequent meniscal tears was 6.7% with median time to diagnosis of 5.8 months. Differences in proportion of subsequent meniscal tears among treatment groups were not significant (p=0.69). Subsequent meniscal tears were diagnosed in 21% of cases without concurrent meniscal tears, but only 1.3% of cases with concurrent meniscal tears (p<0.001). Cases treated with meniscal release did not have subsequent meniscal tears, whereas dogs not treated with meniscal release had a subsequent meniscal tear rate of 11% (p=0.0013). Cases diagnosed and treated for concurrent meniscal tears were 1.3 times more likely to have a successful long-term outcome (p=0.007). Conclusions - Surgical technique did not affect subsequent meniscal tear rate similarly to the CCL disease treated by (1) arthroscopy and TightRope (TR) stabilization; (2) arthroscopy and tibial plateau leveling osteotomy (TPLO); or (3) open arthroscopy and TPLO. Objective - To describe the technique for, and long-term clinical outcome of, a modified hip toggle stabilization using the TightRope® system for coxofemoral luxation repair. Study Design - Retrospective case series. Methods - Medical records (July 2008–July 2010) including radiographs (17 limbs) of dogs that had coxofemoral luxation repaired with the TightRope system were reviewed. Follow-up (≥12 months) was obtained by telephone interview of owners. Six dogs were available for re-evaluation, radiographs, and objective gait analysis. Results - Follow-up (mean, 24 months; range, 12-43 months) by telephone interview was available for 17 dogs. Of these, 6 dogs were re-evaluated (mean, 7.5 months; median 12.5 months; range, 4-24 months) and had gait analysis. Mean duration of luxation before surgical intervention was 7.5 days (median, 7 days; range, 2-44 days). There was a single case of relaxation 27 months postoperatively. One dog died from non-surgical related circumstances. Objective gait analysis showed equal pelvic limb use in all 6 dogs available for re-evaluation. All owners of living dogs reported limb function as being good to excellent, and perceived that their dogs were pain free. Radiographs (mean, 7.5 months; median, 12.5 months; range, 4-24 months post surgery) of 6 dogs showed no progression of osteoarthritis in comparison to immediate postoperative radiographs. Conclusions - Hip toggle with the TightRope system as a prosthetic ligament of the head of the femur produces a favorable clinical outcome with high owner acceptance.

Ability of the Tightrope® and percutaneous lateral fabellar suture techniques to control cranial tibial translation

Biskup JJ, Griffon DJ, Socie M, et al

Objective - To compare the ability of the Tightrope® (TR) cranial cruciate ligament (CCL) technique, percutaneous lateral fabellar suture (pLFS) technique, and normal CCL to control cranial tibial translation (CTT). Study Design - In vitro biomechanical study. Sample Population - Cadaveric canine pelvic limbs (n=18 pairs). Methods - Six small animal surgical residents (1 pair each) and a Diplomate of the American College of Veterinary Surgeons (10 pairs) performed TR and pLFS techniques on paired limbs. Two intact limb pairs served as controls. Limbs were assessed by palpation, radiographs, and dissection before mechanical testing of resistance to CTT. Forces resisted during displacement were compared between groups with a mixed ANOVA and post hoc tests. Results - With 5 mm of displacement, the pLFS resisted 72 ± 45 N and the TR resisted 66 ± 48 N of load. The intact CCL resisted 400 ± 35 N. The intact CCL resisted displacement significantly more than either surgical technique. Conclusions - TR and pLFS had similar ability to resist CTT but neither restored the biomechanical properties of an intact CCL.

Appendicular osteosarcoma in small-breed dogs: 51 cases (1986-2011)

Amsellem PM, Selmic LE, Wypij JM, et al
JAVMA 2014;203-210; doi: 10.2460/javma.245.2.203

Objective - To describe outcomes for small-breed dogs with appendicular osteosarcoma. Design - Multi-institutional retrospective case series. Animals - 51 small-breed dogs. Procedures - Records from participating
Veterinary Society of Surgical Oncology members were searched for dogs that weighed <15 kg (33 lb) with a histologic diagnosis of appendicular osteosarcoma. The Kaplan–Meier method was used to determine median survival times (MSTs), and Cox regression was performed to identify variables associated with survival time. Results - Tumors were most commonly located on the humerus (n = 15) and femur (14). Of the 51 study dogs, 9 were treated nonsurgically, 16 underwent amputation of the affected limb only, and 26 underwent curative-intent treatment, with MSTs of 112, 257, and 415 days, respectively. The MST did not differ significantly between dogs in the amputation-only and curative-intent groups. For dogs in the nonsurgical group, MST decreased significantly as the tumor histologic score increased. For dogs in the amputation-only group, MST decreased as body weight increased. Conclusions and Clinical Relevance - For the small-breed dogs with appendicular osteosarcoma of the present study, tumor histologic grade and mitotic index were subjectively lower and MST following amputation of the affected limb without adjuvant chemotherapy was longer, compared with those for similarly affected larger dogs. Results indicated no significant advantage in MST for dogs that underwent curative-intent treatment versus dogs that underwent amputation only, and further investigation of the importance of adjuvant chemotherapy is warranted.

Evaluation of outcome and prognostic factors for dogs living greater than one year after diagnosis of osteosarcoma: 90 cases (1997-2008)

Culp WTN, Olea-Popelka F, Sefton J, et al
JAVMA 2014;1141-1146; doi: 10.2460/javma.245.10.1141

Objective - To evaluate clinical characteristics, outcome, and prognostic variables in a cohort of dogs surviving >1 year after an initial diagnosis of osteosarcoma. Design - Retrospective case series. Animals - 90 client-owned dogs. Procedures - Medical records for an 11-year period from 1997 through 2008 were reviewed, and patients with appendicular osteosarcoma that lived >1 year after initial histopathologic diagnosis were studied. Variables including signalment, weight, serum alkaline phosphatase activity, tumor location, surgery, and adjuvant therapies were recorded. Median survival times were calculated by means of a Kaplan-Meier survival function. Univariate analysis was conducted to compare the survival function for categorical variables, and the Cox proportional hazard model was used to evaluate the likelihood of death >1 year after diagnosis on the basis of the selected risk factors. Results - 90 dogs met the inclusion criteria; clinical laboratory information was not available in all cases. Median age was 8.2 years (range, 2.7 to 13.3 years), and median weight was 38 kg (83.6 lb; range, 21 to 80 kg [46.2 to 176 lb]). Serum alkaline phosphatase activity was high in 29 of 60 (48%) dogs. The most common tumor location was the distal portion of the radius (54/90 [60.0%]). Eighty-nine of 90 (99%) dogs underwent surgery, and 78 (87%) received chemotherapy. Overall, 49 of 90 (54%) dogs developed metastatic disease. The median survival time beyond 1 year was 243 days (range, 1 to 1,899 days). Dogs that developed a surgical-site infection after limb-sparing surgery had a significantly improved prognosis >1 year after osteosarcoma diagnosis, compared with dogs that did not develop infections. Conclusions and Clinical Relevance - Results of the present study indicated that dogs with an initial diagnosis of osteosarcoma that lived >1 year had a median survival time beyond the initial year of approximately 8 months. As reported previously, the development of a surgical-site infection in dogs undergoing a limb-sparing surgery significantly affected prognosis and warrants further study.

Outcome and prognostic factors for osteosarcoma of the maxilla, mandible, or calvarium in dogs: 183 cases (1986–2012)

Selmic LE, Lafferty MH, Kamstock DA, et al
JAVMA 2014;930-938; doi: 10.2460/javma.245.8.930

Objective - To describe the biological behavior, clinical outcome, and prognostic factors of osteosarcoma of the maxilla, mandible, or calvarium in dogs. Design - Retrospective case series. Animals - 183 client-owned dogs with osteosarcoma of the maxilla, mandible, or calvarium. Procedures - Medical records for dogs treated for osteosarcoma of the maxilla, mandible, or calvarium from 1986 through 2012 were reviewed. Dogs with a histopathologic diagnosis of osteosarcoma and treated for a primary tumor arising from these bones of the head were included. Results - Mean age was 9.3 years, and body weight was 31.8 kg (70.0 lb). Most dogs (124/183 [67.8%]) were purebread, and the most common primary tumor site was the maxilla (80 [43.7%]). Treatments included palliative medical treatment only (11/183 [6.0%]), coarsely fractionated radiation therapy (RT; 12 [6.6%]), fractionated or stereotactic RT (18 [9.8%]), surgery (135 [73.8%]), and both surgery and fractionated RT (7 [3.8%]). Eighty-three (45.4%) dogs received adjuvant chemotherapy. Local recurrence or progression occurred in 80 of 156 (51.3%) dogs, and 60 of 156 (38.5%) dogs developed distant metastases. Median survival time for all dogs was 239 days. Dogs that underwent surgery had a median survival time of 329 days. Histologically tumor-free surgical margins were associated with significantly decreased hazards of progression or recurrence (hazard ratio [HR], 0.4) and death (HR, 0.5). Dogs with osteosarcoma of the calvarium had a significantly greater hazard of local recurrence or progression (HR, 2.0). Conclusions and Clinical Relevance - In this study, tumor excision in dogs with histologically tumor-free margins resulted in better local control and longer survival time than did other treatment types.

Canine soft tissue sarcoma managed in first opinion practice: Outcome in 350 cases

Bray JP, Polton GA, McSparron KD, et al

Objective - To determine outcome of dogs with a diagnosis of soft tissue sarcoma managed in first opinion practice. Study Design - Retrospective, case-controlled study. Animals - Dogs (n=350) with primary occurrence of a soft tissue sarcoma. Methods - A previously validated questionnaire was sent to all veterinarians requesting clinical information and ultimate outcome for all dogs. Histologic sections were reviewed by a single pathologist. Results - Most surgeries were unplanned, with only 15 (4%) dogs having a histologic and 59 (16.8%) dogs having a cytologic diagnosis before surgery. Median survival time for all dogs was not reached with 70% proportional survival at 5 years. Local recurrence developed in 73 (20.8%) cases. The extent of resection performed was not associated with improved survival (P = .2) or tumor recurrence (P = .8). Age <8 years (χ² = 6.1; P = .01), tumors <5 cm in size (χ² = 9.6; P = .002) and discrete tumors (χ² = 16.6; P <.001) had improved survival outcomes. On multivariate analysis, a high tumor grade was significant for recurrence (HR 5.8; P <.001; 95% CI: 2.2–14.8). Evidence of a selection bias towards less aggressive tumors being managed in first opinion practice was confirmed. Conclusions - Wide resection margins are not the primary determinant of outcome for all soft tissue sarcoma. Veterinarians need to better understand the biologic behavior of a suspected soft tissue sarcoma before treatment to allow surgical margins to be adjusted accordingly.
Efficacy of systemic adjuvant therapies administered to dogs after excision of oral malignant melanomas: 151 cases (2001–2012)

Boston SE, Lu X, Culp WTN, et al
JAVMA 2014;401-407; doi: 10.2460/javma.245.4.401

Objective - To determine prognostic factors for and compare outcome among dogs with oral malignant melanoma following excision with or without various systemic adjuvant therapies. Design - Retrospective case series. Animals - 151 dogs with naturally occurring oral malignant melanomas treated by excision with or without adjuvant therapies from 2001 to 2012. Procedures - Case accrual was solicited from Veterinary Society of Surgical Oncology members via an email list service. Information collected from case records included signalment, tumor staging, tumor characteristics, type of surgical excision, histologic diagnosis, adjuvant therapy, and survival time. Results - The overall median survival time was 346 days. Results of multivariate analysis indicated that tumor size, patient age, and intraslesional excision (vs marginal, wide, or radical excision) were considered poor prognostic indicators. All other demographic and clinical variables were not significantly associated with survival time after adjusting for the aforementioned 3 variables. A clear survival benefit was not evident with any systemic adjuvant therapy, including vaccination against melanoma or chemotherapy; however, the number of dogs in each treatment group was small. Ninety-eight dogs received no postoperative adjuvant therapy, and there was no difference in survival time between dogs that did (335 days) and did not (352 days) receive systemic adjuvant therapy. Conclusions and Clinical Relevance - For dogs with oral malignant melanoma, increasing tumor size and age were negative prognostic factors. Complete excision of all macroscopic tumor burden improved survival time. Long-term survival was possible following surgery alone. Although systemic adjuvant therapy was not found to improve survival time, this could have been due to type II error.

Autologous platelet gel to treat chronic decubital ulcers: A randomized, blind controlled clinical trial in dogs

Tambella AM, Attili AR, Dini F, et al

Objective - To determine the efficacy of topical application of the autologous platelet gel (PG) in canine chronic protracted decubital ulcers. Study Design - Prospective, randomized, blind controlled clinical trial. Animals - Dogs (n = 18) with bilateral chronic wounds caused by protracted decubitus ulcers. Methods - For each dog, wound side was randomized to receive either platelet gel (group PG) every 5 days for 5 dressing changes, or paraffin gauzes dressings (group C), as negative control. Wound healing and wound surfaces were compared at admission and then evaluated every 5th day, until day 25. Outcome variables were: open wound area, reduction of open wound surface compared to admission and to each preceding dressing change, time to complete epithelialization. Results - Significant differences in healing process were observed at day 5 and continued throughout the entire study period (p < 0.00001). At 25 days, mean percent reduction in wound area was 93.5% in group PG and 13.2% in group C (p < 0.00001). Conclusions - Appropriately prepared autologous PG, an inexpensive, readily available blood derivative, applied topically results in more rapid healing of chronic non-healing decubital ulcers in dogs than those treated by use of paraffin-impregnated gauzes.

Effects of maropitant citrate or acepromazine on the incidence of adverse events associated with hydromorphone premedication in dogs

Claude AK, Dedaux A, Chiavaccini L, Hinz S
JVIM 2014;28:1414-1417; doi.org/10.1111/jvim.12414

Background - Vomiting is a common complication associated with the use of hydromorphone for pre-emptive analgesia in dogs. The ideal anti-emetic protocol for prevention of this complication has not been established. Hypothesis - Maropitant administered concurrently or before hydromorphone would reduce the incidence of vomiting, signs of nausea, ptalism, and increased panting compared to administration of acepromazine or a 0.9% saline control. Animals - Sixty mixed-breed female dogs scheduled for ovariohysterectomy. Methods - Randomized, blinded, placebo-controlled experimental study. Dogs were assigned to 4 experimental groups with 15 dogs per group. All groups received 0.2 mg/kg of hydromorphone IM. Group “Control” received 0.1 mL/kg saline SC 30–45 minutes before hydromorphone, group “Marop1” received 1 mg/kg maropitant SC 30–45 minutes before hydromorphone, group “Ace” received 0.02 mg/kg IM acepromazine 30–45 minutes before hydromorphone, and group “Marop2” received 1 mg/kg SC maropitant concurrently with hydromorphone. A trained and blinded observer documented adverse events from the time hydromorphone was administered until the time drugs were induced for surgery. Results - Marop1 had significantly less vomiting (0%) compared to Control (87%; p < 0.01) and Ace (53%; p < 0.01). Marop2 had significantly less vomiting (27%) compared to Control (p < 0.01). Marop1 had significantly greater incidence of ptalism (73%) compared to Ace (p < 0.01; 20%). Ace showed significantly less panting (33%) compared to Marop2 (93%; p < 0.01). Conclusions and Clinical Importance - In healthy dogs, maropitant citrate administered before hydromorphone significantly decreases the incidence of vomiting in dogs but does not improve signs of nausea, ptalism, or increased panting.

Use of trazodone to facilitate postsurgical confinement in dogs

Gruen ME, Roe SC, Griffith E, et al
JAVMA 2014;296-301; doi: 10.2460/javma.245.3.296

Objective - To investigate the safety and efficacy of oral administration of the serotonin antagonist and reuptake inhibitor trazodone hydrochloride to facilitate confinement and calming after orthopedic surgery in dogs. Design - Prospective open-label clinical trial. Animals - 36 client-owned dogs that underwent orthopedic surgery. Procedures - Starting the day after surgery, dogs were administered trazodone (approx 3.5 mg/kg [1.6 mg/lb], PO, q 12 h) with tramadol (4 to 6 mg/kg [1.8 to 2.7 mg/lb], PO, q 8 to 12 h) for pain management. After 3 days, administration of tramadol was discontinued, and the trazodone dosage was increased (approx 7 mg/kg [3.2 mg/lb], PO, q 12 h) and maintained for at least 4 weeks. If needed, trazodone dosage was increased (7 to 10 mg/kg [3.2 to 4.5 mg/lb], PO, q 8 h). Owners completed electronic surveys rating their dogs’ confinement tolerance, calmness or hyperactivity level, and responses to specific provocative situations prior to surgery and 1, 2, 3, and 4 weeks after surgery and at the postsurgery evaluation (at 8 to 12 weeks). Results - Most (32/36 [89%]) of owners reported that their dogs, when given trazodone during the 8 to 12 weeks following orthopedic surgery, improved moderately or extremely with regard to confinement tolerance and calmness. Trazodone was well tolerated, even in combination with NSAIIDs, antimicrobials, and other medications; no dogs were withdrawn from the study because of adverse reactions. Owner-reported median onset of action of trazodone was 31 to 45
minutes, and median duration of action was ≥ 4 hours. **Conclusions and Clinical Relevance** - Results suggested that oral administration of trazodone was safe and efficacious and may be used to facilitate confinement and enhance behavioral calmness of dogs during the critical recovery period following orthopedic surgery.

**Clinical outcome after diagnosis of hemophilia A in dogs**

**Methods** - CBC, and 46 healthy controls. **Background** - AMC > 46, and 46 healthy controls. **Hypothesis/Objectives** - To evaluate the clinical course of dogs with hemophilia A (factor VIII deficiency) and to determine whether factor VIII coagulant activity (FVIII:C) was associated with severity of clinical signs and outcome. **Design** - Survey study. **Sample** - Randomized information for 39 client-owned dogs with FVIII deficiency. **Procedures** - Data from 39 dogs (38 males and 1 female) were compiled. **Results** - Data from 39 dogs (38 males and 1 female) were compiled. Mixed-breed dogs, German Shepherd Dogs, and Labrador Retrievers were most commonly affected. In most (34/39) dogs, disease was diagnosed at < 1 year of age. Bleeding associated with teething, minor trauma, vaccination, and elective surgical procedures most commonly prompted FVIII:C testing. Affected dogs had similar signs of spontaneous hemorrhage regardless of the magnitude of FVIII deficiency. Four dogs were euthanized without treatment at the time of diagnosis. Thirty dogs received ≥ 1 blood transfusion; FVIII:C did not appear to influence transfusion requirements. **Conclusions and Clinical Relevance** - Results indicated that dogs with hemophilia A have variations in clinical course of the disease and may have a good long-term prognosis. Residual FVIII:C may not be useful for predicting severity of clinical signs, transfusion needs, or long-term prognosis.

**Platelet volume and plateletcrit in dogs with presumed primary immune-mediated thrombocytopenia**

**Schwartz D, Sharkey L, Armstrong PJ, et al**

**Background** - Mean platelet volume (MPV) and plateletcrit (PCT) are indices used in evaluating immune-mediated thrombocytopenia (IMT) in humans and in dogs with congenital macrothrombocytopenia. These indices may provide clinically valuable information in acquired thrombocytopenia. **Hypothesis/Objectives** - Dogs with presumed primary IMT will have increased MPV, and therefore platelet mass (PCT) will increase faster than platelet count (PLT) during recovery. **Animals** - Forty-nine dogs with automated PLT < 30,000/µL because of presumed primary IMT and hematocrit (HCT), PCT, MPV, and platelet distribution width determined from the same complete blood count (CBC), and 46 healthy controls. **Methods** - Case-control retrospective study; PLT, PCT, MPV, and platelet distribution width (PDW) were recorded from CBCs from 49 dogs, with 45 having data collected on the day of presentation. Fifteen were confirmed to have attained a PLT ≥ 75,000/µL on at least 1 CBC within 15 days after admission. The PCT equivalent to a PLT of 75,000/µL (assuming an average MPV) was calculated for comparison with PLT in terms of time to achieve a threshold of platelet mass by the 2 measures. **Results** - Mean platelet volume was higher in IMT dogs (17.3 fl) than the reference population (10.5 fl) (p < 0.0001). The PDW was not significantly different among the groups. The median time for PCT to reach threshold in confirmed responders was faster (3 days) compared with PLT (4 days). **Conclusions and Clinical Importance** - Immune-mediated thrombocytopenia is characterized by increased MPV. Time to achieve a threshold PCT tended to be shorter than PLT, suggesting that PCT may be a useful platelet parameter for monitoring dogs with IMT.

**Effects of repeated blood donations on iron status and hematologic variables of canine blood donors**

**Ferreira RRF, Gopegui RR, Araujo MMR, et al**

**Design** - Survey study. **Sample** - Information was obtained via a survey distributed to the American College of Veterinary Internal Medicine and American College of Veterinary Emergency and Critical Care email list serves and to the Veterinary Information Network community to identify dogs with hemophilia A (FVIII:C ≤ 20%). Severity of FVIII deficiency was classified as mild (FVIII:C, 6% to 20%), moderate (FVIII:C, 2% to 5%), or severe (FVIII:C < 2%). **Results** - Data for 39 dogs (38 males and 1 female) were compiled. Mixed-breed dogs, German Shepherd Dogs, and Labrador Retrievers were most commonly affected. In most (34/39) dogs, disease was diagnosed at < 1 year of age. Bleeding associated with teething, minor trauma, vaccination, and elective surgical procedures most commonly prompted FVIII:C testing. Affected dogs had similar signs of spontaneous hemorrhage regardless of the magnitude of FVIII deficiency. Four dogs were euthanized without treatment at the time of diagnosis. Thirty dogs received ≥ 1 blood transfusion; FVIII:C did not appear to influence transfusion requirements. **Conclusions and Clinical Relevance** - Results indicated that dogs with hemophilia A have variations in clinical course of the disease and may have a good long-term prognosis. Residual FVIII:C may not be useful for predicting severity of clinical signs, transfusion needs, or long-term prognosis.

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respectively, before hydromorphone; signs of nausea were significantly decreased only when maropitant was administered 60 minutes before hydromorphone. Conclusions and Clinical Relevance - Results indicated that vomiting was significantly decreased and then prevented when maropitant was administered to dogs 15 and 30 minutes before hydromorphone. However, signs of nausea were significantly decreased only when the dosing interval was 60 minutes.

Long-term survival and quality of life in dogs with clinical signs associated with a congenital portosystemic shunt after surgical or medical treatment

Greenhalgh SN, Reeve JA, Johnstone T, et al JAVMA 2014;527-533; doi: 10.2460/javma.245.5.527

Objective - To compare long-term survival and quality of life data in dogs with clinical signs associated with a congenital portosystemic shunt (CPSS) that underwent medical or surgical treatment. Design - Prospective cohort study. Animals - 124 client-owned dogs with CPSS. Procedures - Dogs received medical or surgical treatment without regard to signalment, clinical signs, or clinicopathologic results. Survival data were analyzed with a Cox regression model. Quality of life information, obtained from owner questionnaires, included frequency of CPSS-associated clinical signs (from which a clinical score was derived), whether owners considered their dog normal, and (for surgically treated dogs) any ongoing medical treatment for CPSS. A Mann-Whitney U test was used to compare mean clinical score data between surgically and medically managed dogs during predetermined follow-up intervals. Results - 97 dogs underwent surgical treatment; 27 were managed medically. Median follow-up time for all dogs was 1,936 days. Forty-five dogs (24 medically managed and 21 surgically managed) died or were euthanized during the follow-up period. Survival rate was significantly improved in dogs that underwent surgical treatment (hazard ratio, 8.11; 95% CI, 4.20 to 15.66) than in those treated medically for CPSS. Neither age at diagnosis nor shunt type affected survival rate. Frequency of clinical signs was lower in surgically versus medically managed dogs for all follow-up intervals, with a significant difference between groups at 4 to 7 years after study entry. Conclusions and Clinical Relevance - Surgical treatment of CPSS in dogs resulted in significantly improved survival rate and lower frequency of ongoing clinical signs, compared with medical management. Age at diagnosis did not affect survival rate and should not influence treatment choice.

Analysis of the relationship of extrahepatic portosystemic shunt morphology with clinical variables in dogs: 53 cases (2009-2012)


Objective - To investigate differences in clinical variables among dogs with extrahepatic portosystemic shunts (EHPSSs) of various morphologies. Design - Retrospective case series. Animals - 53 dogs with EHPSSs. Procedures - Medical records of dogs undergoing preoperative CT angiography of an EHPSS over a 3-year period were reviewed. Analysis was performed to investigate relationships of clinical variables with shunt morphology. Morphologies were analyzed individually as well as in several groups. Results - Shunt morphologies included 10 splenocaval, 11 splenophrenic, 11 splenozigos, 10 right gastric-caval, 12 right gastric-caval with a caudal loop, and 1 right gastric-azygos with a caudal loop. Several biochemical variables associated with EHPSS were lowest in dogs with splenocaval shunts. Preoperative clinical signs were more common in dogs that had shunts with vena caval than right azygos vein insertion (36/41 [88%] vs 7/12 [58%]) and insertion caudal to the liver than diaphragmatic insertion (29/32 [91%] vs 14/21 [67%]). Neurologic signs were more common when shunts inserted into the vena cava caudal to the liver than in other locations (21/32 [66%] vs 6/21 [29%]) and were most frequent with splenocaval shunts. Urinary tract signs were more common when shunts had right gastric vein origin than gastroplenic vein origin (14/23 [61%] vs 10/30 [33%]). Conclusions and Clinical Relevance - Splenocaval shunts caused more clinical abnormalities than did other shunt morphologies. Results suggested that dogs with shunt insertion in the caudal vena cava, especially caudal to the liver, were most likely to have clinical signs.

Evaluation of in vivo behavior of ameroid ring constrictors in dogs with congenital extrahepatic portosystemic shunts using computed tomography


Objective - To evaluate the in vivo pattern of ameroid constritor closure of congenital extrahepatic portosystemic shunts in dogs. Study Design - Prospective study. Animals - Dogs (n = 22) with congenital extrahepatic portosystemic shunts. Methods - Contrast-enhanced computed tomography was performed immediately before, and at least 8 weeks after placement of ameroid ring constrictors. Plastic-encased ameroid constrictors were used in 17 dogs and metal constrictors in 5 dogs. Presence of residual flow through the portosystemic shunt, additional anomalous vessels, acquired shunts and soft tissue associated with the ameroid constrictor was recorded. Postoperative internal diameter was recorded for the 17 plastic constrictors. Correlations between internal diameter and pre- and postoperative serum protein concentration were analyzed. Results - No ameroid constrictor closed completely: shunt occlusion was always dependent on soft tissue within the ameroid ring. Residual flow through the shunt was present in 4 dogs (18%), although this caused persistent elevation of shunt fraction in only 1 dog (dog 8). The change in ameroid constrictor internal diameter was not significantly correlated with serum protein concentration. Conclusions - Complete shunt occlusion after AC placement is usually dependent on soft tissue reaction. Ameroid constrictors ≥5 mm diameter may not promote complete shunt occlusion.

Does hepatic steatosis have an impact on the short term hepatic response after complete attenuation of congenital extrahepatic portosystemic shunts? A prospective study of 20 dogs


Objective - To evaluate the relationship between hepatic steatosis and increase in liver size and resolution of shunting after surgical attenuation of congenital extrahepatic portosystemic shunts in dogs. Study Design - Prospective study. Animals - Dogs (n = 20) with congenital extrahepatic portosystemic shunts. Methods - Shunts were attenuated using ameroid ring constrictors. Portal blood flow and liver volume were evaluated using computed tomography before and 8 weeks after surgery. Hepatic steatosis was quantified by stereological point counting of lipid droplets and lipogranulomas (LG) in liver biopsies stained with Oil-red-O. Associations between steatosis and preoperative liver volume, liver growth after surgery, and development of acquired shunts were evaluated. Results - Acquired shunts developed in 2 dogs (10%).
Dogs with larger preoperative liver volumes relative to bodyweight had fewer lipid droplets per tissue point (p = 0.019). LG per tissue point were significantly associated with age: 0.019 ± 0.06 for dogs <12 months versus 0.25 ± 0.49 for dogs >12 months (p = 0.007). There was a significant positive association between liver growth after surgery and the number of LG/month of age in dogs >12 months (p = 0.003). There was no association between steatosis, presence of macrosteatosis, the number of LG or development of acquired shunts. Conclusions - This preliminary study suggests that the presence of hepatic lipidosis and LG has no demonstrable effect on development of acquired shunts or the magnitude of increase in liver volume after attenuation of congenital extrahepatic portosystemic shunts in dogs.

Correlation between liver volume, portal vascular anatomy, and hepatic perfusion in dogs with congenital portosystemic shunt before and after placement of ameroid constrictors

Zwingenberger AL, Daniel L, Steffey MA, et al

Objective - To correlate changes in hepatic volume, hepatic perfusion, and vascular anatomy of dogs with congenital extrahepatic portosystemic shunts, before and after attenuation with an ameroid constrictor. Study Design - Prospective study. Animals - Dogs (n = 22) with congenital extrahepatic portosystemic shunts. Methods - CT angiography and perfusion scans were performed before and after attenuation of a portosystemic shunt with an ameroid constrictor. Changes in hepatic volume, hepatic perfusion, and vascular anatomy were measured. Portal scintigraphy was performed in 8 dogs preoperatively and 22 dogs postoperatively. Results - Dogs with smaller preoperative liver volumes had greater increases in liver volume postoperatively compared with those with larger preoperative liver volumes. Hepatic arterial fraction was increased in dogs preoperatively and returned to normal range after shunt attenuation, and was correlated with increase in liver size and decreased shunt fraction. Three dogs with no visible portal vasculature preoperatively developed portal branches postoperatively. Conclusions - Dogs with smaller preoperative liver volumes had the largest postoperative increase in liver volume. Hepatic arterial perfusion and portal scintigraphy correlate with liver volume and are indicators of successful shunt attenuation. Dogs without visible vasculature on CT angiography had visible portal vasculature postoperatively.

DOGS AND CATS

Sewing needle foreign body ingestion in dogs and cats: 65 cases (2000-2012)

Pratt CL, Reineke EL, Drobatz KJ
JAVMA 2014;302-308; doi: 10.2460/javma.245.3.302

Objective - To characterize clinical signs, diagnostic test results, foreign body location, treatment, and outcome for dogs and cats with sewing needle foreign bodies. Design - Retrospective case series. Animals - 65 dogs and cats with sewing needle foreign bodies. Procedures - Medical records of 27 dogs and 38 cats examined because of sewing needle foreign bodies from January 2000 to February 2012 were reviewed for signalment, medical history, physical examination findings, diagnostic test results, interval from witnessed exposure and radiographic imaging to definitive treatment, definitive treatment, sewing needle location, complications, and outcome. Results - 7 (10.8%) animals had sewing needles in extragastrointestinal locations that were not causing clinical signs. The remaining 58 (89.2%) animals had known sewing needle exposure or acute clinical signs associated with ingestion. The esophageal and gastric regions were the most common location for a sewing needle (10/21 [47.6%] dogs; 19/37 [51.4%] cats), followed by the oropharynx (7/21 [33.3%] dogs; 11/37 [29.7%] cats) and small and large intestines (4/21 [19.0%] dogs; 7/37 [18.9%] cats). Gastrointestinal perforation was detected in 10 of 58 (17.2%) animals (5/21 [23.8%] dogs; 5/37 [13.5%] cats). Sewing needles in the esophagus and stomach were successfully removed endoscopically in 8 of 9 dogs and 18 of 19 cats. Survival rate was 98.1% (51/52) for animals receiving definitive treatment. Conclusions and Clinical Relevance - Endoscopic removal of ingested sewing needles was highly successful and should be recommended to prevent gastrointestinal tract perforation and associated morbidity. Prognosis for dogs and cats receiving definitive treatment for sewing needle foreign body ingestion was excellent.

GREYHOUNDS

Ischemic stroke in Greyhounds: 21 cases (2007-2013)

Kent M, Glass EN, Haley AC, et al
JAVMA 2014;113-117; doi: 10.2460/javma.245.1.113

Objective - To determine the prevalence of ischemic stroke in Greyhounds and determine whether affected dogs had coagulation abnormalities and hypertension. Design - Multi-institutional, retrospective study. Animals - 21 dogs. Procedures - Medical records (including diagnostic testing results) and MRI images of the brain were reviewed for Greyhounds with ischemic stroke that had been evaluated at 4 institutions. The proportion of Greyhounds with ischemic stroke was compared with the proportion of non-Greyhound dogs with ischemic stroke. Demographic information for dogs evaluated at each institution was obtained to determine the proportion of Greyhounds in the hospital populations. Results - 21 Greyhounds with ischemic stroke were identified. Abnormalities in coagulation were not identified in the 14 Greyhounds that underwent such testing. Systemic hypertension was identified in 6 of 14 Greyhounds that underwent such testing. No other abnormalities were identified by means of other routine diagnostic tests for Greyhounds. For all institutions combined, the prevalence of ischemic stroke in Greyhounds was 0.66% (21/3,161 Greyhounds). Greyhounds were significantly more likely to be evaluated because of ischemic stroke, compared with all other dog breeds combined (OR, 6.6; 95% confidence interval, 4.2 to 10.2). Conclusions and Clinical Relevance - Results of this study suggested that Greyhounds were predisposed to ischemic stroke, compared with all other breeds combined. Coagulation abnormalities did not seem to contribute to ischemic stroke. Hypertension may have contributed to the development of ischemic stroke. Greyhounds with ischemic stroke should undergo measurement of systolic arterial blood pressure. Antihypertensive treatments may be warranted for such dogs.

FERRETS

Suspected primary hypoparathyroidism in a domestic ferret (Mustela putorius furo)

de Matos RE, Connolly MJ, Starkey SR, Morrissey JK
JAVMA 2014;419-424; doi: 10.2460/javma.245.4.419

Case Description - A 4-year-old castrated male domestic ferret (Mustela putorius furo) was examined because of a 3-week history of intermittent seizures, signs of depression, hypocalcemia, and hyperphosphatemia. Clinical Findings - Plasma biochemical analysis confirmed hyperphosphatemia (17.7 mg/dL) and low concentrations of total (4.3 mg/dL) and ionized (0.49 mmol/L) calcium. Serum parathyroid hormone concentration (2.30 pmol/L) was low or in the
low part of the reference interval. Treatment and Outcome - Calcium gluconate was administered (2.0 mg/kg/h [0.9 mg/lb/h], IV), followed by a transition to administration of calcium carbonate (53 mg/kg [24.1 mg/lb], PO, q 12 h) and dihydrotachysterol (0.02 mg/kg/d [0.009 mg/lb/d], PO). Attitude of the ferret improved and seizures ceased as blood calcium concentrations increased. The ferret was reexamined because of seizures approximately 1 year after oral maintenance administration of dihydrotachysterol and calcium was initiated. The ferret responded well to emergency and long-term treatment but then was lost to follow-up monitoring. The ferret died approximately 2 years after the initial evaluation and treatment. Hypertrophic cardiomyopathy was diagnosed during necropsy, but the parathyroid glands could not be identified.

Clinical Relevance - To the authors’ knowledge, primary hypoparathyroidism has not previously been reported in a ferret. The condition should be considered for ferrets with hypocalcemia and hyperphosphatemia without azotemia. Treatment with dihydrotachysterol and oral supplementation of calcium appeared to be a viable option for long-term management.

RABBITS

Performance of two portable meters and a benchtop analyzer for blood glucose concentration measurement in rabbits

Selleri P, Di Girolamo N, Novari G

JAVMA 2014;245:87-98; doi: 10.2460/javma.245.1.87

Objective - To evaluate performance of a human portable blood glucose meter (PBGM), a veterinary PBGM, and a veterinary benchtop analyzer for measuring blood glucose concentration in rabbits and to evaluate the effect of sample characteristics on their performance.

Conclusions and Clinical Relevance - Results suggested that use of the human PBGM and with the benchtop analyzer and in 9% (canine setting) to 6.7% (feline setting) of cases with the veterinary PBGM. Clinical errors would have occurred in 0% of cases with the human PBGM and with the benchtop analyzer and in 9% (canine setting) to 6.7% (feline setting) of cases with the veterinary PBGM.
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