Australian
Veterinary Practitioner
Volume 51 (1) March 2021

Management of sleep disordered breathing in a dog using continuous positive airway pressure
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ISSN 0310 - 138X

A few words from the editor

Welcome to the 51st volume of the AVP. Yes, your practitioner-oriented journal is officially 50 years old with this issue!

As I reflected at the beginning of 2020, the purpose of the AVP is the same as it was at its inception, "To be published quarterly to further the interests of veterinary practitioners and students throughout Australasia". This is exemplified in this March issue, where we have 4 interesting case reports and an invited paper (a Creature Feature Article) written by practitioners, and a reprinting of the Australian Small Animal Veterinarians' guidelines for anaesthesia in dogs and cats:

Tso, Indrawirawan and Turner, from the Veterinary Referral Hospital in Dandenong, present a Cavalier King Charles spaniel puppy that suffered from acute respiratory distress after a 2-hour flight from the breeder to the new owners. The cause of the dyspnoea was determined to be an acute manifestation of a congenital pleuroperitoneal hernia thought to have resulted from pressure changes associated with the high-altitude flight. See pp.2-10 for more.

Nguyen, Newman, Tan and Chow, from Sydney Veterinary Emergency & Specialists (Rosebery), report on a Staffordshire bull terrier that was presented following blunt trauma to the chest, which had resulted in rib fractures, mild pneumothorax, pulmonary contusions and acute hepatic haemorrhage. Sounds reasonably straight-forward, but the unusual aspect of this case was that, 3 days after discharge (following 'successful treatment'), the dog developed a bilothorax. What happened? See pp. 11-18.

Wiseman, Rosenblatt and Kung, from Brisbane Veterinary Specialist Centre (Albany Creek) and The University of Queensland School of Veterinary Science, discuss a 4-year-old Cavalier King Charles spaniel presented for evaluation of disruptive apnoeic episodes, experienced every 10-15 minutes during sleep. Brachycephalic obstructive airway syndrome you say? I know the surgery that can assist with that problem. Unfortunately, it was of little assistance. However, the authors then tried using a continuous positive airway pressure device at night. Go to pp. 19-24 to find out the result of this novel treatment.

Brückner and Wagner, European veterinarians, discuss the use of laparoscopic surgery for the removal of cryptorchid testicles in two dogs. The unusual thing about these cases was that they both had torsion of the intra-abdominal testicle, one case presenting with acute pain and the other without such signs. Read more on pp. 26-31.

Gatt, from the Bird & Exotic Animal Clinic in Williamstown (Victoria), discusses the care and husbandry of sugar gliders (pp. 32-38). The article emphasises the importance of trying to emulate conditions in the wild as closely as possible.

The final article in this issue is a reprint (slightly edited from the original) of Anaesthesia guidelines for dogs and cats by **Warne, Bauquier, Pengelly, Neck and Swinney**. These guidelines were first published in the Aust Vet J 96:413-427, 2018. They have since been available from the ASAV website for members. They are being republished in this issue (pp. 39-63) to seek feedback from members in preparation for an update. Have a read and send any comments/ suggestions to Linda North in the ASAV office (linda.north@ava.com.au), with the subject heading "Anaesthesia Guidelines: Comments".

I hope that you find this issue both useful and interesting.

Bruce
Professor emeritus Bruce W. Parry

Acute manifestation of a rare congenital right-sided pleuroperitoneal hernia after a high-altitude flight

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ABSTRACT An 8-week-old female entire Cavalier King Charles spaniel developed acute respiratory distress immediately after a 2-hour flight. Thoracic radiographs and full body computerised tomography revealed a right-sided pleuroperitoneal hernia with most of the alimentary tract, the right caudate liver lobe, and the right kidney located within the right pleural cavity. Most of the small intestine was markedly dilated with gas, causing marked diffuse atelectasis. The hernia was treated successfully surgically, and post-operative recovery was uncomplicated. The puppy was discharged approximately 24 hours after surgery.

KEYWORDS Cavalier King Charles spaniel, congenital, dog, high-altitude flight, pleuroperitoneal hernia

ABBREVIATIONS CT, computerised tomography; IV, intravenous(ly); RI, reference interval

Aust Vet Pract 51 (1): 2-10, 2021

Congenital pleuroperitoneal hernia is rare in dogs, cats, and humans. It is characterised by direct communication between the abdomen and the thoracic cavity via a defect in the diaphragm which allows herniation of abdominal organs into the thoracic cavity. In humans, it occurs in approximately 1 in 3000 live births, and results in high neonatal morbidity and mortality.[1] The most common site of pleuroperitoneal hernia in humans is the left side (75-90% of cases) but the defect has been reported on the right side (10-15% of case) or bilaterally (1-2%).[1] There are limited case reports in dogs, with all being left-sided pleuroperitoneal hernias.[2,3]

The present case discusses an acute presentation of right dorsolateral congenital pleuroperitoneal hernia in an 8-week-old puppy that manifest immediately after flying to its new owner.

CASE REPORT

An 8-week-old female entire Cavalier King Charles spaniel was referred to our hospital after the primary veterinarian made a presumptive diagnosis of a congenital pleuroperitoneal hernia. The puppy was first in the owner's possession about 24 hours prior to her presentation at our hospital. Before her 2-hour flight from the breeder to the owner, she received a standard veterinary check at the airport and was deemed healthy to fly. Upon arrival, the owners noticed her laboured breathing which continued overnight. The next morning, the puppy's respiratory distress worsened. She developed intermittent gagging when eating and became more lethargic. The breeder, staff at the airport and the new owners reported no history or evidence of

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trauma in the last 24 hours. The puppy was deemed healthy at her 6-week-old vaccination examination. Her worming and ectoparasitic history were unknown.

On presentation to her primary veterinarian, she was found to be bright, alert and responsive. Her resting respiratory rate was 60/min, with marked inspiratory effort and paradoxical breathing. One lateral abdominal radiograph was performed (Figure 1). Together with the history and physical examination, a diagnosis of congenital pleuroperitoneal hernia was suspected.

On presentation at the emergency department at the authors' institution, approximately

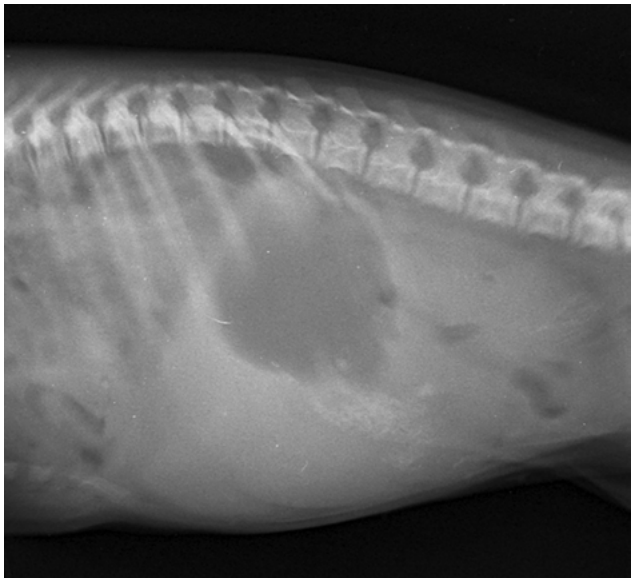
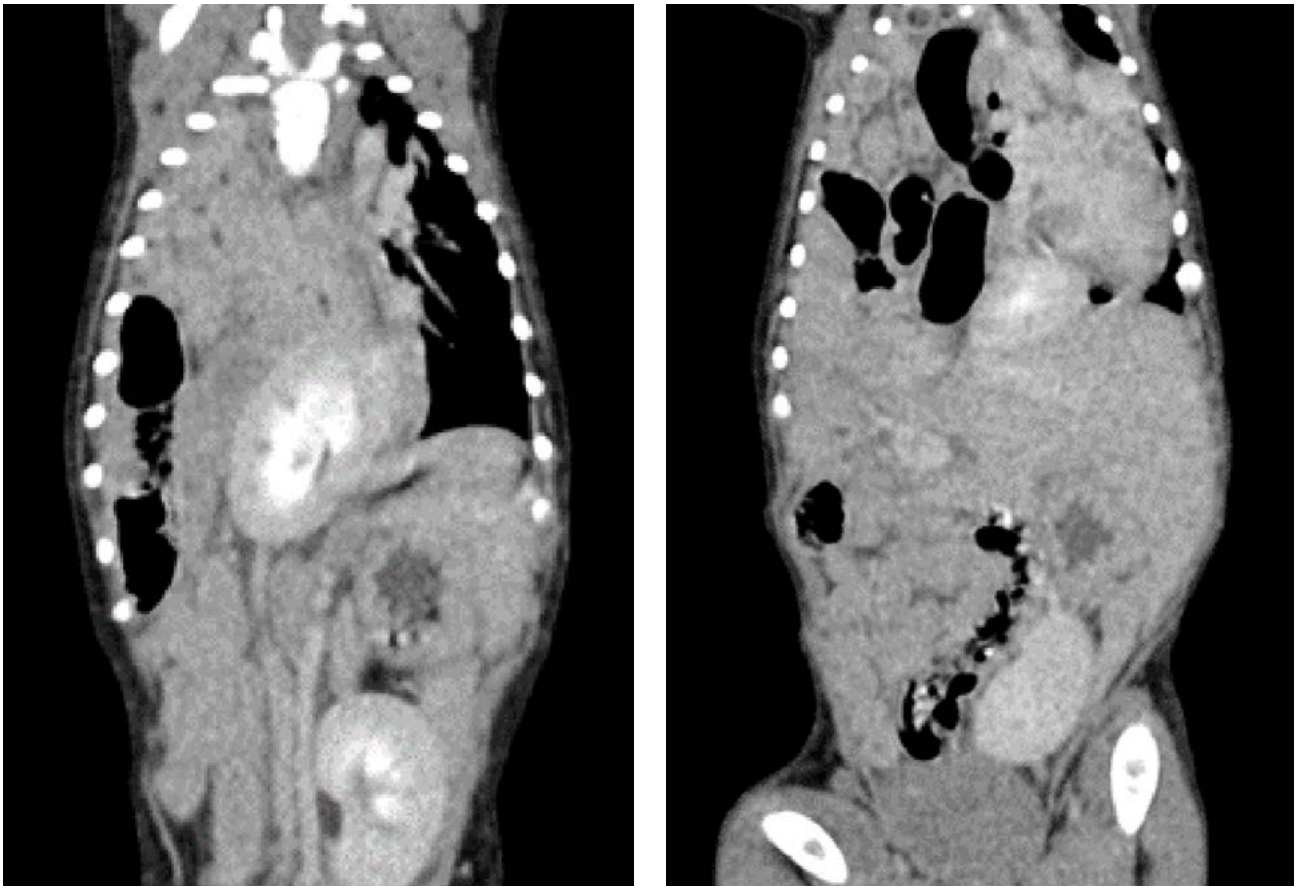


Figure 1. Radiograph showing loops of intestines within the thoracic cavity.

24 hours after the flight, the puppy had a heart rate of 200 beats/min with strong and synchronous femoral pulses, normal heart sound and rhythm, pink and moist mucous membranes with a capillary refill time of <2 sec, temperature 37.5°C, respiratory rate of 80 breaths/min, and a body condition score of 4/9. On thoracic auscultation, there were markedly reduced bronchovesicular lung sounds on both sides of the thorax. On abdominal palpation the abdomen palpated 'empty'.

Computerised tomography (CT) was performed of the thorax and abdomen with the patient in sternal recumbency. A dose of 5 µg/kg fentanyl (Astrazeneca Pty Ltd, North Ryde, NSW) and 0.2 mg/kg midazolam (Apotex, Macquarie Park, NSW) was given intravenously (IV). Three incremental doses of 1 mg/kg of alfaxalone (Jurox Animal Health, Rutherford, NSW) were administered IV to maintain a light plane of sedation. 100% oxygen was administered via a tightly fitted mask during the scan. The study was performed using a 16 slice CT (GE Optima CT520 GE, GE Healthcare Limited, Parramatta, NSW); 100 kVp, modulated mA and a slice thickness of 1.25 mm. The helical acquisition data was reconstructed using both edge-enhancing and smoothing algorithms, and viewed in soft tissue, bone and lung windows. The acquisition was repeated after the administration of iohexol (2 mg/kg IV; GE Healthcare Limited) to acquire a delayed venous phase study. Resultant CT images are shown in Figures 2-5 (overleaf).

The CT revealed a discontinuity of the dorsal aspect of the right hemidiaphragm. The ventral aspect of the right hemidiaphragm was present but caudally displaced. The right kidney, the caudate process of the caudate liver lobe, the stomach, a large portion of small intestine, caecum, transverse colon and associated mesentery were located within the right hemithorax. The herniated viscera and associated vessels extended dorsally over the right liver, giving an hour-glass appearance or 'collar sign'.[4] The small intestines masked the left hemithorax and extended across the midline cranial to the heart. The herniated portions of small intestines were moderately distended by gas with segments containing small granular mineral foci. The abdominally located small intestine was empty. The herniated caecum and transverse colon were moderately to markedly distended with gas. The right kidney was situated at the normal location of the accessory lung lobe.



Figures 2A and B. Dorsal plane of CT images showing the herniated viscera within the pleural cavity.

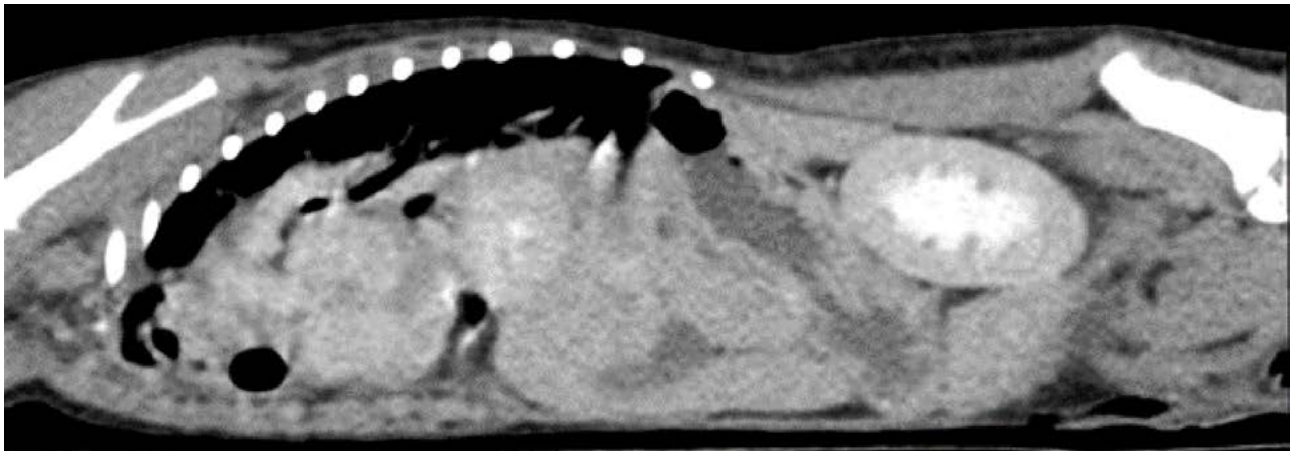


Figure 3. Sagittal CT image: the ventral aspect of the right hemidiaphragm was present but caudally displaced.

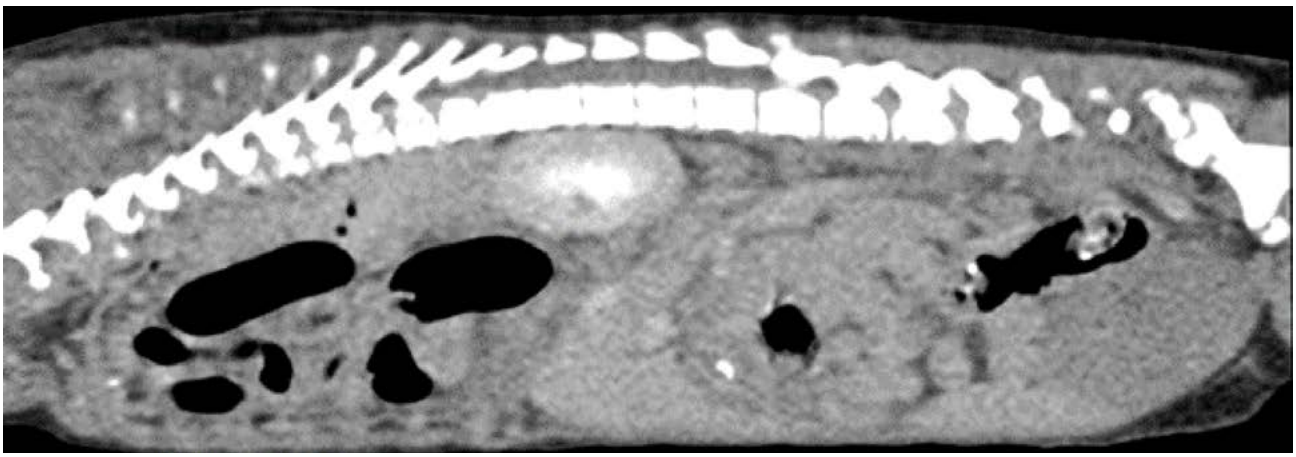


Figure 4. Sagittal CT image showing the herniated caecum and transverse colon were moderately to markedly distended with gas. The right kidney was located within the region of the accessory lung lobe.

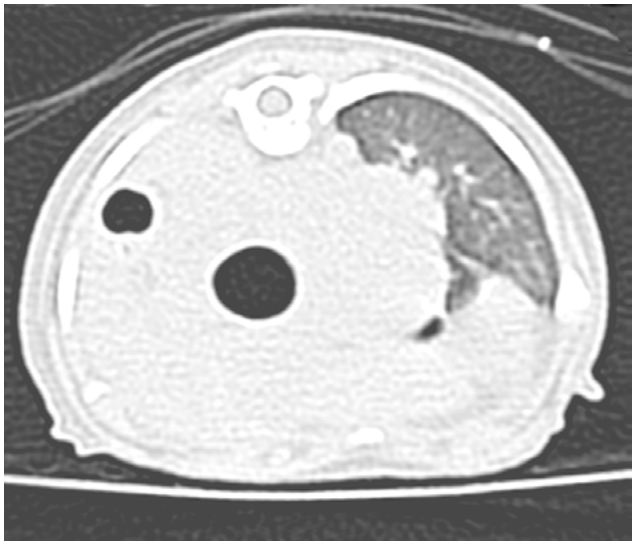


Figure 5. Transverse CT image showing the left lung lobes were aerated but reduced in volume with a diffuse ground glass opacity and focal regions of alveolar pulmonary pattern.

There was reduced serosal detail, but within normal limits for the age of the dog. There was a marked left mediastinal shift. The right lung lobes were completely atelectatic. The left lung lobes were aerated but reduced in volume with a diffuse ground glass opacity and focal regions of alveolar pulmonary pattern.

Based on the history and physical examination findings, an acute manifestation of pleuroperitoneal hernia was suspected. Marked dilation of intestines in the thoracic cavity and evidence of marked atelectasis of all but the left caudal and part of the left cranial lung lobe was evident on CT scan. An immediate surgery was recommended due to the concern of altered intestinal motility and dilation leading to abnormal angles and subsequent obstruction, incarceration and strangulation of the displaced intestine, compressing the lungs and displacing the mediastinum further, in the manner resembling a tension pneumothorax.[5]

Due to delay in getting owner's consent, the puppy was prepared for surgery an hour after completion of imaging. A multimodal anaesthesia was performed with 10 µg/kg fentanyl (Astrazeneca Pty Ltd) IV and 0.2 mg/kg midazolam (Apotex) as premedication. The puppy was positioned with front legs elevated, while the abdomen was surgically clipped and prepared to minimise anaesthetic time. The puppy was pre-oxygenated with flow-by oxygen for 5 minutes prior to induction. A total dose of 3 mg alfaxalone (Jurox Animal Health) IV titrated was given for induction. A size 3.0 endotracheal tube was passed and cuffed. A 30 mg/kg dose of cephazolin (AFT Pharmaceuticals Pty Ltd, North Ryde, NSW) was administered slowly IV at induction. The puppy was maintained under general anaesthesia with isoflurane 1-2% with 100% oxygen. A continuous rate infusion of ketamine (0.02 mg/kg/min; Troy Laboratories Australia Pty Ltd, Glendenning, NSW), fentanyl (0.08-0.2 µg/kg/min; DBL Fentanyl injection, Hospira Australia Pty Ltd, Melbourne, VIC) and midazolam (0.006 mg/kg/min; Apotex) was given. Manual ventilation was performed throughout the anaesthesia, with a maximum inspiratory pressure of 15 mmHg. The puppy was maintained on 5 ml/kg/hr of lactated Ringer's solution (Baxter Laboratories, Toongabbie, NSW) throughout the anaesthesia.

The puppy was placed in oblique dorso-left lateral recumbency and a routine ventral midline coeliotomy was performed. In surgery, a right dorsolateral oval-shaped crural defect (5.5 cm to 3.3 cm) was evident on inspection of the diaphragm. No laceration or haemorrhage could be seen, and the defect margins appeared to be smooth and blade-like. The stomach, proximal duodenum including the right limb of the pancreas were incarcerated within the thoracic cavity. The right limb of the pancreas appeared hyperaemic after being reduced. The distal jejunum, ileum, caecum, ascending and transverse colon were also incarcerated within the right pleural cavity. All the alimentary tract appeared to be viable and only minimally congested. Viscera were gently replaced into the peritoneal cavity. The left limb of

the pancreas remained intact and was at its normal location. The hepatorenal ligament was found to be intact but the right kidney was cranially flexed, torsed and herniated through the diaphragm. The right kidney was only mildly congested on reduction and subsequently improved with time. There was no obvious haemorrhage associated with the capsular vessels. The ureter as well as the renal artery and vein remained intact.

A right nephropexy was performed between the hepatorenal ligament and the transversus abdominis muscle. The caudate process of the caudate lobe of the liver was herniated with the right kidney. A small renal impression was noted on the lobe. The lobe was initially congested on reduction but improved by the time of closure. The lobe was assessed to be viable. The remainder of the liver was normal. The gall bladder, spleen, left kidney and left adrenal gland, both ovaries and uterus were all found to be normal. A scant amount of serosanguineous free fluid was suctioned from within the right pleural cavity. The diaphragmatic defect was closed with two perpendicular simple continuous sutures of 3-0 PDS, in a T-shaped pattern. No tension was noted through the diaphragm after closure. A 14 G chest tube was then placed in the pleural cavity percutaneously using the Seldinger technique. A small amount of air (10 mL) was suctioned via the chest tube immediately after placement. The celiotomy was closed routinely.

General anaesthesia was mostly unremarkable except for a short period (<5 min) of marked hypotension (mean arterial pressure 40 mmHg) immediately after the reduction of the abdominal organs. The blood pressure increased to normal in response to one 10 ml/kg bolus of lactated Ringer's solution.

After surgery, a venous blood sample was taken for routine haematology, biochemistry and blood gas analysis and the puppy was placed in a 50% oxygen chamber to recover. Haematology and biochemistry were unremarkable for a young puppy, with the exception of creatine kinase (984 U/L, reference interval (RI) 73-510), which was likely the result of surgery. A mild respiratory acidosis was noted (pH 7.283, RI 7.320-7.430; pCO_2 53.7 mmHg, RI 37.0-45.0); bicarbonate 21.9 mmol/L, RI 18.0-26.0 mmol/L), which did not warrant further treatment.

Due to a concern for reinflation pulmonary oedema, 20 mL of air was suctioned from the chest drain every 4 hours over 12 hours. An hour after surgery, the puppy was bright, alert and responsive, with respiratory rate 36 breaths/min and SpO_2 98% (in 50% oxygen). Oxygen delivery was tapered to 30%. The chest drain became non-productive of air and fluid 12 hours after surgery. The puppy was fed puppy food once she fully recovered from general anaesthesia. She ate very well overnight without any persistent gagging noted.

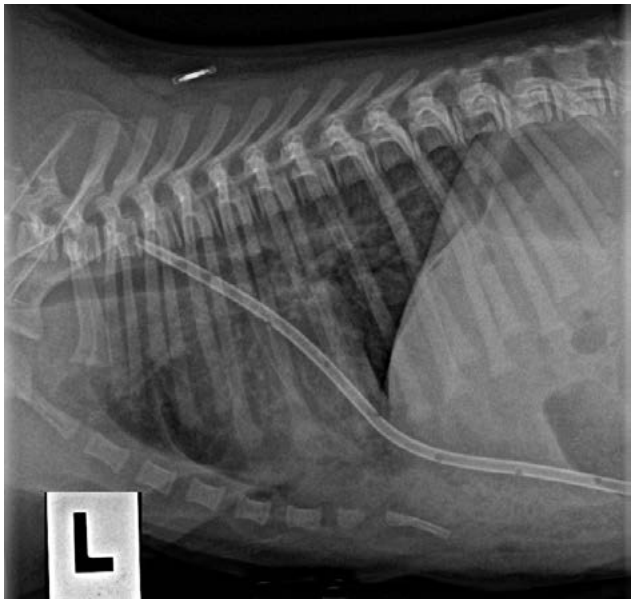


Figure 6. Left lateral thoracic radiograph after surgery; thoracic drain in place.

Fifteen hours after surgery, she remained bright, alert and responsive with mildly increased bronchovesicular lung sounds on thoracic auscultation. Oxygen therapy was

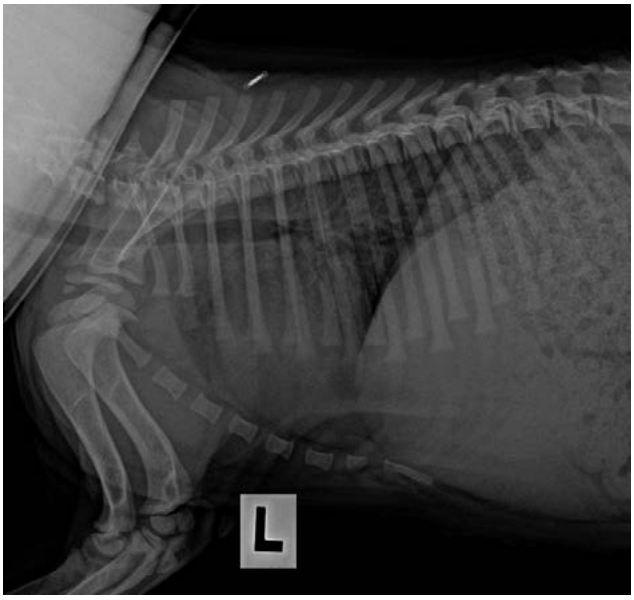


Figure 7. Left lateral thoracic radiograph 10 days after surgery.

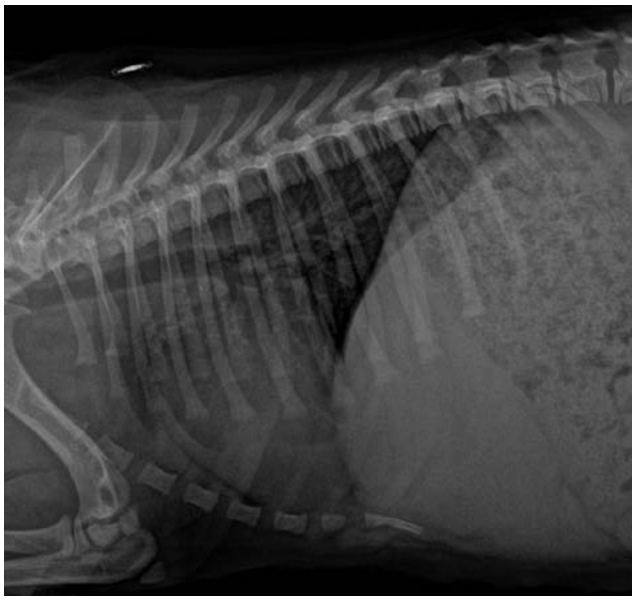


Figure 8. Right lateral thoracic radiograph 10 days after surgery.

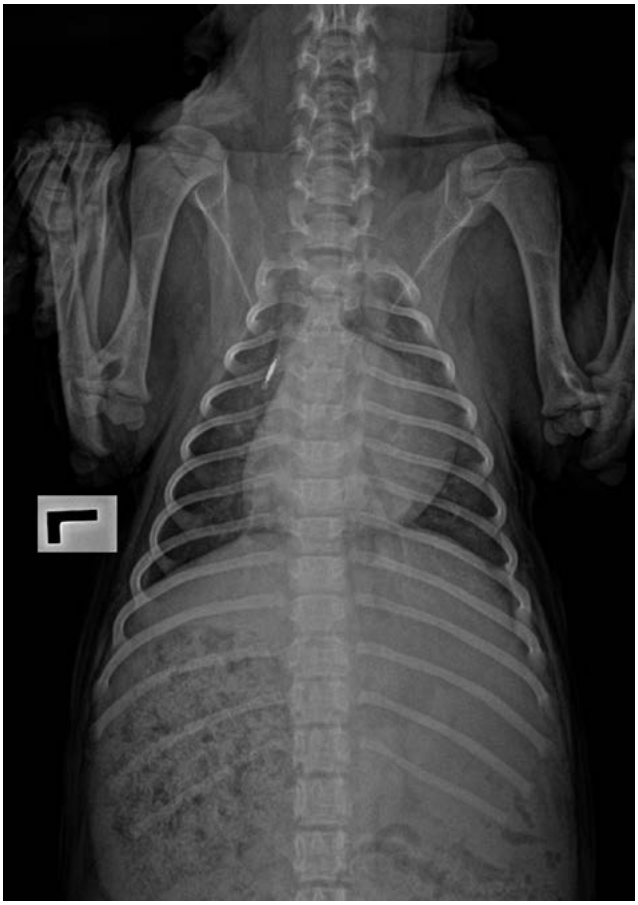


Figure 9. Ventral-dorsal thoracic radiograph 10 days after surgery.

tapered off and analgesia was transitioned to a transdermal fentanyl patch (12 ug/hour; Janssen-Cilag Pty Ltd, Macquarie Park, NSW). Repeated thoracic radiographs confirmed the diaphragm's integrity, and no evidence of pneumothorax or pleural effusion was noted (Figure 6). Puppy was discharged home 7 hours after removal of chest drain.

Ten days after surgery, the puppy re-presented for suture removal. The owners reported no abnormal respiratory signs at home since discharge. The physical examination was unremarkable, with a respiratory rate of 40/min and normal respiratory effort. Thoracic radiographs (Figures 7-9) were unremarkable.

DISCUSSION

There are few case reports of congenital pleuroperitoneal hernia in veterinary science,[6-10] but the embyropathology has been well described.[9] The largest case series was 27 pure-bred golden retrievers from one common sire.[2] The defect persistently involved the left dorsolateral and central portion of the diaphragm. More recently,[5] Cavalier King Charles spaniels with congenital pleuroperitoneal hernia were reported with acute onset of respiratory distress due to

gastrothorax.[3] All 5 were male and all had a left-sided defect. The present case, to the authors' knowledge, is the first report of a right-sided congenital pleuroperitoneal hernia in dog. There has been one case report of a right sided pleuroperitoneal hernia in an asymptomatic cat.[6]

In human medicine, two main types of congenital pleuroperitoneal hernia occur: Morgagni (anteromedial) and Bochdalek (posterolateral), with the latter accounting for about 95% of cases. Furthermore, of the Bochdalek hernias, right-sided hernias represent 13% of cases while left-sided hernias represent 85% of cases.[1,11] People with right-sided hernias have a lower survival rate (50%) than those with left-sided lesions (75%). Bilateral involvement is the most severe and rare form of the disorder (<2% of cases).

With congenital diaphragmatic hernia, it is generally considered that the negative pressure gradient naturally created during inspiration, draws abdominal viscera into the thoracic cavity. Depending on the age and severity of herniation, affected individuals may present very young, later in life after an event disrupts intestinal motility, or herniation may be well tolerated and identified incidentally.[3,9,12] In the present case, it is highly suspected that the intestinal dilation and dysmotility, and possibly the displacement of organs, was related to the flight. Cabin pressure is maintained no lower than 565 mmHg, which is equivalent to an altitude of 8000 feet (2438 m) when the aircraft is at maximum operating altitude.[13] Following Boyle's law, air in the gastrointestinal tract expands by approximately 30% as the atmospheric pressure is reduced during the flight, leading to increased intraabdominal pressure.[5,14] The increased intraabdominal pressure likely forced the abdominal viscera to move cranially through the defect in the diaphragm. The intestinal distension in the enclosed thoracic space, likely lead to further incarceration and obstruction of the small intestines, resulting in significant compression of the already compromised lungs.[5] The ileocolic valve was displaced as the proximal duodenum was dilated and displaced cranially due to the presence of the duodenocolic ligament at the caudal duodenal flexure. It is surmised that the displaced ileocolic valve may have caused a distal jejunal physical obstruction, preventing the release of air via flatulence. As mineral foci are a feature of chronic small intestinal obstruction, the presence of small granular mineral foci within the herniated portions of small intestines further supports the chronicity of the obstruction. Additionally, the severity of atelectasis, supports the suspicion that the puppy had tolerated prolonged reduced thoracic volume prior to this event.

Similar cases have been reported in the human literature. One was a case of acute manifestation of respiratory distress one day after a flight in an 18-year-old female with a congenital pleuroperitoneal hernia.[13] Another was a case of acute respiratory distress in a 6-week-old infant who swallowed a large amount of air, causing a significant increase in intraabdominal pressure, which lead to movement of abdominal visceral into the thorax via a previously asymptomatic congenital pleuroperitoneal hernia.[5]

In humans, if herniation of abdominal contents occurs during foetal development, a common side effect of congenital pleuroperitoneal hernia is pulmonary hypoplasia, resulting from a 'mass effect' of the herniated abdominal contents.[11,15] Not only is the lung volume small, but the lungs also lack a normal bronchial and vascular branching pattern.[15] This leads to increased pulmonary vascular resistance and significant pulmonary hypertension, which is a major contributor to morbidity and mortality in infants with congenital pleuroperitoneal hernia.[15] The prognosis is worse when the

liver is involved.[15] In the present case, no radiographic signs of pulmonary hypoplasia or pulmonary hypertension were noted on thoracic radiographs 2 weeks post-operatively. However, an echocardiogram was not performed at any time, so it is possible that subtle changes were not detected radiographically.

The current recommendation in paediatric medicine for initial management of congenital pleuroperitoneal hernia is to control pulmonary hypertension and to provide oxygen and careful mechanical ventilation without triggering a pulmonary vasospasm crisis or causing further damage to small and underdeveloped lungs.[11,15] Treatment of right-sided heart failure, secondary to severe pulmonary hypertension, is indicated if this has occurred.[11] Extracorporeal life support is a mainstay of rescue support for congenital pleuroperitoneal hernia patients with severe pulmonary hypertension.[1,11,15,16] Surgical repair remains the treatment of choice, however, there is no consensus on the ideal timing for surgery in human medicine.[17] It is often delayed up to a week unless there are signs of bowel ischaemia caused by the hernia.[11,18] In the present case, mechanical ventilation was not attempted prior to surgery, due to the concern of markedly reduced lung volume, since the herniated abdominal visceral occupied three-quarters of the thoracic cavity. It was deemed that the dog required emergency surgery because of the marked atelectasis of most lung lobes and concern of development of secondary bowel ischemia.

Reinflation pulmonary oedema is a well reported complication of treatment for pneumothorax and pleural effusion. The current understanding of its pathophysiology is that there is increased capillary permeability secondary to hypoxic and mechanical damage to the alveolar capillary membrane during atelectasis. Typically this occurs after the lungs have been compressed for more than 3 days.[19,20] The increase in blood supply after re-expansion and relative negative perivascular pressure further exacerbates the oedema.[19] Reinflation pulmonary oedema has been reported in the human literature after surgical repair of a traumatic diaphragmatic hernia.[19] In an attempt to prevent reinflation pulmonary oedema in the present case, we reduced the rate of expansion of the lungs by limiting drainage from the chest tube post-operatively, as suggested in another report.[20]

In a recent study, 5 cases of congenital pleuroperitoneal hernia and gastrothorax were described in male Cavalier King Charles spaniels.[3] A possible breed and sex genetic predisposition was mooted. No familial history of this condition was mentioned by the breeder in the present case.[3]

The outcome in cases of congenital pleuroperitoneal hernia in people is variable, with reported survival rates from 70-90%[1,11] to 32-58% (despite aggressive intervention).[16] Many people with congenital pleuroperitoneal hernia have pathologic gastroesophageal reflux, caused by abnormal positioning of the gastroesophageal junction and requiring long-term acid-suppressive medications. Other reported complications include abdominal compartment syndrome (due to increased intraabdominal pressure), recurrent hernia, increased risk of chronic lung disease and small bowel obstruction and skeletal deformity.[11,21] To the authors' knowledge, there has been no study on prognostic and surgical factors in pleuroperitoneal hernia in veterinary medicine, presumably due to the low number of reported cases. However, most reported cases in which surgery was undertaken had a good outcome in general.[2,3,6-9] Similarly, in the present case, the owners reported no long-term complications at the 2-week post-operative visit or at the 4-week and 6-month post-operative telephone updates.

CONCLUSION

An acute presentation of respiratory distress after a high-altitude flight should alert clinicians to the possibility of sudden manifestation of a pleuroperitoneal hernia. Radiography and CT are useful tools to assist in the diagnosis. Emergency surgery is likely necessary to correct the disorder. With appropriate treatment, longer term prognosis is likely good. As the condition of pleuroperitoneal hernia may have a genetic basis, counselling of owners and breeders in this regard is warranted.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest in this case report.

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An auto-penetrating wound resulting in delayed bilothorax

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ABSTRACT A 13-year-old male neutered Staffordshire bull terrier was presented following blunt trauma from a kick, which resulted in rib fractures, mild pneumothorax, pulmonary contusions and acute hepatic haemorrhage. Exploratory coeliotomy was performed and the hepatic haemorrhage was controlled with pressure and the application of a haemostatic agent. The patient was discharged following a smooth recovery in hospital. The dog presented to the hospital again three days later with signs of lethargy and a mild increase in respiratory effort, following a second event of trauma. A bilious pleural effusion was identified secondary to an auto-penetrating diaphragmatic wound associated with a fractured rib. This also resulted in gall bladder perforation. The dog underwent a diaphragmatic repair and cholecystectomy and recovered uneventfully.

KEYWORDS auto-penetrating wound, bilothorax, biliary effusion, dog, trauma

ABBREVIATIONS AFAST, abdominal focused assessment with sonography for trauma; ALKP, alkaline phosphatase; ALT, alanine aminotransferase: CT, computed tomography; PCV, packed cell volume; TFAST, thoracic focused assessment with sonography for trauma; IV, intravenous(ly); TP, total protein

Aust Vet Pract 51 (1): 11-18, 2021

Bilothorax refers to the accumulation of bile in the pleural space and is a rare differential diagnosis for pleural effusion in animals and humans. There are six canine[1-6] and four feline[7-10] cases that have previously been reported in the veterinary literature. The cause of bilothorax in the majority of cases is due to trauma causing leakage of bile into the thoracic cavity via a diaphragmatic perforation, however, cases without diaphragmatic injury have also been reported. A feline case describing bilothorax secondary to a spontaneous cholecystopleural fistula has also been reported.[10] Similarly, bilothorax is an uncommon cause of pleural effusion in humans. Reported causes in humans include hepatic infection, hepatic trauma, biliary obstruction, and secondary to percutaneous or open biliary surgery.[11,12] The present case is the first report of bilothorax in a dog secondary to an auto-penetrating wound.

CASE HISTORY

A 13-year-old male neutered Staffordshire bull terrier presented for acute abdominal trauma following a reported episode of abuse. Abnormalities found on physical examination included pale mucous membranes, tachycardia (192 beats/min), tachypnoea (56 breaths/min) and hypothermia (rectal temperature of 36.9°C). Thoracic auscultation identified reduced lung sounds on the right side and abdominal palpation identified generalised discomfort. An abdominal focused assessment with sonography for trauma (AFAST) scan identified a small

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amount of free fluid in the cranial abdomen and a thoracic focused assessment with sonography for trauma (TFAST) scan identified a small volume bilateral pleural effusion. All ultrasound scans were performed using a Philips diagnostic ultrasound system (Philips Ultrasound Inc., Bothell, WA, USA). Thoracic radiography was performed, identifying fractures of the right seventh, eighth and thirteenth ribs, mild pulmonary contusions, mild bilateral pleural effusion and mild right-sided pneumothorax. The packed cell volume (PCV) was 0.30 L/L (reference range 0.37-0.55 L/L) and refractometer total protein (TP) was 40 g/L (reference range 50-80 g/L). Serum biochemistry identified increased alanine aminotransferase (ALT), total bilirubin, amylase and lipase (Table 1). The following treatments were commenced: Hartmann's intravenous fluids (10 mL/kg bolus, followed by 5 mL/kg infusion), intranasal oxygen (2L/min), methadone (0.2 mg/kg intravenously (IV) every 4 hours; Methodyne; Jurox Animal Health, Rutherford, NSW, Australia) and tranexamic acid (15mg/kg IV every 8 hours; Ilium Vasolamin S 100; Troy Animal Healthcare, Glendenning, NSW, Australia).

After 8 hours, there was reduction of PCV to 0.27 L/L and TP to 31 g/L and the volume of free abdominal fluid had markedly increased. Abdominocentesis yielded red abdominal fluid consistent with haemoabdomen (abdominal fluid PCV 0.33 L/L and TP 38 g/L, compared to peripheral blood PCV 0.25 L/L and TP 25 g/L). A 350 mL packed red blood cell transfusion was administered, commencing at 1mL/kg/h for the first hour and the remainder of the bag given over 3 hours. After the transfusion, an exploratory coeliotomy was performed. A large, solidified haematoma was removed from the caudal aspect of the right-sided liver lobes and 1.5 L of bloody fluid was drained by suction. There was mild bruising of the bladder and capsular tears in the right medial and lateral liver lobes, with evidence of active haemorrhage. Haemorrhage was controlled with manual pressure and absorbable gelatin foam (Gelfoam; Pharmacia & Upjohn, Kalamazoo, MI, USA). The remainder of the abdomen was normal on gross examination. Abdominal closure was performed with a three-layer closure. The pleural effusion and pneumothorax were monitored and thoracocentesis was not performed.

Forty-eight hours after surgery, the dog's lung sounds were reduced, with recurrence of mild tachypnoea (44 breaths/min). Thoracic radiographs were repeated, identifying a worsening bilateral pleural effusion. Ultrasound-guided right-sided thoracocentesis yielded 350 mL of serosanguineous fluid (PCV 0.05 L/L and TP 40 g/L) and clinical improvement was immediately noted. The dog was monitored for an additional 72 hours in hospital and made an uneventful recovery, with no further development of respiratory abnormalities. Serum biochemistry just before discharge identified normal ALT, and moderately increased alkaline phosphatase (ALKP) and total bilirubin (Table 1).

Three days after discharge the dog was presented for vomiting, panting and lethargy after reportedly falling down a flight of stairs. On examination, he was tachycardic (168 beats/min) with normal lung sounds and increased respiratory effort. PCV was 0.36 L/L and TP was 65 g/L. Serum biochemistry showed a markedly increased ALT, ALKP and total bilirubin compared to the initial presentation (Table 1). Twelve hours after re-admission the dog was hypoxaemic on room air (S_PO_2 = 90%) and required oxygen supplementation. A TFAST was performed and moderate bilateral pleural effusion was identified. An abdominal ultrasound identified mild peritoneal effusion, acute pancreatitis, diffuse hepatopathy, an apparently intact diaphragm, and thickening of the gall bladder wall with a sonographically normal common bile duct. Twenty-four hours after re-admission, the dog's respiratory effort

Table 1: Biochemical findings at the initial admission, prior to discharge and at the second admission

	Units	Reference range	Initial admission	Prior to discharge	Second admission
Glucose	mmol/L	3.89-7.95	5.83		
Creatinine	µmol/L	44-159	39		
Urea	mmol/L	2.5-9.6	5.3		
Phosphate	mmol/L	0.81-2.20	1.14		
Calcium	mmol/L	1.98-3.00	1.99		
Total protein	g/L	52-82	53		
Albumin	g/L	22-39	21		
Globulins	g/L	25-45	32		
ALT	U/L	10-125	1791	93	480
ALKP	U/L	23-212	<10	958	2747
Total bilirubin	umol/L	0-15	32	46	128
GGT	U/L	0-11	7		
Cholesterol	mmol/L	2.84-8.26	2.12		
Amylase	U/L	500-1500	>2500		
Lipase	U/L	200-1800	>6000		

increased and right-sided thoracocentesis was performed at the 8th intercostal space, yielding 400 mL of serosanguineous and brown fluid. Fluid analysis was not performed. A further 2.5 L of fluid was drained over the following 24 hours. Thoracocentesis was performed twice on the left and right sides during that period. The fluid was grossly yellow tinged and total bilirubin of the fluid was 377 mmol/L. The pleural fluid to peripheral blood bilirubin ratio was 9.45, consistent with a bilothorax. By definition, a ratio of greater than one is consistent with a bilious pleural effusion.[4,9]

Computed tomography (CT) of the thorax and abdomen was performed using a 16-slice CT scanner (GE HP60 LightSpeed, GE Healthcare, Waukesha, WI, USA), confirming severe pleural effusion. A small perforation in the right lateral diaphragm was noted in close proximity to the fractured 8th rib. The gall bladder was adjacent to the perforation and appeared flaccid. The gall bladder wall was thickened however no defect was visible (Figure 1). Exploratory coeliotomy was recommended.

At coeliotomy, the surfaces of the intra-abdominal organs and omentum had a yellow discolouration and a moderate amount of green-coloured abdominal fluid was suctioned (a 2-mL aliquot was submitted for analysis and culture). A 15-mm right-sided diaphragmatic perforation and 5-mm perforation on the ventral surface of the gall bladder were identified (Figure 2). The fractured 8th rib was located in close proximity to the perforations. With inspiration and expiration, bile was visualised to flow between the thoracic and abdominal cavities.

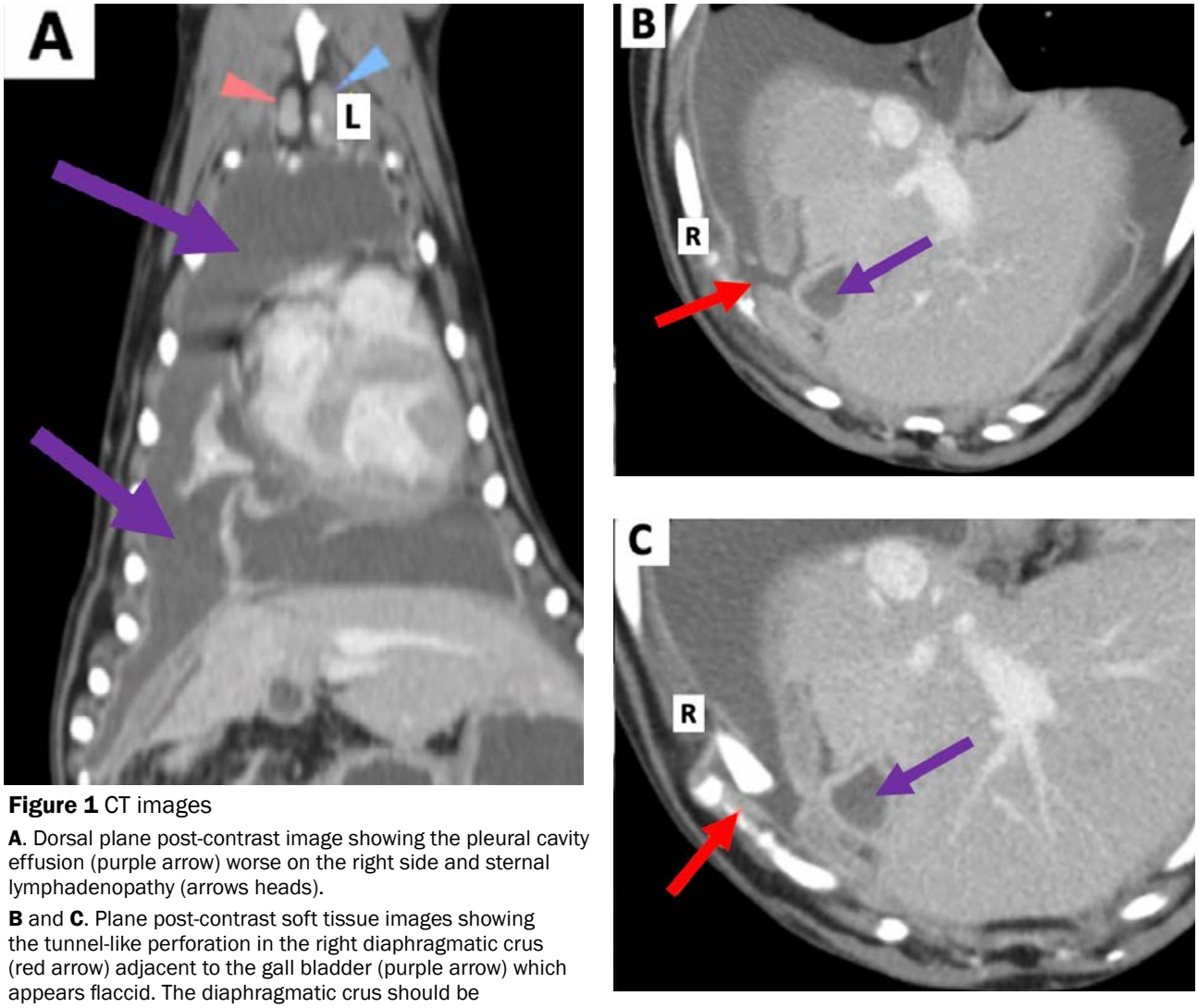

Figure 1 CT images

A. Dorsal plane post-contrast image showing the pleural cavity effusion (purple arrow) worse on the right side and sternal lymphadenopathy (arrows heads).

B and **C**. Plane post-contrast soft tissue images showing the tunnel-like perforation in the right diaphragmatic crus (red arrow) adjacent to the gall bladder (purple arrow) which appears flaccid. The diaphragmatic crus should be one continuous opacity.

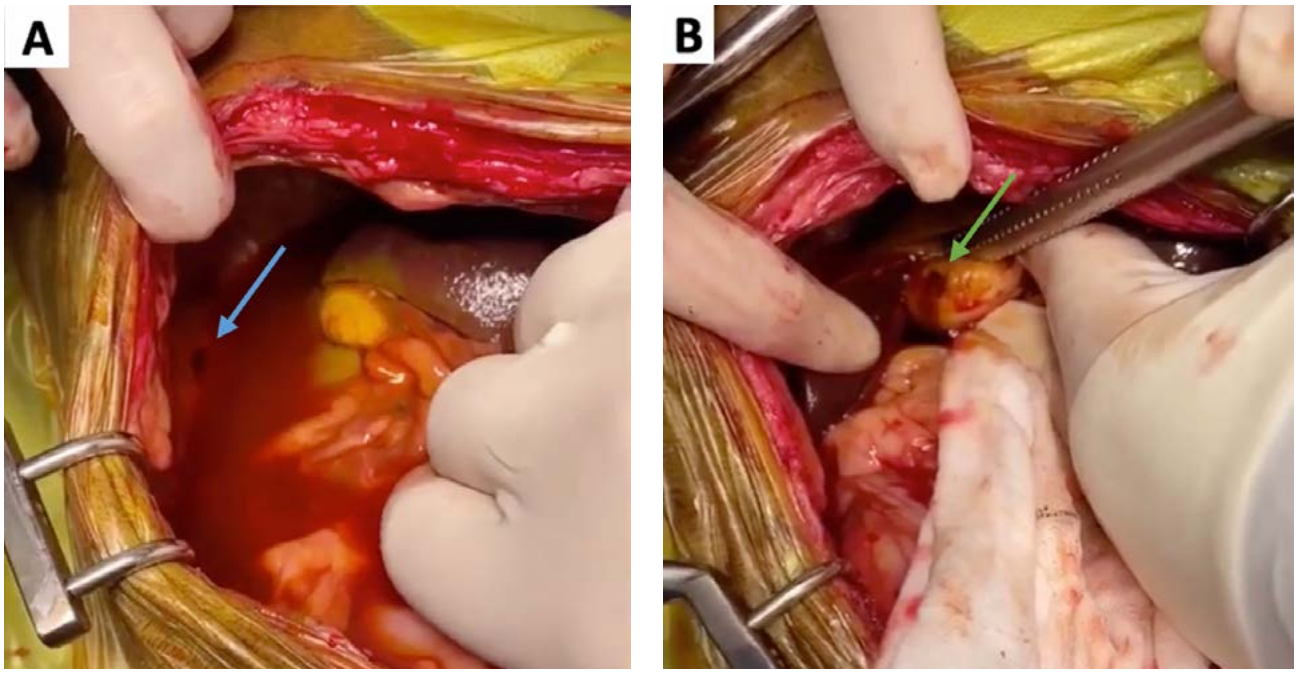

Figure 2 Exploratory celiotomy

A. Free abdominal fluid in the abdomen and a diaphragmatic perforation on the right side (blue arrow).

B. Perforation of the gall bladder on the ventral surface (green arrow).

A cholecystectomy was performed, followed by thoracic and abdominal lavage. A total of 300 mL/kg of warm saline was used. The thoracic cavity was lavaged through the diaphragmatic perforation. Suction was applied to drain the fluid. A 12 Fr, 30 cm MILA guidewire chest drain (MILA International Inc., Florence, KY, USA) and a 15 Fr, 20 cm Jackson-Pratt abdominal drain (MILA International Inc.) were placed. The chest drain was placed on the right side, tunnelling the skin at the 10th intercostal space and entering the thoracic cavity at the 9th intercostal space. The Jackson-Pratt drain exited cranially, to the right of the abdominal incision. The diaphragmatic perforation was repaired via primary closure and the abdomen closed in a three-layer pattern.

Supportive care in the post-operative phase included fluid therapy, fentanyl constant rate infusion (2-5 mg/kg/hr; DBL Fentanyl, Hospira, Lake Forest, IL, USA), cephazolin (30 mg/kg IV every 8 hours; Cefazolin; AFT pharmaceuticals, North Ryde, NSW, Australia) and enrofloxacin (10 mg/kg IV every 24 hours; Ilium Enrotril, Troy Animal Healthcare). Culture results were negative and antibiotic therapy was transitioned to cephalexin (22 mg/kg orally every 12 hours; Cephaforte; Jurox Animal Health). The dog made an uneventful recovery after 5 days of hospitalisation and the drains were removed on day 4 when drain production was less than 2mL/kg/day. The dog was bright and active at the two-week post-operative appointment. A follow-up call was made 6 months post-operatively and the dog was reported to be free of clinical signs of disease.

DISCUSSION

Previous reports of bilothorax in the veterinary literature have been a result of trauma. This report presents an alternative cause of traumatic bilothorax that has not previously been reported. The mechanism of injury is consistent with trauma from the presence of rib fractures in close proximity with the gall bladder and diaphragm. This is suspected to have resulted in an auto-penetrating traumatic diaphragmatic tear and gall bladder perforation. Auto-penetrating herniae have been reported due to traumatic injury in dogs and cats, however, no reports are in association with biliary perforation.[13] With respiration, positive intra-abdominal pressure facilitates movement of bile from the abdomen through the diaphragmatic perforation, however, other routes of bile entry are possible.[2] For bilothorax cases without a diaphragmatic defect, it has been suggested that bile can diffuse via the lymphatic system from the abdomen across the diaphragm.[2,6,14] Another hypothesis suggests that bile diffuses through the diaphragm as a result of negative pressure in the thorax associated with normal respiration.[2,15] No reports of auto-penetrating bilothorax have been described in the human literature.

Several challenges were encountered prior to the final diagnosis of traumatic bilothorax being achieved. The dog re-presented to the hospital after the second event of trauma for vomiting and lethargy. Repeat biochemistry identified an increase in liver enzymes and total bilirubin compared to the values at initial discharge. An abdominal ultrasound also confirmed a diffuse hepatopathy and acute pancreatitis. These findings led to the presumption that the increase in liver parameters and jaundice were associated with previous hepatic trauma and extrahepatic bile duct obstruction from acute pancreatitis. In addition, the diaphragm appeared to be intact, the gall bladder was mildly thickened and the common bile duct was normal. An alternative differential diagnosis was reactive pancreatitis secondary to bile peritonitis, however, this was considered less likely based on the imaging findings at the time. Abdominocentesis of the fluid may have provided a more definitive answer.

There is limited published evidence of the sensitivity of ultrasonographic and CT for detection of traumatic perforation of the gall bladder in dogs and cats. Studies have suggested a sensitivity of 56-85% in using ultrasonography to diagnose a gall bladder rupture.[16-19] Inspection of the gall bladder wall with ultrasound can be limited due to pericholecystic fat obscuring the presence of a tear or perforation.[18] Varying results have also been reported in the human literature. In a study of 11 people with a gall bladder perforation, only 3 patients (37%) were diagnosed ultrasonographically prior to surgery.[20] Similarly, in a study of 13 patients diagnosed with perforation of the gall bladder at surgery, ultrasound identified the defect in 5 patients (39%), while CT did so in 9 patients (69%).[21] In another study, high resolution ultrasound detected a gall bladder defect in 70% of cases (16/23) compared to 78% of cases (14/18) with CT.[22] There is limited literature on the use of CT for diagnosis of traumatic gall bladder perforations in dogs, with one case report of two dogs diagnosed with a breach in the gall bladder wall on CT.[23] Thus the human literature suggests CT is superior to ultrasound for diagnosis of gall bladder defects and similar extrapolations can potentially be made for animals.[21,24] However, interpretation of imaging findings is operator and equipment dependent. Importantly, the absence of ultrasound and CT findings does not exclude the possibility of a gall bladder tear or perforation. Additionally, it may delay the decision for exploratory surgery which would ultimately be required to make a definitive diagnosis.

In the present case, bilothorax developed after the initial surgery based on normal visual findings at the initial exploratory celiotomy. Also, a therapeutic thoracocentesis, performed after surgery due respiratory distress, yielded serosanguineous fluid. Although fluid analysis was not performed at the time, the fluid did not grossly resemble bile. The patient was also clinically well for an additional 3 days prior to discharge with improving biochemical values. Therefore, bilothorax was unlikely to have been present at the time of the initial discharge, because an increasing pleural effusion would have been expected.

In the present case, a concurrent diaphragmatic tear was also present, allowing for movement of bile into the thoracic cavity. In traumatic events, diaphragmatic rupture is most commonly reported as a result of motor vehicle accidents. Herniation occurs due to a rapid increase in intraabdominal pressure and expulsion of air from the lungs. This results in an acute loss of the stabilising effect of air-filled lungs and hence, diaphragmatic rupture.[25] In the present case, an auto-penetrating nature is evident and is a rare cause for bilothorax. The use of ultrasound for diagnosis of diaphragmatic rupture is sensitive, with an accuracy of 93%, however, it was not detected in the present case, likely because of the small size of the perforation.[26] In addition, a late diagnosis of diaphragmatic herniation in humans is also not uncommon, with a missed diagnosis rate of 12-33%.[27]

Bile peritonitis develops due to leakage of bile into the peritoneal cavity resulting in an inflammatory response. Rupture of the gall bladder or biliary tract can occur due to biliary obstruction, inflammatory disease, iatrogenic manipulation and trauma.[28] A review of the causes of extrahepatic biliary tract rupture identified 23/33 cases (70%) with ductal rupture and 25/27 cases (93%) with gall bladder rupture were due to trauma.[29] Isolated traumatic gall bladder rupture is an uncommon cause of bile peritonitis. This is similar in humans in which isolated gall bladder rupture is seen in <1% of cases of blunt abdominal trauma. Case reports in humans also report gall

bladder rupture, if present, is more commonly associated with penetrating trauma. Unfortunately, delay to diagnosis of traumatic bile peritonitis in humans is also common due to the vague clinical signs before clinical deterioration.[30]

Although, we cannot be certain of the nature of the second injury (the owner reported that the dog fell down stairs), there was a history of non-accidental injury at the initial presentation. Recent animal forensic research reported that common sources of trauma in non-accidental injury include kicking, punching, being hit by objects or being thrown into objects.[31] Additionally, rib fractures, head trauma and vertebral fractures were more common in non-accidental injury compared to motor vehicle accidents. Also, rib fractures did not follow a discrete pattern in non-accidental injuries (multiple impacts) whereas they occurred in ribs of close proximity with motor vehicle accidents (single impact). Thoracic trauma commonly led to pneumothorax or pulmonary contusions with no reports of bilothorax in the literature.

The pleural effusion identified on TFAST at the second presentation of the present case was initially presumed to be a result of pleural irritation from the fractured ribs or secondary to trauma. In addition, other ultrasound findings such as acute pancreatitis and a hepatopathy were thought to explain the dog's presenting clinical signs. These interpretations contributed to the delay in diagnosis of bilothorax. Earlier thoracocentesis of the pleural effusion would have been therapeutic and analysis of the fluid would have provided an earlier diagnosis. In humans, it is recommended that thoracocentesis be performed in all patients with a pleural effusion larger than 1 cm on ultrasound examination.[32]

CONCLUSION

Although rare, an increasing number of bilothorax cases have been reported in the literature. Bilothorax should be considered as a differential diagnosis for pleural effusion in dogs and cats with or without an apparent diaphragmatic defect. In cases with a diaphragmatic defect, rib fractures should be considered a potential source of perforation, even if imaging findings are not diagnostic. Surgery may be required for a definitive diagnosis.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest regarding the publication of this case report.

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Management of sleep disordered breathing in a dog using continuous positive airway pressure

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ABSTRACT A 4-year-old Cavalier King Charles spaniel presented for evaluation of disruptive apnoeic episodes, experienced every 10-15 minutes during sleep. Surgical intervention included bilateral tonsillectomy, bilateral laryngeal sacculectomy, and unilateral cricoarytenoid lateralisation. Minimal improvement in the frequency and severity of episodes of sleep disordered breathing were noted. The patient was subsequently fitted with a continuous positive airway pressure device at night and responded well to this intervention, allowing the dog to sleep through the night. Four years after the initial presentation, the patient underwent fluoroscopic large airway assessment and was diagnosed with dynamic pharyngeal collapse. To the authors' knowledge, this is the first report of the use of continuous positive airway pressure in the management of sleep disordered breathing in dogs and represents a novel management strategy for dogs suffering from this condition that respond poorly to airway surgery.

KEYWORDS brachycephalic obstructive airway syndrome, Cavalier King Charles spaniel, continuous positive airway pressure ventilation, dog, dynamic pharyngeal collapse, sleep disordered breathing

ABBREVIATIONS BOAS, brachycephalic obstructive airway syndrome; CPAP, continuous positive airway pressure; DPC, dynamic pharyngeal collapse; SDB, sleep disordered breathing

Aust Vet Pract 51 (1): 19-25, 2021

Sleep disordered breathing (SDB) is a condition documented in both humans and dogs that involves breathing difficulties experienced during sleep, secondary to partial or complete upper airway obstruction.[1,2] This condition is poorly understood in dogs, and there is a paucity of reports in the veterinary literature. In humans, obstructive sleep apnoea syndrome has been associated with a combination of pharyngeal anatomic abnormalities and dilator myopathies.[3] SDB has been reported in the Cavalier King Charles spaniel, English bulldog, and other brachycephalic breeds.[1,4,5] Brachycephalic obstructive airway syndrome (BOAS), which comprises stenotic nares, soft palate elongation and laryngeal saccule eversion, involves changes to the structure and function of the pharynx which causes increased respiratory effort.[4,5] It is also speculated to reduce pharyngeal dilator muscle function and predispose affected dogs to the secondary condition of dynamic pharyngeal collapse (DPC).[4,5]

DPC involves partial or complete collapse of the pharynx resulting in displacement or

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deviation of the soft palate and/or dorsal pharyngeal wall and is a contributing factor in obstructive sleep apnoea syndrome, hypertension, and cognitive dysfunction in humans.[6-8] It has also been previously recognised in a variety of animal species including the dog, cat, and horse.[4,9-12] Dogs with DPC experience coughing, stertorous upper airway noise, gagging, and regurgitation.[11] A canine experimental model of obstructive sleep apnoea syndrome also demonstrated that intermittent airway obstruction led to sustained daytime hypertension compared to control measurements in the same dogs exposed to sleep fragmentation.[13] Additionally, in a videofluoroscopic study of dogs with DPC, 27 of 28 dogs had concurrent airway diseases including principal bronchial collapse, tracheal collapse, and BOAS.[11] The incidence of DPC is disproportionately higher in brachycephalic breeds compared to non-brachycephalic breeds undergoing videofluoroscopy.[10] Thus, DPC is increasingly being investigated as a potential cause of SDB in brachycephalic breeds.[10,11]

Currently, there are limited reports on the management of SDB in dogs in the veterinary literature. One report describes the use of magnetic airway implants to maintain airway patency in an experimental canine model of pharyngeal collapse.[14] A clinical report describes management of SDB using corrective airway procedures (laser turbinectomy, folding flap palatoplasty, tonsillectomy, laryngeal sacculectomy, cuneiform process resection).[1] In the latter case series four out of five Cavalier King Charles spaniels with SDB had complete resolution of signs using a variety of the aforementioned corrective airway surgeries.[1] While the fifth patient experienced a temporary complete resolution of signs, they returned three months post-operatively but remained improved compared to pre-operative assessment.[1]

In humans, there are similarly a range of corrective surgeries available to assist in the management of obstructive sleep apnoea syndrome; however, the first line treatment is often continuous positive airway pressure (CPAP) ventilation.[15] A CPAP device works by delivering air via a pump and through a mask to create a mild positive pressure gradient to a patient's airway, thus maintaining patency and reducing the risk of apnoeic episodes in people suffering from obstructive sleep apnoea syndrome. In the veterinary literature, CPAP has been investigated for use in maintaining patent airways in brachycephalic breeds in the peri-anaesthetic period, as well as a report of the use of CPAP in managing an acute episode of laryngeal paralysis in a cat.[16,17] Additionally, there is a research study assessing the use of CPAP in cats restrained in known pathogenic sleep positions associated with obstructive sleep apnoea syndrome in humans.[18] There is further experimental research in animals demonstrating that CPAP improves oxygenation in acute applications.[19-22] However, to the authors' knowledge, there are no reports of the use of CPAP in the management of naturally occurring SDB in animals. The purpose of the present study is to describe the management of SDB with CPAP in a Cavalier King Charles spaniel whose signs did not resolve following corrective airway surgeries.

CASE REPORT

A 4-year-old female spayed Cavalier King Charles spaniel presented to their primary care veterinarian for assessment of BOAS and apparent episodes of SDB. The dog was observed to sleep for only 10- to 15-minute periods before waking in a dyspneic episode, gasping for air. Inspiratory stertor and severe periodontal disease were noted on examination. The dog was previously diagnosed with eosinophilic stomatitis on tissue biopsy. A trial of doxycycline and long-

acting injectable corticosteroid failed to improve the clinical signs, and the patient was subsequently referred to the Brisbane Veterinary Specialist Centre for further evaluation.

On assessment, the dog was in good body condition (5/9) with marked periodontal disease and a grade I/VI left-sided apical systolic heart murmur. A sedated upper airway examination confirmed stage III laryngeal collapse and bilateral laryngeal paralysis, with bilateral tonsillomegaly and laryngeal saccule eversion. The soft palate was of appropriate length. Stomatitis, pharyngitis and small vesicular lesions on the soft palate were also noted, consistent with the previous diagnosis of eosinophilic stomatitis. Surgical intervention was recommended, though declined by the owner in favour of a trial of prednisolone (1 mg/kg/day orally) and a low allergen diet (Hill's Prescription Diet z/d Canine, Hill's Pet Nutrition, Inc.) to determine whether addressing the oropharyngeal inflammation secondary to eosinophilic stomatitis might improve the clinical signs associated with SDB.

A week later, the dog re-presented after failing the medical trial, and the owner consented to laryngeal sacculectomy and tonsillectomy, but deferred unilateral cricoarytenoid lateralisation for treatment of the laryngeal paralysis. The dog was hypertensive pre-operatively, as assessed by non-invasive blood pressure measurement (145/87, mean arterial pressure 106 mmHg). Surgery and recovery were uneventful and histopathology of the tonsils returned as eosinophilic tonsillitis.

The dog re-presented approximately one month later with minimal improvement in the apnoeic episodes and was still waking approximately every 30 minutes in distress. The owner then consented to unilateral cricoarytenoid lateralisation. Following this surgery, the owner reported an improvement in the quality of the dog's breathing pattern while awake, though she continued to experience apnoeic episodes with a similar frequency during sleep. At that time, computed tomography was recommended to assess any additional structural abnormalities that may have been contributing to her airway disease, but it was declined. Dynamic large airway disease was hypothesised as the cause of SDB episodes, and the idea of fitting the patient with a CPAP was explored.

A CPAP device was sourced (RESmart Auto CPAP System - BMC-680A, BMC Medical Co. Ltd, Beijing, China) and the dog was fitted with a standard veterinary anaesthesia induction mask. A soft muzzle was fitted over the induction mask and used to secure it to the dog's head (Figures 1 and 2 - see overleaf). Positive reinforcement and desensitisation techniques were employed over several weeks to gradually get the dog accustomed to wearing the CPAP apparatus. This involved the use of treats in the muzzle/mask combination and removing the apparatus prior to signs of distress. Eventually, the owner reported that after suffering episodes of SDB, the dog would seek application of the mask/muzzle combination. The owner reported that the CPAP device was run on 'auto' (pressure range 4-20 cm H_2O in 0.5 cm H_2O increments) with intermittent use of the humidifier setting. Without the CPAP device, the dog continued to suffer episodes of SDB every 15-30 minutes during sleep. However, with the CPAP device applied, the owner reported the dog was able to sleep between 4-5 hours each night. The device required no maintenance throughout the night and required removal only in the morning once the dog woke.

Four years later, when the patient was 8 years of age, non-sedated survey neck/thorax radiography and dynamic videofluoroscopy were performed to assess whether the SDB may be attributable to DPC. On both

Figure 1. Induction mask fitted to CPAP hose with a soft muzzle over the induction mask to secure it to the dog.

Figure 2. Patient fitted with CPAP device.

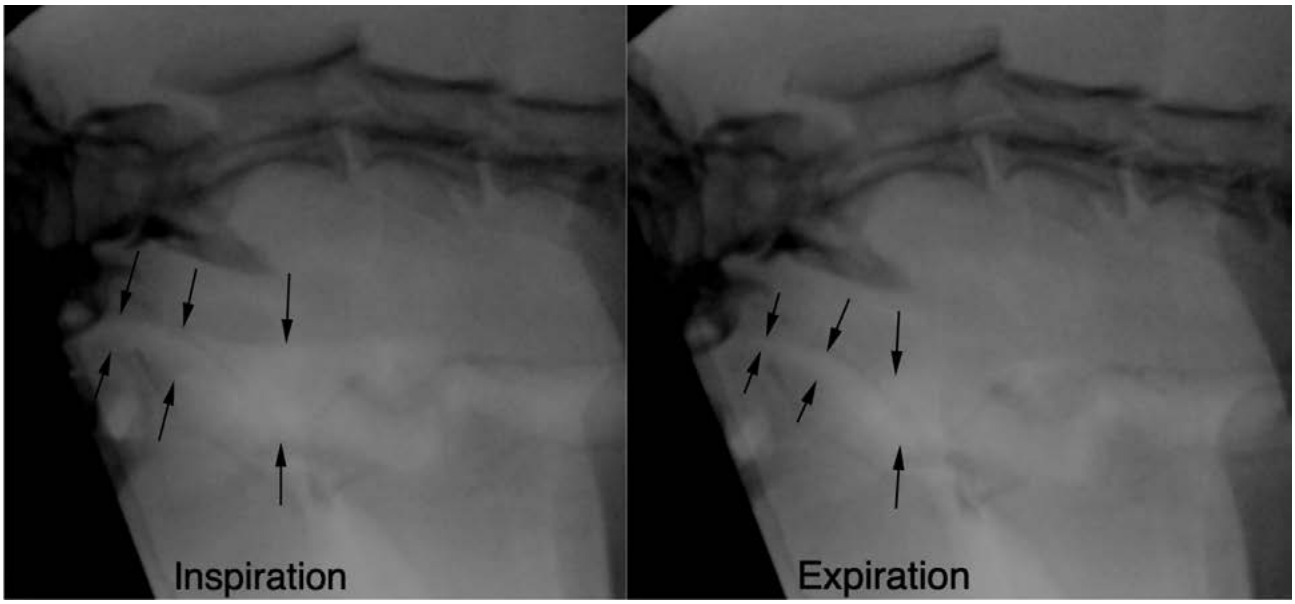

Figure 3. Comparative lateral fluoroscopic images of the throat at inspiratory and expiratory phases of respiration, demonstrating partial pharyngeal collapse (gas-filled pharyngeal lumen depicted by black arrows).

imaging studies, the trachea had relatively uniform luminal diameter within normal limits and no substantial attenuation of the principal bronchi was identified (that is, no evidence of tracheobronchial collapse). The lungs were well inflated with a mild diffuse bronchointerstitial pattern, considered within normal limits given the dog's age and lack of other respiratory signs such as coughing.

Lateral radiographs of the throat obtained at peak inspiration and then during the expiratory pause, and subsequent fluoroscopy, revealed moderate attenuation of the pharyngeal lumen, with ventral displacement of the dorsal pharyngeal wall on expiration when compared to the inspiratory phase of respiration (~60% variation; Figure 3), consistent with partial pharyngeal collapse.[11]

At the time of submission of this article, the dog has been using the CPAP machine daily for nearly 3 years with great success. The dog's ongoing breathing pattern during wakefulness is characterised by mild stertor during moderate levels of activity. No complications or adverse events associated with the use of CPAP have been noted and the dog remains in good health.

DISCUSSION

SDB is a clinical condition recognised in dogs, with brachycephalic breeds over-represented.[1,4,5] In humans, obstructive sleep apnoea syndrome is often attributable to abnormal structure and function of the dilator muscles of the pharynx, resulting in DPC.[3] Similarly, dogs suffer from DPC, with brachycephalic breeds again being over-represented.[10] A recent case series discussed the management of SDB in five Cavalier King Charles spaniels, four of which experienced marked improvement or resolution of their SDB following appropriate corrective airway surgeries for treatment of BOAS.[1] However, one of these dogs did not have resolution of its SDB.

In humans, the use of CPAP facilitates maintenance of a patent airway during sleep, when pharyngeal muscle hypotonia results in exacerbation of pharyngeal collapse in patients with obstructive sleep apnoea syndrome.[7] We hypothesise that our patient continued to suffer SDB, despite corrective airway surgery with good clinical response while awake, due to DPC when sleeping, secondary to muscle hypotonia as seen in humans. In the present case, the owner elected to trial CPAP prior to any further investigation for DPC. The dog responded well to the intervention, supporting our hypothesis. We then retrospectively confirmed partial pharyngeal collapse using videofluoroscopy. A study examining the difference in pharyngeal collapse in humans suffering from obstructive sleep apnoea syndrome in a conscious versus sleeping state found a two-fold increase in the degree of pharyngeal collapse while sleeping.[23] Therefore, it is reasonable to assume that the degree of pharyngeal collapse in the present case during sleep would exceed the 60% percent identified during unsedated, conscious videofluoroscopy.

While the use of CPAP has previously been reported in anaesthetised dogs in a research capacity,[16] and clinically for the acute management of a cat with laryngeal paralysis,[17] it has not been reported for the management of SDB in companion animals. The present case highlights that CPAP can be a well-tolerated and viable treatment for dogs suffering SDB, particularly for those who fail to respond to traditional corrective airway procedures for BOAS. Furthermore, the findings here highlight the complexity and dynamic nature of BOAS in dogs and suggest that patients who respond poorly to traditional surgical intervention may have underlying functional airway disorders such as pharyngeal collapse. Therefore, in addition to considering cross-sectional imaging such as computed tomography to assess for structural abnormalities (e.g. nasopharyngeal turbinates), videofluoroscopy is helpful to evaluate for dynamic large airway disease in these cases.

Some limitations are identified in the present case report. A significant investment on the part of the owner was required during the early phases of the treatment trial, to get the dog accustomed to wearing the CPAP apparatus. Principles of positive reinforcement and desensitisation were employed which involved progressive exposure to the muzzle/mask over time. Certain dogs may not tolerate this intervention, and the level of commitment required of the owner to successfully condition a dog to wearing a CPAP mask during sleep may preclude its use in many instances. Also, the conformation of severely brachycephalic breeds may not

enable the mask to be successfully fitted, although helmets have been reportedly used in the delivery of CPAP to anaesthetised dogs.[16] Additionally, obstructive sleep apnoea syndrome has been associated with hypertension in humans, which was a finding consistent with present case.[8] Unfortunately, blood pressure readings were not available for this patient after instituting CPAP. Computed tomography was declined by the owner for assessment of nasopharyngeal turbinates or additional aberrant airway issues which could have been addressed surgically. Also, this is a single case report and more research is required to determine optimal CPAP protocols and the overall viability of the method. While not specifically assessed in the present case, future investigations into the clinical impact of long-term CPAP use on variables such as oxygenation in an animal suffering from chronic SDB would be worthwhile.

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Laparoscopic cryptorchidectomy in two dogs with testicular torsion

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ABSTRACT Two dogs with unilateral abdominal cryptorchidism were presented for cryptorchidectomy. Abdominal ultrasound was performed in both cases. Case 1 showed a moderately enlarged pampiniform plexus, consistent with spermatic cord torsion, whereas testicular torsion was not evident in Case 2. A laparoscopic-assisted cryptorchidectomy was performed in Case 1, by placing a second cannula directly over the affected testicle, which was then grasped and exteriorised from the abdominal cavity, before extracorporeal ligation. A total laparoscopic approach was performed in Case 2, by placing two additional ports cranial and caudal to the camera port. The cryptorchid abdominal testicle was completely wrapped in omentum and the testicular vessels and ductus deferens were twisted around each other; sealing and transection were performed with the aid of a vessel-sealing device. Portals were connected via a full-thickness incision and the twisted testicle removed. Recovery was uneventful in both cases.

KEYWORDS cryptorchidism, dog, laparoscopy, testicular torsion

ABBREVIATIONS IV, intravenous(ly); VSD, vessel-sealing device

Aust Vet Pract 51 (1): 26–31, 2021

Laparoscopy has been used for cryptorchidectomy in dogs and cats with unilateral or bilateral abdominal cryptorchidism for more than a decade.[1] Various methods, using one to three portals, have been described,[1-3] as well as laparoscopic-assisted techniques.[4-6] For the laparoscopic-assisted approach the testicles are localised during the laparoscopic exploration and then additional portals are inserted directly over the abdominal testicles. The portal sites are then enlarged and the abdominal testicles are exteriorised and ligated extracorporeally.[1,4-6] For the laparoscopic technique, the testicles can be either held up to the abdominal wall with laparoscopic forceps or held in position by a percutaneous needle and suture combination, while the spermatic cord and vessels are sealed with the aid of a vessel-sealing device (VSD) or ligated with endoclips or an extracorporeally tied knot.[1-3] After the testicles are freed from their pedicle, one of the portal sites is enlarged,[1,2] or the single-port device removed, and the testicles removed from the abdominal cavity.[3] Torsion of the spermatic cord or testicular torsion in dogs has been described rarely in the veterinary literature.[7-10] One report described an acute testicular torsion in a puppy, treated via laparoscopy,[11] but laparoscopic-assisted treatment of an acute torsion of the spermatic cord has not been described. Furthermore, to the authors' knowledge, chronic testicular torsion has not been described in dogs.

CASE REPORTS

Case 1 was a 24.7-kg, 9-month-old Labrador retriever with right-sided abdominal cryptorchidism, that presented with signs of an acute abdomen. The physical examination, including a rectal examination, was unremarkable, except for severe pain on palpation of the caudal abdomen. In addition, the patient was reluctant to walk. Haematology and biochemistry profile were unremarkable. Abdominal ultrasonography revealed that the right testicle was located in the caudal abdomen, measuring 2 x 3 cm in diameter, with a slightly inhomogeneous parenchyma and the pampiniform plexus seemed to be moderately enlarged, with no clear Doppler signal (Figure 1). In addition, there was a small amount of free abdominal fluid. All other abdominal organs were within normal limits. The dog was given intravenous (IV) fluids (Ringer-Lactate, B. Braun, Melsungen, Germany) and methadone (0.3 mg/kg IV; Comfortan 10 mg/ml, Eurovet Animal Health BV, Bladel, Netherlands) every four hours. The dog immediately improved and was scheduled for a laparoscopic-assisted cryptorchidectomy and prescrotal castration the next morning.

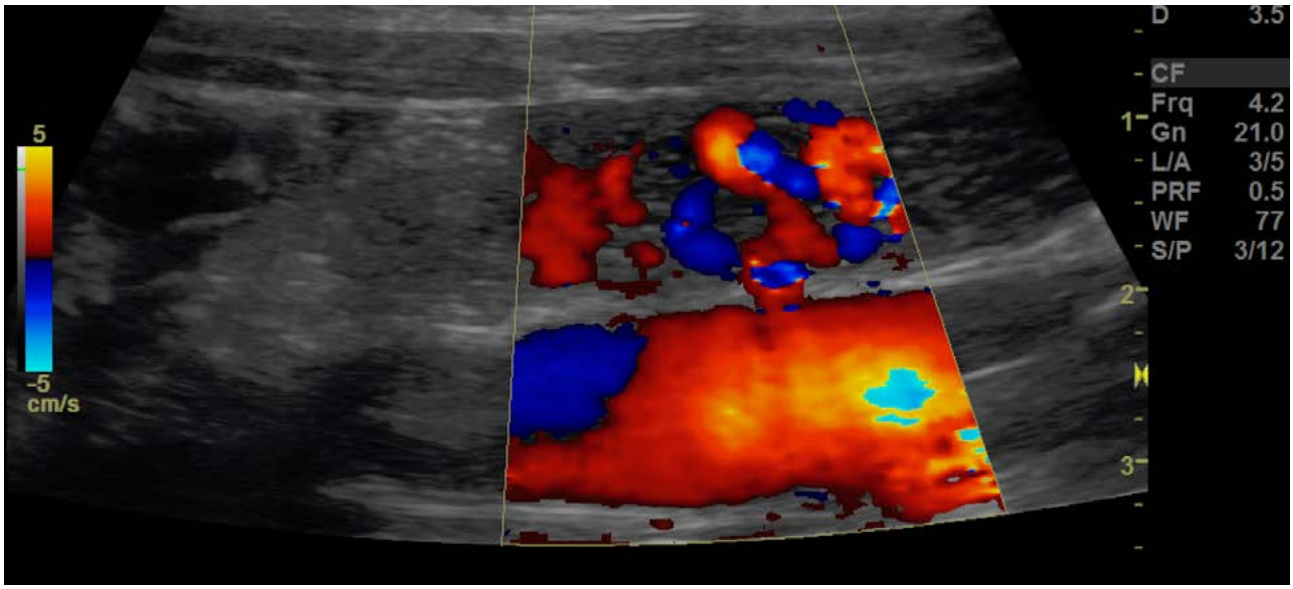

Figure 1: Case 1: Doppler ultrasound images of the abdominal testicle, demonstrating a moderately enlarged pampiniform plexus with no clear Doppler signal. The caudal vena cava and aorta are visible below the pampiniform plexus.

As premedication the dog received 4 µg/kg medetomidine IV (Domitor 1 mg/ml, Orion Corporation, Espoo, Finland) in combination with 0.3 mg/kg methadone and 1 mg /kg ketamine (Ketaminol 100 mg/ml, MSD Tiergesundheit, Wien, Austria). After induction with 3 mg/kg propofol IV (Narcofol 10 mg/ml, CP Pharma, Burgdorf, Germany), the dog was endotracheally intubated and the ventral abdomen clipped, followed by a routine surgical preparation. In a first step, a Ternamian EndoTIP 6-mm cannula (Karl Storz, Tuttlingen, Germany) was placed at the level of the umbilicus with a modified Hasson approach[12] and insufflation with CO_2 to a pressure of 8 mmHg. A second Ternamian EndoTIP 6-mm cannula (Karl Storz) was placed directly over the hemorrhagic infarcted testicle, which was grasped with endoscopic grasping forceps (CLICKline Grasping Forceps, Karl Storz) and exteriorised from the abdominal cavity. The testicular pedicle was swollen and seemed to be twisted around itself. Ligation was performed routinely and the other testicle was removed

via a standard prescrotal approach (Figure 2). Closure of the incision sites was routine. The dog was discharged the same day with oral anti-inflammatory medication (Rimadyl 100 mg once daily, Zoetis Wien, Austria) for three days. The recheck examination after one week was unremarkable. Histopathology revealed haemorrhagic necrosis of the entire organ, which was most likely due to a torsion. An underlying neoplastic disease could not be ruled out definitely, since the specimen consisted mainly of necrotic tissue.

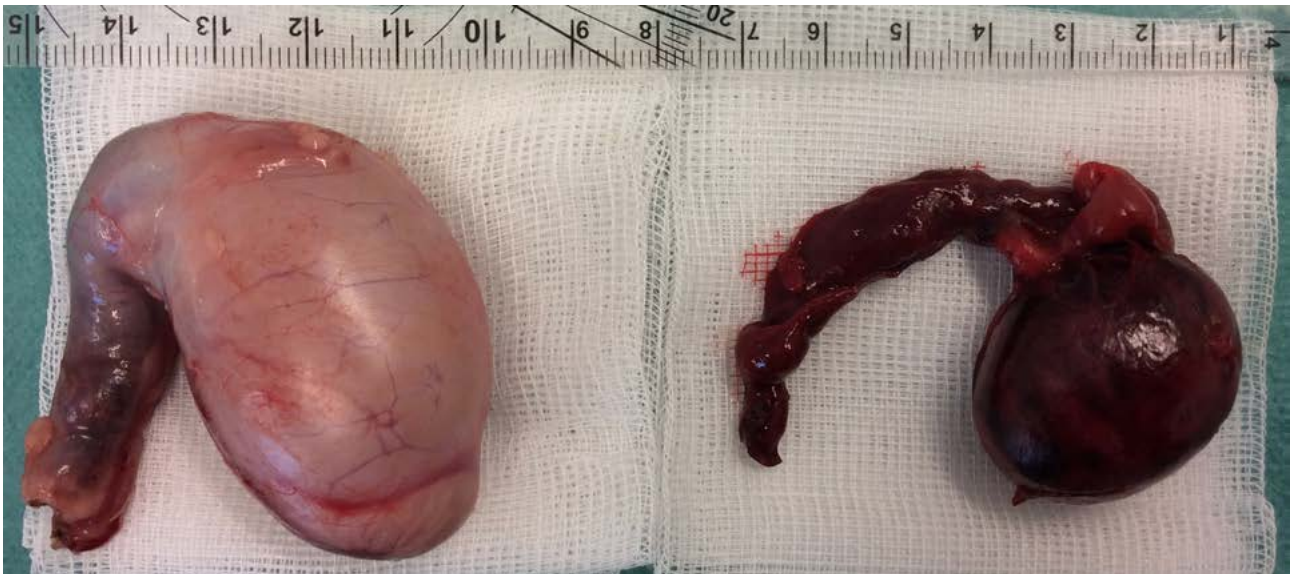

Figure 2: Case 1: Resected testicles. On the left side is the left normal testicle; on the right side, the twisted pampiniform plexus is visible with the hemorrhagic infarcted testicle.

Case 2 was a 36.2-kg, 7-year-old male golden retriever, referred for further investigation of unilateral cryptorchidism. The referring veterinarian had noticed a mass (approximately 5 x 4 cm) in the caudal abdomen and suspected testicular neoplasia. At first presentation to the referral centre the dog was bright, alert and responsive. Physical examination was unremarkable, except for a left-sided cryptorchidism. Abdominal ultrasound confirmed a left-sided abdominal cryptorchidism. The texture of the abdominal testicle was irregular and the structure was severely enlarged, measuring 8 x 6 cm in diameter. The remaining abdominal organs were unremarkable. Neoplasia of the abdominal testicle was suspected and the patient planned for laparoscopic unilateral cryptorchidectomy 4 weeks later.

At the time of surgery the dog was premedicated with diazepam (0.2 mg/kg IV, Diazedor vet, Salfarm Scandinavia, Sweden) and methadone (0.3 mg/kg IV, Semfortan vet, Dechra Veterinary Products, Väsby, Sweden). After induction with propofol (4 mg/kg IV, PropoVet Multidose, Orion Pharma Animal Health, Danderyd, Sweden) the dog was endotracheally intubated and the ventral abdomen clipped and routinely prepared for surgery. With the aid of a Ternamian EndoTIP 6-mm cannula (Karl Storz, Skärholmen, Sweden), a visual entry technique[13] was performed at the level of the umbilicus and insufflation started with CO_2 to a pressure of 8 mmHg. Two additional Ternamian EndoTIP 6-mm cannulas (Karl Storz) were placed cranial and caudal to the first one under visual guidance. Laparoscopic exploration revealed a large structure caudal to the left kidney, measuring about 5 x 8 cm. The structure was completely wrapped by omentum (Figure 3). The omentum was grasped with laparoscopic dissection forceps (CLICKline Dissecting and

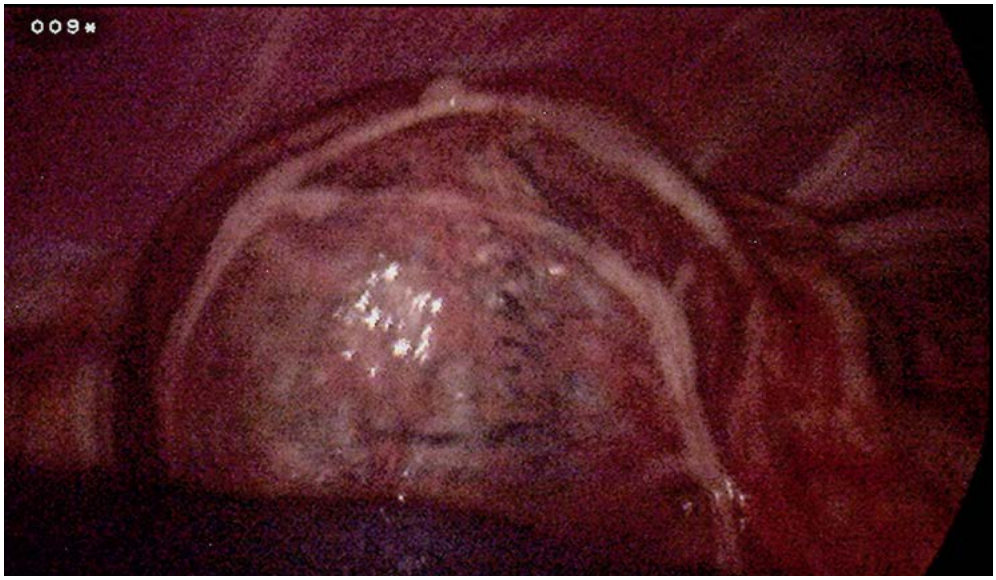

Figure 3: Case 2: Initial laparoscopic view of the abdominal cavity showing the structure within the left caudal abdominal cavity, completely wrapped by omentum.

Grasping Forceps, Karl Storz) and opened by blunt dissection in combination with a VSD (LigaSure 5 mm, Dolphine tip, Covidien, Solna, Sweden). The dissection was continued until the structure was completely freed of all attachments. The structure could then be elevated and it became obvious that the testicular vessels and the ductus deferens were twisted around each other (Figure 4). The vessels and ductus deferens were sealed and transected with the aid of the VSD. Then, the twisted testicle was held in position with the grasping forceps coming through the caudal portal and the endoscope was changed to the most cranial portal. Under visual control the two caudal portals were then connected via a full-thickness incision, which was further enlarged to the cranial portal, so that the twisted testicle could be removed via this enlarged incision. The incision was routinely closed in three layers, including abdominal fascia, subcutis, and dermis. According to the owner's request, the descended testicle was not removed. Due to financial constraints, the resected tissue was not sent for histopathologic analysis. The dog recovered uneventfully and was sent home the same day with a course of oral anti-inflammatory medication (Robenacoxib1, 11 mg/kg once daily, Onsior, Elanco Denmark,

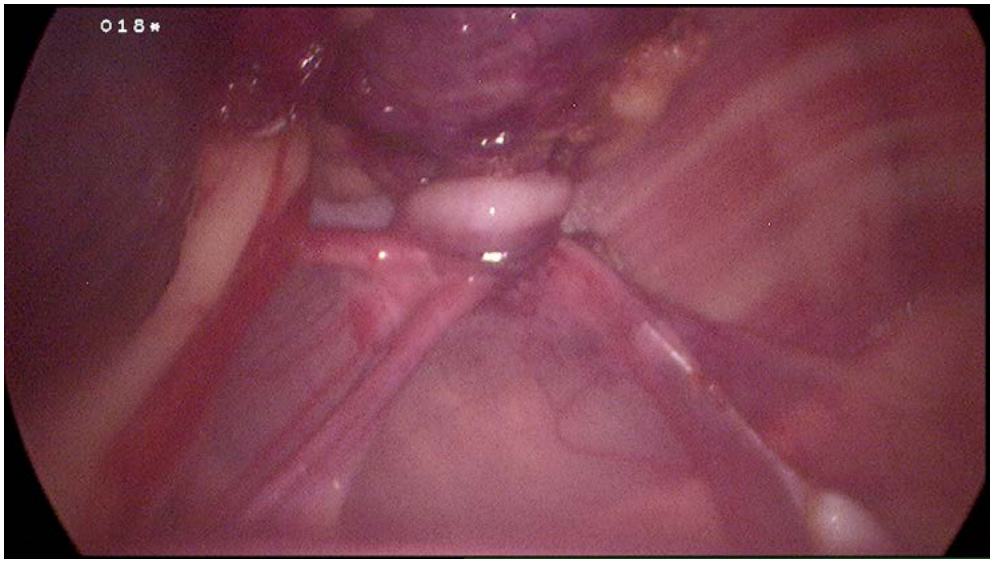

Figure 4: Case 2: The structure is now freed of all omental attachments and lifted up, revealing the completely twisted spermatic cord and testicular vessels.

Ballerup, Denmark) for 3 days. On examination 10 days post-surgery, the surgical site was unremarkable and the dog was doing well.

DISCUSSSION

Acute testicular torsion is an uncommonly described emergency situation in veterinary medicine, with only a few cases published.[7-10] Usually, these patients present with signs of an acute abdomen and require immediate pain relief and surgery as soon as possible.[14] This entity is mainly described in dogs with an abdominal cryptorchid testicle, since those testicles lie free within the abdominal cavity and therefore, are very mobile, predisposing them to torsion.[14] There are many other differential diagnoses for an acute abdomen. These range from gastrointestinal problems (gastric dilatation, volvulus, intussusception, foreign body, pancreatitis) to problems within the urogenital tract (pyelonephritis, ureteral or urethral obstruction, prostatitis), different types of peritonitis (chemical or septic) or splenic torsion.[9,15] To distinguish these causes, an abdominal ultrasound is a valuable diagnostic tool.

Ultrasonography has a sensitivity of 96.6% and a positive predictive value of 100% for detection of intraabdominal testicles,[16] while a 450° rotation of the spermatic cord is required to be able to diagnose that disease via ultrasound.[17] As shown in Case 1, it can be difficult to confirm testicular torsion, even with Doppler ultrasound. Since such a diagnosis was not established, and the patient responded well to the pain medication, the dog was scheduled for a laparoscopic-assisted procedure the next morning.

In Case 2, it seems very likely that the testicle was already twisted at the time of the first presentation, because it did not change in size from the first visit to the second visit (4-week period) and was completely wrapped in omentum, which was strongly adhered to the testicle. Furthermore, the dog did not show any signs of acute abdomen during that time. Because the testicle was not examined by histopathology, the underlying aetiology is speculative. The testicle may have been enlarged due to neoplasia and subsequently twisted on its pedicle, or torsion of a normal abdominal cryptorchid testicle could have caused congestion and an increase in size, until the pressure was too high for more blood to be pumped into the testicle. Ultrasonography was performed on Case 2, but no Doppler examination, since neoplasia was suspected and the dog was not showing any signs of an acute abdomen. Therefore, a testicular torsion was not considered as a potential differential diagnosis.

The reason why a laparoscopic-assisted approach was chosen in the first case is that one goal of minimally invasive surgery is to reduce the number of portals. As shown in one study,[18] there was no significant difference in pain scores post-surgery when single port and two port techniques were compared. Since a single-port system was not available, the laparoscopic-assisted technique was deemed to be most beneficial to the patient. Adding a third portal would have added more soft tissue trauma and pain to the patient and still, there would have been the need to enlarge one of those portals, to be able to remove the cryptorchid testicle from the abdominal cavity. Percutaneous temporary fixation with a needle suture combination was not considered as an option in Case 1, since an underlying neoplastic process could not be entirely ruled out at that point.

CONCLUSION

Laparoscopic or laparoscopic-assisted approaches can be safely used in both acute and chronic torsion of the spermatic cord in cryptorchid dogs. Doppler ultrasonography should be used as part of the examination of a dog presenting with abdominal pain and an intraabdominal testicle. In addition, it may help to verify testicular torsion and/

or testicular neoplasia and will help with the decision of the type of laparoscopic approach and positioning of laparoscopic cannulae.

CONFLICTS OF INTERST AND SOURCES OF FUNDING

The authors declare no conflicts of interest or sources of funding for the work presented here.

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Care and husbandry of sugar gliders (*Petaurus breviceps*)

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ABSTRACT Sugar gliders are becoming increasingly popular as pets in some states in Australia. The present review considers aspects of the life of the free-ranging members of this species and how these can be incorporated into the care and husbandry of captive sugar gliders.

KEYWORDS care, husbandry, Petaurus breviceps, sugar glider

Aust Vet Pract 51 (1): 32-38, 2021

Sugar gliders (*Petaurus breviceps*) are small marsupials, native to Australia, New Guinea and some Indonesian islands. They are becoming increasingly popular as pets, both locally and internationally. In Australia, sugar gliders can be kept, subject to a permit, in the Northern Territory, South Australia and Victoria. However, they are not allowed to be kept as pets in the Australian Capital Territory, New South Wales, Queensland, Tasmania or Western Australia.

Despite their adorable, fluffy appearance these marsupials are actually highly specialised mammals with unique feeding, housing and social requirements that must be met to provide them with a healthy and fulfilling life. As with any non-traditional pet, it is essential to understand the needs of the species free-living counterparts and replicate them as closely as possible.

This review is derived from published literature on the care and husbandry of this species[1-8] and the author's personal experience. It does not consider diseases and their treatment.

Sugar gliders are a nocturnal species of diprotodont marsupial, closely related to possums and other glider species. By day, they sleep in tree hollows, then venture out at night to forage for food (Figure 1). They are social animals that will typically live in groups of 4-6 individuals.

Figure 1. A foraging sugar glider.

The expected life-span of sugar gliders in captivity is 9-12 years.

Healthy adult males weigh 100-160 g and adult females 80-135 g. Their daily food requirements are 15-20% body weight.

A sugar glider's expected "TPR" is: body temperature 35.0-36.0°C, heart rate 200-300/min and respiratory rate 16-40/min. Their thermoneutral zone is 27-31°C.

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DIGESTION

Sugar gliders are omnivorous diprotodonts and possess sharp upper and lower incisors, upper canines, and opposing sets of premolars and molars (3 and 4 teeth, respectively). This allows them to gnaw branches and leaves to obtain gum, sap and pollens, and to crunch insects and small invertebrates for protein. Further details of their nutrition are discussed below.

Sugar gliders are hindgut fermenters that have adapted to use bacteria in their caecum to digest the complex sugars found in tree sap and gum. The digestive tract terminates at the cloaca, a shared chamber with the reproductive and urinary tracts, that exits through a single common opening known as a 'vent'.

Reproduction

The female reproductive tract comprises two lateral vaginas, a central vaginal canal and two uteri. There is a ventral abdominal pouch containing four teats.

Males have a long, forked penis with 2 openings (proximal for urine, distal for seminal fluid) and a pedunculated scrotum containing 2 testicles.

Females are seasonally polyoestrous, with an oestrus cycle of 29 days. After a gestation period of 15-17 days, females give birth to tiny joeys (usually two, each weighing about 0.2 g) that ascend from the cloacal opening into the pouch, where they stay for 70-74 days. On emerging from the pouch, the joeys stay in the nest/hollow until weaning at 110-120 days of age, eventually leaving their colony in search of a new one at 7-10 months of age.

Puberty occurs at 8-12 months in females and 12-15 months in males.

BEHAVIOUR

Sugar gliders are highly social animals. They prefer to live in harem-style groups, with one dominant male and several cohabitating females, that may or may not be related.

Males have prominent scent glands that produce dark brown, musky scented secretions. These glands are positioned on top of the head (frontal gland), under the chin and throat, on the cranial thorax (sternal gland) and adjacent to the vent (paracloacal gland). The last one is also found in females. These scent glands, along with urine spraying, are used for marking territory and mates and make gliders quite fragrant a lot of the time!

In a captive situation, sugar gliders are best kept in small groups. The author does not recommend keeping a single individual as a pet, as it is so far from the 'normal' way of life for this species and is arguably a source of chronic stress for a lone animal. Housing them at least in a pair should be considered imperative to good glider welfare.

These intelligent and social creatures form strong bonds to cage mates and can become very trusting and tolerant of regular handling and interaction with their humans (Figure 2).

Figure 2. Sugar gliders can become very trusting of their human contacts.

Positive reinforcement through regular, gentle handling during the animal's active hours (evening, night or dawn) and offering treats such as meal worms can help build on this relationship.

Distressed or scared sugar gliders make a chattering sound, called "crabbing", which should signal to the handler to slow down and give the animal time to adjust to what is happening. A frightened animal that feels trapped will likely try to scramble away, causing many scratches and the occasional bite.

HOUSING

The native habitat of the sugar glider is dense woodland and scrub, allowing them to lead an arboreal lifestyle. Their very specialised anatomy and gliding membrane (patagium) allows them to save energy by gliding between trees whilst hunting insects for food.

In a captive setting, a large aviary style enclosure (PVC-coated stainless-steel wire) or a tall wire-sided cage can be used to provide space for climbing and jumping. A maximum width of 10 mm between bars is advised, with horizontal bar position preferred, making many of the standard large parrot cages useful for gliders too. Cages should be placed on a stand, or elevated to eye level, to mimic a raised position within a tree, and should be away from direct sunlight and draughts. Given that these are nocturnal animals, it is also suggested to keep the cage away from thoroughfares within the home to avoid disturbing their sleep pattern.

Furniture within the cage should include a number of nesting boxes and pouches, anchored at various heights and positions. Wood, plastic, wicker and fleece fabrics are all suitable. Shredded paper, strips of fabric and even old socks can serve as bedding within these spaces. Wood shavings should be avoided due to their moisture absorbing and ammonia releasing properties, which may predispose to respiratory disease.

Climbing branches and ledges can be put together using native tree branches, rope perches (sisal fibre), sea grass mats and non-treated pine shelves (Figure 3). Generally, bird-safe toys made of natural, non-toxic materials make good sugar glider equipment, with items such as ladders, hanging paper toys and colourful plastic beads being popular choices. It is important to note that no matter how large and well equipped the enclosure, sugar gliders should also be provided with supervised time outside of their cage to explore and exercise. For those who are really committed, there are a number of excellent climbing frames and suspended play gyms that can be constructed to hang from the ceiling and mimic an arboreal canopy for gliders. Pinterest has some fabulous ideas!

Figure 3. An example of a large sugar gliders' enclosure.

When sugar gliders need to be transported, for instance to the veterinary clinic, a soft, comfortable pouch will help to make them feel secure and allow for safe transport and handling (Figure 4).

Figure 4. An example of a 'travel' pouch to help a sugar glider to feel secure and allow for safe transport and handling.

NUTRITION

Perhaps the most crucial thing to get right with sugar gliders is their diet. This is the subject of extensive research and debate about the best way to deliver the required nutrients, whilst avoiding issues with obesity and nutritional deficiencies.

The diet of wild sugar gliders is complex, consisting of a combination of eucalyptus sap, acacia gum, manna, nectar, pollen and insects. The captive diet needs to mirror the latter. The wild diet also varies with the season, as flowering plants emerge and insect abundance varies, which further adds to the difficulty of getting a captive diet right.

The basic components of the wild diet are:

- Protein- derived from insects and arachnids.
- Carbohydrates - sugars as complex polysaccharides in tree sap, gum and secretions of sap-sucking insects.
- Fibre - leafy greens and plant matter.

An example of a balanced captive diet (Figure 5) includes:

- 65% vegetable and fruit mix (carbohydrates), with 25% fruit, 25% leafy greens and 50% vegetables.
- 20% nectar mix (sugars).
- 15% insects and carnivore mix (protein).

The diet can be formulated using a combination of Wombaroo products[6] to try and replicate the natural diet of the sugar glider in a captive setting.

Figure 5. Food preparation is an essential part of good sugar glider husbandry. It needs to include fruit and vegetables (front row), protein (middle row) and nectar (back row).

Historically, the Leadbeater's possum formula has been a popular base diet that was developed by Australian zoos to help provide the variety of nutrients needed for their captive gliders. Various items of fresh food and insects can be added to achieve a similar goal, though the precise nutrition is less well documented.

Ingredients for the Leadbeater's possum formula are:

150 mL warm water
150 mL honey
1 shelled hard-boiled egg
25 g high protein baby cereal
1 tsp multivitamin/mineral supplement

Recipe:

- Mix warm water and honey. Blend egg in a separate container until evenly mixed.
- To the egg, gradually add the honey/water and blend until smooth.
- Repeat for multivitamin supplement, then baby cereal, blending after each addition.
- The formula can be refrigerated (use within 2-3 days) or frozen in ice cube trays for use later.

Fresh, clean water must be available at all times. It can be offered in a bowl or sipper bottle, depending on the glider's preference. It is advisable to have more than one water station available as active animals may accidentally knock things over during the course of their nightly adventures.

WELLNESS CARE

Annual health checks are recommended as part of routine wellness care for this species. The purpose is to review the diet and husbandry and perform a physical examination (Figure 6), including a weight check, dental check and faecal examination (flotation and direct smear) for parasites. For animals 5 years and older, 6-monthly wellness checks are advised, including annual blood tests to monitor for underlying issues and provide a baseline for the individual should future issues arise. Depending on the patient and any other concerns, urinalysis and radiographs could also be considered as part of the patient's database.

It is recommended that blood samples are collected under general anaesthesia. This can be achieved using a mask and isoflurane in oxygen (4-5% isoflurane for induction and 1-2% for maintenance at an oxygen flow rate of 1 L/min). Blood (0.5-1 mL) may be collected from the cranial vena cava or the jugular vein. Reference values for haematology and biochemistry have been published (Tables 1 and 2 respectively).

Figure 6. Performing a physical examination

CONCLUSION

Veterinarians and owners are responsible for meeting the specialised needs of sugar gliders in their care, and ultimately ensuring their welfare is maintained. It is essential that the veterinary practitioner understands the basic biology and needs of the wild sugar glider in order to guide their clients on how best to adapt them for a captive lifestyle. Only by providing adequate care and husbandry to these pets will we be able to mitigate the onset of captivity-induced disease.

Table 1. Haematology reference values for sugar gliders[5]

Analyte	Units	Reference values
Haemoglobin	g/L	130-150
Haematocrit	L/L	0.45-0.53
Erythrocytes	x 10^{12}/L	5.1-7.2
MCHC	g/L	300-330
Total leukocytes	x 10^{9}/L	5.0-12.2
Neutrophils	x 10^{9}/L	1.5-3.0
Lymphocytes	x 10^{9}/L	2.8-9.2
Monocytes	x 10^{9}/L	0.06-0.20
Eosinophils	x 10^{9}/L	0.02-0.14
Basophils	x 10^{9}/L	0

MCHC mean corpuscular haemoglobin concentration

Table 2. Serum biochemistry reference values for sugar gliders[5]

Analyte	Units	Reference values
Total protein	g/L	56-69
Albumin	g/L	30-35
Globulin	g/L	22-36
ALT	U/L	50-106
AST	U/L	46-179
Creatine kinase	U/L	210-589
Urea	mmol/L	6.4-8.6
Creatinine	µmol/L	18-44
Glucose	mmol/L	7.2-10.0
Sodium	mmol/L	135-145
Potassium	mmol/L	3.3-5.9
Calcium	mmol/L	1.7-2.1
Phosphorus	mmol/L	1.2-1.4

ALT Alanine aminotransferase

AST Aspartate aminotransferase

ACKNOWLEDGEMENT

Most of the material in this paper was originally presented at the Unusual Pets and Avian Veterinarians conference in Melbourne, Australia, in November 2019. This article has not been peer-reviewed.

CONFLICTS OF INTEREST

The author declares no conflicts of interest relating to this review.

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Anaesthesia guidelines for dogs and cats

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EDITOR'S NOTE These guidelines were first published in the Australian Veterinary Journal 96:413-427, 2018; doi: 10.1111/avj.12762 as "Standards of Care. Anaesthesia guidelines for dogs and cats". They were subsequently produced as a guideline document for practitioners by the Australian Small Animal Veterinarians. The latter group intends to update the guidelines and is reproducing them here to enable practitioners to comment on their content. Please refer such comments to the ASAV office (details at the front of this issue).

ABBREVIATIONS ACVAA, The American College of Veterinary Anesthesia and Analgesia; APL, adjustable pressure limiting/"pop-off" (scavenging valve); ASA American Society of Anesthesiologists; BMBT, buccal mucosal bleeding time; CRT, capillary refill time; ECG, electrocardiograph(y); ET, endotracheal (tube); IM; intramuscular(ly); IV, intravenous(ly); MAC, minimun alveolar concentration; NSAID, non-steroidal anti-inflammatory drug; PCV, packed cell volume; SC, subcutaneous(ly); TS, total solids; USG, urine specific gravity; VIC, vaporiser in circuit (system); VOC, vaporiser out of circuit (system); vWD, von Willebrand disease; WHO, World Health Organization.

Aust Vet Pract 51 (1) 39-63, 2021

The Australian Veterinary Association has prepared these guidelines to support veterinarians in offering the highest standards of care to their patients. The guidelines set out in this document detail the ideal standards of anaesthetic care for dogs and cats within a general practice setting. The Australian Veterinary Association believes that owners should be advised of the best available care as set out in these guidelines. These guidelines have been based on the latest peer-reviewed scientific research, surveys of Australian veterinarians and broad consultation within the veterinary profession. Anaesthesia is a continually evolving discipline, with frequent advances in pharmacology and technology. It is mandatory for all members of the anaesthesia team to periodically undergo training to refresh their knowledge. Referral to a board-certified veterinary anaesthesiologist should be considered for cases that are beyond the practitioner's level of expertise or comfort.

These guidelines will be reviewed and updated based on feedback from veterinarians and the publication of relevant new research.

PREANAESTHETIC EVALUATION

Signalment

Information pertaining to species, age, breed, neutering status and demeanour should be noted for each patient. An understanding of age and breed characteristics may provide information about additional anaesthetic concerns and prompt further diagnostic tests. Geriatric animals may pose higher anaesthetic risk due to reduced cardiac, hepatic and renal function, which may not be apparent on routine physical examination. Similarly, certain breed differences can lead to greater risks for airway obstruction, increased responsiveness to anaesthetic drugs, and delayed recovery, all of which can result in increased anaesthesia-related morbidity and mortality. Individual genetic variability can trigger unexpected and adverse responses to anaesthetic drugs, which need to be identified by good record keeping and consistent patient monitoring. Although genetic differences are typically held responsible for prolonged recoveries and

Table 1: Selected breed-related anaesthesia concerns

Category	Concern	Precaution
Brachycephalic breeds (e.g. Pug, bulldog, Boxer, Pekingese, Persian cats, etc.)	Brachycephalic airway syndrome; increased risk of upper airway obstruction. Consideration should be given to delaying the elective procedure until corrective airway surgery has been performed.	• Preoxygenate • Caution with excessive sedation • Diligent monitoring from admission to discharge • Prepare for difficult intubation • Provide supplemental oxygen until extubation • Extubate ONLY after patient is bright and alert with a gag-reflex present • Monitor breathing pattern and oxygenation status post extubation • Be prepared to re-anaesthetise and re-intubate the patient in recovery if required
Sighthounds (e.g. greyhound, whippet, Borzoi, Saluki, Afghan hound, Irish wolfhound)	Delayed drug metabolism, possible delayed recovery from drugs such as barbiturates, propofol and acepromazine; lower body fat percentage; hypothermia; stress-induced hyperthermia; myopathy	• Use low-dose acepromazine (0.005-0.02 mg/kg IM) • Avoid barbiturates • Administer propofol or alfaxalone SLOWLY to effect • Monitor patient temperature and actively warm as required • Treat stress/pain-induced hyperthermia with prompt anxiolytics, analgesics, cooling • Provide adequate padding during long procedures

Table 1: Selected breed-related anaesthesia concerns

Category	Concern	Precaution
Herding breeds (e.g. collie, Border collie, Australian shepherd dog, Shetland sheepdog)	*ABCB1* (MDR1) mutation causes defect in P-glycoprotein pump, resulting in accumulation of certain drugs into the central nervous system, causing excessive prolonged sedation	• Diligent monitoring following sedation • Reduce premedication/ sedation dose by at least 25% • Consider using drug which can be reversed/antagonised • Consider genetic screening prior to anaesthesia for elective procedures
Toy breeds (e.g. Chihuahua, pomeranian, Shih Tzu, Brussels griffon)	Hypothermia due to large body surface area to volume ratio; difficulty monitoring; hypoglycaemia	• Active patient warming and diligent monitoring of body temperature • Utilise Doppler blood pressure monitoring device, along with ECG, pulse oximetry, temperature • Monitor blood glucose concentrations prior to and during anaesthesia as well as recovery; supplement as required
Giant breeds (e.g. Saint Bernard, Newfoundland)	Increased response to sedatives	• Dose drugs based on lean body mass or allometric scaling (e.g. α2-adrenergic agonists)
Dobermann	Predilection for dilated cardiomyopathy, von Willebrand disease	• Evaluate coagulation status • If von Willebrand disease suspected, administer desmopressin prior to surgery • If NSAID are required, administer COX-2-selective drug
Boxer dog	Acepromazine-induced vagal response, marked hypotension, and bradycardia has been reported in Boxer dogs of UK lineage; Boxer cardiomyopathy	• Avoid acepromazine in Boxer dogs if possible and reduce acepromazine dose (0.005-0.01 mg/kg IM) if alternative sedation agents are not available • Thorough patient history and physical examination including ECG analysis prior to anaesthesia

increased drug responsiveness, true genetic sensitivity has been demonstrated in only a handful of breeds, including the greyhound and the collie (Table 1). Demeanour should be taken into consideration when planning individual anaesthetic drug protocols, reducing patient perioperative stress and ensuring safety for veterinary personnel.

History and Reason for Anaesthesia

A detailed, accurate history should be considered an essential part of a preanaesthetic evaluation. Previous conditions take on significance when their residual effects compromise the patient while under anaesthesia or become exacerbated as a result of the stress of anaesthesia and recovery. For example, a patient with cardiac disease may be more intolerant of fluid therapy or a brachycephalic breed may have a higher risk of recovery complications due to brachycephalic airway syndrome. The history should include pertinent information regarding prior and concurrent drug therapy and whether the patient has had any adverse reactions or sensitivities to medications or anaesthetic agents. When possible, the anaesthetic record of a patient that has been anaesthetised previously should be retrieved and reviewed thoroughly for any adverse responses during induction, maintenance, or recovery from anaesthesia.

Physical Examination

A thorough physical examination of all body systems should be performed prior to general anaesthesia and sedation. For further information please refer to REGULAR HEALTH CHECK GUIDELINES FOR DOGS AND CATS, from the ASAV office.

Clinical Diagnostics

Preanaesthetic diagnostics are a consideration to allow detection of underlying disorders that may influence the management of the patient or influence the prognosis associated with any given disorder. The decision regarding when to perform preanaesthetic diagnostics and which tests to include is a decision that needs to be based on the patient's history, as well as physical examination, and addressed individually by each practice on a case-by-case basis. Evaluation of patients at least one day prior to an elective procedure can help avoid time conflict between the surgery schedule and the benefit of additional clinical diagnostics. Recommended diagnostic testing for specific conditions are discussed later.

Preanaesthetic Laboratory Evaluation

Preanaesthetic diagnostic testing is an adjunct to a detailed patient history and thorough physical examination to aid in the detection of disease. While there can be no doubt that preanaesthetic biochemical and haematological analysis are valuable for certain patient groups (e.g. geriatrics) and any patient that is clinically unwell, questions have been raised as to whether they are justified for every patient.[1] The use of extensive laboratory screening of patients has not been shown to significantly improve patient outcomes or prompt changes in anaesthetic technique.[2] Despite this conclusion, preanaesthetic blood analysis was found to have resulted in a reclassification to a higher American Society of Anesthesiologists (ASA) status (see below) in 8% of dogs and additional preoperative treatments in 1.5% of dogs which would have otherwise not been evident from the patient history or physical examination and should therefore be considered for all patients undergoing anaesthesia.[2] Given increased understanding of the limitation of population based reference intervals, there is value in comparing results of preanaesthetic biochemical parameters to previous results for that individual, to see if there have been significant changes, even if the values remain within the population based reference intervals.[3] Recent publications have shown

that significant changes can be detected by diagnostic tests in patients which would have otherwise been considered to be in good health based on history and physical examination alone (6.2% of dogs and 19.2% of cats).[4] The type and timing of diagnostics should be determined by the veterinarian based on previously mentioned factors, as well as any changes in patient status or the presence of concurrent disease. There is no evidence to indicate the minimum timeframe prior to anaesthesia within which laboratory analysis should be performed. However, the timing should be such as to best reflect current changes that may impact anaesthetic risk.

When faced with financial or technical limitations which prevent extensive preanaesthetic biochemical and haematological analysis the authors recommend the following minimal preanaesthetic screening be mandatory: packed cell volume (PCV), total solids (TS), blood glucose, blood urea and urine specific gravity (USG). Prior to major surgical procedures the authors also recommend performing a peripheral blood film evaluation to enable prompt identification and characterisation of conditions such as anaemia and thrombocytopenia.

Advanced Laboratory Evaluation

von Willebrand disease (vWD): Breeds that have a high incidence such as Dobermanns should have a buccal mucosal bleeding time (BMBT) performed prior to anaesthesia for any surgical procedure. A finding of prolonged BMBT indicates further testing for vWD is required.

Platelet counts and coagulation panels: Indicated for patients exhibiting signs of unexplained/easy bruising, petechiae or ecchymosis and for procedures where significant haemorrhage is possible.

Arterial blood gas, acid base and electrolyte analysis: Indicated for patients with suspected pathophysiologic abnormalities that can alter gas exchange or acid-base disturbances. Interpretation of a blood gas profile should be accompanied with consideration of electrolyte concentrations. This should focus on recognition of acid-base and electrolyte patterns; and while this rarely leads to a specific diagnosis, it allows for tailoring of fluid therapy and specific intervention which can be lifesaving (e.g. addition of potassium in hypokalaemic patients).

Diagnostic Imaging

Suspected or known conditions requiring diagnostic imaging for the purpose of evaluating anaesthetic risk include cardiac and/or respiratory disease. Investigative techniques include thoracic radiographs and, if indicated, echocardiography or ECG.

Anaesthetic Risk and Physical Status

Following completion of a thorough patient history, clinical examination and interpretation of findings from ancillary diagnostics, the ASA Physical Status Classification System can be assigned (Table 2). The practice of assigning an ASA Status to a patient provides a framework for the clinician to summarise their preanaesthetic evaluation and encourages the clinician to think about the physical status of the patient rather than solely focusing on the procedure itself. It is also a useful standardised method of documenting patient risk from a medico-legal standpoint as high ASA scores have been shown to be predictive of anaesthetic morbidity and mortality in veterinary patients.[5]

Owner Comprehension and Informed Consent

Prior to anaesthesia clients should be fully informed of all relevant known risks associated with the planned procedure including the anaesthesia and associated

loco-regional anaesthesia techniques, and signed consent should be obtained. In addition, this discussion should include consented instruction as to what degree of intervention should be performed in the event of a life-threatening emergency such as a cardiac arrest. Utilisation of a colour-coded system has been recommended for clarity within the practice setting, for example:

- CODE Red - No resuscitation should be attempted
- CODE Amber - Resuscitation should be attempted but should be limited to basic life support techniques such as closed chest cardiac compressions - unless the abdomen is open and trans-diaphragmatic direct cardiac massage is feasible
- CODE Green - All available means of resuscitation should be employed including open-chest direct cardiac massage (when trained personnel capable of performing such techniques are present).

Table 2: ASA* Physical Status Classification System

1 - A normal healthy patient
2 - A patient with mild systemic disease
3 - A patient with severe systemic disease
4 - A patient with severe systemic disease that is a constant threat to life
5 - A moribund patient who is not expected to survive without the operation
If the procedure is an emergency, the physical status classification is followed by "E" (for emergency), for example "3E" would be for a patient with severe systemic disease undergoing emergency anaesthesia.

* American Society of Anesthesiologists

Should more patient information be obtained (e.g. from additional diagnostic tests), the risk assessment associated with anaesthesia may change. The owners should be made fully aware of such changes.

PREPARATION FOR ANAESTHESIA

Individual Patient Plan

An individualised anaesthetic and analgesic plan should be constructed for the management of each patient based on risks identified in the preanaesthetic evaluation and incorporating the staffing, equipment and drug resources available. Contingencies should be made for potential adverse events, and emergency drugs should be available, and doses calculated.

Stabilisation

Where possible concurrent disease or conditions which may contribute to an elevated anaesthetic risk should be addressed prior to anaesthesia. Elective procedures should not proceed whilst concomitant and untreated risk factors exist. Patients requiring emergency procedures should receive optimal stabilisation prior to anaesthesia.

Fasting

It is recommended that healthy dogs and cats are fasted for at least 6 hours prior to being anaesthetised, whenever possible, to reduce the risk of regurgitation and aspiration pneumonia.[6] Water should not be restricted until just prior to anaesthesia at the time of premedication.[6] Exceptions to this would

include patients undergoing gastric surgery and those with megaoesophagus. Dogs and cats aged less than 8 weeks or weighing less than 2 kg are at greater risk of hypoglycaemia and should not be fasted for greater than 1-2 hours.[6]

Premedication and Analgesic Plan

The importance of a well-planned premedication and analgesic plan cannot be understated. The choice of premedication will be influenced by signalment, temperament, concurrent disease, the procedure to be performed, drug availability, personal familiarity and preference. The appropriate choice of premedication drugs should aim to achieve the characteristics outlined below. No individual drug possesses all of the following properties, and as such various combinations of agents are used to best achieve these characteristics:

Sedation and stress reduction - Calm or immobilise a patient to enable minor procedures (e.g. catheterisation, clipping etc.), reduce stress and decrease anaesthetic drug requirement during induction of anaesthesia and reduce adverse arrhythmogenic autonomic activity.[6]

Safe handling - Facilitate safe patient handling and preanaesthetic preparation (e.g. catheterisation, clipping etc.) for both the animal and personnel.

Analgesia is a key component to well-balanced anaesthesia for all surgical and potentially painful procedures. It is also important to note that appropriate preanaesthetic stabilisation of a patient should involve the treatment of any pre-existing pain. Experimental studies have shown a reduced requirement for post-operative analgesia when analgesics are administered “pre-emptively”.[7] Typically an opioid forms the analgesic component of the premedication for surgical procedures. The selection of the particular opioid and initial dose rate should be made on the basis of:

- the expected intensity of the pain
- the duration of action required
- the desired speed of onset
- relevant side effects of the particular drug.

For a detailed and practical resource designed to assist practitioners in recognising, assessing, and treating pain, refer to The WSAVA Global Pain Council Guidelines available at https://wsava.org/global-guidelines/global-pain-council-guidelines/

Balanced anaesthesia - By providing analgesia and sedation the premedication should also facilitate a dose reduction of other potentially more physiologically compromising drugs (e.g. inhalational agents) used to produce anaesthesia.

Calm recovery - Both the analgesic and sedative components of the premedication drug combination should be present at the time of emergence from anaesthesia to promote a calm recovery. If the duration of therapeutic effect of the individual agents is not sufficient to achieve this, redosing of one or both components may be required prior to the conclusion of the anaesthetic procedure.

Premedications are typically administered intramuscularly (IM) or subcutaneously (SC) 15-45 minutes before induction of anaesthesia. Whenever permissible by drug labelling guidelines, premedicants should be administered IM, as this route affords more reliable drug absorption, compared with SC administration.[8]

Venous Catheterisation

An intravenous (IV) catheter is the patient's lifeline while under the effects of general anaesthesia and sedation. It allows direct administration and rapid uptake of anaesthetic, analgesic and emergency drugs as required perioperatively. Various indwelling catheters placed using an ‘over-the-needle’ technique are appropriate for peripheral veins

and the catheter choice depends on animal size and personal preference. To reduce resistance-to-flow and clot formation, the largest possible gauge catheter appropriate for the vessel to be catheterised should be selected. The catheter type and size, along with the time, date and site of insertion should be noted in the anaesthetic/medical record.

The cephalic vein of the thoracic limbs or the lateral and medial saphenous veins of the pelvic limbs are the most common peripheral veins catheterised. With placement of catheters, strict asepsis is important. Appropriate clipping of the area and an aseptic preparation of the skin (with a 1-2% iodine tincture, iodophors, chlorhexidine or 70% alcohol solution) is necessary. Appropriate hand hygiene must be applied, and ideally sterile gloves worn, particularly for long-stay catheters.

Topical anaesthetic agents (e.g. Emla® cream) may be applied 45-60 minutes prior to cannulation to reduce stress and facilitate catheter placement in anxious or hyperaesthetic patients. Cover the topical anaesthetic with an occlusive dressing after application to ensure it is not ingested. Constant supervision is advised.

To decrease risk of catheter-related complications, sterile placement, daily inspection and rewrapping of the catheter site is advised. When possible, administration of irritating or hypertonic solutions into a peripheral vein should be avoided. Regular inspection enables early identification of complications including, but not limited to:

- phlebitis, or inflammation of the vessel
- thrombosis or formation of a thrombus on the catheter or vessel wall
- embolism
- catheter breakage
- subcutaneous fluid infiltration.

Tight taping or bandaging, leading to swelling distal to the catheter, may contribute to resistance to the flow of fluids and/or drugs being given via the IV catheter. There is little evidence from human patients that the type of dressing significantly reduces the incidence of catheter infection compared with catheters left exposed and kept clean and dry.[9] Furthermore, transparent “breathable” dressings have not been shown to offer significant advantage for human patients over gauze dressings unless impregnated with chlorhexidine (e.g., 3M™ Tegaderm™ CHG Chlorhexidine Gluconate I.V. Securement Dressing), and do not adhere effectively to animal skin.[10,11]

Although peripheral IV catheters are typically removed after 72 hours in human patients, there is no evidence in veterinary medicine that this is necessary if there are no identified complications.[12] Venous catheters should be flushed and inspected every 6 hours and rewrapped every 12 hours. If the catheter site becomes wet or soiled, the catheter should be removed, and another placed aseptically. While monitoring an indwelling catheter, record the date, catheter site and type, and comments daily. Catheters used for continuous fluid therapy do not need to be flushed routinely. Unused IV catheters should either be removed or flushed every 6 hours to maintain patency. This should be done using 0.9% sodium chloride with or without heparin. There is little evidence to support the use of heparinised saline over normal saline to maintain patency of venous or arterial catheters.[13,14]

Equipment Preparation

In order to reduce the risks of anaesthesia, foreseeable problems must be prevented. Patient related problems will be identified by the preanaesthetic evaluation. Checking equipment will help to identify and prevent problems due to technical faults or errors. It is critical that all members of the anaesthetic team (both veterinarians and nursing

personnel) are familiar with the anaesthetic machine, monitoring systems and related equipment. All members of the team should be able to troubleshoot common equipment dysfunctions.

Equipment Checklist

The checks outlined below should be carried out prior to each anaesthetic procedure. Servicing of anaesthesia delivery systems should be performed regularly, at specified intervals in accordance with the manufacturer's documented service requirements. Servicing requirements should be noted on a label and displayed on the equipment in a prominent position. The label should list the date of the most recent service and the due date for the next service.

Anaesthetic Machine

- For piped gases: Connect the oxygen, nitrous oxide or medical air to the correct gas pipelines and perform a 'tug test' (pull to check the integrity of the connection to the anaesthetic machine). Check and ensure that there is a second supply of oxygen for emergencies (a full oxygen cylinder safely secured near the anaesthetic machine).
- For oxygen cylinders: Check there are two oxygen cylinders accessible, one 'in use' cylinder and one 'full' reserve cylinder.
- Turn the oxygen flowmeter on to maximum and off again, check the ball/bobbin is rising/rotating freely respectively. Repeat this step for nitrous oxide and medical air if required.
- Check the machine for leaks: Turn on the oxygen to 4 L/min. Occlude the fresh gas outlet and check that the oxygen flowmeter bobbin/ball drops. This should be performed with the vaporiser you wish to use in place on the back bar and repeated if a different vaporiser is placed on the anaesthetic machine. *NB: Refer to anaesthesia machine's documentation for specific leak-checking procedures applicable to that machine.*
- Operate the emergency oxygen bypass control (flush). Ensure flow occurs and ceases when control is released.
- Switch the oxygen off.

Vaporiser

- Check that the vaporiser(s) for the required volatile agent(s) is/are sitting correctly on the back bar of the anaesthetic machine and locked in place.
- Check that the dial turns fully through the full range. Turn dial off.
- Check that the vaporiser(s) are adequately, but not over, filled and that the filling port is tightly closed.

Scavenging

- For active scavenging systems: Check that the scavenging pipe is connected to the scavenging system outlet. Ensure that the active scavenging system is turned on.
- Ensure that the end of the scavenging system is correctly attached to the breathing system.
- For passive scavenging systems: Ensure that the scavenging hose is connected to a charcoal absorber. The charcoal absorber should be weighed weekly to assess the degree of absorption and replaced when the maximum weight is reached (as recommended by the manufacturer).

Breathing system

- Select the appropriate breathing system (see Selecting Breathing Systems and Fresh Gas Flow Rates) and check that the reservoir bag is an appropriate volume for the patient (tidal volume [10 to 20 mL per kg] x 5).

- Connect the breathing circuit to the fresh gas outlet, ensuring all connections are secured tightly.
- Connect the scavenging tube to the circuit.
- Perform a leak test by closing the APL (Adjustable Pressure Limiting/"pop-off") scavenging valve, and turning on the oxygen flowmeter.
- Occlude the "to the patient" end of the circuit with your hand. Let the reservoir bag fill completely with oxygen.
- Turn off the flowmeter when the pressure gauge registers 30 cm H_2O or when the reservoir bag is fully inflated and slightly distended if the anaesthetic machine does not have a pressure gauge.
- If the pressure holds steady the system is leak-free, but if the pressure drops (or the bag deflates when squeezed) the system will need to be checked for leaks.
- **Open the APL valve** to depressurise the system prior to removing your hand from the patient end of the breathing system.
- When using a rebreathing circuit, the soda lime should be checked for signs of exhaustion (according to manufacturer's recommendations) and the unidirectional valves checked to ensure they are moving freely.
- When using a non-rebreathing circuit (e.g. Bain) check the integrity of the inner tube by performing an occlusion test of the inner tube. Occlusion of the inner tube while oxygen is flowing should cause the flowmeter bobbin/ball to drop temporarily.
- All circuits with adjustable scavenging valves should be checked to ensure the valves open and close; **then leave the valve open** prior to use.

<u>Ventilator</u>

- Check that the ventilator is configured correctly for its intended use.
- Ensure that the ventilator tubing is securely attached.
- Set the controls for use and ensure that adequate pressure is generated during the inspiratory phase.
- Check that alarms are working and correctly configured.
- Check that the pressure relief valve functions correctly at the set pressure.
- Two-bag test: A two-bag test should be performed after the breathing system, vaporisers and ventilator have been checked individually.

    1. Attach the patient-end of the breathing system (including angle piece and filter) to a test lung or rebreathing bag.
    2. Set the fresh gas flow to 5 L/min and ventilate manually. Check the whole breathing system is patent and the unidirectional valves are moving (if present).
    3. Check the function of the APL valve by squeezing both bags.
    4. Turn on the ventilator to ventilate the test lung. Turn off the fresh gas flow or reduce to a minimum. Open and close each vaporiser in turn. There should be no loss of volume in the system

Breathing systems should be protected with a test lung or rebreathing bag when not in use to prevent intrusion of foreign bodies. Conduct a check before every procedure.

What if a leak occurs?

- **Reservoir bag** - If leak occurs, replace reservoir bag.
- **Breathing circuit** - Install new breathing circuit or obstruct inhalation/exhalation openings to determine if leak originates from the breathing circuit.
- **Vaporiser fittings** - Verify fittings and tubing are securely attached.
- **Absorber canister gaskets** - Check for loose absorbent grains between canister housing gaskets and verify that the canister is seated properly.
- **APL Valve** – Remove valve and obstruct opening to determine if leak originates from the APL valve.

Ancillary equipment

Endotracheal tubes

- Select the appropriate size (diameter and length) and type of tube. Prepare three tubes of different sizes for each patient.
- Check the cuffs for leaks. Inflate the cuff and leave it inflated for a few minutes to detect leaks. Deflate cuffs.
- Ensure that the lumen of the tube is clean and free of debris.

Laryngoscope

- Attach the appropriate length and type of blade to the handle. Typically, a Miller style straight blade is most appropriate for small animal patients.
- Open/engage the blade to ensure that the light source is working properly.
- Laryngoscope blades should be washed between patients.

Monitoring equipment

- Check that the monitor is switched on and in the correct “work” mode rather than standby.
- Check the required monitoring cables are correctly connected.
- Check that the available monitoring probe types and sizes are suitable for the patient.

Cleaning and reuse of anaesthetic equipment

- Currently, there are no official guidelines for veterinary anaesthesia related to the cleaning and reusing of anaesthesia breathing circuits, endotracheal tubes and ancillary equipment between patients, with the cost-benefit ratio of using sterilised breathing systems or filters remaining questionable. We recommend that all anaesthesia equipment that has direct patient contact (e.g. endotracheal tubes, laryngoscope blades, pulse oximeter clips, oesophageal stethoscope, thermometer, etc.) should be thoroughly cleaned in mild soap and water, rinsed thoroughly, dried and disinfected between patients.
- Anaesthetic circuits and endotracheal tubes be routinely changed, ideally between individual patients but at a minimum on a daily basis. If visibly contaminated, circuits, endotracheal tubes and ancillary equipment should be changed and thoroughly cleaned between patients. Circuits and endotracheal tubes used for highly infectious cases should be safely discarded.
- Regular culturing of breathing circuits, absorbent canisters, and rebreathing bags should be performed for both bacterial and fungal contaminants.

NB: Vaporiser In Circuit (VIC) Systems

Most modern vaporisers are agent-specific, concentration-calibrated, out of the circuit, and high-resistance vaporisers that are compensated for temperature, flow, and back-pressure. Non-precision, in the circuit vaporisers are still occasionally found in veterinary practice, however, without proper specific training and inhalant anaesthetic agent monitoring these vaporisers pose unnecessary risks during anaesthesia and should not be used.[15] All vaporisers mentioned within this document will, unless otherwise stated, refer to vaporiser agent-specific, concentration-calibrated, temperature-compensated, out of the circuit (VOC) vaporiser systems.

Selecting Breathing Systems and Fresh Gas Flow Rates

Anaesthetic gas exits the anaesthesia machine (via the common gas outlet) and then enters a breathing circuit. The function of the circuit is to deliver oxygen and anaesthetic gases to the patient and to eliminate carbon dioxide. The carbon dioxide may be eliminated by gas inflow (**non-rebreathing systems**) or by soda lime absorption (**rebreathing systems**).

Breathing systems have been classified using various schemes without consensus and uniformity (i.e., open, semi-open, and semi-closed). This inconsistency in nomenclature found within the literature and teaching institutions is a source of confusion and inconsistency. For this reason, it has been suggested that these terms be abandoned. For clarity it is easiest to classify the breathing system into one of two groups: those designed for rebreathing of exhaled gases (**rebreathing systems**) and those designed to be used under circumstances of minimal to no rebreathing (**non-rebreathing systems**).[16] It has also been suggested that in addition to describing the design of the breathing system, the fresh gas flow (in mL/kg/min) needed to prevent or enable rebreathing to occur should be stated to fully describe how the system is being used.[17]

The amount of rebreathing that occurs with any particular anaesthetic breathing system depends on four factors:

- the design of the individual breathing circuit,
- the mode of ventilation (spontaneous or controlled),
- the fresh gas flow rate and
- the patient's respiratory pattern.

Circuits may eliminate rebreathing either by ensuring an adequate flow of fresh gas which flushes the circuit clear of expired alveolar gas, or additionally, in the case of a circle system, by the use of soda lime, which absorbs the carbon dioxide so that lower fresh gas flows may be used.

Rebreathing Systems

A rebreathing system allows for the rebreathing of the exhaled gases. The expired carbon dioxide is removed and the fresh gas mixture along with inhalant anaesthetic vapour is continually added. The components of a rebreathing system include: the fresh gas inlet; absorber circuit; manometer; rebreathing bag; hoses; Y-piece; unidirectional valves (inspiration and expiration); APL valve; and a scavenger system. These components increase the resistance to the movement of the gas mixture in the system as well as the total volume of the system compared to a non-rebreathing system. Human adult size rebreathing circuits are typically used for patients weighing > 10 kg. It is advisable that patients in the weight range 2.5-10 kg be placed on paediatric size rebreathing systems with a reduced circuit diameter. There is no minimum patient size for using a rebreathing system accepted among anaesthesiologists;

however, the minimum patient size is generally suggested as not less than 2.5 kg, when using a modern anaesthesia machine with light weight unidirectional valves. The following are recommendations for selecting fresh gas flow rates in rebreathing systems:

Following Induction (with an injectable agent)

For patients induced with an injectable anaesthetic agent and subsequently intubated and connected to an anaesthetic machine with a rebreathing system, the initial flow rates should be relatively high (**50-100 mL/kg/min**) to facilitate rapid inspired concentration increases in anaesthetic agent within the system and to replace the anaesthetic vapour that is dissolving into patient tissues during the initial uptake period of anaesthetic delivery (e.g., the first 10-20 min following induction).[15]

During Maintenance

Once the patient has reached a satisfactory depth of anaesthesia, the flow rate may be reduced to a maintenance level. Rebreathing systems require relatively lower flow rates compared with non-rebreathing systems during the anaesthetic maintenance period because carbon dioxide is removed from the expired gases, which are then returned to the patient. Provided that there are no leaks in the system and the carbon dioxide absorber is functional, the carrier gas and anaesthetic can be recycled continuously, with only a small amount of additional fresh gas required. Following the initial uptake period of anaesthetic delivery (e.g. the first 10-20 min following induction) flow rates are reduced.

Lower flow rates are beneficial in conserving moisture and heat. During this period flow rates of **20-50 mL/kg/min** are recommended for small animal patients.[15]

Flow Rates When Making Changes in Anaesthetic Depth: Higher fresh gas flow rates (50-100 mL/kg/min) are also recommended to facilitate more rapid equilibration of anaesthetic concentration within the breathing circuit, and ultimately the patient's depth if a patient is judged as being too "deep" or too "light", and the vaporiser dial setting has been adjusted accordingly.

Low-Flow Anaesthesia: The minimum safe fresh gas flow during maintenance supplies enough oxygen to satisfy the patient's metabolic oxygen consumption. To economise on gas use and waste during the maintenance period, the oxygen flow rate can be reduced to equal the metabolic oxygen requirement of the patient (5-10 mL/kg/min), plus enough additional gas flow to replace gases lost because of leaks in the circuit and/or via a side-stream gas analyser (See Appendix 1). The anaesthetist should be aware that at flow rates less than 250 mL/min, some precision vaporisers and flowmeters may not accurately deliver the dialled vaporiser concentration and oxygen flow rate.

NB: Specific technical training in this technique and/or the use of gas analysers to monitor inspired and expired oxygen and anaesthetic partial pressures is recommended.

During Anaesthetic Recovery/Emergence

Immediately after the vaporiser has been turned off, increase the fresh gas flow rate to **200-300 mL/kg/min** (or as close to non-rebreathing flow rates as possible). Then with the APL valve open, apply gentle pressure to evacuate the reservoir bag, repeating this process when it refills. Where such high flow rates are applied and the fresh gas flow rate matches or exceeds the patient's minute ventilation (tidal volume x respiratory rate), a rebreathing system can be made to function as a non/minimal-rebreathing system. This strategy will wash out waste anaesthetic gas, increase oxygen concentration in the breathing circuit, and hasten the

patient's emergence from anaesthesia. It is recommended that these high flow rates be maintained until the patient is extubated.

NB: See Appendix 2.

IMPORTANTLY the APL valve should be fully open at all times when the patient is spontaneously breathing and only closed temporally during manually (bag) assisted ventilation by the anaesthetist.

Non-Rebreathing Systems

The distinguishing feature of non-rebreathing circuits is that elimination of carbon dioxide is accomplished by removing all expired gases from the system and venting them to the atmosphere. This is normally achieved by using the fresh gas flow from the anaesthetic machine to direct the expired gases out of the circuit via a valve or other arrangement. In general, non-rebreathing systems provide good control of the inspired gas concentrations, since the anaesthetic vaporiser concentration setting closely matches the inspired isoflurane concentration at all times and fresh gas delivered from the anaesthetic machine is inspired in each breath. They are, however, less economical to use than rebreathing systems because the minute volume of ventilation (or more) must be supplied to the patient to prevent rebreathing, and they contribute more to the problem of atmospheric pollution with anaesthetic agents. Appropriate scavenging of anaesthetic waste gases is mandatory.

Non-rebreathing systems have traditionally been recommended for patients <5 kg, due to lower resistance during spontaneous breathing, less equipment deadspace, and smaller total circuit volume compared to adult rebreathing circuits.[15] However, by using newer paediatric and neonatal rebreathing systems, many of the aforementioned advantages of non-rebreathing systems are negated and it is possible to maintain patients <5 kg safely using rebreathing systems providing that the patient's tidal volume is sufficient to operate the unidirectional valves.[15]

There is no patient size recommendation for selecting a non-rebreathing, paediatric/neonatal rebreathing or adult rebreathing system that has been universally accepted among anaesthesiologists. It is however generally accepted that non-rebreathing systems are advisable for patients <2.5 kg. Patients ranging from 2.5-10 kg are well suited to paediatric/neonatal rebreathing systems; and patients >10 kg adult rebreathing systems.

The precise fresh gas flow rate to minimise the rebreathing of carbon dioxide in non-rebreathing systems differs with each individual patient and type of system. Ranges have been reported from 100-600 mL/kg/min for patients <7 kg. In general, an approximate range of 100-300 mL/kg/min with a minimum of 500 mL/min and a maximum of 3 L/min is recommended for patients <6.8 kg.[18] Continuous monitoring of expired carbon dioxide values (by capnography) is the ideal modality to determine an individual patient's fresh gas flow requirement.

NB: *IMPORTANTLY* the APL valve should be fully open at all times when the patient is spontaneously breathing and only closed temporarily during manually (bag) assisted ventilation by the anaesthetist.

Anaesthetic Waste Gas Scavenging

It is advised that practitioners familiarise themselves with relevant Australian state/territory-based legislation pertaining to occupational health and safety procedures or requirements, concerning control of waste anaesthetic gases. Veterinary Surgeons Boards within each state/territory can provide information regarding complying with all relevant standards.

For a detailed resource outlining recommendations for the control of waste anaesthetic gases in the workplace please refer to ***Commentary and recommendations on control of waste anesthetic gases in the workplace*** produced by The American College of Veterinary Anesthesia and Analgesia.

INDUCTION AND MAINTENANCE OF ANAESTHESIA

Team approach

All personnel involved in the delivery of anaesthesia should be fully informed of the details and plan related to the patient's scheduled procedure, relevant clinical history, and be aware of their individual role and responsibilities. It is also essential for all members of the anaesthesia and procedural team to be familiar and practised in the delivery of any potential emergency contingencies. All team members should be familiar with emergency protocols and supplies (including emergency drug doses calculated) for each individual patient prior to anaesthetic induction. A full description of the most current clinical cardiopulmonary resuscitation (CPR) guidelines, including new algorithms and drug dosing charts are available online: https://www.veccs.org/recover-cpr/

A diligent anaesthetist who is ready for varying contingencies can intercept developing problems before they reach the "crisis" stage. Diligent monitoring and timely, well thought out responses to changes in patient status are crucial in avoiding adverse events.

The maintenance period is made easier by prior planning. For those practices which may not have a dedicated anaesthetist, make sure that everything required for the procedure is organised prior to induction (e.g. surgical kit, suture material etc.) This will enable personnel responsible for the maintenance of anaesthesia to be able to spend more time dedicated to monitoring of the patient and less time spent performing other tasks during this period.

Surgical safety checklist

The management of risk and patient safety are major drivers in the implementation of surgical safety checklists. Checklists designed to ensure effective communication as well as improve collaboration and the delivery of patient care have been implemented in both human and veterinary healthcare sectors. The World Health Organization (WHO) surgical safety checklist has been shown to decrease mortality and complications and has been adopted worldwide.[19] The WHO surgical safety checklist has been modified to achieve veterinary relevance and successfully implemented by many practices and institutions. An example of one such checklist can be found in Appendix 3. Development of a surgical safety checklist is strongly recommended and should be tailored to individual needs and work practices. The development of such a surgical safety checklist should involve all members of the perioperative team in order to ensure engagement and compliance.

Patient preparation

Preparation of a patient for anaesthesia should include the following:

- Place an IV catheter (mandatory).
- Connect monitoring equipment and record the patient's pre-anaesthesia baseline blood pressure, heart rate, haemoglobin oxygen saturation (pulse-oximetry) and evaluate the ECG for any anomalies.
- Manage/optimise any detected cardiovascular or respiratory problems. ***Anaesthesia for elective procedures should be postponed until a patient is appropriately stabilised***. Cardiovascular stabilisation and ongoing maintenance of

patients, may include, but is not limited to:

- Administration of IV fluids. Hypovolaemic patients may require isotonic crystalloids, colloids, and/or hypertonic saline to optimise intravascular hydrostatic pressure, improve venous return, cardiac output, and improve tissue perfusion. Dehydration should be corrected in animals that are moderately to severely dehydrated, prior to anaesthesia if time permits. Only 75% to 80% of the dehydration deficit should be replaced during the 24 hours before anaesthesia to avoid fluid overload. The remaining fluid deficit due to dehydration can gradually be replaced over 24-48 hours after surgery.[20] In addition, preoperative volume loading of normovolaemic patients is not recommended, and, in humans, blood volume was unchanged after overnight fasting.[21-25]

- Managing cardiac arrhythmias (when feasible based on the patient's condition and the urgency of the procedure to be performed).

- Administration of blood products (when indicated and available). Anaemia and coagulation disorders can decrease the delivery of oxygen to tissues, and hypoalbuminaemia can alter drug transport and binding and effect fluid balance.

- Preoxygenate patient with 100% oxygen via a firm fitting face mask (demeanour permitting) for a minimum of 3 minutes prior to induction of anaesthesia. Preoxygenation for 3 minutes prior to induction of anaesthesia increases the time to desaturation of haemoglobin by a factor of 3 to 4 compared to non-preoxygenated patients and is particularly beneficial if a prolonged or difficult intubation is expected.[26] Removal of the rubber diaphragm from the facemask may increase patient tolerance.

- When the patient is as deemed to be as stable as possible, proceed according to the individual patient plan.

Anaesthetic Induction Phase

Anaesthesia is best achieved via IV administered induction agents carefully titrated slowly to effect. Mask or chamber inductions increase patient anxiety, are stressful on the cardiovascular and respiratory systems, delay airway control, and increase the risk of environmental and personnel exposure to gaseous anaesthetics.[27] It is strongly advised to reserve these techniques as options of "last resort" where other alternatives are not suitable.

Endotracheal Intubation

Equipment:

- 3-10 mL syringe for cuff inflation. It is recommended not to use greater than a 3 mL syringe for cuff inflation in cats.

- Endotracheal (ET) tubes in varying sizes - the correct ET tube should be measured from a point immediately rostral to the upper incisors to the thoracic inlet. The safest endotracheal tubes are the high-volume, low-pressure cuffs because they generate less pressure per given volume of inflation with less risk of tracheal trauma.

- Gauze squares (to grasp the tongue).

- Laryngoscope - While it is possible to intubate a cat without laryngoscopy, the visualisation is poor and therefore the risk of trauma is much greater. If there is not a laryngoscope in your practice, you may choose to purchase one as the time and stress it will save is invaluable. There are various models available from

most veterinary equipment suppliers. Alternatively, a good light source and a tongue depressor could be used (head torches can be useful).

- Facemask and oxygen supply.
- Lubricant (preferably sterile).
- Supplies to secure the endotracheal tube (i.e. rolled gauze to tie the tube to the upper jaw, lower jaw, or behind the head).
- Stylet - Recommended for use with small, delicate or flexible silicon tubes. Use a stylet with a flexible tip to avoid tracheal trauma. Once the stylet has been inserted slightly beyond the vocal chords, the tube should be advanced over the stylet rather than advancing a protruding stylet into the trachea, which may result in tracheal trauma, especially if the patient is lightly sedated and coughs or the neck is bent down during intubation.

Steps:

1. Ideally, patients should have been pre-oxygenated with an oxygen mask for a minimum of 3 minutes before intubation. Oxygen flow-by should always be available. ***Two team members should be involved in the procedure.***

2. Induction agent is administered until a suitable plane of anaesthesia is reached; manifested by a loss of righting reflex. Patients should typically be placed in sternal recumbency; however, team members should also prepare for and be able to intubate patients in lateral and dorsal recumbency. When intubating a patient in sternal recumbency, an assistant should place one hand behind the base of the head (occipital bone), while the opposing hand holds the maxilla (upper lips) and extends it in a dorsal direction. This should fully extend the head and neck as close to a horizontal position as possible.

3. When the patient has received an adequate amount of anaesthetic to sufficiently relax the muscles of the jaw and decrease the gag reflex; the anaesthetist should gently pull the tongue forward, and down between the lower incisors, thus pulling the lower jaw down and the epiglottis forward. This should be done in a gentle manner as the hypoglossal/glossopharyngeal nerves can be stretched leading to neuropraxia and problems with the tongue post-anaesthesia.

4. The blade of the laryngoscope should be placed just in front of the epiglottis and downward pressure applied to depress the tongue, so that the epiglottis is drawn forward and the soft-palate disengaged, allowing visualisation of the larynx. Assess the glottal diameter to determine an appropriate ET tube size. If possible, avoid directly contacting the epiglottis as this can cause damage leading to swelling and laryngeal spasm.

5. Spray the larynx with a single spray of lignocaine (ALL cats, AND dogs with a sensitive reactive/inflamed larynx). Test the spray before use to ensure the pump mechanism is loaded.

6. Allow at least 60-90 seconds for the local anaesthetic to take effect. Initially the larynx may spasm due to irritation from the spray but once the local anaesthetic has taken effect the larynx should relax. During the waiting period the tongue should be relaxed and the laryngoscope removed from the oral cavity.

7. Flow-by oxygen should be provided, and additional induction agent administered if further relaxation of the jaw or larynx is required.

8. Lightly lubricate the balloon of the selected ET tube avoiding the lumen and side-port (Murphy-Eye) of the ET tube to prevent obstruction. Cuff lubrication has been shown to reduce pulmonary aspiration.[28]

9. Reposition the patient, with their head and neck extended (to allow better visualisation and personal comfort this can be at more of an angle to the table than before). Use the laryngoscope as before to depress the tongue. With the laryngoscope in place, there should be a clear view of the arytenoids. Guide the ET tube over the epiglottis, aiming the bevel at the most ventral point of the laryngeal opening. Gently advance the tip of the ET tube through the laryngeal opening during inspiration (the laryngeal folds should abduct). A gentle twisting motion can be employed to aid passage of the tube. It is very important to visualise the tube passing between the arytenoids and into the glottal opening. It is not sufficient simply to pass the tube over the epiglottis as this will most likely result in the tube passing into the oesophagus. Other methods to assure proper placement include observing condensation in clear tubes as the patient breathes, auscultation of lung sounds and observation of a capnography trace.

10. Once correctly positioned, the ET tube should be secured by tying rolled gauze first around the tube then in a bow behind the canine teeth and around upper or lower jaw; or behind the base of the head in cats and brachycephalic dog breeds.

11. Connect the ET tube to the breathing circuit. Remember that the cuff can cause shear damage to the wall of the trachea if twisted, so during patient repositioning the ET tube should be disconnected from the breathing system.

12. Inflate the ET tube cuff to achieve an appropriate tracheal seal. An inflatable cuff has a pilot tube with a port for a syringe. It is ***important not to over-inflate*** the cuff as tracheal ischaemic necrosis or rupture can result.[29] To ensure correct inflation pressure, attach a syringe full of air to the pilot tube; inflate the lungs by forcing air down the endotracheal tube (by squeezing the reservoir bag) and listen for air escaping around the tube; slowly inject air into pilot tubing, inflating the cuff until you no longer hear air escaping. Once the cuff has sealed the trachea, the leak will cease, and an optimal airway pressure of 20-30 cm H_2O should be maintained (if an airway manometer is used).[30] Some pilot tubes have a valve, while others have to be clamped to keep the cuff inflated. A reliable method for ensuring that cuff pressures are within the recommended range is to use a cuff monitor to inflate cuffs. A cuff monitor is essentially a low-pressure manometer similar to those used for Doppler blood pressure measurement that is attached to the pilot balloon of the cuff and provides a measure of intra-cuff pressure.

13. Remember to assess your patient's vital parameters immediately after intubation.

Anaesthetic Maintenance Phase

Positioning

During anaesthesia patients should, if possible, be positioned in a normal physiological position. The head should be extended to promote a 'free' airway and reduce the likelihood of endotracheal tube kinking. The eyes should be protected from contact with abrasive surfaces and corneal lubricant should be applied following induction to protect the eyes from corneal ulceration.

Temperature Regulation

Anaesthesia invariably effects temperature

regulation. This will typically manifest as hypothermia, however, during warmer weather large hairy dogs may overheat. Small animals with large surface area to volume ratios lose heat rapidly under anaesthesia and sedation but all animals may develop clinically relevant hypothermia. Hypothermia has many physiological effects and will delay greatly a patient's recovery from anaesthesia.[31] Care should be taken to monitor body temperature and, when required, provide supplemental heating to maintain a patient's temperature to as close to normal during anaesthesia. Thermal support may include warm IV fluids, use of a fluid line warming device, insulation on the patient's extremities (e.g. bubble wrapping of the feet), heated conductive warming blankets, circulating warm-water blankets, and warm air circulation systems. Do not use supplemental heat sources that are not designed specifically for anaesthetised patients as they can cause severe thermal injury.[32]

Intravenous Fluids

The provision of fluids during anaesthesia, as well as the type and volume used, should be based on the individual patient's signalment, physical condition, and the length and type of procedure. The dogma of administering crystalloid fluids at 10 mL/kg/hr perioperatively, with higher volumes for anaesthesia-induced hypotension is not evidence-based and should be re-evaluated. Such high fluid rates are likely contributors to adverse patient outcomes, including increased body weight and lung water; abnormal pulmonary function; coagulation deficits; reduced gut motility; reduced tissue oxygenation; increased infection rate; decreased wound healing; and decreased PCV and TP.[33,34]

In the absence of evidence-based anaesthesia fluid rates for veterinary patients and taking into consideration the growing weight of evidence relating to the adverse effects of hypervolaemia, the authors suggest that rarely should there be a need to exceed 3 mL/kg/hr of balanced crystalloids in dogs and cats undertaking surgical procedures. Fluid volumes administered above this rate should be for the purpose of resuscitation (i.e. replacing blood volume as it is lost from the intravascular space).

Experimental data in isoflurane anaesthetised dogs, and clinical studies in people, suggest that crystalloid fluid replacement ratios from 4-5 mL or greater for each mL of lost blood (4:1 or 5:1) are required.[20,35-37]

Monitoring

Clinical observation and assessment by a vigilant anaesthetist are essential for safe patient care during the peri-anaesthetic period. During the maintenance period good monitoring is critical. The person monitoring the patient should be suitably trained and skilled and should not be the practitioner performing the procedure. Clinical monitoring should be supplemented with monitoring devices as necessary, to assist the practitioner responsible for the anaesthesia. The anaesthetist, whose sole responsibility is the provision of anaesthetic care for that patient, must be constantly present from induction of anaesthesia until the patient has recovered. In exceptional circumstances brief absences of the anaesthetist primarily responsible for managing anaesthesia may be unavoidable. In such circumstances, observation, including recording of observations of the patient and a plan for responding to significant changes in monitored physiological variables, must be temporarily delegated to a suitably trained and skilled colleague who is judged to be competent for the task.

Minimum Monitoring Standards

The monitoring of a patient undergoing any type of anaesthesia should include regular assessment and accurate recording keeping of physiological parameters, including circulation,

oxygenation, ventilation and temperature, as well as diligent post-anaesthetic care in the recovery period. The following guidelines for anaesthesia monitoring are adapted from existing standards developed by The American College of Veterinary Anesthesia and Analgesia (ACVAA).

Circulation

Objective: to ensure adequate circulatory function.

Methods:

- Palpation of peripheral pulse to determine rate, rhythm and quality, and evaluation of mucous membrane colour and capillary refill time (CRT).
- Auscultation of heart beat (stethoscope, oesophageal stethoscope or other audible heart monitor). Continuous (audible heart or pulse monitor) or intermittent monitoring of the heart rate and rhythm.
- Pulse oximetry to determine the % haemoglobin saturation.
- ECG continuous display for detection of arrhythmias.
- Blood pressure: either non-invasive (indirect), such as by oscillometry or Doppler ultrasonic flow detection; or invasive (direct) by arterial catheter connected to an aneroid manometer or to a transducer and oscilloscope.

Recommendations: Continuous awareness of heart rate and rhythm during anaesthesia, along with gross assessment of peripheral perfusion (pulse quality, mucous membrane colour and CRT) are mandatory. Blood pressure and ECG should also be monitored.

Oxygenation

Objective: to ensure adequate oxygenation of the patient's arterial blood.

Methods:

- Pulse oximetry (non-invasive estimation of haemoglobin saturation).
- Arterial blood gas analysis for oxygen partial pressure (PaO_2).

Recommendations: Assessment of oxygenation should be done whenever possible by pulse oximetry, with blood gas analysis being employed when necessary for more critically ill patients.

Ventilation

Objective: to ensure that the patient's ventilation is adequately maintained.

Methods:

- Observation of thoracic wall movement or observation of breathing bag movement when thoracic wall movement cannot be assessed.
- Auscultation of breathe sounds with an external stethoscope, an oesophageal stethoscope, or an audible respiratory monitor.
- Capnography (end-expired CO_2 measurement).
- Arterial blood gas analysis for carbon dioxide partial pressure ($PaCO_2$).
- Respirometry (tidal volume measurement).

Recommendations: Qualitative assessment of ventilation is essential as outlined previously, and capnography is required when using continuous mechanical/mandatory ventilation, with blood gas analysis as necessary.

Temperature

Objective: to ensure that patient does not encounter serious deviations from normal body temperature.

Methods:

- Rectal thermometer for intermittent measurement.
- Rectal or oesophageal temperature probe

for continuous measurement.

Recommendations: Temperature should be measured periodically (ideally at least every 15 minutes) during anaesthesia and recovery; and if possible checked within a few hours after return to the wards (more frequently if temperature abnormalities are present).

Record keeping

Objectives:

- To maintain a legal record of significant events related to the anaesthetic period.
- To enhance recognition of significant trends or unusual values for physiologic parameters and allow assessment of the response to intervention.

Recommendations:

- Record all drugs administered to each patient in the peri-anaesthetic period and in early recovery, noting the dose, time, and route of administration, as well as any adverse reaction to a drug or drug combination.
- Record monitored variables on a regular basis (minimum every 5 minutes) during anaesthesia. The minimum variables that should be recorded are heart rate and respiratory rate, as well as oxygenation status and blood pressure.
- Record heart rate, respiratory rate, and temperature in the early recovery phase.
- Any untoward events or unusual circumstances should be recorded for legal reasons, and for reference should the patient require anesthesia in the future.

RECOVERY PERIOD

Half of all anaesthesia related deaths occur during the recovery period (47% in dogs, 60% in cats), most frequently during the first 3 hours.[38] ***It is critical that patients are diligently monitored during this period*** to ensure a safe and comfortable recovery from anaesthesia. Extubation should ONLY be performed once the patient has regained a gag-reflex and no patient should be left intubated and unattended during the recovery period.

Patient care during recovery from anaesthesia

The frequency with which patients are monitored following anaesthesia will vary depending on the individual patients' procedure, signalment, disease process, pain profile, and on-going medical and/or surgical management requirements. The following recommendations are considered mandatory for the provision of care during the period following extubation or recovery from anaesthesia:

- Observation of respiratory pattern.
- Observation of mucous membrane colour and CRT.
- Palpation of pulse rate and quality.
- Measurement of body temperature, with appropriate warming or cooling methods applied as indicated. Do not use supplemental heat sources that are not designed specifically for anaesthetised patients as they can cause severe thermal injury.
- Re-apply eye lubrication during the recovery period until an appropriate blink reflex is present, particularly if an anticholinergic drug was administered.
- Manually express a patient's urinary bladder if distended to minimise any associated discomfort.
- Observation of any behaviour that indicates pain, emergence delirium or dysphoria, with appropriate

pharmaceutical intervention as necessary. Re-assess the patient's pain level and, if necessary, adjust the pain management plan.

- Other measurements as indicated by patient's medical status, e.g. blood glucose, pulse oximetry, PCV, TP, blood gases, etc.

Patient discharge

Discharge of patients having undergone anaesthesia should only occur after the patient is awake, aware, warm, comfortable and ambulatory (if the patient's condition allows). Evaluate the animal for its responses and its ability to interact safely with owners and maintain physiologic homeostasis.

Provide written instructions for owners, outlining the dose and potential side effects of analgesics and other medications to be given to the patient at home.

ACKNOWLEDGEMENTS

These guidelines were not peer reviewed.

CONFLICTS OF INTEREST

Dr Warne received personal fees from IDEXX during the conduct of the study. The authors report no other conflicts of interest or sources of funding for the work presented here.

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Appendix 1: Recommendations for performing low-flow anaesthesia

During Induction

- Use a total fresh gas flow that approximates minute ventilation and increase the fresh gas flow only if the measured (inspired) concentrations of oxygen and anaesthetic vapour are less than the set concentrations on the vaporiser.
- Decrease vaporiser setting as the difference between exhaled and inspired anaesthetic concentrations diminish. Monitor the exhaled concentration of anaesthetic vapour to ensure adequate anaesthetic depth.

During Maintenance (When expired anaesthetic concentration approaches the inspired concentration)

- Estimate patient oxygen consumption to be 5 mL/kg/min.
- Set total oxygen flow (oxygen + 21% of air flow if used) to equal estimated oxygen consumption.
- Add 200 mL/min to total oxygen flow if using a sidestream gas analyser that does not return sampled gas to the circuit.
- Add the volume of any estimated/ quantifiable leaks in the system to the total oxygen flow.
- Oxygen flow can be reduced in 50 mL/ min increments until the inspired oxygen concentration begins to decrease.
- Monitor inspired oxygen concentration and set low oxygen concentration alarm at desired level.
- Monitor exhaled anaesthetic vapour concentration to ensure adequate minimum alveolar concentration (MAC). Set alarm for desired level of expired vapour concentration.

During Recovery/Emergence (When expired anaesthetic concentration approaches the inspired concentration)

- Increase the total fresh gas flow to greater than 200 mL/kg/min (effectively converting the system to a non/minimal-rebreathing circle system). This will promote the scavenging of exhaled anaesthetic agents from the system.

NB: Specific technical training in this technique and/or the use of gas analysers to monitor inspired and expired oxygen and anaesthetic partial pressures is recommended.

Appendix 2

Quick Reference Chart: Oxygen flow rate guidelines for rebreathing systems (L/min)				
Weight (kg)	During induction, and changes in depth (50-100 mL/kg/min)	During maintenance (20-50 mL/kg/min)	Low-flow anaesthesia During maintenance (5-10 mL/kg/min)	During Emergence – Minimal rebreathing system† (200-300 mL/kg/min)
2.5	0.25-0.3	0.25	0.25*	0.5-0.8
5	0.3-0.5	0.25	0.25*	1.1-1.5
10	0.5-1	0.25-0.5	0.25*	2-3
15	0.8-1.5	0.3-0.75	0.25*	3-4.5
20	1.0-2	0.4-1	0.25*	4-5
25	1.3-2.5	0.5-1.25	0.25*	5
30	1.5-3	0.6-1.5	0.25-0.3*	5
40	2-4	0.8-2	0.25-0.4*	5
50	2.5-5	1.0-2.5	0.25-0.5	5
60	3-5	1.2-3	0.3-0.6	5
70	3.5-5	1.4-3.5	0.35-0.7	5
80	4-5	1.6-4	0.4-0.8	5
90	4.5-5	1.8-4.5	0.45-0.9	5
100	5	2.0-5	0.5-1.0	5
150	5	3.0-5	0.75-1.5	5
>150 kg Switch to large animal anaesthesia machine				

* At flow rates < 250 mL/min, some precision vaporiser and flowmeters may not accurately deliver the dialled vaporiser concentration and oxygen flow.

† Minimal rebreathing occurs only when the oxygen flow matches or exceeds the patient's minute ventilation.

Appendix 3

The University of Melbourne U-Vet Animal Hospital Surgical Safety Checklist

BEFORE INDUCTION

Surgeon: _____ **Anaesthetist:** _____ **Nurse:** _____

- SURGEON AND ANAESTHETIST +/- NURSE CONFIRM **PATIENT DETAILS**
  - ☐ Identity
  - ☐ Procedure
  - ☐ **CPR CODE** confirmed
  - ☐ **TPR** AND **BLOOD WORK** performed and reviewed
  - ☐ CONFIRM **SURGERY SITE** L / R, front/hind limb
  - ☐ Are there second surgery sites?
  - ☐ Is a scope or other imaging planned?
- ANAESTHESIA **SAFETY CHECK** COMPLETE
  - ☐ Anaesthesia machine checked
  - ☐ Monitoring equipment checked
  - ☐ Ventilator checked
- **PRE-EXISTING CONDITIONS** OR SURGICAL/ANAESTHESIA **RELATED RISKS** IDENTIFIED AND REVIEWED
  - ☐ Yes
  - ☐ No
- ARE **ANTIBIOTICS** NEEDED?
  - ☐ Yes, and ready to administer
  - ☐ No
- IS **PRE-OP OR INTRA-OP IMAGING** REQUIRED?
  - ☐ Yes, Request form submitted Yes No
  - ☐ No
- NURSE AND SURGEON
  - ☐ CONFIRM PATIENT **POSTITIONING**, SPECIAL **EQUIPMENT**, **IMPLANTS** and **OR SETUP**

Patient Identification Label

BEFORE SKIN INCISION

Surgeon: _____ **Anaesthetist:** _____ **Nurse:** _____

- ☐ CONFIRM ALL **TEAM MEMBERS** HAVE INTRODUCED THEMSELVES BY **NAME** AND **ROLE**
- SURGEON AND NURSE VERBALLY CONFIRM
  - ☐ Patient Identity
  - ☐ Procedure
  - ☐ Surgery site
- Ask ANAESTHETIST has **ANTIBIOTIC PROPHYLAXIS** been given at least 30 minutes previously?
  - ☐ Yes
  - ☐ No
- SURGEON AND ANAESTHETIST VERBALLY CONFIRM **ANTICIPATED CRITICAL EVENTS**
  - ☐ Blood test results reviewed
  - ☐ What are the critical steps?
  - ☐ What is the anticipated surgical time?
  - ☐ What is the anticipated blood loss?
  - ☐ Are blood products available if blood loss anticipated?
  - ☐ Are there any anaesthetic concerns?
- NURSE REVIEW
  - ☐ Has sterility (indicators) been confirmed?
  - ☐ Confirm implants / equipment in the OR
  - ☐ Surgical sponges counted and reported
- IS **IMAGING DISPLAYED IN OR?** SURGEON AND NURSE
  - ☐ Yes
  - ☐ No or Not applicable
- SURGEON ASKS ANAESTHETIST
  - ☐ Is anaesthetic depth adequate for surgery?
  - ☐ Is additional induction agent available?

BEFORE PATIENT LEAVES SURGERY

Surgeon: _____ **Anaesthetist:** _____ **Nurse:** _____

- NURSE CONFIRMS
  - ☐ Name of procedure has been recorded
  - ☐ Record if sterility was compromised, how?
  - ☐ Sponge count reported and correct
  - ☐ Any equipment concerns
- IS **POST-OP IMAGING** IS REQUIRED?
  - ☐ Yes
  - ☐ No
- CONFIRM **LOCATION OF RECOVERY**
  - ☐ Wards or ICU
- SURGEON AND ANAESTHETIST **REVIEW KEY CONCERNS FOR RECOVERY** AND **POSTOP MANAGEMENT PLAN**
  - ☐ Were there any anaesthetic concerns?
  - ☐ Were there any surgical concerns?
  - ☐ Analgesia
  - ☐ Antibiotics
  - ☐ Intravenous fluid rate
  - ☐ Particular medication
- SURGEON AND NURSE
  - ☐ Confirm responsibilities for submission of samples collected
  - ☐ CONFIRM **WHO WILL CALL THE OWNER** and **WHEN**

Prior to the patient progressing to the next stage, a minimum number of signatures must be obtained to ensure successful completion of each stage of the checklist. A signature from the surgeon and anaesthetist are mandatory at each stage.

DATE: _____

Abstracts

These Abstracts have been sourced from the following journals: Journal of the American Veterinary Medical Association, Journal of Exotic Pet Medicine, Journal of Feline Medicine and Surgery, Journal of Veterinary Emergency and Critical Care, Journal of Veterinary Internal Medicine, Journal of Small Animal Practice, Veterinary Surgery and New Zealand Veterinary Journal.

These abstracts are arranged under the headings Cats, Cats and Dogs, Dogs and Exotics, and within those headings by broad subject area.

CATS

Analgesia

Feline procedural sedation and analgesia: When, why and how

Simon BT and Steagall PV

J Feline Med Surg 22:1029-1045, 2020; https://doi.org/10.1177/1098612X20965830

Practical Relevance: Procedural sedation and analgesia (PSA) describes the process of depressing a patient's conscious state to perform unpleasant, minimally invasive procedures, and is part of the daily routine in feline medicine. Maintaining cardiopulmonary stability is critical while performing PSA.

Clinical challenges: Decision-making with respect to drug choice and dosage regimen, taking into consideration the cat's health status, behavior, any concomitant diseases and the need for analgesia, represents an everyday challenge in feline practice. While PSA is commonly perceived to be an uneventful procedure, complications may arise, especially when cats that were meant to be sedated are actually anesthetized.

Aims: This clinical article reviews key aspects of PSA in cats while exploring the literature and discussing complications and risk factors. Recommendations are given for patient assessment and preparation, clinical monitoring and fasting protocols, and there is discussion of how PSA protocols may change blood results and diagnostic tests. An overview of, and rationale for, building a PSA protocol, and the advantages and disadvantages of different classes of sedatives and anesthetics, is presented in a clinical context. Finally, injectable drug protocols are reported, supported by an evidence-based approach and clinical experience.

Cardiovascular System

Risk indictors in cats with preclinical hypertrophic cardiomyopathy: a prospective cohort study

Ironside VA et al

J Feline Med Surg 23:149-159, 2021; https://doi.org/10.1177/1098612X20938651

Objectives: This study aimed to identify indicators of the risk of progression of preclinical hypertrophic cardiomyopathy (HCM).

Methods: This was a prospective cohort study following a population of cats with preclinical HCM. Cats serially underwent physical examination, blood pressure measurement, blood sampling and echocardiography. Development of congestive heart failure (CHF), arterial thromboembolism (ATE) or sudden death (SD) were considered cardiac-related events. Associations between factors recorded at baseline, and on revisit examinations, and the development of a cardiac-related event were explored using receiver operator characteristic (ROC) analysis.

Results: Forty-seven cats were recruited to the study and were followed for a median period of 1135 days. Fifteen cats (31.9%) experienced at least one cardiac-related event; six CHF, five ATE and five SD. One cat experienced a cardiac-related event per 10.3 years of patient follow-up. Cats with increased left atrial (LA) size and higher concentrations of N-terminal pro B-type natriuretic peptide (NTproBNP) at baseline were more likely to experience an event. Cats with a greater rate of enlargement of LA size between examinations were also more likely to experience an event.

Conclusions and Relevance: Factors easily measured, either once or serially, in cats with preclinical HCM can help to identify those at greater risk of going on to develop clinical signs.

Digestive System

Anemia, iron deficiency, and cobalamin deficiency in cats with chronic gastrointestinal disease

Hunt A and Jugan MC

J Vet Intern Med 35: 172-178, 2021; https://doi.org/10.1111/jvim.15962

Background: Iron deficiency and cobalamin deficiency, as sequelae to chronic gastrointestinal (GI) disease, could result in anemia and increased morbidity in cats with chronic enteropathies.

Objective: To evaluate iron deficiency in cats with chronic GI disease and its relationship with hypocobalaminemia, anemia, and disease severity.

Animals: Twenty client-owned cats with primary GI disease.

Methods: Prospective, cross-sectional study. Cats were enrolled at the time of evaluation for chronic GI disease, after exclusion of comorbidities. CBC with reticulocyte indices, iron metabolism (serum iron and ferritin concentrations, total iron binding capacity [TIBC]), serum methylmalonic acid (MMA), cobalamin, and folate concentrations, pancreatic lipase and trypsin-like immunoreactivity, and disease severity were evaluated.

Results: Anemia (hematocrit <30%), iron deficiency, and cobalamin deficiency were diagnosed in 4/20, 7/20, and 8/20 cats, respectively. Hematocrit ($r_s = -0.45$; $P < 0.05$) and body condition score ($r_s = -0.60$; $P < 0.01$) negatively correlated with MMA. Median TIBC was lower in cats with increased vs normal MMA (218 µg/mL; range, 120-466 µg/mL vs 288 µg/mL; range, 195-369 µg/mL; $P = 0.02$). Hematocrit ($r_s = 0.51$; $P = 0.02$), reticulocyte MCV ($r_s = 0.52$; $P = 0.02$), reticulocyte hemoglobin content ($r_s = 0.71$; $P < 0.001$), and percent transferrin saturation ($r_s = 0.79$; $P < 0.0001$) positively correlated with serum iron concentration.

Conclusions and Clinical Importance: Functional iron deficiency was common in cats with chronic GI disease. Associations between hypocobalaminemia, iron parameters, and hematologic parameters warrant further investigation on the impact of iron deficiency on chronic GI disease morbidity in cats.

Feline comorbidities: What do we really know about feline triaditis?

Cerna P et al

J Feline Med Surg 22:1047-1067, 2020; https://doi.org/10.1177/1098612X20965831

Practical Relevance: Feline triaditis describes concurrent pancreatitis, cholangitis and inflammatory bowel disease (IBD). The reported prevalence is 17-39% in ill referral patients. While the aetiology is poorly understood, it is known to include infectious, autoimmune and physical components. What is not known is whether different organs are affected by different diseases, or the

same process; indeed, triaditis may be part of a multiorgan inflammatory disease. Feline gastrointestinal tract anatomy plays its role too. Specifically, the short small intestine, high bacterial load and anatomic feature whereby the pancreatic duct joins the common bile duct before entering the duodenal papilla all increase the risk of bacterial reflux and parenchymal inflammation. Inflammation may also be a sequela of bowel bacterial translocation and systemic bacteraemia.

Diagnostic Challenges: Cholangitis, pancreatitis and IBD manifest with overlapping, vague and non-specific clinical signs. Cholangitis may be accompanied by increased serum liver enzymes, total bilirubin and bile acid concentrations, and variable ultrasonographic changes. A presumptive diagnosis of pancreatitis is based on increased serum pancreatic lipase immunoreactivity or feline pancreas-specific lipase, and/or abnormal pancreatic changes on ultrasonography, though these tests have low sensitivity. Diagnosis of IBD is challenging without histopathology; ultrasound findings vary from normal to mucosal thickening or loss of layering. Triaditis may cause decreased serum folate or cobalamin (B12) concentrations due to intestinal disease and/or pancreatitis. Triaditis can only be confirmed with histopathology; hence, it remains a presumptive diagnosis in most cases.

Evidence Base: The literature on feline triaditis, pancreatitis, cholangitis and IBD is reviewed, focusing on histopathology, clinical significance and diagnostic challenges. Current management recommendations are provided. Further studies are needed to understand the complex pathophysiology, and in turn improve diagnosis and treatment.

The use of hydrolysed diets for vomiting and/or diarrhoea in cats in primary veterinary practice

Kathrani A et al

J Small Anim Pract 61:723-731, 2020; https://doi.org/10.1111/jsap.13214

Objective: To describe responses of cats prescribed a hydrolysed diet with or without concurrent medication for chronic vomiting and/or diarrhoea of undetermined aetiology.

Materials and Methods: Anonymised records of 512,213 cats under UK veterinary care in 2016 from the VetCompass database were searched using relevant terms for hydrolysed diets. The records of 5000 (90%) of 5569 cats with evidence of receiving a hydrolysed diet were randomly reviewed for gastrointestinal indication, prior and concurrent medication and response after hydrolysed dietary intervention. A poor response was defined as evidence of receiving antibiotic or glucocorticoid treatment for vomiting/diarrhoea at visits after the onset of the diet or death from gastrointestinal signs for at least 6 months follow-up.

Results: Of 977 cats prescribed a hydrolysed diet for chronic vomiting/diarrhoea, 697 (71%) were first prescribed the diet without concurrent antibiotics or glucocorticoids while 280 (29%) first received the diet with these medications. Thirty-four per cent of cats in the former group and 61% in the latter had a poor response. Cats older than 6 years and cats prescribed antibiotic and/or glucocorticoid for vomiting/diarrhoea before and concurrently with the diet had higher odds of poor response.

Clinical Significance: Although variations in our observations may reflect severity of signs or prescribing habits of primary-care veterinary surgeons, our study suggests there is merit in trialling a hydrolysed diet first as a sole therapy in cats with chronic vomiting/diarrhoea when diagnostic investigations do not reveal a cause, before resorting to antibiotic and/or glucocorticoid therapy for cases that respond poorly.

Endocrinology

Feline comorbidities: Recognition, diagnosis and management of the cushingoid diabetic

Cook AK and Evans JB

J Feline Med Surg 23:4-16, 2021; https://doi.org/10.1177/1098612X20979507

Practical Relevance: Diabetes mellitus (DM) is a common feline endocrinopathy, and is often driven by underlying insulin resistance with associated pancreatic beta (β)-cell dysfunction. Although spontaneous hyperadrenocorticism (HAC) with hypercortisolemia (hypercortisolism) is relatively uncommon in cats, it is a well-established cause of insulin resistance and is routinely associated with DM in this species.

Clinical Challenges: Many of the clinical signs associated with feline HAC are subtle and may be attributed to concurrent DM or the aging process. Failure to recognize HAC in the diabetic cat can impact patient wellbeing and predispose the patient to progressive compromise. Unfortunately, it can be difficult to establish a diagnosis of HAC, as test results may be influenced by poor diabetic regulation, and protocols are different to those used in canine patients. Treatment options depend on the underlying cause, and often require careful, ongoing assessment and modulation of both adrenal function and insulin requirements. However, various approaches have been shown to either improve glycemic control in cats with sustained insulin dependence, or facilitate diabetic remission.

Evidence Base: This review summarizes the current literature on feline HAC, with a particular focus on cats with concurrent DM. The clinical findings that suggest HAC are discussed, along with an outline of diagnostic options and their limitations. Published outcomes for various medical options, surgical procedures and radiation therapy are provided. The authors also share their thoughts on the safe and effective management of cats with HAC and DM, with an emphasis on the anticipation and recognition of changing insulin requirements.

Renal System

A review of phosphorus homeostasis and the impact of different types and amounts of dietary phosphate on metabolism and renal health in cats

Laflamme D et al

J Vet Intern Med 34:2187-2196, 2020; https://doi.org/10.1111/jvim.15961

Summary: Elevated concentrations of serum phosphate are linked with progression and increased case fatality rate in animals and humans with chronic kidney disease. Elevated concentrations of serum phosphate can be a risk factor for development of renal and cardiovascular diseases or osteoporosis in previously healthy people. In rodents, an excess intake of dietary phosphorus combined with an inverse dietary calcium : phosphorus ratio (<1:1) contributes to renal calcification. Renal injury also has occurred in cats fed experimental diets supplemented with highly soluble phosphate salts, especially in diets with inverse calcium : phosphorus ratios. However, not all phosphorus sources contribute similarly to this effect. This review, which focuses on cats, summarizes the published evidence regarding phosphorus metabolism and homeostasis, including the relative impact of different dietary phosphorus sources, and their impact on the kidneys. No data currently shows that commercial cat foods induce renal injury. However, some diets contain high amounts of phosphorus relative to recommendations and some have inverse Ca : P ratios and so could increase the risk for development of kidney disease. While limiting the use of highly soluble phosphates appears to be important, there are insufficient data to support a specific upper limit for phosphate intake. This review also proposes areas where additional research is needed in order to

strengthen conclusions and recommendations regarding dietary phosphorus for cats.

Risk factors associated with disturbances of calcium homeostasis after initiation of a phosphate-restricted diet in cats with chronic kidney disease

Tang P-K et al

J Vet Intern Med 35:321-332, 2021; https://doi.org/10.1111/jvim.15996

Background: Dietary phosphate restriction improves survival in cats with chronic kidney disease (CKD). However, feeding a phosphate-restricted diet may disrupt calcium homeostasis leading to hypercalcemia in some cats.

Objectives: To identify risk factors associated with increasing plasma total calcium (tCa) concentration after transition to a phosphate-restricted diet and to explore its role in CKD-mineral and bone disorder (CKD-MBD) in cats.

Animals: 71 geriatric (≥9 years) euthyroid client-owned cats with International Renal Interest Society (IRIS) stage 2 to 3 azotemic CKD.

Methods: Retrospective cross-sectional cohort study. Changes in plasma tCa concentration in the first 200 days of diet transition were assessed using linear regression. Binary logistic regressions were performed to identify risk factors for increasing calcium concentration. Changes in clinicopathological variables associated with CKD-MBD over time were explored using linear mixed model and generalized linear mixed model analyses.

Results: Lower baseline plasma potassium (odds ratio [OR] = 1.19 per 0.1 mmol/L decrease; P = 0.003) and phosphate (OR = 1.15 per 0.1 mmol/L decrease; P = 0.01) concentrations remained independent risk factors for increasing plasma tCa concentration. Plasma creatinine (β = 0.069 ± 0.029 mg/dL; P = .02), symmetric dimethylarginine (β = 0.64 ± 0.29 μg/dL; P = 0.03), phosphate (β = 0.129 ± 0.062 mg/dL; P = 0.04), and ln[FGF23] (β = 0.103 ± 0.035 pg/mL; P = 0.004) concentrations had significantly increased rates of change in cats with increasing plasma tCa concentration over time.

Conclusion and Clinical Importance: Lower plasma potassium or phosphate concentrations or both at the time of transition of cats with CKD to a phosphate-restricted diet are independently associated with increased risk of an increase in plasma tCa concentration. Increasing plasma tCa concentration is associated with progression of CKD.

Plasma symmetric dimethylarginine and creatinine concentrations and glomerular filtration rate in cats with normal and decreased renal function

Brans M et al

J Vet Intern Med 35:303-311, 2021; https://doi.org/10.1111/jvim.15975

Background: Glomerular filtration rate (GFR) is the gold standard in assessing renal function but is impractical. Serum creatinine (sCr) has limited sensitivity in identifying early chronic kidney disease (CKD), whereas symmetric dimethylarginine (SDMA) has been commercialized as more accurate biomarker. Studies comparing SDMA and sCr with GFR in cats are limited.

Objectives: To further investigate the diagnostic performance of SDMA in nonazotemic and azotemic cats.

Animals: 49 client-owned cats: 17 cats with CKD, 15 cats with diabetes mellitus (DM), and 17 healthy cats.

Methods: Retrospective study using spare blood samples from cats with documented sCr and GFR results for SDMA analysis. Diagnostic performances of SDMA and sCr

were evaluated using correlation coefficients, sensitivities, specificities, and receiver operator characteristic curves.

Results: Compared to healthy cats and cats with DM, CKD cats had significantly higher SDMAplasma (26.7 ± 9.9 µg/dL) and sCr (249.7 ± 71.6 µmol/L [2.8 ± 0.8 mg/dL]; both $P < 0.001$) values. SDMAplasma ($\tau B = -0.57$; $P < 0.001$) and sCr ($\tau B = -0.56$; $P < 0.001$) were significantly correlated with GFR. SDMAplasma ($\tau B = 0.52$; $P < 0.001$) had a significant relationship with sCr. SDMAplasma and sCr had similar sensitivity (76%-94% and 71%-88%, respectively) in detecting reduced renal function. Creatinine had higher specificity (94%-96%) than SDMAplasma (75%-76%) ($P < 0.05$).

Conclusion and Clinical Importance: In this study of azotemic and nonazotemic cats, SDMA was a reliable marker to identify decreased GFR. However, superiority of SDMA over sCr could not be confirmed.

Clinicopathologic and pathologic characteristics of feline proteinuric kidney disease

Rayhel LH et al

J Feline Med Surg 22:1219-1229, 2020; https://doi.org/10.1177/1098612X20921056

Objectives: The aim of this study was to describe the causes, clinicopathologic features and outcomes of feline protein-losing nephropathy (proteinuria secondary to glomerular disease [PLN]).

Methods: Kidney biopsy/necropsy samples from proteinuric cats submitted to the International Veterinary Renal Pathology Service were retrospectively reviewed. Diagnoses based on histopathology were categorized by primary disease compartment. Clinicopathologic variables at diagnosis, development of hypoalbuminemia, anemia, hypertension, azotemia and effusion/edema, and survival were compared between cats with immune-complex glomerulonephritis (ICGN) and other causes of PLN.

Results: Fifty-eight percent (n = 31/53) of proteinuric cats had ICGN and 74% (n = 31/42) of cats with PLN had ICGN. Cats with glomerular diseases other than ICGN had a higher median urine protein:creatinine ratio than ICGN cats (14.5 vs 6.5; $P < 0.001$). Onset of PLN occurred at a young age; median age at diagnosis was 3.5 years in ICGN cats vs 1.3 years in cats with other glomerular diseases ($P = 0.026$). Development of complications such as hypoalbuminemia, anemia, hypertension, azotemia and effusion/edema were common, regardless of the cause of PLN, and were not different between ICGN and cats with other glomerular diseases. Male cats were over-represented in the ICGN group ($P = 0.003$). Median survival time (MST) for all cats with PLN was 94 days (range 3-1848 days). Survival was not different between cats with ICGN and cats with other glomerular diseases. MST in ICGN cats that developed effusion was shorter (94 days) than cats that did not (700 days; $P = 0.035$). MST in IGCN cats that received immunosuppressive medications was longer (244 days) than cats that did not (17 days, $P = 0.039$).

Conclusions and Relevance: Taken together, these data suggest that clinical suspicion for glomerular proteinuria should increase in young, male cats with higher degrees of proteinuria, and immune-mediated disease is common. Further studies are needed to determine the effect of immunosuppression on morbidity and mortality in cats with ICGN.

Effects of low-dose meloxicam in cats with chronic kidney disease

KuKanich K et al

J Feline Med Surg 23138-148, 2021; https://doi.org/10.1177/1098612X20935750

Objectives: Meloxicam therapy may benefit cats with degenerative joint disease, and retrospective studies suggest it could slow

kidney disease progression and increase survival. This study aimed to prospectively evaluate the renal effects of low-dose meloxicam treatment (0.02 mg/kg/day) over 6 months in cats with chronic kidney disease (CKD).

Methods: Twenty-one cats with stable International Renal Interest Society stage 2 or 3 CKD were recruited and randomized to placebo or meloxicam groups. Cats were evaluated at baseline and at 1, 3 and 6 months, including blood pressure, chemistry, symmetric dimethylarginine (SDMA), glomerular filtration rate (GFR), urinalysis, urine protein:creatinine ratio (UPC), urine transforming growth factor-beta (β):creatinine ratio, urine clusterin, urine cystatin B and serum inosine.

Results: No statistical difference was observed in systolic blood pressure, blood urea nitrogen, creatinine, SDMA, GFR, urine transforming growth factor-β:creatinine ratio, urine clusterin, urine cystatin B or serum inosine in cats receiving meloxicam vs placebo. Mean UPC was greater in the meloxicam group (0.33) than the placebo group (0.1) at 6 months ($P = 0.006$). Four cats had meloxicam discontinued owing to potential (mainly gastrointestinal) adverse effects.

Conclusions and Relevance: No decline in renal excretory function was observed when meloxicam was administered to cats with CKD. However, gastrointestinal adverse effects were observed, and cats that received meloxicam had greater proteinuria at 6 months than cats that received placebo. As proteinuria is associated with negative outcomes (progression of azotemia and hypertension) in cats with CKD, this finding suggests that meloxicam should be used with caution in cats with CKD and UPC monitored. Until further research is available, clinicians should weigh the risk of potential increased proteinuria against quality of life benefits when considering meloxicam for analgesia in cats with renal disease.

Factors affecting survival to discharge in 53 cats diagnosed with uroabdomen: a single-centre retrospective analysis

Hornsey SJ et al

J Feline Med Surg 23:115-120, 2021; https://doi.org/10.1177/1098612X20932267

Objectives: The aim of this study was to assess outcomes in cats diagnosed with uroabdomen at a single referral centre.

Methods: Fifty-three cats diagnosed with uroabdomen at a veterinary teaching hospital were identified between June 2003 and September 2016. Data collected included signalment, presenting signs, aetiology, location of rupture, presence of concurrent injury, outcome of urine culture, presence of uroliths and packed cell volume (PCV)/creatinine/potassium levels at presentation. Cats managed medically and surgically were included, and the use of urinary catheters, cystotomy tubes and abdominal drains were recorded. It was determined if patients survived to discharge or if they were euthanased or died.

Results: Seventy-four percent ($n = 39$) of cats survived to discharge. Elevations in creatinine ($P = 0.03$) were shown to be significantly correlated with survival to discharge. Sex, age, location of rupture, presence of uroliths, outcome of urine culture, presence of concurrent injury, potassium at presentation and PCV at presentation were not associated with survival to discharge. There was no difference in survival between cats that were medically or surgically managed.

Conclusions and Relevance: Cats that develop uroabdomen have a good chance of survival. Electrolyte and biochemistry

values should be assessed at the time of presentation, in addition to the presence of concurrent injury.

Survival and complications in cats treated with subcutaneous ureteral bypass

Kulendra NJ et al

J Small Anim Pract 62:4-11, 2021; https://doi.org/10.1111/jsap.13226

Objectives: To report the complications and factors affecting outcome for cats following placement of a subcutaneous ureteral bypass (SUB™).

Materials and Methods: In this retrospective study, complications, the presence of a urinary tract infection and survival time were recorded following subctutaneous ureteral bypass placement. Factors affecting survival time were assessed using a Kaplan Meier curve and log rank test.

Results: 95 cats had 130 subcutaneous ureteral bypasses placed. Ten cats did not survive to discharge. Forty cats died or were euthanised after discharge (42%); the median survival time of these cats was 530 days (range 7 to 1915). Minor complications occurred in 18 cats (19%) and major complications occurred in 46 cats (48%), the majority of which were after hospital discharge. Twenty-seven cats were diagnosed with a urinary tract infection (UTI) post-operatively. A significant association between long-term survival and creatinine at presentation was identified. The median survival time for cats presenting with creatinine concentration ≥440 µmol/L (International Renal Interest Society stage acute kidney injury (AKI) 4 and 5) was 530 days (95% CI 273-787 days), compared to a median survival time of 949 days (95% CI 655-1243 days; Log Rank P=0.024) for those cats presenting with creatinine <440 µmol/L (International Renal Interest Society stage AKI 1-3).

Clinical Significance: In this population of cats, subcutaneous ureteral bypass placement was associated with an approximately 10% in-hospital mortality and a high complication rate. Most complications were manageable, resulting in an overall median survival time of over 2 years.

Transfusions

Transfusion transmissible pathogens are prevalent in healthy cats eligible to become blood donors

Mesa-Sanchez I et al

J Small Anim Pract 62: 107-113, 2021; https://doi.org/10.1111/jsap.13257

Objectives: This study aims to determine the prevalence of subclinical infectious agents considered core pathogens for worldwide screening in healthy, client-owned, indoor cats eligible to become blood donors in Spain and Portugal.

Materials and Methods: Blood samples of healthy, indoor, domestic cats selected to be potential blood donors were tested for feline leukaemia virus antigens, feline immunodeficiency virus antibodies and polymerase chain reactions for Mycoplasma haemofelis, Candidatus Mycoplasma haemominutum, Candidatus Mycoplasma turicensis, feline leukaemia virus provirus, Leishmania spp. and Bartonella spp. Not all donors were tested for all agents.

Results: Overall, 5105 healthy indoor cats were tested and 8.1% (414/5105) had at least one subclinical infectious agent that is transmissible through blood product transfusion. 1.5% (77/5105) were positive for feline leukaemia virus antigens and 2.9% (148/5105) were positive for feline immunodeficiency virus antibodies, therefore they were excluded as donors. The overall prevalence of haemoplasmas in feline leukaemia virus and feline immunodeficiency

virus SNAP-negative feline blood donors was 3.7% (181/4880) [1.3% (63/4880) were positive for Mycoplasma haemofelis; 2.3% (112/4880) to Candidatus Mycoplasma haemominutum and 0.12% (6/4880) to Candidatus Mycoplasma turicensis]. The prevalence of feline leukaemia provirus was 5.2% (9/173) and of Bartonella spp. was 0.2% (2/1122). None of the 173 cats were positive for Leishmania spp.

Clinical Significance: The prevalence of many transfusion transmissible pathogens was relatively high in this healthy, client-owned, indoor cats eligible to become blood donors. Performing an extended screening panel that includes at least polymerase chain reactions for Mycoplasma haemofelis, Candidatus Mycoplasma haemominutum, Candidatus Mycoplasma turicensis, feline leukaemia virus provirus, and Bartonella spp., in addition to feline leukaemia virus antigens and feline immunodeficiency virus antibodies, is recommended in pet blood banks from analogous regions.

CATS AND DOGS

Abdominal cryptococcosis in dogs and cats: 38 cases (2000-2018)

Johnston L et al

J Small Anim Pract 62: 19-27, 2021; https://doi.org/10.1111/jsap.13232

Introduction: To report the clinical presentation, laboratory and imaging findings, treatment and outcome of abdominal cryptococcosis in dogs and cats in Australia.

Materials and Methods: Canine and feline cases from Australia were retrospectively identified (2000 to 2018) via laboratory and referral centre searches for abdominal cryptococcosis diagnosed by cytology (needle aspirates) or histopathology (biopsy or necropsy) of abdominal organs/tissues. Signalment, presenting complaints, clinical signs, laboratory findings, medical imaging, latex cryptococcal antigen agglutination test (LCAT) titres, treatment and outcome data was collected.

Results: Thirty-eight cases were included (35 dogs, three cats) in the study. Median age of presentation was 2 years for dogs and 6 years for cats. Common presenting complaints included vomiting (23/38), lethargy (19/38) and inappetence/anorexia (15/38). Abdominal ultrasound (25/38 cases) revealed mesenteric and intestinal lesions in most of the cases. On surgical exploration, seven cases had an intestinal lesion associated with an intussusception. Nineteen cases had a pre-treatment LCAT performed, with a median initial titre of 1:2048 (range 1:2 to 65,536). Twenty-four cases (23 dogs, one cat) received treatment, either medical, surgical or both. Median survival time for cases with combined medical and surgical treatment, surgical treatment alone or medical treatment alone was 730, 140 and 561 days, respectively. Eleven remain alive at the time of follow up.

Clinical Significance: Abdominal cryptococcosis although rare should be a considered as a diagnostic possibility in an especially young dog presenting with gastro-intestinal signs. Older dogs can also present with this condition and should not be euthanised based on imaging alone due to the likenesses with neoplasia. With appropriate treatment and monitoring many dogs may have a prolonged survival period and some may be cured.

Period prevalence and mortality rates associated with hypocholesterolaemia in dogs and cats: 1,375 cases

Tan AWK et al

J Small Anim Pract 61: 669-675, 2020; https://doi.org/10.1111/jsap.13204

Objectives: To determine the period prevalence of hypocholesterolaemia and the associated mortality rates in dogs and cats at a university teaching hospital. The secondary

aim was to identify disease processes associated with hypocholesterolaemia.

Materials and Methods: Medical records over a 5-year period were reviewed to determine the severity of hypocholesterolaemia and its associated mortality rate. Medical records of animals with moderate to severe hypocholesterolaemia (<2.59 mmol/L in dogs, <1.81 mmol/L in cats) were analysed further. Animals with hospital-acquired hypocholesterolaemia were identified.

Results: Among 16,977 dogs and 3,788 cats that had at least one cholesterol measurement, the period prevalence of hypocholesterolaemia was 7.0% in dogs and 4.7% in cats. The mortality rate of hypocholesteraemic dogs and cats was 12% in both species which was significantly higher than that of animals with normal serum cholesterol. The degree of hypocholesterolaemia was significantly associated with mortality. Dogs, but not cats, with hospital-acquired hypocholesterolaemia had a higher mortality rate than those presenting with hypocholesterolaemia. Disease of hepatic, gastrointestinal and lymphoreticular systems were most commonly associated with hypocholesterolaemia, and infectious and neoplastic disease were the most commonly associated pathophysiologic processes in both species. Lymphoma was over-represented in dogs with neoplasia.

Clinical Significance: Hypocholesterolaemia is not a frequent abnormality but was associated with mortality in this study and may be a negative prognostic indicator. It is not known if hypocholesterolaemia is simply a marker for disease severity, or if it has active physiologic effects contributing to poor outcomes.

DOGS

Abdominal cavity

Diagnostic utility of abdominal ultrasonography for evaluation of dogs with nontraumatic hemoabdomen: 94 cases (2014-2017)

Cudney SE et al

J Am Vet Med Assoc 258:290-294, 2021; https://doi.org/10.2460/javma.258.3.290

Objective: To evaluate the utility of abdominal ultrasonography (AUS) to detect grossly evident masses in dogs with nontraumatic hemoabdomen.

Animals: 94 client-owned dogs.

Procedures: Electronic medical records from 2014 to 2017 were searched to identify dogs with nontraumatic hemoabdomen that had an AUS performed by a radiologist and subsequently underwent gross evaluation by surgery or necropsy. Ultrasonography, surgery, and histology reports were reviewed, and descriptive statistics were performed. Sensitivity of ultrasonography to detect grossly identifiable masses was calculated.

Results: Differences were identified between AUS and surgical or necropsy findings for 51 of 94 (54%) dogs. Splenic masses were most commonly identified as the cause of hemoabdomen. Sensitivity of AUS was 87.4%, 37.3%, and 31.3% for masses in the spleen, liver, and mesentery, respectively. Five dogs had more lesions identified with AUS than were found on gross evaluation; 0 of 6 dogs with peritoneal diffuse nodular metastasis had lesions detected by AUS.

Conclusions and Clinical Relevance: In this sample of dogs, the utility of AUS to detect grossly identifiable lesions in dogs with nontraumatic hemoabdomen was limited, with the highest and lowest sensitivity found for splenic masses and diffuse nodular metastasis, respectively.

Anaphylaxis-related hemoperitoneum in 11 dogs

Hnatusko AL et al

J Vet Emerg Crit Care 31:80-85, 2021; https://doi.org/10.1111/vec.13017

Objective: To describe the unique complication of hemoperitoneum associated with anaphylaxis.

Design: Retrospective case series from September 2012 to August 2017.

Setting: Two private emergency and specialty referral hospitals.

Animals: Eleven client-owned dogs diagnosed with anaphylaxis and hemoperitoneum upon presentation or referral.

Interventions: None.

Measurements and Main Results: Inclusion criteria included clinical signs consistent with anaphylaxis (hypotension, tachycardia, vomiting, diarrhea, weakness, collapse, with or without the presence of dermal signs) due to witnessed or unwitnessed presumed bee sting, an elevated alanine aminotransferase (ALT), performance of abdominal FAST (AFAST) examination with an abdominal fluid score, the sonographic presence of gallbladder wall edema, and hemoperitoneum.

All dogs (n=11) were managed medically without surgical intervention. 91% (n=10) of dogs survived to discharge.

Conclusions: Hemoperitoneum development can be seen with anaphylactic reactions, though the exact mechanism remains to be fully understood. Medical therapy is warranted and can be successful in these patients; surgery is not indicated to address hemoperitoneum.

Digestive System

Hyperlipasemia in critically ill dogs with and without acute pancreatitis: Prevalence, underlying diseases, predictors, and outcome

Prümmer JK et al

J Vet Intern Med 34:2319-2329, 2020; https://doi.org/10.1111/jvim.15902

Background: Hyperlipasemia is frequent in critically ill people without evidence of acute pancreatitis (AP), and has been associated with increased morbidity and mortality.

Objective: To evaluate the prevalence of hyperlipasemia at admission and development of hyperlipasemia during hospitalization in critically ill dogs, explore factors associated with hyperlipasemia, and evaluate association with outcome.

Animals: Critically ill, client owned dogs (n = 1360), presented on emergency and admitted to the intensive care unit, that had 1,2-o-dilauryl-rac-glycero-3-glutaric acid-(6′-methylresorufin) ester (DGGR) lipase activity measured within 24 hours of admission.

Methods: Retrospective cross-sectional study of clinical and laboratory records.

Results: The DGGR lipase activity was increased >3× the upper reference limit at admission in 216/1360 (16%) dogs, of which 70/216 (32%) had a clinical diagnosis of AP. Other primary conditions associated with hyperlipasemia were renal, endocrine, and immune-mediated diseases, and upper airway obstruction. Predictors of hyperlipasemia at admission were prior glucocorticoid administration, vomiting and abdominal pain, increased age, plasma bilirubin and creatinine concentrations, and decreased hematocrit. Of dogs with repeat measurements, 78/345 (23%) had significantly increased lipase during hospitalization, of which 13/78 (17%) had a clinical diagnosis of AP. Other primary conditions associated with in-hospital

hyperlipasemia were renal and immune-mediated disorders. Predictors of developing hyperlipasemia during hospitalization were hemodialysis events, increased plasma bilirubin and creatinine concentrations, and decreased hematocrit. Hyperlipasemia both at admission and during hospitalization was associated with longer hospitalization and higher mortality.

Conclusions and Clinical Importance: Significant DGGR-hyperlipasemia is frequent in critically ill dogs and associated with a variety of nonpancreatic conditions and negative outcome.

Endocrinology

Symmetric dimethylarginine concentrations in dogs with hypothyroidism before and after treatment with levothyroxine

Di Paola A et al

J Small Anim Pract 62: 89-96, 2021; https://doi.org/10.1111/jsap.13212

Objectives: To evaluate the serum symmetric dimethylarginine (SDMA) and serum creatinine concentrations in a population of hypothyroid dogs at the time of diagnosis and after treatment.

Materials and Methods: Serum SDMA and serum creatinine were measured in serum samples of 24 healthy dogs and 24 hypothyroid dogs, at the time of diagnosis (T0) and after supplementation with levothyroxine (T1).

Results: The mean SDMA concentrations (reference intervals [RI] <18 µg/dL and <14 µg/dL depending on the source) were 11.7 ± 3.5 µg/dL, 13.8 ± 3.1 µg/dL and 11.83 ± 2.87 µg/dL in healthy dogs, and in the hypothyroid dogs at T0 and T1, respectively. The SDMA concentrations were higher in the hypothyroid dogs at T0 in comparison with the healthy dogs. Of the hypothyroid dogs, 1 out of 24 had an SDMA concentration above 18 µg/dL and 12 out of 24 above 14 µg/dL at T0. At T1, none of the hypothyroid dogs had SDMA concentrations above 18 µg/dL and two of them had SDMA concentrations above 14 µg/dL. The serum creatinine concentration was higher in the hypothyroid dogs at T0 as compared to the healthy dogs. At T0, 8 out of 24 hypothyroid dogs had serum creatinine concentrations above the RI (>1.4 mg/dL). In all but one dog, serum creatinine normalised after treatment.

Clinical Significance: The SDMA and serum creatinine concentrations were higher in hypothyroid dogs at diagnosis as compared to healthy dogs. Serum creatinine concentrations were increased in one-third of the hypothyroid dogs and in the majority of cases normalised after levothyroxine supplementation. SDMA concentrations were rarely above the upper limit of the RI when the higest (<18 µg/dL) cut-off was employed. The diagnostic accuracy of SDMA in dogs with thyroid dysfunction requires additional evaluation.

Changes in systolic blood pressure in dogs with pituitary dependent hyperadrenocorticism during the first year of trilostane treatment

San José PG et al

J Vet Intern Med 35:130-141, 2021; https://doi.org/10.1111/jvim.15978

Background: Systemic hypertension (SH) is common in dogs and humans with hypercortisolism and can persist after treatment.

Objectives: To evaluate changes in prevalence of SH and systolic blood pressure (SBP) in dogs with pituitary-dependent hyperadrenocorticism (PDH) during the first year of trilostane treatment, its relationship with disease control and selected laboratory variables, and their response to antihypertensive treatment.

Animals: Fifty-one dogs with PDH treated with trilostane Q12h.

Methods: Prospective case series study. Dogs were evaluated at diagnosis (T0) and 1, 3, 6, and 12 months (T12). Dogs were classified as nonhypertensive (SBP < 160 mm Hg) or hypertensive (SBP≥160 mm Hg) and subclassified according to target organ damage (TOD) risk. Hypertensive dogs were treated with benazepril and, if control of SH was not achieved, amlodipine was added.

Results: Prevalence of SH decreased from T0 (36/51) to T12 (17/37; P = 0.01). Changes in SBP during the study were influenced by the risk of TOD at T0. In severely hypertensive (SBP ≥ 180 mm Hg) dogs, the decrease in SBP was more pronounced whereas in normotensive (SBP < 140 mm Hg) dogs SBP increased slightly (P = 0.00). Blood pressure was not associated with disease control. Antihypertensive treatment was needed in 31/51 dogs, and in 13/31 dogs additional SH control with amlodipine was required. One third of nonhypertensive dogs at T0 required treatment with benazepril because SH developed during follow-up.

Conclusions and Clinical Importance: In dogs with PDH, SBP should be measured at every visit, regardless of disease control or SBP at diagnosis. More than 1 drug may be necessary to manage SH in affected dogs.

Haematology

Effect of dilution of canine blood samples on the specificity of saline agglutination tests for immune-mediated hemolysis

Sun PL and Jeffery U

J Vet Intern Med 34:2374-2383, 2020; https://doi.org/10.1111/jvim.15945

Background: Saline agglutination tests (SATs) are widely recommended for diagnosis of immune-mediated hemolytic anemia in dogs, but there are frequent false-positive results.

Objectives: Specificity of SATs will improve at higher saline-to-blood ratios.

Animals: 150 dogs treated at a veterinary referral hospital with hematocrits ≤30%.

Methods: Prospective diagnostic accuracy study. Immune-mediated hemolysis (IMH) was considered present if a gel direct antiglobulin test (DAT) was positive and there was clinical evidence of hemolysis (n = 9), absent if another mechanism for anemia was identified and the DAT was negative or there was no hemolysis (n = 138), and if IMH status was unclear, dogs were excluded (n = 3). Saline agglutination tests were prepared at 1:1, 4:1, 9:1, and 49:1 saline-to-blood ratios, and microscopic agglutination was considered a positive result.

Results: Specificity for IMH increased from 29% (95% confidence interval 20-38) at a 1:1 dilution to 97% (93-99) at a 49:1 dilution. Sensitivity was 88% (47-100) at 1:1 and 4:1 dilutions and 67% (30-93%) at 9:1 and 49:1 dilutions. Diagnostic accuracy increased from 33% (24–42) at 1:1 dilution to 95% (90-98) at 49:1 dilution.

Conclusions and Clinical Importance: If performed using a 49:1 saline-to-blood ratio, SATs achieve high specificity for IMH. Based on a gold standard of positive DAT and evidence of hemolysis, lower saline-to-blood ratio results should not be used because false-positive results are common.

In vitro iatrogenic hemolysis of canine packed red blood cells during various rapid transfusion techniques

Weeks JM et al

J Vet Emerg Crit Care 31:25-31, 2021; https://doi.org/10.1111/vec.13020

Objective: To evaluate which rapid blood administration technique causes the least iatrogenic hemolysis in canine packed red blood cells (pRBCs) as determined by plasma free hemoglobin (fHb) and percent hemolysis (% hemolysis).

Design: Prospective in vitro randomized study.

Setting: Private referral center.

Animals: None.

Interventions: Thirteen units of canine pRBCs were divided equally into 5 aliquots, resulting in 65 trials. The aliquots of each unit were subjected to the following administration techniques: gravity-driven (control), an infusion pump at maximal rate, application of a pressure bag, manual compression, and syringe bolus. Plasma fHb and % hemolysis were recorded before and after each trial. Rate of administration (mL/s) was calculated for each method.

Measurements and Main Results: Compared to the control, there were no significant increases in % hemolysis or plasma fHb noted among any of the trial methods. The manual compression and syringe bolus methods resulted in the fastest transfusion rates, whereas the infusion pump was not faster than the gravity-driven method. Despite a storage time of ≤14 days, 15% of pRBC units had unsuitable (>0.8%) hemolysis before even being subjected to the trials.

Conclusions: Commonly used rapid infusion techniques in small animal transfusion medicine do not cause significant iatrogenic hemolysis of canine pRBCs in vitro, although a significant risk is present in stored blood. This suggests that if an expedited transfusion is needed, any method described in this study could be considered, although stored pRBCs should be tested for unsuitable levels of hemolysis prior to transfusion.

ORTHOPAEDICS

Influence of radiographic examination findings on recommendations made during routine clinical re-evaluation of dogs with uncomplicated tibial plateau leveling osteotomy

Alexander A et al

Vet Surg 50: 44-52, 2021; https://doi.org/10.1111/vsu.13533

Objective: To determine the influence of follow-up radiographic examination on recommendations made during routine clinical re-evaluation of dogs that had undergone uncomplicated tibial plateau leveling osteotomy (TPLO).

Study design: Retrospective multi-institutional case series.

Animals: Client-owned dogs (N = 1010) that underwent uncomplicated TPLO.

Methods: Records from 11 institutions were searched for dogs that had been treated with unilateral TPLO and had no history of postoperative complications before their routine follow-up examination. The frequency of change in further clinical recommendations resulting from client- or clinician-voiced concerns or radiographic abnormalities was investigated.

Results: Follow-up evaluation was performed at a median of 6 (range, 4-15) weeks after TPLO. Radiographic examination findings contributed to a change in recommendations in 4.15% (38/915) of dogs presented without client concerns and without abnormalities at orthopedic examination. Abnormal radiographic findings alone influenced the management of 3.76% (38/1010) of dogs. An association was detected between clinical features and radiological findings leading to a change in recommendations ($P < .0001$). Administration of analgesia at the time of follow-up was associated with radiographic abnormalities ($P = .017$) and change in postoperative plans ($P = .0007$).

Conclusion: Radiographic examination findings at follow-up did not influence the management of most dogs with uncomplicated TPLO.

Clinical significance: Radiographic examination findings are unlikely to

influence the treatment of dogs that seem to be recovering uneventfully from an uncomplicated TPLO without concerns from clients, analgesia, or abnormal findings on thorough orthopedic examination by a surgical specialist, at the time of the planned clinical re-evaluation.

RESPIRATORY SYSTEM

Nebulization of epinephrine to reduce the severity of brachycephalic obstructive airway syndrome in dogs

Franklin PH et al

Vet Surg 50: 62-70, 2021; https://doi.org/10.1111/vsu.13523

Objective: To determine the preoperative and postoperative effect of nebulized epinephrine on brachycephalic obstructive airway syndrome (BOAS) severity in dogs.

Study design: Prospective clinical study.

Sample population: Thirty-one client-owned pugs, French bulldogs, and English bulldogs with moderate to severe BOAS.

Methods: Whole body barometric plethysmography was used to determine BOAS severity (BOAS index; 0%-100%) prior to and after nebulization with 0.05 mg/kg epinephrine diluted in 0.9% saline preoperatively. The same protocol was repeated postoperatively (within 24 hours of surgery).

Results: Five dogs were excluded because they did not tolerate nebulization, and postoperative data were available for 13 dogs. Epinephrine nebulization resulted in a decreased BOAS index across all breeds of dog both before (9.6% [3.1% to -30.2%], n = 26) and after surgery (14.3% [0.9% to 24.3%], n = 13). The preoperative reduction in BOAS index was greater (17.3% [1.8% to -27.4%]) in dogs with a baseline BOAS index >70% (P = .006) and in pugs (16.9% [0.8% to -27.4%]) compared with French bulldogs (5.2% [3.1% to -30.2%], P = .03). Simple linear regression was used to identify a positive relationship between baseline BOAS index and reduction in BOAS index for pugs (n = 10, P = .001). Nausea was noted as a side effect in four dogs.

Conclusion: Nebulized epinephrine reduced the BOAS index of dogs in this study. This effect was clinically significant in preoperative dogs with a BOAS index >70% and in dogs recovering from surgery.

Clinical significance: This study provides evidence to support the use of nebulized epinephrine in the perioperative management of BOAS-affected dogs.

Utility of point-of-care lung ultrasound for monitoring cardiogenic pulmonary edema in dogs

Murphy SD et al

J Vet Intern Med 35:68-77, 2021; https://doi.org/10.1111/jvim.15990

Background: Point-of-care lung ultrasound (LUS) is an effective tool to diagnose left-sided congestive heart failure (L-CHF) in dogs via detection of ultrasound artifacts (B-lines) caused by increased lung water.

Hypothesis/Objectives: To determine whether LUS can be used to monitor resolution of cardiogenic pulmonary edema in dogs, and to compare LUS to other indicators of L-CHF control.

Animals: Twenty-five client-owned dogs hospitalized for treatment of first-onset L-CHF.

Methods: Protocolized LUS, thoracic radiographs (TXR), and plasma N-terminal pro-B-type natriuretic peptide were performed at hospital admission, hospital discharge, and recheck examinations. Lung ultrasound findings were compared between timepoints and to other clinical measures of L-CHF.

Results: From time of hospital admission to discharge (mean 19.6 hours), median number of LUS sites strongly positive for B-lines (>3 B-lines per site) decreased from 5 (range, 1-8) to 1 (range, 0-5; P < .001), and median total B-line score decreased from 37 (range, 6-74) to 5 (range, 0-32; P = .002). Lung ultrasound indices remained improved at first recheck (P < .001). Number of strong positive sites correlated positively with respiratory rate (r = 0.52, P = .008) and TXR edema score (r = 0.51, P = .009) at hospital admission. Patterns of edema resolution differed between LUS and TXR, with cranial quadrants showing more significant reduction in B-lines compared to TXR edema score (80% vs 29% reduction, respectively; P = .003).

Conclusions and Clinical Importance: Lung ultrasound could be a useful tool for monitoring resolution of pulmonary edema in dogs with L-CHF.

TOXICITIES

Retrospective evaluation of Vitis vinifera ingestion in dogs presented to emergency clinics in the UK (2012-2016): 606 Cases

Croft R et al

J Vet Emerg Crit Care 31:74-79, 2021; https://doi.org/10.1111/vec.13025

Objective: To assess key presenting signs in dogs following Vitis vinifera fruit (VVF) ingestion (grapes, raisins, currants, and sultanas), outcome, and the incidence of acute kidney injury (AKI).

Design: Retrospective study (2012-2016).

Setting: Out-of-hours clinics in the United Kingdom.

Animals: The study population included 606 dogs attending 53 emergency clinics across the United Kingdom following ingestion of VVF.

Interventions: None.

Measurements and Main Results: Vitis vinifera fruits were found in vomit after more than 12 hours after ingestion. There was an increased incidence in December. Serum creatinine or urea was measured in 338 dogs; all were within reference intervals. In this cohort of dogs with VVF ingestion, there is no evidence for significant AKI in 32 of 33 cases within 24 hours of admission where supportive measures were introduced and repeated assessments of creatinine concentration were performed.

Conclusions: All dogs survived to discharge. One out of 33 cases with repeated creatinine concentration developed IRIS AKI grade I within 24 hours following VVF ingestion, when current emergency treatment guidelines were followed in whole or part.

Retrospective evaluation of albuterol inhalant exposure in dogs: 36 cases (2007–2017)

Meroni ER et al

J Vet Emerg Crit Care 31:86-93, 2021; https://doi.org/10.1111/vec.13012

Objective: To describe the clinical features, clinicopathological features, treatment, and outcome of dogs presented for albuterol exposure.

Design: Retrospective case series from January 2007 to December 2017.

Setting: Tertiary veterinary facility.

Animals: 36 client-owned dogs presenting for known or suspected albuterol exposure secondary to chewing on albuterol metered-dose inhalers (MDIs).

Interventions: None.

Measurements and Main Results: All dogs presented with clinical signs attributable to albuterol exposure. The most common physical examination abnormality was sinus tachycardia, noted in 34 of 36 (94%) dogs.

Twenty-seven patients (75%) were admitted to the hospital for therapy, with a median length of hospitalization of 20.5 hours (16.75–24.5). Thirty-two of 36 dogs had serum electrolytes evaluated at admission, with 22 of 32 (69%) presenting with hypokalemia ([K+] < 3.62 mmol/L]). Hyperlactatemia ([lactate] > 2.80 mmol/L) was noted in 23 of 28 (82%) dogs. A negative correlation was found between serum lactate and potassium (r = –0.64, r2 = 0.40, P = 0.0003). Hyperglycemia ([glucose] > 6.44 mmol/L) was noted in 20 of 30 (67%) dogs. Beta antagonist therapy was utilized in 20 of 36 (56%) of dogs.

Conclusions: Although uncommon, albuterol intoxication can lead to significant clinical and electrolyte abnormalities. Albuterol-induced hypokalemia and associated tachyarrhythmias can be successfully managed, and albuterol intoxication has an excellent prognosis for survival to discharge. A minimum database should be evaluated in all dogs presenting for suspected albuterol exposure, with lactate and glucose monitored carefully in dogs with moderate or severe hypokalemia given the correlation found.

EXOTICS

Chelonians

Prognostic value of packed cell volume and blood glucose concentration in 954 client-owned chelonians

Colon VA and Di Girolamo N

J Am Vet Med Assoc 257: 1265-1272, 2020; https://doi.org/10.2460/javma.257.12.1265

Objective: To evaluate the prognostic value of PCV and blood glucose concentration in chelonians presented for veterinary care and to develop risk categories on the basis of the interaction of these analytes.

Animals: 954 client-owned chelonians (34 genera).

Procedures: Medical records of 1,059 client-owned chelonians presented to 2 veterinary institutions between 2014 and 2018 were reviewed. Logistic regression models were developed to evaluate factors associated with death, including PCV and blood glucose concentrations.

Results: There were 954 chelonians (34 genera) for which the data required to be included in the analysis were available. Both PCV and blood glucose concentration were significant prognostic indicators of death. Odds of death for chelonians with severe anemia (PCV, < 10%) and moderate anemia (PCV, 11% to 20%) were 6.8 times (adjusted odds ratio [aOR], 6.8; 95% CI, 3.8 to 12.3) and 1.6 times (aOR, 1.6; 95% CI, 1.01 to 2.7), respectively, the odds of death for chelonians with PCV within reference range. Odds of death for chelonians with severe hypoglycemia (< 30 mg/dL), moderate hyperglycemia (91 to 150 mg/dL), and severe hyperglycemia (> 181 mg/dL) were 5.3 times (aOR, 5.3; 95% CI; 2.4 to 11.4), 3 times (aOR, 3.0;95% CI, 1.4 to 6.3), and 4.3 times (aOR, 4.3; 95% CI, 2.4 to 7.6), respectively, the odds of death for chelonians with blood glucose concentration within reference range. Five risk categories were identified on the basis of PCV and blood glucose concentration.

Conclusions and Clinical Relevance: Derangements in PCV and blood glucose concentration in client-owned chelonians were associated with increased odds of death. On the basis of these results, more aggressive diagnostic testing and treatments may be indicated in chelonians with similar alterations.

FERRETS

Reference interval determination of venous blood gas, hematologic, and biochemical parameters in healthy sedated, neutered ferrets (Mustela putorius furo)

DanielaYuschenkoff D et al

J Exotic Pet Med 36:25-27, 2021; https://doi.org/10.1053/j.jepm.2020.10.007

The purpose of this study was to determine reference ranges for the Nova tabletop blood gas analyzer laboratory parameters in ferrets. Blood was collected from 40 clinically healthy ferrets, ranging in age from 1 to 5 years old (28 neutered males, 12 spayed females). The evaluated parameters were measured using whole blood and included hematologic (hematocrit, hemoglobin), biochemical (lactate, glucose, Na+, K+, iCa++, iMg++, Cl−, BUN/Urea, creatinine, tCO2, anion gap), and blood gas values (pH, PCO2, PO2, SO2%, BEecf, BEb, HCO3−). Laboratory reference intervals were established for 20 hematologic, biochemical, and blood gas values. The reference intervals are as follows: pH 7.274-7.415, PCO2 35.2-59.1 mm Hg, PO2 30.0-69.2 mm Hg, SO2 55.3-93.3%, Hematocrit 33-57%, Hemoglobin 11.0-18.9 g/dL, Na 147.4-154.8 mmol/L, K 3.81-5.37 mmol/L, Cl 109.2-119.3 mmol/L, iCa 1.14-1.35 mmol/L, iMg 0.44-0.66 mmol/L, Glu 74-152 mmol/L, Lac 0.3-1.5 mmol/L, BUN 10-33 mg/dL, Cr 0.4-0.9 mg/dL, TCO2 22.6-31.6 mmol/L, Anion gap 7.8-14.9 mmol/L, BEecf -5.2 to 3.8 mmol/L, BEb -3.9 to 3.5 mmol/L, HCO3− 21.3-30.0 mmol/L. Results varied slightly from previously established values in ferrets and include reference intervals for blood gas analytes that have not been previously reported. The reference intervals provided in this manuscript will aid veterinarians in the evaluation of acid-base balance of ferrets.

LIZARDS

Prevalence and risk factors for dental disease in captive Central bearded dragons (Pogona vitticeps) in the United Kingdom

Mott R et al

J Exotic Pet Med 36:1-7, 2021; https://doi.org/10.1053/j.jepm.2020.09.002

Despite periodontal disease being recognized as a common condition in captive bearded dragons, there is a lack of data regarding the prevalence. A soft diet has previously been cited as the main risk factor linked to the disease, although there has been little research conducted into the etiology since the disease was first described. The aims of this study were to investigate the prevalence of dental abnormalities and disease in captive Central bearded dragons in the United Kingdom, and to begin to investigate the risk factors affecting the presence and increased severity of disease in this species. Data collection was conducted from 20 veterinary practices across the United Kingdom from March to October 2018. All bearded dragons presented to participating practices during this time period were assessed for the presence of dental disease, and for each animal a standardized data collection form was completed to provide information concerning the animal's signalment, diet, and health status. Severity of any dental disease was also graded in a subset of bearded dragons (n = 147) by two of the authors using a grading system from 0 to 5. The prevalence of dental abnormalities and disease was 50% in the sampled population of 304 bearded dragons. Increasing age, an abnormal body condition score, presence of concurrent disease, as well as presence of fruit in the diet were all significant risk factors for the presence of dental abnormalities and disease. Contrary to previous reports, neither presence of different live foods in the diet, nor presence of vegetable matter in the diet had any significant associations with dental abnormalities and disease, challenging some of the assumptions made to date about the etiology of dental disease in Central bearded dragons. This study instead found that fruit could be the main dietary risk factor for dental disease and should be excluded from the

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