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ENDOLOOP BASED THORACOSCOPIC BLEB RESECTION FOR THE TREATMENT OF SPONTANEOUS PNEUMOTHORAX IN DOGS: A REPORT OF 2 CASES

Introduction
Surgical resection of lung lesions is the only treatment that effectively prevents recurrence (1-3). Lateral thoracotomy and median sternotomy however, require extensive tissue dissection and retraction at the surgical site (4, 5). Minimally invasive thoroscopic surgery has challenged these traditional approaches, although lung tissue extraction using standard thoroscopic techniques can be problematic and make conversion to open thoracotomy necessary. Endoloop-based thoroscopic bleb resection circumvents this problem and may prove to be a more elegant solution in certain situations. The aim of this report was to describe the thoroscopic endoloop technique for bleb or lung apical segmental resection and the outcome in two dogs with spontaneous pneumothorax.

Case description
Two Belgian shepherds of 8 and 3 years old were presented with bilateral spontaneous pneumothorax. Thoracic computed tomography showed multiple blebs in the left ventral tip of the cranial lung lobe in the older dog and one large bleb in the apex of the right cranial lung lobe in the younger dog. Both dogs underwent video-assisted thoracoscopic surgery (VATS) to identify and remove blebs. After localization of the blebs, either the blebs were ligated using an Endoloop or the affected lung lobe apex was ligated with an Endoloop and then partially resected using a LigaSure™ vessel-sealing device. No surgical complications were observed, nor was conversion to open thoracotomy necessary. Both dogs were discharged 30 hours after surgery and had full recovery without recurrence of pneumothorax during the follow-up period of 3 months after surgery.

Conclusions: Our findings indicate that VATS combined with the use of the Endoloop technique is safe and effective for the resection of blebs in spontaneous pneumothorax in dogs. Since the Endoloop is flexible and avoids the use of large incisions it is a discreet, minimal invasive technique further reducing post-operative pain and morbidity.

References
OCCURRENCE OF PERITONEOPERICARDIAL DIAPHRAGMATIC HERNIA AND OCCULT SPINA BIFIDA IN A WEIMARANER

Introduction
Peritoneopericardial diaphragmatic hernia (PPDH) is the most frequent congenital abnormality of the pericardium. It has been suggested that Weimaraners are predisposed (1). It is often associated with defects of the abdominal wall, sternum or cardiac congenital anomalies (2,3).

Clinical Case
A 2-year-old male entire Weimaraner presented with acute onset of unproductive vomiting and abdominal distension. Based on physical examination, a gastric dilatation and volvulus syndrome was suspected. After initial stabilization, a lateral abdominal radiography was taken. The radiograph showed severe accumulation of gas in the stomach and intestines, with no sign of gastric compartmentalization. However, there was a circular gas opacity within the caudal aspect of the pericardial sac that was a continuation of the stomach. In the thoracic vertebrae, doubling of the spinous process was noted for T1, T2, T3 and possibly T4. The ventrodorsal view confirmed spina bifida. A PPDH with herniation of the stomach was diagnosed. Thoracic ultrasonography revealed that at least part of the liver was also herniated. Emergency surgery was performed and the defect corrected.

Results and Conclusion
This is the first report of the coexistence of a PPDH and spina bifida. PPDH is thought to occur from an abnormal development of the septum transversus or a failure of the pleuroperitoneal membranes to fuse during embryogenesis (2,3). Other congenital defects have been associated (cardiac, sternal or abdominal wall anomalies) and they are thought to develop secondary to a defect at the same time during embryogenesis (2,3). It is possible that spina bifida could be associated with the presence of PPDH in this case. As a conclusion, it is important to evaluate animals diagnosed with PPDH for the presence of other possible congenital anomalies.

References
COLONIC HEMANGIOSARCOMA IN A CAT ASSOCIATED WITH THE PRESENCE OF A METALLIC FOREIGN BODY

Introduction
Hemangiosarcoma (HSA) is a malignant tumor of endothelial cells, identified most commonly in dogs than in cats (1). The reported prevalence of feline HSA (visceral and non-visceral forms) is 1.1-1.7%, with small and large intestine occurrence in 31% of visceral cases (2). Development of a sarcoma, usually liposarcoma or fibrosarcoma, has been documented at the site of foreign bodies (FB) and associated to previous traumatic events in dogs and cats (3-5).

Clinical case
A 13-year old neutered female Domestic shorthaired cat was evaluated for lethargy, hyporexia, weight loss, vomiting, and diarrhea of 3 weeks duration. On physical examination, pale mucous membranes and abdominal distension were detected. Abdominal radiographs provided by the referring vet showed loss of peritoneal detail and the presence of 3 metallic FB compatible with gunshots. Abdominal ultrasound revealed peritoneal effusion, hepatic nodules, and a hypoechoic, moderately vascularized mass close to the intestine, with a central hyperechoic line with mild acoustic shadowing. Differential diagnosis for the mass included neoplasia and granuloma. During exploratory laparotomy, ileocolic obstruction and a hemorrhagic abdominal mass were observed. Enterectomy was performed and biopsies from the mass, liver, and spleen obtained. The cat died during the postoperative period.

Results and conclusion
The necropsy revealed that the intestinal obstruction was caused by a colonic HSA. In the center of the colonic mass, one of the gunshots was found. Peritoneal sarcomatosis and pulmonary metastasis were also diagnosed. Although it can’t be confirmed, it is possible that the presence of the FB could have predisposed this cat to develop the intestinal HSA, as it happens with other types of sarcoma. To our knowledge, this is the first description of a HSA arising at the site of a metallic FB in a cat.

References
MEGAESOPHAGUS ASSOCIATED WITH ADDISON’S DISEASE IN A DOG

In the adult dog, most cases of megaesophagus are classified as idiopathic. Acquired megaesophagus may be due to 1) neuromuscular disorders such as myasthenia gravis, SLE, and polymyositis/polymyopathy, 2) esophageal obstruction, 3) toxins such as lead and organophosphates, and 4) miscellaneous causes such as hypoadrenocorticism, hypothyroidism, esophagitis, and thymoma.

This report describes a 5-year-old, neutered female Basset hound, presented with a 4 weeks' history of lethargy, decreased appetite, weight loss and polyuria/polydipsia. A few days ago she vomited once. Treatment with broad spectrum antibiotics had had no effect. Routine laboratory examination revealed mild non-regenerative anemia, leukocytosis, hypoproteinemia and hypoalbuminemia. A thoracic radiograph, performed in the non-anesthetized dog, showed a megaesophagus.

Based on history, findings at physical exam, blood work and diagnostic imaging many causes of megaesophagus could be excluded or were considered less likely. Additional diagnostics revealed no adrenocortical stimulation after ACTH administration and high basal plasma ACTH concentration, indicating Addison's disease. Within a few days after start of the treatment for Addison's disease the dog showed full clinical recovery; hematological and biochemical abnormalities were normal at 5 weeks after start of the therapy.

For the idiopathic form and some acquired forms of megaesophagus, treatment is only supportive and symptomatic. A risk factor is development of aspiration pneumonia, and this is the reason for the guarded to poor prognosis (1). Although rare, Addison's disease has been reported as an underlying cause for megaesophagus (2-4). Treatment of Addison's disease often results in disappearance of megaesophagus (2). Therefore, dogs with megaesophagus should be screened for Addison's disease.

SEVERE PULMONARY HYPERTENSION AND RIGHT-SIDED CONGESTIVE HEART FAILURE CAUSED BY AN INFECTION WITH ANGIOSTRONGYLUS VASORUM IN A YOUNG DOG

A six month old male crossbreed dog was presented for assessment of lethargy and abdominal distension. He was up-to-date with anthelmintic prophylaxis and vaccination and had never been outside the Netherlands. Clinical examination findings included tachycardia, tachypnea, weak peripheral pulses, a positive undulation test, jugular venous distension, and a right apical, grade 1/6 holosystolic heart murmur. Thoracic radiographs showed diffuse interstitial and alveolar infiltrates and right-sided cardiomegaly. Echocardiography showed right ventricular concentric and eccentric hypertrophy, dilatation of the main pulmonary artery, systolic flattening of the interventricular septum and a moderate tricuspid regurgitation. Application of the modified Bernoulli equation to the velocity of the tricuspid regurgitation jet showed an estimated systolic pulmonary artery pressure of 88 mmHg. The severe pulmonary hypertension and consecutive right-sided congestive heart failure were suspected to be caused by a pulmonary pathology. Hematological and biochemical blood testing did not show any indications for thromboembolic or inflammatory lung disease. Faecal examination revealed a severe infection with Angiostrongylus vasorum. Treatment with fenbendazole and pimobendan resolved the symptoms of right-sided congestive heart failure and improved the general clinical status of the dog. This case report shows that Angiostrongylus vasorum is native in the Netherlands and that the possibility of an infection with Angiostrongylus vasorum should always be considered in dogs with respiratory and/or (right-sided) cardiac symptoms, even if deworming seems adequate.
CLINICAL NUTRITION IN A DOG WITH RENAL FAILURE

A 6-year-old dog was presented with acute vomiting, dehydration and lethargy. Clinical examination, urinalysis and blood work revealed that the dog was suffering from acute renal failure. The dog was referred to the ICU for fluid therapy. During the hospitalization the dog was offered a recovery food but did not eat sufficient amounts to fulfill resting energy requirements (RER), and the dog kept on vomiting, so the dog was put on parenteral nutrition. During the fluid therapy and parenteral nutrition, the dog improved and was offered a liquid recuperation supplement. Finally the dog started eating a recovery diet and the blood levels were stable. The dog was send home with a renal diet. After 2 weeks the dog came in for a control visit. The dog refused to eat several commercial renal diets, so the dog was referred to the nutritionist to provide a homemade recipe. After 6 months being fed the homemade recipe, the dog was still performing well and blood levels remained stable. This case report reflects the different approaches needed for management of a dog with renal failure. Nutritional support is important during the hospitalization phase because hospitalized animals are in a catabolic phase, characterized by increased needs for energy and protein. After a switch from the catabolic to the anabolic phase, the dietary plan has to be re-evaluated, especially in case of renal failure. Compliance to, and acceptance of, a renal diet is challenging. If dogs refuse to eat several commercial renal diets, homemade recipes, provided by a board certified nutritionist, can be very helpful.
SECONDARY ACROMEGALY IN A BOXER

Acromegaly, i.e. hypersecretion of growth hormone (GH) in the adult dog, is a syndrome characterized by overgrowth of connective tissue, bone and viscera. A seven-year-old male intact Boxer was presented for thick interdigital skin folds of one year duration. The dog also displayed exercise intolerance and was lethargic. Physical examination revealed an increased body condition score, weak peripheral pulse, bradycardia, pharyngeal stridor, local proliferation of the gingiva, symmetrical alopecia at the base of the tail and thick skin folds in the interdigital spaces, head and neck area.

The dog was suspected of acromegaly, which was later confirmed by an elevated plasma insulin-like growth factor-1 concentration. Excess of GH can be caused by a somatotroph adenoma of the pituitary gland, progesterone-induced hypersecretion of GH in the mammary gland and by primary hypothyroidism. As the dog was a male that never had received exogenous progestins, progesterone induced acromegaly could be ruled out. The history and physical examination were suggestive of hypothyroidism. Blood examination showed an immeasurably low total plasma thyroxine concentration and a normal plasma thyroid stimulating hormone concentration. Scintigraphic imaging of the thyroids, using radioactive pertechnetate, justified the diagnosis of primary hypothyroidism. The dog was treated with synthetic levo-thyroxine and after 3 months the skin folds almost completely had disappeared, and all other problems had resolved.

Primary hypothyroidism can bring about adenohypophysyal changes that result in acromegaly, is easily treated and should always be considered in a dog with acromegaly.

References

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FANCONI SYNDROME IN A MALTESE. QUANTITATIVE APPROACH TO THE ACID-BASE ANALYSIS

Introduction
Fanconi syndrome (FS) is a rare disorder which is associated with a resorption defect of the proximal renal tubule, resulting in an excessive loss of bicarbonate, glucose, sodium, potassium, phosphate, calcium, water and amino acids1.

Clinical Case
A 3-year-old female Maltese dog was presented for a 1 month history of inappetence, polyuria and polydipsia, and weight loss. Diagnostic tests showed euglycaemic glucosuria, hypophosphatemia, hypernatremia, hypokalemia, hyperchloremic metabolic acidosis, proteinuria, aminoaciduria and increased urinary excretion of electrolytes, consistent with Fanconi syndrome1. Quantitative approach to acid-base analysis2 on initial presentation showed that metabolic acidosis was mostly due to hypernatremia (increased free water effect) and bicarbonate deficit (increased chloride effect). Sodium bicarbonate and potassium gluconate were established as treatment. Follow-up quantitative acid-base analyses were performed 1, 2 and 5 months after treatment initiation showing progressive improvement in metabolic acidosis. Seven months after initial presentation, the dog was examined for anorexia, vomiting, and severe dehydration. Quantitative approach to the acid-base analysis showed severe metabolic acidosis mostly due to hypoperfusion (increased lactate effect), unmeasured anions, and hypernatremia (increased free water effect). Intravenous sodium chloride and sodium bicarbonate were given to correct dehydration, hypernatremia and metabolic acidosis. It was discharged 48 hours after presentation. The dog died 8 months after initial examination. The necropsy showed diffuse renal tubular necrosis.

Discussion and Conclusion
Conventional acid-base analysis characterizes the magnitude of metabolic abnormalities, but the relative contribution of individual metabolic processes is not further characterized 2. This is the first report of quantitative approach to the metabolic acidosis in FS. This approach could be a good tool to understand the individual factors that affect the acid-base balance in FS, to development of an appropriate therapeutic plan for each patient, and to monitoring treatment response.

References
Cobalamin deficiency in a border collie

A 3-year-old, intact male Border collie was referred to the Department of Clinical Sciences of Companion Animals of Utrecht University because of pale mucous membranes, tachycardia, lethargy, elevated temperature, enlarged lymph nodes, skin infection, diarrhea and lethargy. Biochemistry and CBC revealed hypoglycemia, neutropenia and anemia. Additional findings were a bacterial cystitis and an elevated cPLI.

The dog was treated symptomatically (infusion, glucose and antibiotics) and recovered clinically, but he still had a non-regenerative anemia.

Four weeks later, the dog presented with hypovolemic shock after another episode of diarrhea. With IV infusion therapy, the dog completely recovered, but still had a non-regenerative anemia. Additionally, a toxocara canis infection was found and treated.

To work out non-regenerative anemia additional blood examination was performed, after bone marrow biopsy revealed no diagnosis, with measuring the serum cobalamine concentration. Cobalamin was 32 pmol/l (reference range: 166 - 635 pmol/l).

In Border collies four case reports are described with cobalamine deficiency causing unspecific signs and a non-regenerative anemia (America, Great Britain and Switzerland) 1,2,3,4. A selective intestinal cobalamin malabsorption is suspected, as only parenteral treatment is successful.

Parenteral treatment caused a complete improvement; the dog did not have periods of diarrhea anymore, he was more energetic and the hematocrit returned within the reference range.

In Border Collies with non-regenerative anemia and unspecific clinical signs cobalamin deficiency should be taken into account. The diagnosis is suspected with low serum cobalamin concentration and can be supported by other abnormalities like erythroblastemia and methylmalonic aciduria. Administration of lifelong parenteral cobalamin resolves clinical symptoms and restores laboratory abnormalities.

References