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EMERGENCY & CRITICAL CARE

Updates on Non-Traumatic Hemoabdomen Management

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Emergency clinic veterinarians know the feeling when a 12-year-old Labrador retriever walks into the clinic with a presenting complaint such as “wobbly in back legs, not eating.” Said Labrador is then triaged, and veterinarians find pale mucous membranes leading to a fast scan that identifies ascites that aspirates as blood. The non-traumatic hemoabdomen (spontaneous hemoperitoneum (SHP)) has been an area of extensive study as this is not an uncommon case to encounter during an emergency shift. It is also one that comes with high emotional weight, so it is important that we understand what is available to these patients. This article will concentrate

on updates over the past five years regarding the management of non-traumatic hemoabdomen and prognostic indicators.

If a client expresses interest in going to surgery, a veterinarian will likely start to ponder the value of pre-operative imaging of the abdomen. Most commonly, if available, clinicians will reach for an abdominal ultrasound (AUS) to identify the source of bleeding and evaluate for gross metastasis. But how helpful is this?

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CARDIOLOGY

Use of Focused Cardiac Ultrasound in the Small Animal Practice

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Cardiac disease is a common cause of morbidity and mortality in dogs and cats. Focused cardiac ultrasound (FCU) is a tool that allows one to identify certain ultrasonographic features of heart disease, such as chamber enlargement, cavitory effusions, or systolic dysfunction, which can ultimately help guide clinical decision-making, typically in an emergency setting. FCU, as defined by

the American Society of Echocardiography, refers to a “focused examination of the cardiovascular system performed by an appropriately trained clinician (typically not a cardiologist) by using ultrasound as an adjunct to the physical examination to recognize specific ultrasonic signs that represent a narrow list of potential diagnoses in specific clinical settings.” FCU findings should not be used in isolation but rather be interpreted in

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In February 2021, a study⁵ evaluated the diagnostic utility of ultrasound to answer this question. Medical records from 94 client-owned dogs were evaluated for differences between AUS, surgical, and necropsy findings. The study identified that splenic masses were most commonly identified as the cause of the SHP. The sensitivity of AUS was 87.4%, 37.3%, and 31.3% for masses in the spleen, liver, and mesentery, respectively. Notably, zero of six dogs with peritoneal diffuse nodular metastasis had lesions detected by AUS. As such, it can be concluded that AUS is not very sensitive for nodular metastasis but is for splenic masses (which is the most common). For most clients, an ultrasound report with nodular metastasis would half the plan for surgical treatment. With that said, only six of the 94 dogs had nodular metastasis, so it might be considered somewhat rare in SHP cases. However, the risk must be weighed, as clinicians will not fully be able to answer the question of metastasis on a pre-operative ultrasound, and the client should be warned of such risk. Additionally, it can be considered to forego the pre-operative abdominal ultrasound if it is not available (or a client has limited finances) as it is not a 100% sensitive modality for assessing metastasis. Gross evaluation during surgery is the only way to evaluate fully.

As clinicians gather point-of-care information on the patient, they will likely run a PCV/TP as part of their database. The decision of when or if to consider a packed red blood transfusion (pRBC) can be one of debate. A study² published in 2022 reviewed admission total plasma protein as a predictor of red blood cell transfusion requirements in both traumatic and non-traumatic hemoabdomens. Ninety dogs were retrospectively evaluated. A total of 47 dogs (traumatic hemoabdomen 11/26; non-traumatic 36/64) received pRBC transfusions. For each 1 g/dL unit decrease in TP, dogs had an increased odds ratio of 2.14 of receiving a transfusion. Lower PCV, bicarbonate values, base excess, and higher lactate were also associated with increased transfusion rate. As such, it was concluded that lower admission

TP values, independent of low PCV, were associated with increased red blood cell transfusion. Clinically, you might consider preparing for red blood cell transfusion or even pro-actively administering a pRBC transfusion if the total protein is low despite the PCV. In this paper, a TP of <5.6 g/dL was associated with an increased rate of pRBC administration vs a TP of >6.1 in those that did not. This ultimately may lead to better post-operative and surgical outcomes if we stay ahead of blood loss. PCV/TP is also a technically accessible and cost-effective test for running point of care and is part of a workup for any SHP case.

The last update to touch on is tranexamic acid (TXA), a synthetic derivative of the amino acid lysine that inhibits fibrinolysis. This is still an area of debate and study, but this author will highlight a few papers on this topic published recently. Hyperfibrinolysis (HF) is when the body dissolves hemostatic fibrin, resulting in excessive or recurrent bleeding. It has been established that, likely in the initial diagnosis of SHP, dogs have a hyperfibrinolytic state, but the prevalence and duration were unknown. A study¹ published in 2022 showed that HF occurs in some dogs with hypovolemic shock due to SHP but resolves rapidly following surgical control of bleeding. This was determined by measuring PCV, total plasma protein, platelet count, and thromboelastography with 50 U/mL plasminogen activator at presentation and then every eight hours post-operatively until 72 hours, discharge or death. This proves that post-operatively, TXA is not likely to be of benefit to

patients since HF resolves with surgical treatment. But what about pre-operatively? Another study in 2020³ reviewed the use of TXA in critically ill dogs and cats. Administration of TXA before or after pRBC transfusion did not significantly affect the median dose of pRBC administration. Adverse reactions were mild and rare in this study; the average dose was 10 mg/kg. While these conclusions help determine that TXA is a well-tolerated drug, clinicians do not know if it truly contributes to better clinical outcomes. This paper shows that the need for transfusion was not significantly lower. In human medicine, in 2013, the CRASH 2 study⁴ has been cited, which showed that early administration for adults with traumatic bleeding reduced the risk of death. This may be extrapolated to cases of traumatic hemoabdomen, but in veterinary medicine, doctors do not have a clinical trial for comparison. All this is to say that clinically, TXA is well tolerated and may have a benefit, but none has been proven. It likely does not have a benefit for post-operative use but may be considered pre-operatively as clinicians have not fully evaluated its benefit pre-op.

Cases of SHP can be some of the most rewarding with good stabilization techniques and surgical correction. Consider ultrasound's sensitivity in pre-operative metastasis evaluation for a future case and factor this into the conversation with clients. Another suggestion is to double-take at a low TPP and prepare for a pRBC transfusion. Also, maybe consider the use of pre-operative TXA if the patient is showing evidence of shock. But likely not post-operatively, as it has been proven the

> <https://todaysveterinarypractice.com/radiology-imaging/imaging-essentials-small-animal-abdominal-ultrasonography-spleen/>



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hyperfibrinolytic state resolves with surgical removal of the bleeding mass. It will be exciting to see ongoing research areas on this topic over the next five years as clinicians continue to learn how to approach it best.

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CARDIOLOGY

Use of Focused Cardiac Ultrasound in the Small Animal Practice

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conjunction with the patient's signalment, history, physical examination findings, and other imaging modalities, namely thoracic radiography.

This article highlights some of the more common clinical indications of FCU in small animal practice. The interested reader is encouraged to peruse the review articles cited under references for further information. While there are no available guidelines that outline specific training needed to perform FCU proficiently, there is a growing body of resources and hands-on training opportunities through continuing education courses that can help improve the confidence and accuracy of FCU.

Clinical Indication 1: The Dyspneic Cat

Treating the dyspneic feline patient is challenging. Obtaining thoracic radiographs on presentation can be difficult due to the fragile nature of these patients, and the clinician is often faced with the difficult task of deciding how to stabilize the patient with minimal information. FCU can help differentiate cardiac vs. non-cardiac causes of dyspnea and has several advantages in this setting. It can be performed rapidly with portable equipment and typically does not require significant restraint. FCU can also be easily incorporated into other point-of-care ultrasound assessments, namely thoracic and abdominal focused assessment with sonography for trauma, triage, and tracking (TFAST and AFAST), as well as veterinary bedside lung ultrasound exam (Vet BLUE).

For example, identification of an enlarged left atrium to aorta (LA/Ao) ratio of 2:1, in conjunction with B-line artifacts on Vet BLUE lung ultrasound (which can support the presence of pulmonary edema) as well as the presence of pleural and/or pericardial effusion, would dramatically increase the suspicion of congestive heart failure, and prompt the clinician to administer a furosemide bolus as the first step in stabilizing the patient. The author reminds the reader that such a patient should ultimately receive a comprehensive diagnostic work-up, including blood work, thoracic radiographs, and, if and when possible, a comprehensive echocardiogram performed by a cardiologist. Alternatively, identification of a normal LA/Ao ratio of 1.2:1 on FCU in a dyspneic feline patient with a history of intermittent coughing episodes may increase the suspicion of primary lower airway disease.

Clinical Indication 2: The Asymptomatic Cat with a Heart Murmur

Another common indication of FCU is the feline patient with an incidental heart murmur in need of general anesthesia. While elective procedures (such as a routine dental procedure) can often be safely delayed for the patient to be first evaluated by a cardiologist, there are many instances in which the clinician has to proceed without a comprehensive cardiac evaluation, such as with emergency procedures. FCU can be advantageous in this setting in that it can quickly help determine the patient's risk of volume overload and, for the practitioner more experienced in FCU, it may even allow for assessment of the left ventricular wall thickness and systolic function, which provide additional information and may prompt the practitioner to avoid certain anesthetic drugs. In a recent study by Loughran et al., evaluation of the left ventricular wall thickness using FCU by a trained general practitioner was significantly correlated with echocardiographic measurements in cats.

Clinical Indication 3: The Dyspneic Dog

Left-sided congestive heart failure (LCHF) is common in dogs with heart disease, such as myxomatous mitral valve disease (MMVD) or dilated cardiomyopathy (DCM). FCU can be helpful to increase or lower one's suspicion of left-sided congestive heart failure, which in dogs manifests as pulmonary edema. Key FCU features include an increased LA/Ao ratio (Figure 1), in combination with numerous B-line artifacts on Vet BLUE lung ultrasound, which, in dogs with LCHF, typically manifests as bilateral, diffuse distribution of infinite coalescing B-lines. Ultimately, thoracic radiographs

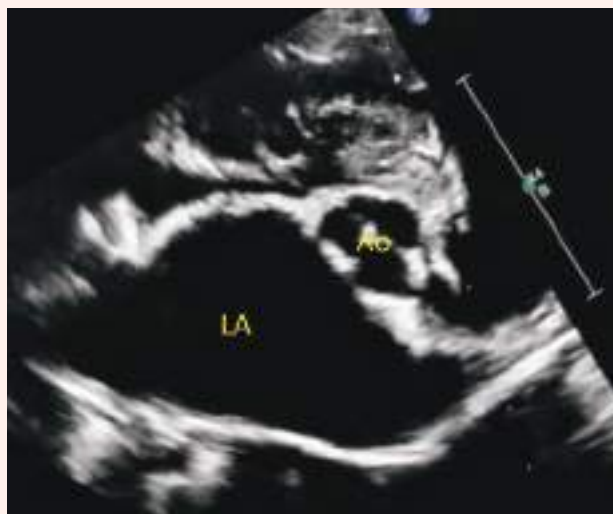
should be performed to confirm the suspicion of LCHF and monitor the patient's response to treatment. The reader is also encouraged to pay particular attention to the patient's signalment and physical exam findings. Typically, advanced MMVD is most common in older, small-breed dogs and is accompanied by a loud left apical systolic murmur. Conversely, DCM is more common in larger-breed dogs, and in these patients, the heart murmur may only be soft or even be absent. Key FCU findings in DCM include left ventricular dilation and severe left ventricular systolic dysfunction.

Dogs with active LCHF are often tachycardic on presentation. However, patients with concurrent sinus node dysfunction or diseases associated with high vagal tone (such as primary respiratory, GI, neurologic, or urinary disease) may not always present with tachycardia.

Another important disease that deserves mention in this section is pulmonary hypertension (PH). While a comprehensive discussion on PH is beyond the scope of this article, dogs with pulmonary hypertension may present with dyspnea, exercise intolerance, and/or syncope, similar to dogs with LCHF. In these cases, however, FCU may identify subjective right heart enlargement and, in some cases of pre-capillary PH without significant concurrent left heart disease, normal LA/Ao ratios.

Figure 1

> An enlarged left atrium in a dog with advanced MMVD and LCHF. In this case, the LA/Ao ratio was severely increased at 2.3:1. LA, left atrium; Ao, aorta.



Limitations of FCU

As mentioned, there are currently no published guidelines on the amount of training required for a practitioner to perform FCU with high proficiency. Additionally, it is vital that the imaging views be obtained in proper imaging planes and the measurements be performed accurately; therefore, FCU performed by an inexperienced clinician may result in inaccurate diagnoses. However, there is growing literature in both human and veterinary medicine showing that, with reasonable training, emergency clinicians, as well as general practitioners, can utilize FCU with clinically relevant accuracy in certain settings.

While highly useful in aiding clinical decision-making, FCU should not replace the value of thoracic radiography and a comprehensive echocardiogram.

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Summary of the ACVIM Consensus Statement on Pancreatitis in Cats

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Introduction

In 2021, eight experts, including five internists, one radiologist, one clinical pathologist, and one anatomic pathologist, summarized current literature regarding the etiology, pathogenesis, diagnosis, and management of pancreatitis in the etiology pathogenesis, diagnosis, and management of pancreatitis cats.¹ The main goal was to offer clinically relevant suggestions for veterinarians based on evidence, and where such evidence was lacking, recommendations were based on the consensus of the experts in the field.

Pancreatitis in cats, once thought rare, is a fairly common disease. In one study of 115 cats undergoing necropsy at UC Davis², 66.1% had evidence of pancreatic inflammation. While acute lesions were found in 6.1% of cats, 50.4% had evidence of chronic pancreatitis, and 9.6% had both acute and chronic findings. However, 45% of healthy cats also had evidence of inflammation, suggesting the possibility of subclinical pancreatitis in many asymptomatic cats. Increased frequency of a diagnosis of pancreatitis over the last two decades is likely due to increased awareness and availability of minimally invasive diagnostics. Management of pancreatitis, however, remains challenging and definitive treatments are unavailable.

Definition

Acute pancreatitis is characterized by completely reversible inflammation after removal of the inciting cause, while chronic pancreatitis results in irreversible histopathologic changes. Clinically, however, it is impossible to distinguish acute pancreatitis from an exacerbation of chronic pancreatitis. Chronic cases commonly exhibit milder symptoms, while acute cases tend to be more severe.

Etiology

Pancreatitis has no age, sex, or breed predisposition, and no associations with body condition score, dietary indiscretion, or drugs have been established in cats. Infectious diseases rarely associated with feline pancreatitis include *Toxoplasma gondii*, coronavirus, parvovirus, herpesvirus, calicivirus, and certain parasites. Hypotension during surgery is likely a more critical risk factor in developing pancreatitis than surgical manipulation of the pancreas. Autoimmune pancreatitis occurs uncommonly in people, and there is some suspicion of immune-mediated etiology in cats, but the evidence is lacking. Pancreatitis in cats has been associated with several concurrent diseases, including diabetes mellitus, chronic enteropathies, hepatic lipidosis, cholangitis, nephritis, and immune-mediated hemolytic anemia (IMHA). Whether these conditions cause or are risk factors for pancreatitis is unknown.

In conclusion, 95% of cases of pancreatitis in cats are idiopathic with no specific etiology.

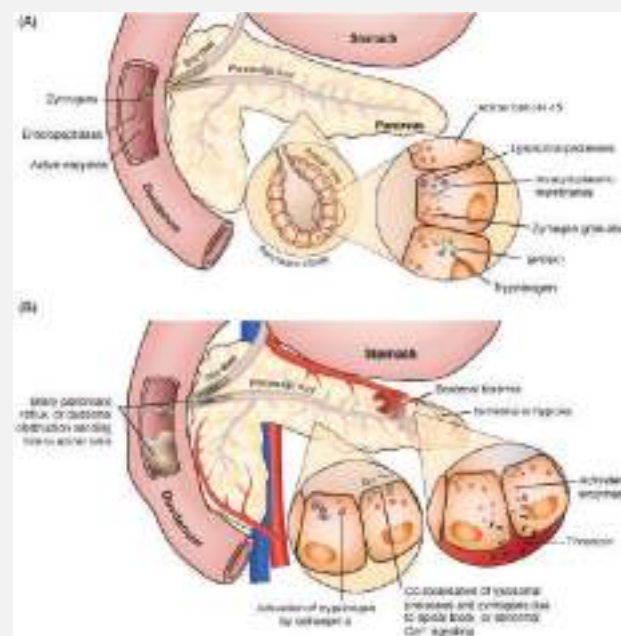
Pathophysiology

Premature activation of pancreatic digestive enzymes or zymogens (inactive forms of digestive enzymes) results in pancreatic autodigestion. The pancreas has several protective mechanisms to prevent this process (Fig. 1A). Hypotheses for spontaneous development of pancreatitis include activation of trypsinogen during biliary pancreatic reflux or duodenal obstruction, activation of zymogens by thrombin during bacterial toxemia, co-localization of lysosomal proteases and zymogen granules due to an apical block of zymogen granule secretion, or abnormal calcium signaling (Fig. 1B). Researchers have concluded that trypsinogen activation is the initiating event for acute pancreatitis; however, the exact mechanism remains unknown. Recent hypotheses are suspected of abnormal calcium signaling and early activation of the nuclear factor kappa B pathway. Physiologic sequelae include an influx of neutrophils, increased vascular permeability, and loss of paracellular barriers.

The unique anatomy of the cat, with shared entry of the common bile duct and the pancreatic duct into the duodenum, may explain the association between acute bacterial cholangitis and pancreatitis.

Figure 1A and B

► Mechanisms protect the pancreas from premature activation of zymogens (A). Events can lead to acute pancreatitis due to the premature activation of trypsinogen (B).



INTERNAL MEDICINE

Summary of the ACVIM Consensus Statement on Pancreatitis in Cats

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Figure 2

› Sagittal plane abdominal ultrasound image of the left pancreatic limb in a cat with acute pancreatitis showing enlargement, decreased echogenicity, and surrounding halo of hyperechoic mesentery.



In contrast to acute pancreatitis, trypsin activation is not the inciting cause of chronic pancreatitis. Cholecystokinin and oxidative stress likely alter calcium signaling and cause mitochondrial damage. Low-grade inflammation leads to the activation of stellate cells, which are the source of fibrosis. Ductal obstruction and irritation bypassing choleliths may be a significant factor, as suggested by a recent study from Cornell where 51% of cats with suspected idiopathic pancreatitis had passing choleliths documented by ultrasound or at the time of surgery.³ Some of them were microcholeliths, less than 3mm in size.

Clinical signs

Clinical signs are nonspecific; 51% to 100% of cats show lethargy, 62% to 97% present with anorexia, only 35% to 2% have vomiting, and 11% to 38% have diarrhea.

Figure 3

› Sagittal plane abdominal ultrasound image of the left pancreatic limb in a cat with chronic pancreatitis. The pancreas is mildly enlarged at 1.5cm (< 0.9cm), heterogeneous, and has mottled echotexture. The surrounding mesentery is unremarkable.



Diagnostic imaging

Abdominal radiography is neither sensitive nor specific for this disease. Recent studies established the normal and abnormal features of the feline pancreas imaged by multiphase contrast-enhanced computed tomography and magnetic resonance. Ultrasonography (US) remains the most used imaging modality and is considered part of the minimum database in these patients. Despite some limitations, US is important to detect comorbidities of the intestines, liver, and gallbladder. In cats, sonographic findings in acute pancreatitis may reveal pancreatic enlargement, decreased echogenicity, hyperechoic surrounding mesentery, and focal abdominal effusion (Fig. 2). The sensitivity of these findings for diagnosing acute pancreatitis in cats is usually less than 67%, depending on the severity and operator's experience. Features of chronic pancreatitis are even less defined and include hyperechoic or heteroechoic pancreas, a dilated common bile duct, pancreatic enlargement, and irregular margins (Fig. 3).

Ultrasound-guided fine-needle aspiration of feline pancreases was safe with diagnostic sampling obtained in 67% of cats.

Clinical pathology

Chemistry findings are also variable. Abnormalities on CBC may show increased hematocrit due to dehydration and an inflammatory leukogram with a left shift. Increases in hepatic enzymes and bilirubin may be due to concurrent inflammation of the biliary tree, extrahepatic biliary obstruction, hepatic lipidosis, or some combination of these. In cats with severe pancreatitis, azotemia and a low urine specific gravity can result from an acute kidney injury secondary to hypoxemia, impaired renal microcirculation, or hypovolemia. Azotemia, hypocalcemia, and hypoglycemia have been associated with poor outcomes. Hypertriglyceridemia is not clinically significant in feline pancreatitis.

Measurement of pancreatic lipase to diagnose pancreatitis is complicated by the presence of many types of lipases in the body. DGGR-based assays (PSL by Antech) measure pancreatic lipase, hepatic and lipoprotein lipase, and even hemoglobin. Another measure of lipase is serum pancreatic lipase immunoreactivity (fPLI), which can be measured by a commercial ELISA test (Spec fPL). Most of the data suggest high sensitivity and specificity of this test in evaluating for the presence of pancreatitis. Sensitivity is higher for severe cases than for mild cases. A positive Spec fPL indicates pancreatitis, but a negative result does not entirely rule out this disease. SNAP fPL is a semiquantitative test that correlates well with the Spec fPL. Cats with a "normal" result are unlikely to have pancreatitis, while those with "abnormal" results might have pancreatitis or a Spec fPL in the equivocal range. Few studies showed discordance between Spec fPL and PSL.

Management of acute pancreatitis

The mortality rate in cats with acute pancreatitis has been reported to be between 9% and 41%, depending on the severity and associated comorbidities. Management is predominantly supportive and symptomatic and is extrapolated from humans and dogs. Identification of comorbidities (e.g., diabetes mellitus (DM), diabetic ketoacidosis (DKA), cholangitis, and chronic enteropathy) as well as management of complications (e.g., hepatic lipidosis, cholestasis, acute kidney injury, pneumonia, shock, myocarditis, disseminated intravascular coagulation (DIC), or multi-organ failure) play an important role in therapeutic success. There are no proven disease-specific treatments in humans that change the natural progression of acute pancreatitis, although the research is ongoing. A new leukocyte function-associated antigen-1 antagonist was recently approved in Japan in dogs.

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Summary of the ACVIM Consensus Statement on Pancreatitis in Cats

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Figure 4

↳ Naso-esophageal (A) and esophagostomy (B) tubes are the most practical tubes for alimentary support of cats with acute pancreatitis.



Supportive therapy includes intravenous fluids because establishing normovolemia can limit tissue damage by improving pancreatic perfusion and oxygen delivery. Early, aggressive hydration with Lactated Ringers Solution (LRS) hastens clinical improvement in humans. Avoiding over-hydration is equally important as fluid overload is associated with increased morbidity and mortality. Anti-emetics are used to minimize fluid losses and reduce the risk of regurgitation and esophagitis. The most commonly used anti-emetics in cats are maropitant and ondansetron, which work by different mechanisms and can be combined. Metoclopramide has questionable anti-emetic effects but, when administered as a continuous rate infusion (CRI), increases gastric emptying and decreases gastric atony. Pain is challenging to evaluate in cats and is likely underestimated in cats with pancreatitis. Opioids should be used as the first line of analgesia; buprenorphine is adequate in most cats, while methadone or fentanyl can treat more severe pain. Maropitant was shown to offer some visceral analgesia and anti-inflammatory activity. Appetite stimulants play an essential role in preventing malnutrition and impairment of the gastrointestinal barrier secondary to anorexia. Mirtazapine is used most commonly, but capromorelin has been recently approved in stable cats with CKD. Capromorelin has been reported to cause hypotension and bradycardia in cats, so the use of this medication should only be reserved for use in non-hospitalized clinically stable cats.

Nutritional support plays a central role in managing acute pancreatitis in people and companion animals. Lack of enteral nutrition leads to impaired gastrointestinal motility, intestinal villous atrophy, compromised intestinal blood flow, altered barrier function, and disruption of the normal intestinal microbiota. Cats have high dietary protein requirements and a higher tolerance for dietary fat than dogs. Highly digestible diets, often labeled "gastrointestinal" diets, are recommended. Placement of a feeding tube (Fig. 4) is indicated for cats that fail to respond to appetite stimulants within 48 hours.

These cases are characterized by severe dehydration (often 8% to 10% dehydrated or more), failure to respond to medical management, hypotension, hypoglycemia, and ionized hypocalcemia. Severe complications include systemic inflammatory response syndrome, cardiovascular shock, DIC, pulmonary thromboembolism (PTE), or multi-organ failure. Cats with severe pancreatitis should be referred to specialty hospitals equipped to provide critical care support and monitoring.

Fresh frozen plasma is not recommended as a standard treatment in humans or cats and should be reserved for cats with coagulopathy. Antibiotics are not recommended for non-complicated cases of pancreatitis in cats unless a solid clinical indication or sepsis is present.

There is insufficient evidence to recommend glucocorticoids in cats with acute pancreatitis, as no studies have evaluated their use.

Dyspnea is a common complication of severe pancreatitis in cats with multifactorial origin (pleural effusion or pulmonary edema due to volume overload, acute lung injury, acute respiratory distress syndrome, congestive heart failure, pulmonary thromboembolism, or pain). Thoracic radiographs and echocardiography often permit rapid diagnosis and guide treatments.

Additional therapeutic strategies, including proton pump inhibitors, trypsin inhibitors, antisecretory agents, and antioxidants, have not been proven efficacious, and their use is not recommended. No clinical studies support the use of (hyperbaric oxygen therapy) HBOT.

Management of chronic pancreatitis

Little research is available on the clinical therapy of chronic pancreatitis in cats. Cats with chronic pancreatitis often have other diseases, and treatment of these conditions usually takes priority. However, if chronic pancreatitis is causing clinical symptoms and decreases the patient's quality of life, it should also be managed. Traditional analgesics may not be effective for visceral pain mediated by cytokines, substance P, and neurokinin A. Acute exacerbation can be treated with buprenorphine; however, chronic pain may be better controlled with gabapentin, tramadol, and maropitant.

Dietary recommendations are controversial. The majority of the panel members did not have concerns about the dietary fat content in cats with chronic pancreatitis, given the lack of scientific evidence suggesting fat should be avoided. Anti-emetics and appetite stimulants should be used as needed, and, similar to treatment recommendations for the acute form of this disease, antibiotics are not indicated.

Prednisolone is a commonly used anti-inflammatory and immunosuppressive drug with potentially antifibrotic effects. Given the lack of scientific evidence to recommend its use, the risks should be weighed against the benefits for an individual patient. The most concerning side effects of steroid use in these patients are enhanced peripheral insulin resistance and diabetes mellitus.

Some experts use prednisolone only in cats that are not hyperglycemic and only at anti-inflammatory dosages (i.e., 0.5 to 1 mg/kg PO q24h on a tapering schedule). Other panel members use immunosuppressive dosages (e.g., 2 mg/kg q12h for five days, then 1mg/kg q12h for six weeks and taper after that) with close monitoring (i.e., clinical re-evaluation and measurement of fPLI after two to three weeks). If hyperglycemia develops or is pre-existing, cyclosporine can alternately be used (5mg/kg q24h for six weeks) with close monitoring (i.e., clinical re-evaluation and measurement of fPLI after two to three weeks).

If prednisolone or cyclosporine do not improve clinical signs and a reduction in fPLI, they should be discontinued. In cats that show improvement on anti-inflammatory/immunosuppressive treatment, continued therapy may be necessary. Cyclosporine has been reported to unmask latent toxoplasmosis, which needs to be considered in cats exposed to raw meat.

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Nero's Law: Training and Implementation

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Most states do not allow EMTs (emergency medical technicians) to transport police dogs injured in the line of duty to veterinary hospitals. This was also the case in Massachusetts before the passage of Nero's Law in February 2022.

Nero was a Belgian Malinois police dog who worked with Sergeant Sean Gannon for the Yarmouth Police Department. In April 2018, both Nero and Sergeant Gannon were shot while serving an arrest warrant. Tragically, Sergeant Gannon died of his wounds, and Nero was severely injured. At the time, the Law did not allow Nero to be transported for medical care in an ambulance, so he had to be taken in a police cruiser to the nearest veterinary hospital.

It is common for states to have laws that prevent non-veterinarians from practicing veterinary medicine. However, these laws also prevent first responders (EMTs/Ambulances/fire departments) from legally transporting or treating any veterinary patients. There has been a substantial push in several states to change these laws and create training programs for human first responders so they can treat and transport injured working dogs to appropriately outfitted veterinary hospitals. This has also been the case in Massachusetts since the passage of Nero's Law over two years ago.

Nero's Law allows for the limited treatment and transport of working dogs owned by police departments from the site of injury to one of about a dozen hospitals across the state, including the MSPCA-Angell Boston and the MSPCA-Angell West in Waltham. These dogs may be injured while fulfilling

› Sergeant Sean Gannon and Nero



› Nero's Law training session at Gillette Stadium.



their role as police dogs, scent detection dogs, or K-9 community resource officers. This Law does not cover working dogs owned by other agencies or the public (Seeing Eye dog, medical alert dogs, etc.) and emotional support dogs and animals.

Nero's Law requires that all first responders in Massachusetts must attend a half-day course (1.5 hours lecture and 1.5 hours lab) on the emergency management of canine trauma/illness. There are approximately 20,000 EMTs and firemen/women in the state who need to receive this training. The lab training sessions must be offered by veterinarians or registered veterinary technicians. So far, it has been a long time since this author has found enough trainers to satisfy the need for all these training sessions.

The lecture component of Nero's Law training is a 90-minute online lecture completed by first responders on their own or sometimes in a group setting. This component introduces the need for and history of Nero's Law and summarizes medical techniques that will be performed in the hands-on lab portion. The lab training is usually done at the first responders' place of work. Four modules are presented during the lab: physical exam/assessment, restraint and handling, hemorrhage control/shock, and CPR. During their training, EMTs will practice CPR on a K-9 CPR mannequin, be introduced to an actual working police dog and their handler, use K-9 specific bandages and bandaging techniques, and perform a physical exam on a non-police dog.

The scope of medical intervention permitted under Nero's Law remains fairly limited. The first writing of the Law aimed to allow life-saving measures and transport of an injured K-9 dog within an ambulance, but not to permit more advanced care that would be better employed at the destination referral veterinary hospital. As such, first responders can bandage wounds, administer first aid, treat with epinephrine or naloxone, or perform CPR (without intubation). They are not permitted to place IV catheters, give fluid, or perform

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Nero's Law: Training and Implementation

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intubation and advanced life support during CPR. These limitations have proven frustrating to some EMTs with extensive medical training and enviable experience in handling emergent patients. But Nero's Law is currently undergoing some changes in its second iteration that will hopefully allow more advanced treatments that can better take advantage of the skillset of the human first responders helping these dogs.

The implementation of Nero's Law has been primarily organized by David Schwarz, DVM, former president of the Massachusetts Veterinary Medical Association (MVMA) and State of Massachusetts Animal Resource Team (SMART) Team Leader; Sean Majoy, DVM DACVECC, Tufts University School of Veterinary Medicine; and Jamie Falzone, MVMA executive director. They have organized multiple 'Train the Trainer' sessions at Tuft's Cummings School of Veterinary Medicine and created online training opportunities through the MVMA website.

More information about Nero's Law and Nero's Law training sessions are available on the MVMA website (<https://www.massvet.org>) under Advocacy and Legislative Affairs.

› Dr. Kiko Bracker of Angell's Emergency and Critical Care service training a group of EMTs from Boston EMS.





Wound Dehiscence: Causes, Prevention, Management

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Common Causes of Wound Dehiscence

Wound dehiscence, or the disruption of apposed surfaces of a wound, is the result of several factors, either alone or in combination. Dehiscence is one of the most common postoperative complications encountered by veterinarians.

The resultant costs associated with wound care and closure can easily double the cost of the original surgery. Fortunately, most of the causes of wound dehiscence are preventable.

The following are the most common causes noted by the author:

1. Wound closure under excessive tension, with suture “cut-out,” secondary to ischemic necrosis
2. Suture placement too close to the incisional border with “cut-out” (collagenase activity within 5 mm cutaneous zone bordering skin incision)
3. Improper suture material selection (size, tensile strength, suture pattern, placement, rate of resorption/degradation)
4. Closure of severely compromised skin with subsequent necrosis
5. Suture placement, compromising cutaneous circulation
6. Moisture accumulation contributes to tissue overhydration and maceration
7. An underlying pocket of infection, necrosis, foreign body, or neoplasia
8. Lack of postoperative protection/support from motion, licking, and external trauma
9. Premature suture removal
10. Delayed healing precipitated by corticosteroids and other agents

> Bite wound on dog with closure of compromised tissue



11. Suture placement in scar tissue, which has poor suture-holding ability
12. Excluding the above causes, an underlying healing disorder or delay in healing should be suspected

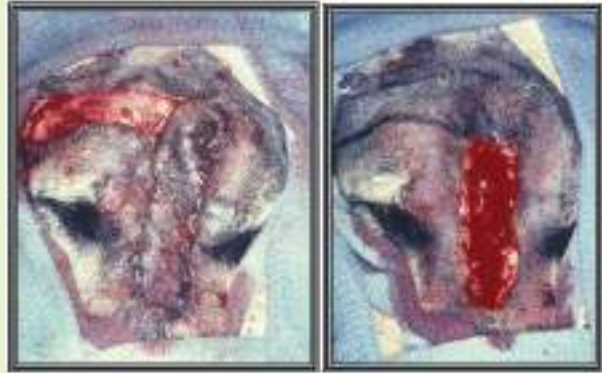
> Directing skin to reduce incisional tension



Key Clinical Points

Critical evaluation of the separated wound can assist the surgeon in determining the most likely cause(s) of dehiscence. Incisional gaps with the absence of sutures suggest the premature removal of sutures, usually by the patient. The presence of the sutures oftentimes demonstrates a suture “cut-out” from one side of the incision, with an irregular right-angle tear from the suture hole to the incision. An intact suture with the presence of an incisional gap is usually associated with stretching or deformation of suture material (from incisional tension or sutures deliberately placed loosely). Often, a veterinarian may place sutures loosely when edema of the skin is evident or anticipated. When the swelling subsides, the incisions retract, leaving an incisional gap.

> Transposition flap closure (before and after)



Suture “cut-out” can occur from placement too close to the incision. Because collagenase activity remains high within 5 mm of the skin incision, sutures placed near or within this zone are more likely to cut-through the weakened dermal collagen weave, especially in the presence of motion and incisional tension. Larger suture bites are less likely to pull out. Cutting needles have a cutting edge within the curvature of the needle. Under tension, with placement in susceptible skin, the suture is more likely to cut along the “V” shape cut created by the suture in the direction of the incision. Today, most surgeons use *reverse cutting needles* (cutting edge on the outer curvature of the needle) almost exclusively since the flat edge of the needle hole facing the incision line is more resistant to tissue tear.

For example, delays in healing from inflammation, trauma, or malnutrition will further increase the likelihood of dehiscence. Suture placement, pattern, and selection are particularly important under these circumstances. At the time of traumatic wound closure, it is not always clear whether the onset of necrosis resulted from “dying skin” or additional ischemia precipitated by tight suture placement. Large areas of skin necrosis would suggest preceding trauma was primarily responsible for dehiscence. In areas with ample regional skin, aggressive debridement could prevent dehiscence by removal of compromised or potentially compromised tissues. Necrosis limited to the suture site would be compatible with suture compromise to the skin.

Selecting an elastic suture material that can stretch to a limited degree to accommodate possible tissue swelling but is of sufficient size and strength to prevent permanent stretch deformation is useful. Suture material size and selection should be tailored to the skin thickness, durability, location, and the anticipated postoperative incisional forces that require neutralization. In more difficult wound closures, complete familiarity with the physical properties of a given suture material is essential for more consistent success in wound closure.

Closure of an incision under moderate tension increases the likelihood of dehiscence. Closure under tension, with the addition of motion, can further promote suture serration of the skin and “cut-out.” Techniques to offset skin tension should be considered in concert with immobilization of the area to promote uncomplicated healing. *[Closure of wounds under excessive tension is the most common cause of wound dehiscence.]*

Underlying pockets of infection, necrotic tissue, or foreign bodies will result in persistent wound drainage at the expense of incisional closure until the underlying causes are alleviated. Future continuous discharge and prolonged moisture exposure can hydrate or soften the skin, enhancing the likelihood of

suture cut-out. As a result, failure to abide by basic wound management principles can negatively affect wound healing and closure until the problems are recognized and remedied.

Proper bandage and external support can be critically important to preventing dehiscence in areas subject to motion or weight-bearing. Care and proper bandage care are used to avoid circulatory compromise during bandage application.

Surgical Options to Reduce Surgical Tension

- Careful undermining
- Release or relaxing incisions
- Load cycling with skin hooks — intraoperative skin stretching
- Tacking sutures, including walking sutures
- Skin flaps
- Skin stretchers (Contact Dr. Pavletic if needed)

Open Wound Management

The area of dehiscence is thoroughly examined to determine what factors likely led to this complication. Those identifiable causes will need to be eliminated.

With wound dehiscence, the condition of the tissues is evaluated for tissue necrosis and the presence of infection. Debridement and a variable period of open wound management may be required before surgical closure can be attempted. Depending on the size and location of the wound, healing by second intention may be the best option.

As noted, closure under tension is the most common cause of wound dehiscence. Surgical methods to reduce tension of the closure should be considered, along with the appropriate immobilization of the area if motion is a contributing factor.

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Marijuana Toxicity in Dogs

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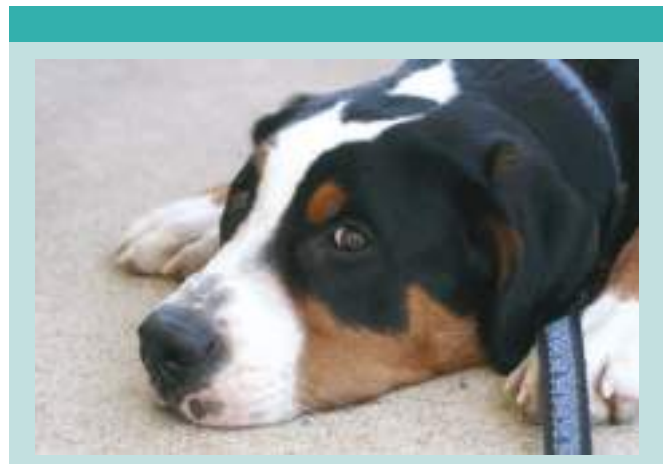
The recreational, psychogenic drug, marijuana, is formed from the leaves and tops of the hemp plant (*Cannabis sativa*) and has been used in many different cultures for a variety of different recreational, religious and medical purposes for over 5,000 years (Krietzer, 2009). Interestingly enough, marijuana was actually one of the most commonly prescribed medications in the United States until it was made illegal in the early 1900s and then declared a Class II controlled substance (Burns, 2006). Over the last few decades, marijuana's effectiveness in pain control and appetite stimulation has led to legalization for medical use in several states. As many may already know, the people of Massachusetts voted to legalize marijuana for recreational use in November 2016, which went into effect on December 15, 2016, making marijuana and marijuana products (i.e, edibles) more readily available to the public.

So, what does this mean for pets? Well, marijuana ingestion/toxicity is actually one of the more common toxicities seen in dogs through the ER, perhaps aside from chocolate and raisins/grapes. The active, psychogenic ingredient in marijuana is delta 9-tetrahydrocannabinol (THC), which can vary greatly in concentration depending on the product (much higher concentration in hashish and hashish oils). This should be taken into account in cases of toxicity.

Over 99% of cases following ingestion of marijuana present with neurologic signs consistent with Central Nervous System (CNS) depression, including ataxia (incoordination when walking), head bobbing, lethargy/listlessness, dilated or pinpoint pupils, slow or rapid heart rate (depending on amount of marijuana ingested) and often urinary incontinence (dribbling urine). In some, but not all cases, low blood pressure and body temperature may also be seen. Onset of signs varies based on the individual patient's metabolism, but most commonly is within one to three hours of ingestion and can last anywhere from 30 minutes to 96 hours (based on the amount and type of marijuana ingested) with the average being 18 to 24 hours. Ingestion of hashish or marijuana butter tends to result in more severely affected animals, who can have serious enough CNS depression to result in seizures or coma and the need for temporary mechanical ventilation.

Initial treatment recommendations for any pet having ingested marijuana will vary based on timing and/or degree of clinical signs present. If less than 30 minutes have passed since known ingestion of marijuana or a marijuana product, emesis (vomiting) can be induced to physically remove the compound from the system. If more than 30 minutes have passed since known ingestion, the anti-nausea effects of the THC will likely be strong enough that it will be difficult to induce vomiting. Additionally, vomiting should not be induced in any clinically depressed dogs for risk of aspiration pneumonia. Although not always necessary, administration of activated charcoal by mouth every eight hours for 24 hours can help to reduce the severity and duration of signs; often times, exposure is minor enough that this is not necessary (Donaldson, 2002).

For mildly affected patients, simple outpatient care with subcutaneous fluids (to encourage faster excretion of the toxin) and minimizing stimulation (i.e, provide a quiet, dark environment) at home is recommended, with most animals recovering within 12 to 24 hours. For more severely affected animals,



hospitalization for intravenous fluid therapy, close monitoring of heart and respiratory rate, as well as sedatives for agitation are sometimes needed. Uncommonly, CNS depression can be severe enough to induce coma, necessitating temporary mechanical ventilation until the drug can be metabolized and excreted (Osweiler et al).

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Non-infectious Diseases of Psittacines

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When breaking down topics related to pet bird medicine, several unrelated yet common conditions fall under non-infectious diseases. Many present as emergencies, given that bird illnesses can progress quickly. The focus of this article is to discuss common presentations and the management of these various conditions.

allows the metal to dissolve and be absorbed by the small intestine. Clinical signs encountered depend on the exposure dose.

An acute ingestion of a large volume results in anorexia, depression, PU/PD, hematuria, paresis, and death. When a smaller amount is ingested at once or more chronic exposure over a more extended period, you are more likely to encounter weight loss, regurgitation, paresis, and nonspecific signs related to secondary immunosuppression. Typical findings on CBC include leukocytosis characterized by heterophilia and hypochromic regenerative anemia. The chemistry panel may reveal elevations in uric acid, CK, and LDH, as well as hypoproteinemia. Radiographs can be suggestive but not definitive. As many of these patients are unstable, a “box shot” is often obtained to look for evidence of opacities more opaque than bone. It is important to note that certain non-toxic materials can appear opaque, like metal. Paint chips are not always visible and can be comparable to mineral opacity. Confirmation of the diagnosis should be performed on serum or tissue submissions. Treatment plans include basic stabilization and removal of the metal source if possible. The most commonly used chelator is calcium disodium salt of ethylenediaminetetraacetic (CaEDTA), which is administered parenterally and tends to be more effective at eliminating lead stored in bone. Stable complexes with lead are formed, which are then excreted through urine. Meso 2,3-dimercaptosuccinic acid (DMSA) is often used as the oral chelation of choice but is more effective at eliminating lead from soft tissue sources. Regurgitation has been noted even at standard therapeutic doses.



Heavy Metal Toxicity

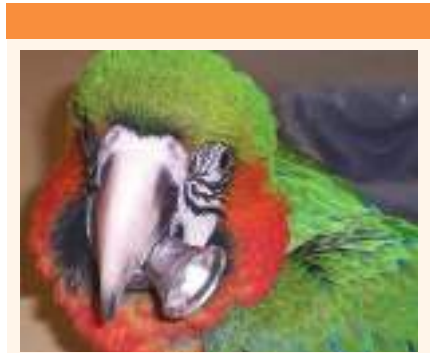
Lead toxicity has long been recognized as the most common cause of toxicosis in birds. Lead paint is one of the most commonly encountered sources. Despite banning lead paint in 1978, many older homes still have lead paint beneath the surface. Given the capacity of flight and potential for destruction with a hook bill, many parrots become exposed without their owner’s knowledge. Stained glass windows, costume jewelry, curtain weights, grout, caulking, and linoleum are just a few other sources of toxicity noted in practice. Once ingested, the low pH of the proventriculus



examines the most useful for diagnosis. Treatment includes removing the metallic object and parenteral treatment with CaEDTA.

Inhalant Toxins

The avian respiratory system is particularly sensitive to inhalant toxins. Teflon, or polytetrafluoroethylene (PTFE), is a common non-stick coating on pans, cooking utensils, waffle irons, drip pans, and specific tin foils.



Zinc toxicity is the second most common heavy metal toxicity encountered in captive birds. Exposure is often through ingesting nuts, bolts, galvanized items, or pennies minted after 1982. Elevated zinc levels result in red blood cell hemolysis, secondary anemia, hemoglobinuria, and potentially renal failure. Nonspecific signs similar to lead toxicity, such as regurgitation, ataxia, paresis, weight loss, and death, can be encountered. Concentration levels in the pancreas and kidneys make sampling them in post-mortem



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When heated above 536°F (260°C), the coating will depolymerize, releasing fumes resulting in arrhythmias or heart failure. Exposed birds will die acutely or present with severe respiratory signs and ataxia. Treatment after mild exposure has been attempted with oxygen supplementation, diuretics, bronchodilators, NSAIDs, and antibiotics. Pulmonary congestion and hemorrhage are typically noted on necropsy.

Avocado Feather Damaging Behavior

There are a few exceptions to foods suitable for parrots to share with their owners. Some of the most toxic foods include avocado, raw onion, garlic, and chocolate. Persin is the toxic substance found in an avocado tree's leaves, fruit, bark, and seeds. The leaves have the highest concentration and tend to be the most toxic. The most common exposure is through ingestion of the fruit. Clinical signs develop between 24 to 96 hours after ingestion. Clinical signs are attributed to myocardial insufficiency and will present as dyspnea, cyanosis, cough, or acute death. Initial treatment should focus on decontamination of the crop and charcoal administration to reduce absorption. Oxygen therapy and diuretics may be useful. Post-mortem findings consist of myocardial necrosis and hemorrhage.

Feather Damaging Behavior

This condition encompasses a broad spectrum of behaviors that damage or remove feathers. Distinguishing changes in feather appearance and excess loss from normal molting is essential. Most parrots undergo a significant turnover of feathers one to two times per year. Light cycles, nutrition, and hormones affect the time frame. Feather damaging behavior (FDB) includes over-preening, feather chewing, and feather plucking. When damage to the skin occurs, this is referred to as self or automutilation. These abnormal behaviors are more common in birds kept in captivity. While every species has the capacity for FDB, it is more common in Cockatoos and African Greys. FDB is multifactorial but is broadly classified as either medical or behavioral. A detailed history is



helpful in an acute presentation of FDB to better pinpoint contributing factors. A medical workup, including a thorough physical exam, baseline blood work, three-view radiographs, and protein electrophoresis, is suggested to get an overall picture of the patient's health. Additional testing should be considered based on the risk factors determined in the history. Treatment is based on medical, socioenvironmental, and psychological assessment.

Egg Binding

Egg binding occurs when an egg is not laid in the expected period of time through the oviduct. Most parrots produce an egg every other day, while 80% of that time is spent in the shell gland at the distal aspect of the oviduct. This is the most common reproductive emergency and is more often seen in smaller species of parrots. Presenting complaints can include straining, weakness, dyspnea, trouble standing, and general malaise. A palpable egg is often identified within the coelomic window. Standing radiographs should be performed to confirm the diagnosis and better characterize the size, shape, and number of eggs present. A thorough history will often reveal poor nutrition in combination with a recent history of excessive egg production. However, egg binding can occur with any prior reproductive history — implementation of supportive care, including heat support, subcutaneous fluids, calcium supplementation, and nutritional support. Pain medications or antibiotics are indicated if secondary salpingitis or tissue trauma is suspected. If no egg is produced after the first 24 to 48 hours of support, assisted delivery under

general anesthesia is recommended. If the egg cannot be removed with gentle pressure, then percutaneous or transoviductal collapse can be achieved by removing the contents with a needle and syringe. Surgical intervention is recommended if the shell has become adhered or torsion is suspected.

Prolapse

Tissue protruding from the vent can be identified as cloacal, oviduct, or colon. Oviductal prolapse is often related to recent egg production, excessive straining, or adhesion of an egg to the shell gland. This tissue is best identified by longitudinal striations and a central lumen that accommodates the passage of an egg. Cloacal prolapse has been associated with behavioral and nutritional issues, infection, neoplasia, papillomatosis, and secondary reproductive complications. Evaluation for tissue damage and viability will impact prognosis. Lubrication and gentle replacement can improve viability. Lateral sutures to reduce the vent opening may be required to keep the tissue in place while the swelling resolves. This reduces the vent aperture, allowing feces to pass without tissue prolapse. Addressing the underlying etiology is necessary to prevent temporary resolution. Permanent surgical solutions should be considered for patients with recurrent prolapse secondary to behavioral or reproductive causes. Salpingohysterectomy, cloacopexy, and ventplasty should be considered. Behavioral, environmental, and nutritional modifications are necessary to improve long-term success after surgical intervention.

Iron Storage Disease

Rhamphastids, Mynahs, Birds of Paradise, and certain frugivorous parrot species (Lories, Lorikeets) are prone to hemochromatosis. Many of these species consume low-iron foods in areas with low mineral density in the soil. As a result, they are highly efficient at extracting and storing minerals. These species have no way of downregulating absorption and end up storing excessive iron in the hepatocytes as hemosiderin. Adding ascorbic and citric acid to the diet also



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aids translocation across the intestine and subsequent storage. Therefore, it is recommended to feed less than 100 ppm of iron per day and low-acid food items to susceptible species. This can be achieved by feeding select fruits and a commercial pellet specific for low-iron species. Clinical signs are nonspecific but generally reflect reduced hepatic function. Weight loss, ascites, dyspnea, and acute death have all been reported. Antemortem diagnosis remains difficult as blood tests are not sensitive or specific. Total iron binding capacity and serum iron levels have not been correlated with hepatic iron storage. Imaging abnormalities on survey radiographs or ultrasounds can increase suspicion but are non-confirming. Definitive diagnosis is most reliable through histology of liver biopsy using Prussian blue stains. Toxicology levels can also be determined on liver samples submitted. Treatment is based on diet modification, serial phlebotomy, and chelation. Diet modification is best used as a preventative measure, but reducing iron content to 100 ppm should be implemented. Phlebotomy is the mainstay of treatment in other species. This is contraindicated in patients who develop anemia or are diagnosed with concurrent heart disease. Chelation with deferoxamine (100mg/kg SQ Q24 x 16 weeks) has been reported and found to reduce hepatic iron storage through serial biopsies.

Atherosclerosis

Heart disease was once considered uncommon in parrot species but has since been shown to be a significant contributing factor to shorter life spans in captive species. Atherosclerosis is a chronic degenerative disease of the arteries through an accumulation of fat and cholesterol and inflammatory debris within the lumen of the vessel. Over time, this reduces the diameter and elasticity of the vessels, predisposing them to stenosis, thrombosis, and ischemia. While this is by far the most common post-mortem cardiac finding in companion psittacines, it is also suspected to be the main underlying cause for the non-infectious manifestation of heart disease. The most common places to find lesions are within the ascending aorta, brachycephalic trunks, and pulmonary arteries. Although less common, lesions such as the coronary and carotid arteries can also develop in peripheral vasculature. While any species can develop atherosclerosis, there is a predilection for African Grey parrots, Amazons, and Cockatiels. Macaws and cockatoos appear to be less susceptible. One study also found a positive correlation with advanced age, female sex, female reproductive disease, and concurrent hepatic disease. High-fat diets and reduced exercise are also contributing factors. Preventative measures are focused on diets high in pellets and fresh foods and encouraging exercise through flight and foraging activities.

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(W/B) Services available at both our Waltham and Boston locations

*Boston-based pathologists and radiologists serve both Boston and Waltham locations

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› Courtesy Shuttle for Patients Needing Further Specialized Care

Angell Animal Medical Center offers the convenience of our MSPCA-Angell West facility in Waltham, MA. The Waltham facility offers Urgent Care and specialized service appointments. If needed, an oxygen-equipped courtesy shuttle can transport animals to Boston for further specialized care and then return them to Waltham. Whether in Boston or in Waltham, our specialists regularly collaborate and plan treatments tailored to our patients' emergency, surgical, and specialty needs.

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