#SIENTELAEXPERIENCIA

MEMORIAS

LAVC'18

La Conferencia De Educación Continua En Medicina Veterinaria Más Importante De Latinoamérica

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MEDICINA DE ANIMALES DE COMPAÑÍA Y MASCOTAS EXÓTICAS

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Acute Abdomen Diagnosis Surgery and Post Op Care Final

PRESENTATION: *Acute abdomen is defined as the sudden onset abdominal discomfort or pain due to a variety of conditions involving intraabdominal organs. Many animals present with the primary complaint of lethargy, anorexia, ptyalism, vomiting, retching, diarrhea, hematochezia, crying out, moaning, or abnormal postures. Abnormal postures can include generalized rigidity, walking tenderly or as if "on eggshells", or a prayer position in which the front limbs are lowered to the ground while the hind end remains standing. In some cases, it may be difficult to initially distinguish between true abdominal pain or referred pain from intervertebral disk disease. Rapid progression and decompensation of the patient's cardiovascular status can lead to stupor, coma, and death in the most extreme cases, making rapid assessment, treatment and definitive care extremely challenging.*

SIGNALMENT AND HISTORY

Often the patient's signalment and history can increase your index of suspicion for a particular disease process. A thorough history is often overlooked or postponed in the initial stages of resuscitation of the patient with acute abdominal pain. Often, asking the same question in a variety of methods can elicit an answer from the client that may lead you to the source of the problem and the reason for acute abdominal pain. Important questions to ask the client include: What is your chief complaint or reason that you brought your animal in on emergency? When did the signs first start or when was your animal last normal? Do you think that the signs have been the same, better, or getting worse? Does your animal have any ongoing or past medical problems? Have similar signs occurred in the past? Does your animal have access to any known toxins or does he or she run loose unattended? Has your animal ingested any garbage, compost, or table scraps recently? Are there any other animals in your household and are they acting sick or normal? Has your animal been vaccinated recently? Has there been any change in your pet's appetite? Have you noticed any weight loss or weight gain? Have you noticed any increase or decrease in water consumption or urination? Does your animal chew on bones or toys? Have you noticed any toys, socks, underwear, or other items missing from your household? Is there a possibility of any trauma including being hit by a car or kicked by a larger animal or person? Have you noticed a change in your pet's defecation habits? Have you seen any vomiting or diarrhea? What does the vomitus or diarrhea look like? Is the vomitus in relation to eating? Is there any blood or mucous in the vomitus or diarrhea? When was the last time your animal vomited or had diarrhea? When your animal vomits, do they actively retch with abdominal contractions, or is it more passive like regurgitation? What is the color of the feces? Is it black or red in color? Does the vomit smell malodorous like feces?

IMMEDIATE ACTION

Physical Examination As with any other emergency, the clinician must follow the

A,B,C's of therapy, treating the most life-threatening problems first. First, a perfunctory physical examination should be performed. Examination of the abdomen should ideally be performed last, in case inciting a painful stimulus precludes you from evaluating other organ systems more thoroughly. Briefly observe the patient from a distance. Are there any abnormal postures? Is there respiratory distress? Is the animal ambulatory and if so, do you observe any gait abnormalities? Do you observe any ptyalism or attempts to vomit? Auscult the patient's thorax for crackles that may signify aspiration pneumonia secondary to vomiting. Examine the patient's mucous membrane color and capillary refill time, heart rate, heart rhythm, and pulse quality. Many patients in pain have tachycardia that many or may not be accompanied by dysrhythmias. If a patient's heart rate is inappropriately bradycardic, consider hypoadrenocorticism, whipworm infestation, or urinary obstruction or trauma as a cause of hyperkalemia. Assess the patient's hydration status by evaluating skin turgor, mucous membrane dryness, and whether the eyes appear sunken in their orbits. A brief neurologic examination should consist of whether the patient is actively having a seizure, or whether mental dullness, stupor, coma, or nystagmus are present. Posture and spinal reflexes can assist

in making a diagnosis of intervertebral disk disease versus abdominal pain. Perform a rectal examination to evaluate for the presence of hematochezia or melena. Finally, examination of the abdomen should proceed, first with superficial, then deeper palpation. Visually inspect the abdomen for the presence of external masses, bruising, or penetrating injuries. Reddish discoloration of the periumbilical area is often associated with the presence of intraabdominal hemorrhage. It may be necessary to shave the fur to visually inspect the skin and underlying structures for bruising and ecchymoses. Auscult the abdomen for the presence or absence of borborygmi to characterize gut sounds. Next, perform percussion and ballottement to evaluate for the presence of a gas-distended viscus or peritoneal effusion. Finally, perform first superficial, then deep palpation of all quadrants of the abdomen, noting abnormal enlargement, masses, or whether focal pain is elicited in any one area. Once the physical examination has been performed, initial therapy in the form of analgesia, fluid resuscitation, and antibiotics should be implemented.

TREATMENT

Treatment for any patient with acute abdomen and shock is to treat the underlying cause, maintain tissue oxygen delivery, and prevent end-organ damage and failure.

Analgesia The administration of analgesic agents to any patient with acute abdominal pain is one of the most important therapies in the initial stages of case management. A list of analgesic drugs for use in the patient with acute abdomen is listed in Table 1.

Fluid Resuscitation Many patients with acute abdominal pain are clinically dehydrated or are in hypovolemic shock secondary to hemorrhage. Careful titration of intravenous crystalloid and colloid fluids including blood products are necessary based on the patient's perfusion parameters including heart rate, capillary refill time, blood pressure, urine output and packed cell volume. Fluid therapy should also be based on the most likely differential diagnoses, with specific fluid types administered according to the primary disease process. In dogs, a shock volume of fluids is calculated based on the total blood volume of 90 ml/kg/hour. In cats, shock fluid rate is based on plasma volume of 44

ml/kg/hour. In most cases, any crystalloid fluid can be administered at an initial volume of ¼ of a calculated shock dose, then titrated according to whether the patient's cardiovascular status responds favorably or not. In cases of acute abdomen secondary to known or suspected hypoadrenocorticism, severe whipworm infestation, or urinary tract obstruction or rupture, 0.9% sodium chloride fluid without added potassium is the fluid of choice. When hemorrhage is present, the administration of whole blood or packed red blood cells may be indicated if the patient is clinical for anemia, and showing clinical signs of lethargy, tachypnea, and weakness. Fresh frozen plasma is indicated in cases of hemorrhage secondary to Vitamin K antagonist rodenticide intoxication or hepatic failure, or in cases of suspected disseminated intravascular coagulation (DIC).

Antibiotics The empiric use of broad-spectrum antibiotics is warranted in cases of suspected sepsis or peritonitis as a cause of acute abdominal pain. Ampicillin sulbactam (22 mg/kg IV Q6 – Q8 hours) and enrofloxacin (10 mg/kg once daily) are the combination treatment of choice to cover gram-negative, grampositive, aerobic, and anaerobic infections. Alternative therapies include a 2nd-generation cephalosporin such as Cefotetan (30 mg/kg IV TID) or Cefoxitin (22 mg/kg IV TID), or added anaerobic coverage with Metronidazole (10 – 20 mg/kg IV TID). Aminoglycoside antibiotics should be avoided in a hypotensive, hypovolemic or dehydrated patient due to the risk of causing acute kidney injury.

Oxygen Supplementation Tissue oxygen delivery is dependent on a number of factors including arterial oxygen content and cardiac output. If an animal has had vomiting and subsequent aspiration pneumonitis, treatment of hypoxemia with supplemental oxygen in the form of nasal, nasopharyngeal, hood, or transtracheal oxygen administration is extremely important.

DIAGNOSTIC PROCEDURES

Complete Blood Count A complete blood count should be performed in all cases of acute abdominal pain to determine if life-threatening infection. Coagulating testing (Prothrombin time, activated partial thromboplastin time) should be considered if a coagulopathy or disseminated intravascular coagulation are suspected. In cases of sepsis, infection, or severe non-septic inflammation, the white blood cell count may be normal, elevated, or low. A peripheral blood smear should be examined and evaluated for the presence of toxic neutrophils, eosinophils, atypical lymphocytes, nucleated red blood cells, platelet estimate, anisocytosis, and blood parasites. A falling PCV in the face of red blood cell transfusion is suggestive of ongoing hemorrhage.

Biochemistry Panel A biochemistry panel should be performed to evaluate organ system function. Azotemia with elevated BUN and creatinine may be associated with pre-renal dehydration, impaired renal function, or post-renal obstruction or leakage. The BUN can also be elevated when gastrointestinal hemorrhage is present. Serum amylase may be elevated with decreased renal function, or in cases of pancreatitis. A normal amylase, however, does not rule out pancreatitis as a source of abdominal pain. Serum lipase may be elevated with GI inflammation or pancreatitis. Like amylase, a normal lipase does not rule out pancreatitis. Total bilirubin, alkaline phosphatase, and ALT may be elevated with primary cholestatic or hepatocellular diseases, or due to extrahepatic causes including sepsis.

Urinalysis A urinalysis should be obtained via cystocentesis whenever possible, except in cases of suspected pyometra or transitional cell carcinoma. Azotemia in the presence of a nonconcentrated (isosthenuric or hyposthenuric) urine is suggestive of primary renal disease. Secondary causes of apparent renal azotemia and lack of concentrating ability also occur in cases of hypoadrenocorticism and Gram-negative sepsis. Renal tubular casts may be present in cases of acute renal ischemia or toxic insult to the kidneys. Bacteriuria and pyuria may be present with infection and inflammation. When a urinalysis is obtained via free catch or urethral catheterization, the presence of bacteriuria or pyuria may also be associated with pyometra, vaginitis, or prostatitis/prostatic abscess. Lactate Serum lactate is a biochemical indicator of decreased organ perfusion, decrease oxygen delivery or extraction, and end-organ anaerobic glycolysis. Elevated serum lactate > 6 mmol/L has been associated with increased sensitivity in cases of GDV, and increased patient morbidity and mortality in other disease processes. Rising serum lactate in the face of adequate fluid resuscitation is a negative prognostic sign.

ABDOMINAL RADIOGRAPHS

Abdominal radiographs should be performed as one of the first diagnostic tests when deciding whether medical versus surgical management should be pursued. The presence of gastric dilatationvolvulus, linear foreign body, pneumoperitoneum, pyometra, or splenic torsion warrants immediate surgical intervention. If a loss of abdominal detail secondary to peritoneal effusion, additional diagnostic tests including abdominal paracentesis (abdominocentesis) and abdominal ultrasound should be performed to determine the cause of the peritoneal effusion.

Abdominal Ultrasound Abdominal ultrasonography is often useful in place of or in addition to abdominal radiographs. The sensitivity of abdominal ultrasonography is largely operator dependent. Indications for immediate surgical intervention include loss of blood flow to an organ, linear bunching or plication of the intestinal tract, intussusception, pancreatic phlegmon or abscess, a fluid-filled uterus suggestive of pyometra, gastrointestinal obstruction, intraluminal GI foreign body, dilated bile duct, or gall bladder mucocoele, or gas within the wall of the stomach or gall bladder (emphysematous cholecystitis). The presence of peritoneal fluid alone does not warrant immediate surgical intervention without cytologic and biochemical evaluation of the fluid present.

ABDOMINOCENTESIS

Abdominal paracentesis (abdominocentesis) often is the deciding factor in whether to perform immediate surgery or not. Abdominocentesis is a sensitive technique for detecting peritoneal effusion when >6 ml/kg of fluid is present within the abdominal cavity. Abdominal effusion collected should be saved for bacterial culture, and evaluated biochemically and cytologically based on your index of suspicion of the primary disease process. If creatinine, urea nitrogen (BUN) or potassium is elevated compared with that of serum, uroabdomen is present. Elevated abdominal fluid lipase or amylase compared with serum supports a diagnosis of pancreatitis. Elevated lactate compared with serum lactate, or an abdominal fluid glucose < 50 mg/dL is highly sensitive and specific for bacterial/septic peritonitis. The presence of bile pigment or bacteria is

supportive of bile and septic peritonitis, respectively. Free fibers in abdominal fluid along with clinical signs of abdominal pain strongly support gastrointestinal perforation, and requires immediate surgical exploration.

Diagnostic Peritoneal Lavage (DPL) In the event of a negative abdominocentesis, but peritoneal effusion, or bile or gastrointestinal perforation are suspected, a diagnostic peritoneal lavage can be performed. Peritoneal dialysis kits are commercially available, but are often expensive and impractical.

DIFFERENTIAL DIAGNOSES

Differential diagnoses for acute abdominal pain, vomiting, and diarrhea are numerous. Broad categories include primary gastrointestinal disease (viral, bacterial, parasitic, pancreatitis, gastrointestinal foreign body/obstruction, inflammatory bowel disease, intussusception, neoplasia, gastrointestinal ulceration/perforation, gastric or colonic torsion), versus other diseases (liver lobe torsion, hepatic abscess, bile peritonitis, gall bladder mucocoele, ureteral obstruction, uroabdomen, pyelonephritis, splenic torsion, pyometra, prostatitis, prostatic abscess, neoplasia, hemoabdomen).

MANAGEMENT

Animals that present with acute abdominal pain can be divided into three broad categories, depending on the primary cause of pain, and the initial definitive treatment. Some diseases warrant a nonsurgical, medical approach to case management. Other conditions require immediate surgery following rapid stabilization. Other conditions can initially be managed medically until the patient is hemodynamically more stable, then may or may not require surgical intervention at a later time.

EXPLORATORY LAPAROTOMY/CELIOTOMY

Whenever there is penetrating abdominal injury, evidence of intra- and extracellular bacteria in peritoneal fluid, pneumoperitoneum on radiographs, or biochemical evidence of septic peritonitis, uroabdomen or bile peritonitis, an exploratory laparotomy should be performed. The best means to accurately and thoroughly explore the abdominal cavity is to open the abdomen on midline from the level of the xyphoid process caudally to the pubis for full exposure, then perform evaluation of all organs in every quadrant in a systematic manner. Specific problems such as gastric or splenic torsion, enteroplication, foreign body removal, etc. should be addressed, then the abdomen copiously lavaged with warmed sterile saline or Lactated Ringers solution. The fluid should be suctioned thoroughly from the peritoneal cavity so as to not impair macrophage function. In cases of septic peritonitis, the abdomen should have a closed suction drain placed for further drainage during the post-operative period. The routine use of antibiotics in irrigation solutions is contraindicated, as the antibiotics can irritate the peritoneum and delay healing.

Anesthesia and Pain Management of the Critical Patient Final

PRESENTATION: *General anesthesia involves the careful and judicious use of compounds that induce sensory deprivation to noxious stimuli, muscle relaxation, and in most cases, unconsciousness. In critically ill patients, there often is a delicate balance between loss of consciousness and cardiovascular and respiratory compromise, requiring careful monitoring techniques to ensure patient safety. Fortunately, many animals that present to you in an emergency setting will be young, healthy animals that may require anesthesia to repair minor trauma. Other cases, however, will present to you with potentially life-threatening critical illnesses, making anesthesia more challenging and somewhat risky. Many anesthetic agents induce some degree of cardiovascular and respiratory depression. The goal of this presentation is to describe anesthetic protocols for both healthy and unhealthy animals.*

THE PHYSICAL EXAMINATION

The physical examination is one of the most important aspects of preparation prior to inducing anesthesia. In critically ill patients, a careful physical examination should be performed just prior to inducing anesthesia, as clinical status may have changed dramatically since initial presentation, often changing your choice of anesthetic protocols. Questions to ask yourself include: Is the patient's airway patent? Does the animal require mechanical or assisted ventilation? Is the animal morbidly obese or have an intraabdominal mass that will change efficacy of ventilation once the patient is placed in dorsal recumbancy? Does the animal have a sucking chest wound, rib fractures, pleural effusion, or potential for pneumothorax? Are there pulmonary contusions that may be affected by large volumes of intravenous fluids? Are the animal's respirations effective or ineffective? Is the animal in a state of circulatory shock? Is there a normal circulating blood volume? Is the heart beating efficiently, or is there a cardiac murmur or dysrhythmia? What is the clinical status of the animal? Is there an adequate blood pressure? Is there any sign of ongoing hemorrhage or severe electrolyte loss? In most cases, the answers to these questions can be obtained from a thorough and systematic physical examination, starting with the basic ABC's of emergency medicine. The animal must have a patent Airway, to deliver oxygen into the lungs while Breathing, and the oxygen can be delivered to tissues during the process of normal Cardiac function and Circulation. Once the ABC's have been evaluated and stabilized, other diagnostics can then be performed.

BLOODWORK AND ELECTROLYTES ABNORMALITIES

Following physical examination, bloodwork should be performed to evaluate the patient's oxygen carrying capacity, renal and hepatic function, and coagulation status. Every effort should be made to normalize values prior to inducing anesthesia. However, in many cases, until the underlying problem is definitively addressed, patient clinical status may not improve despite very aggressive efforts.

These types of cases present the most challenge for the veterinary practitioner in deciding how to induce general anesthesia without potentially doing more harm. Patients with traumatic uroabdomen should be stabilized prior to inducing general anesthesia. Every effort should be made to decrease serum potassium to less than 7 mmol/L before any anesthesia is induced. Treatment protocols include administering regular insulin (0.25 units/kg IV) and dextrose (2 gm dextrose IV per unit of insulin, followed by 2.5 – 5% dextrose CRI to prevent hypoglycemia), intravenous sodium bicarbonate (0.25 – 1.0 mEq/kg), or calcium gluconate (0.5 – 1.0 ml/kg 10% solution IV). The electrocardiogram should be monitored closely for the appearance of atrial standstill and inappropriate bradycardia. Secondly, drainage of intraabdominal fluid can be accomplished by placement of an intraabdominal catheter attached to a closed collection system. A red rubber tube or an Argyle chest tube can be placed using a local anesthetic such as lidocaine (0.5 – 1.0 mg/kg). Once secured, the drain can be left in place until definitive repair of the urinary tract can be performed at the time of surgery. Anemic or hemorrhaging patients should have a combination of crystalloid and colloidal support. In cases of hemoabdomen and gastric dilation-volvulus, synthetic hemoglobin can be administered as a bolus or as a slow trickle (1 – 2 ml/kg/hour) to provide both colloidal support and improve oxygen carrying capacity.

Preanesthetic Agents There are several rationales for using pre-anesthetic medications. One of the most important reasons for using premedications is to decrease the total amount of anesthesia required to induce and maintain general anesthesia. The use of a balanced anesthetic approach provides many benefits for the patient, particularly those that are critically ill. Anticholinergic drugs such as atropine and glycopyrrolate increase heart rate by inhibiting vagal stimulation. Glycopyrrolate though, has less of a chance of inducing tachyarrhythmias. Atropine reduces respiratory secretions and can cause second-degree heart block. Atropine crosses the blood-brain and placental barriers, while glycopyrrolate does not. This has important implications when providing anesthesia for the periparturient dam in need of a C-section, anesthesia that can potentially affect the outcome of the neonates. Opioids, used in combination with a phenothiazine tranquilizer such as acepromazine, provide neuroleptanalgesia. Morphine provides excellent analgesia without inducing severe cardiovascular compromise. Potential complications of morphine and other opiate drugs include bradycardia (which can be reversed or prevented by using an anticholinergic agent), and hypoventilation. Morphine administration can also induce vomiting and ileus in ambulatory patients. In recumbent patients, though, the use of morphine is justified by decreasing doses of induction agents and inhalant drugs required to maintain general anesthesia. Butorphanol, a mu antagonist, kappa agonist, also can be used as a premedication when used in combination with a phenothiazine tranquilizer such as acepromazine. Used alone, however, butorphanol's sedative effects are fairly unreliable and short-lived. Additionally, due to its receptor affinity, using butorphanol early in the course of anesthesia may prevent more potent drugs such as morphine and fentanyl from providing adequate analgesia in the early post-operative time period, depending on the length of surgery. For these reasons, this author does not routinely use butorphanol, favoring more potent and more reliable opioids such as morphine and fentanyl. Fentanyl, a pure opioid agonist, is potent opioid with a very short duration of action. It should be used in very critical patients for analgesia, then as part of an

induction protocol. When used as a premedication, induction of general anesthesia should occur shortly thereafter, within approximately 20 minutes, or the drug should be given as a constant rate infusion until general anesthesia is induced. Phenothiazine tranquilizers, namely acepromazine, should be given to healthy animals as part of the premedication protocol. Acepromazine's antagonism of alpha-adrenergic activity can potentially induce vasodilation with subsequent hypotension, so should be used with caution. Other untoward side effects that have been reported include reduction of seizure threshold in predisposed animals. Thus, its use is relatively contraindicated in such patients. A potentially beneficial side effect of acepromazine is decreasing catecholamine-induced dysrhythmias. Alpha-2 agonists such as xylazine and medetomidine induce intense peripheral vasoconstriction, AV nodal block, bradycardia, and decrease in cardiac output. For these reasons, alpha-2 agonists should never be administered to emergency and critical care patients for absolutely any reason. The alpha-2 agonists may have their place in healthy animals, but should not be used in emergent settings.

ANESTHETIC INDUCTION

Anesthetic induction should occur rapidly. The most critically ill patients often will benefit from preoxygenation prior to induction. An intravenous catheter should be in place prior to induction, for maintenance of vascular access. Benzodiazepene tranquilizers such as diazepam (0.3 – 0.5 mg/kg IV) or midazolam can be used. Diazepam induces more reliable tranquilization and is less expensive than the more costly midazolam. If given alone, diazepam can potentially induce excitement; therefore, this drug is often used in combination with dissociative agents such as ketamine (5.5 mg/kg IV, 10 – 30 mg/kg IM), opioids such as fentanyl (10 mcg/kg IV), or in combination with etomidate (0.5 – 1.5 mg/kg IV). Ketamine is a dissociative anesthetic agent that will initially cause a catecholamine-induced increase in cardiac output. In critically ill patients that have maximized sympathetic tone, however, ketamine can decrease cardiac contractility; therefore its use is relatively contraindicated. Ketamine, when used pre-intra- and post-operative, can decrease activation of NMDA receptor-mediated "wind-up" and decrease post-operative pain even days after surgery. Its use in combination with other analgesic agents is therefore beneficial, especially in controlling post-operative orthopedic pain. Propofol (4 – 7 mg/kg IV) is another drug that can be used to induce general anesthesia. Unrelated to other pharmacologic agents, propofol induces rapid anesthesia. Recovery from Propofol is also very rapid in most cases. Potential untoward effects of this drug include vasodilation and hypotension, and apnea. In cats, Propofol should not be used on consecutive days due to the potential for

development of Heinz body anemia. In the most critically ill patients, Etomidate can be administered along with Diazepam with minimal effects on cardiovascular status.

ANESTHESIA MAINTENANCE AND MONITORING

Immediately after successful induction of anesthesia, anesthesia should be maintained with an appropriate gas anesthetic agent. For short procedures in cardiovascularly stable patients, Propofol can be administered as a constant rate infusion (6 – 20 mg/kg/hour). It must be remembered, however, that Propofol has no analgesic properties; therefore, if a painful procedure is to be performed,

appropriate analgesia should be administered prior to recovery from anesthesia. Proper anesthetic monitoring including pulse oximetry, electrocardiogram, temperature, capnography, and blood pressure should be performed and recorded for even "apparently routine" procedures. In many critical patients, pre-, intra- and post-anesthetic hypotension is a potential hazard that should be carefully addressed. First, the level of gas anesthesia should be decreased. Secondly, a fluid bolus (5 – 10 ml/kg) can be administered IV. All patients under general anesthesia should have vascular access. If a patient is hypotensive and hemodilution or true anemia is a concern, synthetic colloids such as Hydroxyethyl starch can be administered as an IV bolus (5 ml/kg). Alternatively, component blood products such as whole blood, packed red blood cells, or fresh frozen plasma can be administered depending on the primary problems at hand. If decreasing anesthetic depth and fluid bolus does not sufficiently increase blood pressure, use of a positive inotrope such as dobutamine (2 – 20 mcg/kg/min IV CRI) can be administered. Dopamine at lower doses (2 – 10 mcg/kg/min IV CRI) stimulates cardiac contractility through beta-adrenergic stimulation. Dobutamine, primarily a beta- agonist, stimulates cardiac contractility and as such, indirectly increases blood pressure. Dobutamine increases blood pressure more reliably than dopamine. Ephedrine is a synthetic sympathomimetic drug that stimulates both alpha- and beta-adrenergic receptors to stimulate catecholamine release. Bolus injection of ephedrine (0.1 – 0.25 mg/kg IV) has a longer duration of action than dobutamine, thus does not require administration as a constant rate infusion. If none of the above options are successful, vasopressor agents such as dopamine (> 10 mcg/kg/minute IV CRI), epinephrine (0.05 – 0.4 mcg/kg/min IV CRI), and norepinephrine (0.05 – 0.4 mcg/kg/min IV CRI) can also be administered. It is important to remember that agents that induce peripheral vasoconstriction may increase blood pressure but not necessarily improve renal, cerebral, and coronary blood flow. In any patient with tachy- or bradyarrhythmias such as AV block, bradycardia, or sinus or ventricular tachycardia, attempts should be made to treat the dysrhythmia. Anticholinergic drugs such as atropine or glycopyrollate should be administered to treat cardiovascularly unstable bradyarrhythmias. In some cases, if the heart rate is 50, and the animal's blood pressure is stable and normal, treatment may not be necessary, However, if the animal is bradycardic and hypotensive, interventions should be implemented. Sinus tachycardia can adversely affect blood pressure by decreasing the amount of time the heart has to fill. Decrease in diastolic filling will result in a decreased cardiac wall stretch available for rebound, the force necessary for normal myocardial contraction. Therefore, filling the cardiovascular space with fluids, and in some cases, INCREASING anesthetic depth if the animal is actually feeling the procedure, may be necessary to decrease heart rate. Ventricular dysrhythmias should be treated with a combination of crystalloid/colloid therapy, oxygen, and drugs such as lidocaine (2 mg/kg IV bolus, followed by 50 – 100 mcg/kg/minute IV CRI) or procainamide. Procainamide can contribute to refractory hypotension, and is not the antiarrhythmic of choice in a patient with hypotension.

POST-OPERATIVE ANALGESIA

Post-operative analgesia should be performed with the thought that the patient should never ever be allowed to be painful. Constant rate infusions of fentanyl (1 – 7 mcg/kg/hour), morphine (0.1

mg/kg/hour in dogs, 0.01 mg/kg/hour in cats) can be administered with ease. Additionally, at the suggested doses, the drugs cause minimal cardiovascular depression or ileus. Intermittent bolus injections can also occur, provided that the drugs are administered according to an exact schedule, not given when the patient begins acting apparently painful or "PRN". Local anesthetic agents can be placed in the wounds prior to wound closure for post-operative analgesia. Additionally, intrapleural lidocaine (1 mg/kg) or bupivacaine (0.5 – 1.0 mg/kg) can be administered via thoracostomy tubes for additional pain relief. Transdermal fentanyl patches are effective in controlling patient discomfort, but do not immediately take effect, thus requiring other types of analgesia until adequate blood levels of fentanyl are reached, usually 16 – 24 hours after placement of the patch. Some clinicians advocate placing the fentanyl patch on the evening prior to surgery, to ensure adequate blood levels are obtained for the immediate post-operative period. Using combination multimodal approach to therapy such as a constant rate infusion of ketamine (0.5 mg/kg IV bolus at time of anesthetic induction, then 10 mcg/kg/minute intraoperatively, then 2 mcg/kg/minute post-operatively as a CRI), a nonsteroidal anti-inflammatory drug such as carprofen or ketoprofen, epidural morphine, along with opioids helps to ensure that the patient never becomes painful. At the time of discharge, a combination of opioids and/or Nonsteroidal anti-inflammatory drugs should be considered for continued analgesia in the post-operative recovery period. It is sometimes difficulty to distinguish between pain, anxiety, and opioid dysphoria. Physical examination parameters such as heart and respiratory rate, pupil size and responsiveness, should all be checked, but are often indistinguishable from pain, anxiety, need to urinate or defecate, or attention seeking behavior. When I suspect an animal may be painful or dysphoric, I perform a thorough physical examination, including palpation of the surgical site. If the patient reacts to surgical site manipulation, I immediately give an additional dose of analgesia. If no apparent pain is present with surgical site manipulation, but the patient quiets when being handled, anxiety or attention-seeking behavior is diagnosed. I will add an anxiolytic agent to the treatment protocol, provided that hypotension is not a concern. If the patient neither responds to pain nor attention and anxiolytic agents, I make sure that the patient's urinary bladder is empty and there isn't a need to urinate or defecate. Only after all of these choices have failed will I consider reversing an opioid drug with naloxone.

CONCLUSIONS: "WHAT'S THE BOTTOM LINE?"

In the emergent situation, there may not be enough time to take a "wait and see if this works" kind of approach. One of the most important things to remember is that gas is poison. Many animals, particularly those that are critically ill, are exquisitely sensitive to the cardiorespiratory effects of inhalant anesthetic gases. For this reason, when an animal is hypotensive, one of the first things to consider is turning down the anesthetic vaporizer. If there is concern about an animal waking up during the anesthesia and surgery, balanced anesthesia with constant rate infusions of fentanyl or fentanyl and ketamine can be administered, to decrease the total amount of anesthetic gas required to maintain an adequate plane of anesthesia without causing hypotension and cardiovascular compromise. Next, (or sometimes simultaneously), a crystalloid (10 ml/kg) or colloid (5 ml/kg) fluid bolus can be administered, to fill up the vasodilated vascular beds. When a blood

vessel dilates, a state of relative hypovolemia occurs, in which there is inadequate circulating volume to maintain vascular tone and cardiac preload. If there is insufficient myocardial stretch, the force of contraction is limited, and thus, can result in impaired cardiac output and a decrease in systemic blood pressure. Many anesthetic agents render the cardiovascular system incapable of compensatory changes such as vasoconstriction, so blood pressure and thus tissue perfusion and oxygen delivery become compromised. If decreasing the anesthetic depth and administration of fluids does not cause an increase in blood pressure, then positive inotropes (dobutamine and/or ephedrine) and vasopressors (dopamine) can be administered. Having an "anesthetic book" that contains charts of all of the necessary drugs, instructions on how to dilute each drug, resulting concentration, and volume of the diluted drug to administer to each patient based on body weight can save a lot of time and quick arithmetic in an emergent situation.

References available from author upon request.

Emergency Management of Congestive Heart Failure final

PRESENTATION: *Congestive heart failure (CHF) is unfortunately a common problem that presents to the veterinary small animal practitioner. Clinical signs may include weakness and exercise intolerance, cough, lethargy, inappetance, vomiting, diarrhea, and syncope or collapse. A presumptive diagnosis often is made on the patient's primary presenting complaints, signalment, a thorough history, and physical examination findings. One of the most important concepts to remember in the diagnosis and management of any patient with CHF is to minimize patient stress and do no harm.*

PHYSICAL EXAMINATION

A careful physical examination is essential in the diagnosis and management of the patient in CHF. In some cases, the patient should be placed in an oxygen cage or receive flow-by oxygen supplementation, and observed carefully from a distance. Observe the patient's respiratory rate and effort, and posture from afar. Next, approach the patient, and perform a systematic examination, and evaluate the mucous membrane color and capillary refill time? Look carefully at the thoracic inlet and jugular groove for jugular venous distension or a jugular pulse? Auscult the heart for murmurs or dysrhythmias. Simultaneously palpate the inguinal region for a femoral pulse, checking for synchrony and pulse quality. Auscult all lung fields for pulmonary crackles or wheezes. Palpate the abdomen for hepatomegaly and a ballotable fluid wave. Does compression of the cranial abdomen and liver result in jugular venous distension? Palpate the distal extremities. Are they warm to the touch, or do they feel cold due to poor peripheral circulation? Patients with fulminant pulmonary edema from left sided CHF may have blood-tinged fluid coming from the nares and mouth and have concomitant pulmonary crackles and a rapid restrictive respiratory pattern. A cardiac murmur or dysrhythmia is often present in cases of severe mitral insufficiency, but in some cases, the heart may be difficult to hear beyond harsh pulmonary crackles. Pulse quality may be supportive of low output cardiac failure. Pulses may be absent in cases of severe low output failure, or in cases of arterial embolism. Jugular venous distension and jugular pulses may be visible in cases of right-sided heart failure. Heart sounds may be muffled to absent in cases of pleural or pericardial effusion. Hepatomegaly and a fluid wave may be present on abdominal palpation in cases of right-sided heart failure. Distal extremity coolness and hematochezia on rectal examination may be present due to low cardiac output.

EMERGENCY THERAPY

Emergency management of the patient in CHF consists improving systemic oxygen delivery and minimizing patient stress.

Oxygen delivery is a function of both oxygen uptake by the respiratory system, cardiac output, and hemoglobin concentration. The mainstay of therapy for treatment of congestive heart failure is to provide supplemental oxygen and decrease fluid buildup within the lungs.

Oxygen

Flow-by oxygen should be administered to patients with congestive heart failure as a physical examination is taking place. Flow-by oxygen is well-tolerated, and requires minimal physical restraint. Because flow-by is a relatively inefficient method of providing an increase in the fraction of inspired oxygen, other methods including oxygen hoods, oxygen cages, nasal, nasopharyngeal and tracheal oxygen insufflation should be considered for longterm therapy. Humidified oxygen flow rates can be administered at 50 – 100 ml/kg/minute.

Diuretics

Aside from supplemental oxygen supplementation, furosemide is one of the most important therapies for management of the patient with congestive heart failure and pulmonary edema. Furosemide can be administered as a bolus (4 – 8 mg/kg IV or IM), or as a constant rate infusion (0.66 – 1 mg/kg/hour IV) to promote diuresis and decrease pulmonary vascular overload and pulmonary edema. The goal of diuretic treatment is to repeat the therapy every 30 – 60 minutes until the patient's body weight has decreased by 5 – 7%. Once the patient's respiratory rate and effort has normalized, oral furosemide can be started.

Nitric Oxide Donors

Nitric oxide donors should be initiated as a primary initial therapy in any patient with fulminant congestive heart failure. Nitric oxide donors cause dilation of the pulmonary vasculature and a relative decrease in pulmonary vascular pressures. Nitroglycerine paste is not absorbed as readily across the skin as was once previously thought, and has largely fallen out of favor for use in veterinary patients with CHF. In patients with refractory pulmonary edema that is not responding to traditional diuretic therapy, sodium nitroprusside should be considered, as long as the patient is not hypotensive. Sodium nitroprusside is a balanced arteriolar and venous dilator that decreases both pulmonary and systemic vascular resistance. The drug is administered as a constant rate infusion (2 – 10 mcg/kg/minute IV, titrated to effect). Because of its potent hypotensive effects, arterial blood pressure must be monitored closely throughout the infusion.

Morphine

Morphine is an opioid agonist that is useful in patients with congestive heart failure. In dogs, low dose (0.025 – 0.05 mg/kg IV) morphine dilates the splanchnic vasculature and increases venous capacitance, allowing drainage of fluid from the pulmonary parenchyma. Morphine provides the additional benefits of allowing slower, deeper respirations, and decreasing anxiety in patients with congestive heart failure.

Positive Inotropes Dobutamine is a synthetic beta-adrenergic agonist that is used as a positive inotrope, specifically in patients with DCM. At lower doses, dobutamine improves cardiac contractility with minimal effects on chronotropy or heart rate. At higher, doses, however, dobutamine can be pro-arrhythmogenic. Dobutamine can be administered at a dose of 2 – 20 mcg/kg/minute. Potential side effects include tachyarrhythmias (at higher doses), facial twitching, and seizures. In animals with severe myocardial dysfunction, treatment often includes 48 hours on a dobutamine constant rate infusion (5-10 mcg/kg/min). Pimobendan

(0.25 mg/kg PO BID) has been used with success dogs for both emergency and long-term management of dogs with CHF secondary to DCM and mitral valve insufficiency. Pimobendan is a phosphodiesterase-III inhibitor that sensitizes the myocardium to calcium, and improves inotropic activity in addition to causing arteriolar and venous dilation. In addition to its use as a long-term inodilator in the treatment of dogs with CHF, Pimobendan is also recommended for use in emergency therapy of CHF, as it can have an onset of effects within one hour.

CONCLUSIONS

Irrespective of the underlying cause, patients with CHF must be managed carefully and aggressively following initial diagnosis. Supplemental oxygen, potent diuretics, and nitric oxide donors continue to be the mainstay of therapy in both cats and dogs during the initial management of CHF. Patients that do not respond to standard therapies may require additional drug protocols, including positive inotropic and intravenous vasodilatory drugs. Careful monitoring of the patient's heart rate and rhythm, arterial blood pressure, respiratory rate and effort, and pulse oximetry or arterial oxygen saturation should be performed to evaluate the patient's response to therapy.

Emergency Management of Head and Spinal Trauma Final

INTRODUCTION *Severe head and spinal injury are among the most challenging problems that present to the small animal practitioner. Often, neurologic injury is accompanied by other serious life-threatening problems including pneumothorax, pulmonary contusions, hemoabdomen and fractures. Many of the animals have sustained multiple trauma and are in hypovolemic shock at the time of presentation. Head injury may be so severe that localization of head or spinal lesions, then considering an accurate prognosis are difficult. The treatment of any patient with head and spinal injury involves making an accurate assessment to identify the extent of injury, prevention of further damage to the brain and spinal cord, and maintenance of cerebral perfusion pressure and end-organ function. Many cerebral injuries and spinal injuries with intact deep pain perception can carry an overall favorable prognosis with aggressive nursing care and tincture of time. Other types of injuries such as thoracolumbar spinal injury with loss of deep pain sensation are more clear-cut, but offer a less favorable prognosis.*

TRIAGE AND INITIAL STABILIZATION OF THE HEAD/SPINAL TRAUMA PATIENT

The assessment and treatment approach for any patient that has sustained severe trauma should be based on the ABC's, or "airway, breathing, and circulation". Examine the patient for a patent airway. Note the patient's respiratory status and listen carefully to thorax. The absence of lung sounds with rapid restrictive respiration may be associated with a pneumothorax or diaphragmatic hernia. Harsh crackles with cyanosis and respiratory difficulty can be associated with pulmonary contusions. Tachycardia can be associated with pain, anxiety, and hypovolemic shock. The patient's blood pressure should be measured, and intravenous fluids should be administered to restore circulating blood volume and cerebral perfusion pressure. The maintenance of cerebral perfusion pressure and cerebral oxygen delivery is of paramount importance in any head trauma patient. Intravenous crystalloid and colloid fluids should be titrated based on the patient's arterial blood pressure and response to treatment. Careful administration of intravenous crystalloid fluids at ¼ of the calculated shock volume of fluids (shock volume is 90 ml/kg in dogs and 44 ml/kg in cats) should be administered as rapidly as possible with careful and frequent reassessment of the patient's blood pressure. Alternatively, hydroxyethyl starch (5 ml/kg IV) can be administered as a rapid bolus. The goal is to restore systolic blood pressure to 90 – 100 mm Hg, diastolic blood pressure to > 40 mm Hg, and mean arterial blood pressure to 60 – 80 mm Hg. This approach serves to restore cerebral blood flow and flow to vital organs such as the heart and kidneys, as well. Hypertonic saline has also been used in combination with synthetic colloids such as hydroxyethyl starch in the successful treatment of head trauma. The potent hyperosmolar effect of IV hypertonic saline acts to pull fluid from

the intracellular and interstitial fluid compartments into the intravascular space within 3 minutes of administration. The effect is very short-lived, and lasts approximately 20 – 30 minutes. When used in combination with a colloid such as hydroxyethyl starch, the fluid is retained in the intravascular space for a longer period of time. Hypertonic saline (7.5%, 3 - 5 ml/kg for dogs, and 2 – 4 ml/kg in cats hypertonic saline with 5 – 10 ml/kg colloid) has been shown to restore extracellular sodium concentrations and decrease neutrophil chemotaxis to limit secondary brain injury in the head trauma patient. The patient's oxygenation status should be monitored closely with pulse oximetry or arterial blood gas analyses. Ideally, the patient's arterial partial pressure of oxygen (PaO2) should be maintained above 80 mm Hg, and oxygen saturation as measured by pulse oximetry above 90% SaO2. Supplemental oxygen should be administered by mask, hood, or flow-by in any patient with head trauma. The placement of nasal oxygen catheter(s) can result in patient discomfort and subsequent sneezing. Sneezing can increase intracranial pressure, so nasal oxygen catheters should be avoided. A minimum data base of PCV/TS, glucose, azostick, and lactate can be useful for baseline measurements and may help predict outcome. Hyperglycemia has been shown to be a negative prognostic indicator in humans and dogs with severe head injury. Glucose acts as a substrate for anaerobic metabolism during periods of cerebral ischemia, and can lead to cerebral acidosis. For this reason, the administration of glucose-containing fluids (D5W, 0.45% NaCl + 2.5% dextrose) and any steroid is contraindicated in the head trauma patient.

Steroid use has not been documented to provide any benefit in traumatic brain injury, and can cause hyperglycemia that can potentially worsen secondary brain injury and cerebral edema. One exception to the use of steroids in head injury are those patients with severe facial, oropharygeal, and ocular trauma in which steroid use is necessary to decrease edema to maintain a patent airway. Any trauma patient that exhibits abnormal neurologic postures should be confined to a backboard for stabilization and to prevent further injury during the initial triage period. Extensor rigidity of the forelimbs with flaccid paralysis of the hindlimbs is characteristic of Schiff-Sherrington posture, and is commonly associated with an injury between T3 and L3. The loss of deep pain perception in such patients carries a grave prognosis. Intact deep pain perception with Schiff-Sherrington has been associated with spinal shock and not necessarily due to transaction of the spinal cord, and can carry a slightly more favorable prognosis, depending on the exact location and extent of the injury. Flaccid paralysis of the forelimbs with the hindlimbs tucked up close to the body and opisthotonus is associated with severe injury to the cerebellum, and is known as decerebellate rigidity. Extensor rigidity of all four limbs and opisthotonus is known as decerebrate rigidity, and carries a very grave prognosis.

NEUROLOGIC EXAMINATION

Once the patient's ABC's have been accurately assessed and problems addressed. A complete neurologic examination can be performed. Pupil size and response to light, presence or absence of menace, physiologic nystagmus, mentation, ambulation, and reflexes should be evaluated in a step-bystep approach, starting from nose to tail. Mental status is often difficult to accurately assess until hypovolemic shock has been successfully treated and perfusion has normalized. A patient's mentation can be categorized as normal, depressed, obtunded, stuporous, or comatose. A depressed patient may

appear mentally dull and be slow to react to external stimuli including noise and touch. As mental status worsens, an obtunded patient will be depressed, and slower to respond to external stimuli. A stuporous patient appears unconscious, but will respond to painful or noxious stimuli. Coma is the most severe change in mentation, in which the patient is unconscious and completely unresponsive to a noxious stimulus. The presence of coma alone does not necessarily mean a poor long-term prognosis. The patient's mentation should be evaluated in combination with the patient's pupil size when gauging severity of condition in order to make a prognosis. Miotic pupils are associated with forebrain lesions. Mydriatic dilated pupils or loss of papillary light reflexes are associated with a rostral brainstem lesion, and carry a much more guarded prognosis when in combination with stupor or coma. Anisocoria, or unequal pupil size, can be associated with either intracranial lesions, or extracranial lesions of the eye, brachial plexus, or vagosympathetic trunk. For example, a patient with normal mentation, miosis, and anisocoria can potentially have anterior uveitis secondary to a corneal abrasion, brachial plexus injury, or injury to the lateral neck affecting the vagosympathetic trunk on the side ipsilateral to the miotic pupil, with no intracranial lesion at all. Animals with Schiff-Scherrington posture should be placed on a flat backboard or other flat surface and strapped down to prevent movement and potential disruption of a partially displaced spinal fracture/luxation. Procedures to assess balance and muscle strength such as hopping and wheelbarrowing should not be performed until spinal trauma has been completely ruled out. Reflexes and deep pain perception should be evaluated. Withdrawal of the hind limbs in a patient with SchiffScherrington posture is a local reflex arc only, and should not be interpreted as perception of pain and functioning motor and sensory tracts through the spinal cord to the cerebral cortex. Additionally, if the patient attempts to move during the evaluation, motion at the fracture site can be perceived as painful without functioning sensory pathways to the rear limbs. Less than 5 – 8% of animals with loss of deep pain perception regain motor function and continence, and thus, prognosis for return to function must be considered poor, at best. Radiographs should be performed if depressed skull or spinal fractures are suspected. Patients with suspected spinal fractures should never be moved from lateral position in order to take dorsoventral or ventrodorsal radiographs. A lateral radiograph of the suspected fracture site should be performed. In many cases, disruption of the articular facets, compression fractures, or obvious disruption of the spinal column can be visualized. Some injuries, however, may be difficult to accurately assess without an orthogonal view. Rather than move the patient and potentially cause further disruption and injury to the fracture, a ventrodorsal radiograph view can be obtained by turning the bucky at a 90 degree angle and placing an x-ray cassette behind the patient. Radiographs are a sensitive imaging modality for diagnosis of intracranial hemorrhage or edema. The use of computed tomography (CT) and magnetic resonance

imaging (MRI) are more sensitive at detecting intracranial lesions. An MRI is considered to be the best imaging modality for detection of fibrocartilagenous emboli (FCE).

TREATMENT OF HEAD AND SPINAL TRAUMA

The various forms of recommended treatment for head and spinal trauma remains a subject of wide debate and

controversy. The treatment of severe head trauma in a patient that is obtunded, stuporous, or in a coma involves maintenance of cerebral perfusion and oxygen delivery and decreasing cerebral edema. Mannitol (0.5 – 1.0 g/kg IV over 10 - 15 minute) acts both as an osmotic diuretic and free radical scavenger in the patient with traumatic brain injury, and serves to decrease cerebral edema and secondary brain injury after the time of impact. The use of mannitol had fallen out of favor in the past because of the potential risk of worsening intracranial hemorrhage. The benefits of mannitol far outweigh the unsubstantiated risks, particularly in the traumatically head injured patient. Mannitol administration is not necessary in patients that are normal or depressed with an obvious skin abrasion or laceration on the face or head. This author reserves mannitol use to the patient with traumatic head injury that is obtunded, stuporous, or comatose, or exhibits a decline in mental status despite aggressive treatment to restore and maintain intravascular volume and normal blood pressure. Steroid use has dramatically fallen out of favor for the treatment of traumatic brain injury. Glucocorticosteroids were thought to stabilize neuronal membranes. However, steroids also decrease immune defense mechanisms, disrupt glucose homeostasis, contribute to negative nitrogen balance and insulin resistance, and can worsen intracranial acidosis. Additionally, steroid use has demonstrated equivocal results. The benefits of steroid use are unsubstantiated, and are far outweighed by their risks, and as such, are contraindicated at this time.

Fluid Therapy Its More Than Just Lactated Ringers Final

Total body water constitutes approximately 60% of a patient's body weight in normal individuals, although this value can vary slightly with age, gender, and obesity. Approximately 67% of total body water is located intracellularly. The remaining 33% of total body water is located extracellularly, in the intravascular and interstitial extravascular spaces. A very small amount of fluid, known as transcellular fluid, is located in the compartments of the gastrointestinal tract, within synovial fluid of joints, and the cerebrospinal tract. Within the body, all fluid is in a constant state of flux in between compartments. The movement of fluids from space to space is largely governed by the concentration of electrolytes, proteins, and other osmotically active particles relative to the amount of fluid within each compartment. The balance of fluids and electrolytes are necessary for normal body functioning and cellular processes. Normally, fluid intake is in the form or drink and foodstuffs. Water is also produced during the oxidation of food materials. Fluid can be lost during excessive panting, vomiting, diarrhea, and urination. Sensible fluid losses in the form of urine, vomit, and feces can be measured, and constitute approximately 2/3 of the body's daily maintenance fluid requirements. Insensible fluid loss is largely estimated from evaporation from the respiratory tract. Insensible losses can be excessive in situations of excessive panting, salivation, or from evaporation or hemorrhage from surgical sites. In normal individuals, fluid intake and excretion are kept in balance by the activity of sodium and chloride and serum osmolality. Osmoreceptors in the hypothalamus sense sodium and chloride concentration in the vascular space. As serum sodium rises due to increased sodium intake or fluid loss in excess of solute, serum osmolality rises. An increase in serum osmolality stimulates the release of arginine vasopressin (antidiuretic hormone) to be released from the hypothalamus. Antidiuretic hormone stimulates the opening of water channels in the collecting duct of the renal tubules, and thus stimulates water reabsorption. Once water is retained in the vascular space, sodium, urea, and glucose, the major contributors of serum osmolality, are diluted, and serum osmolality decreases. Hypothalamic excretion of ADH ceases once serum osmolality returns to normal. During a state of equilibrium, a patient's daily water intake equals water loss, creating no net loss or gain of fluid under normal conditions. Daily fluid requirements are based on the metabolic water requirements of a patient in a state of equilibrium. For each kilocalorie of energy metabolized, 1 ml of water is consumed. Metabolic energy requirements are calculated based on the linear formula: Kcal/day = [(30 x body weightkg) +70] By substituting Kcal for 1 mL H2O, the following formula can be used to estimate a patient's daily metabolic water requirements: ml/day = [(30 x body weightkg) + 70] Recent studies have indicated that metabolic energy

requirements rarely increase during states of critical illness except in cases of sepsis. Because our patients frequently pant and may have excessive evaporative losses or sensible fluid losses in the form of vomiting, diarrhea, wound exudates, body cavity effusions, daily fluid requirements may be greater than that calculated above. The formula should be used as a guideline, and careful assessment and measurement of ongoing losses should be added to the patient's daily fluid therapy as needed, to prevent further dehydration. The degree of interstitial dehydration can subjectively be estimated based on a patient's body weight, mucous membrane dryness, skin turgor, degree of sunkeness of the eyes, and mentation. Subjectively, if a patient has a history of fluid loss in the form of vomiting or diarrhea, but no external evidence of mucous membrane dryness of skin tenting, dehydration estimate is less than 5%. A patient is said to be 5% dehydrated when mild skin tenting, and mucous membrane dryness is present. Clinically, 7% dehydration is manifested as increased skin tenting, dry oral mucous membranes, and mild tachycardia with normal pulse quality. A patient is 10% dehydrated with increased skin tenting, dry oral mucous membranes, tachycardia, and decreased pulse pressure is present. Finally, a patient is said to be 12% dehydrated when skin tenting, and mucous membrane dryness is markedly increased, the eyes appear dry and sunken, and alteration of consciousness is observed. The parameters are largely subjective, because they can also be affected by loss of body fat and increased age.

The later stages of dehydration are also accompanied by parameters consistent with hypovolemic shock. Other factors, including hemorrhage and third spacing of body fluids can also result in a decrease in intravascular circulating volume, resulting in signs of hypovolemia. With severe hypovolemia of more than 15% of circulating volume, transcompartmental fluid shift from the interstitial to intravascular compartments occurs within one hour of fluid loss. When fluid loss is so severe that intravascular fluid volume is affected, hypovolemia can result in clinical signs of tachycardia, prolonged capillary refill time, decreased urine output, and hypotension. The vascular space is very sensitive to changes in the amount of circulating volume. During states of normovolemia, the degree of wall tension is sensed by baroreceptors in the carotid body and aortic arch, sending a pulsatile continuous feedback via vagal afferent stimuli to decrease heart rate. In the early stages of hypovolemic shock, a decrease in vascular wall stretch or tension is sensed by baroreceptors in the carotid body and aortic arch, causing blunting of tonic vagal stimulation, and allows sympathetic tone to increase heart rate and contractility to normalize cardiac output in the face of decreased circulating volume. Later, decreased blood flow and delivery of sodium to receptors in the juxtaglomerular apparatus of the kidneys cause activation of the reninangiotensin-aldosterone axis, stimulating sodium and fluid retention to replenish intravascular volume.

FLUID REPLACEMENT: HYPOVOLEMIA VERSUS DEHYDRATION

When clinical signs of hypovolemic shock are present, intravascular fluids must be replaced in an emergency phase of fluid resuscitation. Calculated shock volumes of fluids are 90 ml/kg/hour for dogs, and 44 ml/kg/hour for cats. A simple guideline to follow is to replace ¼ of the calculated intravascular fluid deficit, or the "shock volume" as rapidly as possible, then reassess perfusion parameters- heart rate, blood pressure, capillary refill time, and urine output. In dogs, a simple method to calculate ¼ shock volume i is to take the

animal's weight in pounds and add a zero, giving you the amount of fluid in milliliters to administer as a bolus as quickly as possible. Approximately 80% of the crystalloid volume fluid infused will re-equilibrate and leave the intravascular space within 1 hour of its administration. A constant rate infusion of crystalloid is recommended to provide continuous fluid support in patients that are dehydrated and have ongoing losses. In some cases, the fluid required to restore intravascular and interstitial volume can cause hemodilution and dilution of oncotically active plasma proteins, resulting in interstitial edema formation. In such cases, a combination of a crystalloid fluid along with a colloid containing fluid can help restore colloid oncotic pressure and prevent interstitial edema. Once immediate life-threatening intravascular fluid deficits are replaced, additional fluid is provided based on the estimated percent interstitial dehydration and maintenance needs. Dehydration estimates can be calculated based on the fact that 1 milliliter of water weighs approximately 1 gram. Dehydration estimates in liters can then be calculated by the formula: Body weight in kg x estimated percent dehydration x 1000 ml/liter. This provides you with the number of liters deficit. A frequent mistake when replenishing fluid deficits is to arbitrarily multiply a patient's daily water requirement by a factor of 2 or 3 to replenish intravascular and interstitial deficits. This practice frequently underestimates the patient's actual fluid needs, and does little to treat intravascular volume depletion and interstitial dehydration. Instead, it is better to perform the calculation and add this to daily maintenance fluid requirements and ongoing losses, to maintain hydration in your hospitalized patients. Eighty per cent of the calculated fluid deficit can be replaced in the first 24 hours. After successful treatment of hypovolemic shock and replacement of estimated dehydration volumes, maintenance fluids can be supplemented, provided that no signs of dehydration or ongoing fluid loss are present. An objective way of assessing whether fluids volume is adequate is to assess body weight in a regular basis throughout the day. Acute losses in body weight are commonly associated with fluid losses, and can be used to determine whether the patient is at risk of once again becoming dehydrated.

ISOTONIC FLUIDS, HYPOTONIC FLUIDS, AND HYPERTONIC FLUIDS

There is a wide variety of fluids are available for use by the veterinary practitioner. A crystalloid fluid contains crystals or salts that are dissolved in solution. Specific crystalloid fluids are indicated in certain disease states, and may be contraindicated in others. Therefore, whenever a crystalloid fluid is used, one must carefully consider it to be another drug in the armamentarium, and justify its use or potential disuse in each patient. Basic categories of crystalloid fluids include isotonic, hypotonic, and hypertonic solutions, depending on the concentration and type of solute present relative to normal body plasma. Isotonic fluids have tonicity, or solute relative to water, similar to that of plasma. Examples of isotonic fluids include 0.9%

(normal) saline, Lactated Ringer's solution, Normosol-R, and Plasmalyte-A. Isotonic fluids are indicated to restore fluid deficits, correct electrolyte abnormalities, and provide maintenance fluid requirements. Hypotonic solutions are fluids whose tonicity is less than that of serum. Examples of hypotonic fluid solutions include 0.45% saline, 0.45%NaCl + 2.5% dextrose, and 5% dextrose in water (D5W). Hypotonic fluids are indicated when treating a patient with diseases processes that cause sodium and water retention, namely, congestive heart

failure and hepatic disease. Infusion of hypotonic fluids is also indicated when severe hypernatremia exists and you need to slowly correct a free water deficit. To calculate a patient's free water deficit, use the following formula: Free water deficit = 0.4 x lean body weight x [patient serum Na/140 – 1] The free water deficit should be corrected slowly, to not cause iatrogenic cerebral edema. Ideally, the patient's sodium should not decrease by more than 15 mEq/L during a 24-hour period. Hypertonic solutions act to draw fluid from the interstitial fluid compartment into the intravascular space to correct hypovolemia. Their use is absolutely contraindicated if interstitial dehydration is present. Hypertonic solutions such as 3% or 7% saline have solute in excess of fluid relative to plasma. Hypertonic saline should be administered in bolus increments of 3 – 7 ml/kg as a rapid infusion. Because the net effect of hypertonic saline solution lasts only approximately 20 minutes, hypertonic saline must always be infused along with a crystalloid solution to prevent further interstitial dehydration.

Electrolyte Composition (mEq/L) of Commonly Used Isotonic and Hypotonic Crystalloid Fluids

	0.9% Saline	0.45% NaCl	Lactated Ringer's	Normosol-R
Sodium	154	77	130	140
Chloride	154	77	109	98
Potassium	0	0	4	5
Calcium	0	0	3	0
Magnesium	0	0	0	3
pH	7.386	5.7	6.7	7.4
Buffer	none	none	lactate 28	acetate 27 gluconate 23

Colloids

A colloid solution contains negatively charged large molecular weight particles that are osmotically active, drawing sodium around their core structures. Wherever sodium is, water follows. By drawing sodium around the particle, water is thus held within the vascular space. Colloids replace intravascular fluid deficits only. Therefore, colloids are always administered along with crystalloids, to restore both intravascular and interstitial fluid volume. Examples of artificial colloids include Hetastarch, Vetstarch, Pentastarch, and Voluven. Whenever a colloid is administered along with a crystalloid, calculated crystalloid fluid requirements should be decreased by 25% - 50%, to avoid intravascular volume overload. Natural colloid solutions include whole blood, packed red blood cells, and plasma. Fresh whole blood is indicated when loss of both red blood cells and plasma has occurred. The Rule of Ones states that one ml of fresh

blood infused per one-pound body weight will increase the patient's packed cell volume by one per cent, provided that no ongoing losses are present. Packed red blood cells can be administered when anemia is present in sufficient quantity to cause clinical signs of anemia, including lethargy, inappetance, tachycardia and tachypnea. Fresh frozen plasma can be administered at 10 – 20 ml/kg/day to replenish clotting factors and provide antiprotease activity during inflammatory conditions. Fresh frozen plasma can be used to replace small amounts of albumin, in cases of hypoalbuminemia, however, is not efficient as administering purified concentrated canine-specific (when available) or 25% human albumin. Frozen or fresh frozen plasma (20 ml/kg IV) needs to be infused for every 0.5 g/dL

increase in plasma albumin, provided that no ongoing losses are present. The goal of albumin administration is to raise the patient's serum albumin to 2.0 g/dL, then provide the remainder of colloidal support with synthetic colloids. Hetastarch is a polymer of amylopectin suspended in a lactated ringer's solution. The average molecular weight of Hetastarch is 69,000 Daltons. Larger particles are broken down by serum amylase, and last in circulation for approximately 36 hours. Because Hetastarch can bind with von Willebrand's factor, mild prolongation of a patient's APTT and ACT may be observed, but do not contribute to or cause clinical bleeding. Hetastarch should be administered in incremental boluses of 5 – 10 ml/kg in dogs, and 5 ml/kg in cats. Because rapid administration of hetastarch can cause histamine release and vomiting in cats, the bolus should be administered slowly over a period of 15 – 20 minutes. Some authors recommend that the total daily dose of hetastarch should not exceed 20 – 30 ml/kg/day. Following the administration of hetastarch boluses, it should be administered as a constant rate infusion (20 – 30 ml/kg/day IV) until the patient is able to maintain its albumin and colloidal support on its own. Concentrated human albumin and concentrated canine albumin solutions are now available for use in veterinary patients. Both immediate and delayed rare Type 3 hypersensitivity reactions have been documented in healthy and hypoalbuminemic dogs following administration of concentrated human albumin. Reactions that occurred that include fever, vomiting, acute anaphylaxis, urticaria, angioneurotic edema, and delayed vasculitis, polyarthopathies, glomerulonephritis and death in both healthy and critically ill animals. Although there are studies which have demonstrated adverse reactions and the development of anti-human albumin antibodies after concentrated human albumin infusion in dogs, there also have been studies which have documented improved clinical outcome when concentrated human albumin was infused into critically ill animals that were refractory to other more mainstream therapies, including pressors, synthetic colloids, and fresh frozen plasma transfusions. Concentrated 25% human (2 ml/kg IV in dogs over 4 hours; pre-treat with 1 mg/kg diphenhydramine IV). should be considered in any patient with refractory hypoalbuminemia (< 2.0 g/dL) or hypotension unresponsive to other synthetic colloids, pressors, and inotropes. The perceived benefits of albumin infusion and risks of not infusing albumin must be weighed against the potential risks of its administration. Clients must be aware of the potential risks of complications.

Hypoadrenocorticism Insidious and Deadly Final

INTRODUCTION : *The hypothalamic-pituitary-adrenal axis is a key player in the maintenance of protein metabolism, mineral and acid-base homeostasis, immune function, blood pressure, gastrointestinal function, erythropoiesis, and mentation. Hypoadrenocorticism (Addison's disease) can result from either lack of cortisol production from the adrenal glands, or result from lack of CRF or ACTH stimuli from the hypothalamus or pituitary glands. The majority of cases of hypoadrenocorticism in dogs result from immune-mediated lymphocytic plasmacytic destruction of the adrenal cortex. Adrenocortical destruction typically results in a lack of both glucocorticoid from the zona fasciculata and lack of mineralocorticoid production from the zona glomerulosa. However, in some dogs, destruction of the zona fasciculata can occur before destruction of the zona glomerulosa, resulting in clinical signs of glucocorticoid deficiency alone before clinical signs associated with loss of mineralocorticoids. This is known as "atypical primary hypoadrenocorticism". Secondary adrenocortical deficiency ("secondary Addison's") is associated with a loss of either corticotropin releasing factor (CRF) from the hypothalamus, or lack of production or release of adrenocorticotropic hormone (ACTH) from the posterior pituitary gland. In secondary adrenocortical deficiency, loss of trophic stimulation of the zona fasciculata results in a loss of glucocorticoid (cortisol) release and spares the zona glomerulosa. Thus, mineralocorticoid activity is maintained. Secondary hypoadrenocorticism has been described following craniocerebral trauma, or can occur spontaneously.*

ALDOSTERONE

The actions of aldosterone serve to promote expansion of intravascular volume. Sodium is reabsorbed in exchange for potassium in the distal convoluted tubule, promoting sodium and water resorption and potassium excretion. Lack of stimulation of aldosterone release, either from destruction of the zona glomerulosa or from lack of trophic stimuli from secondary hypoadrenocorticism results in a decreased ability to resorb sodium and water, as well as impaired potassium excretion. Loss of sodium and water cause eventual hyponatremia and hypovolemia. Retention of potassium causes hyperkalemia and can result in cardiac dysrhythmias as serum potassium becomes increasingly elevated. Chronic sodium loss depletes the concentration gradient of the renal medullary interstitium, further impairing the kidney's ability to reabsorb sodium, chloride and water. Eventually, intravascular volume depletion can result in decreased stroke volume, decreased cardiac output, and hypotension.

Hyperkalemia develops due to decreased excretory ability in the distal convoluted tubules of the kidney. Hydrogen ion excretion, too, is impaired, leading to metabolic acidosis. Hyperkalemia is exacerbated by glucocorticoid deficiency. Hyperkalemia causes a decrease in myocardial excitability, decreased or delayed conduction, and a prolonged refractory period. Atrial cells are especially sensitive to the effects of hyperkalemia, and develop an idioatrial rhythm, often called atrial standstill. Characteristic electrocardiographic changes associated

with hyperkalemia include prolonged P-R interval, widened QRS complexes, spiked t-waves, and eventual loss of p waves.

Glucocorticoids Loss of glucocorticoid activity has numerous detrimental effects on homeostasis, including abdominal pain, weight loss, muscle atrophy, vomiting, diarrhea, impaired hepatic gluconeogenesis and glycogenolysis, impaired erythropoiesis, and lack of ability to handle stress. The net effect of decreased or absent glucocorticoid activity is a resting hypoglycemia, vomiting, anorexia, weight loss, peripheral muscle wasting, possible regurgitation secondary to megaesophagus, normocytic normochromic nonregenerative anemia from bone marrow suppression, weakness, dehydration, and in extreme cases, hypotension and collapse. This is of particular importance in dogs with primary atypical hypoadrenocorticism, characterized by lack of glucocorticoid activity alone, with normal serum electrolyte levels.

PRESENTATION AND CLINICAL SIGNS

The most severe form of hypoadrenocorticism is associated with severe acute vomiting, diarrhea, weakness, and collapse, sometimes with a waxing and waning history of nonspecific clinical signs. Some clients will complain about "not doing well", intermittent inappetance or "she's a picky eater", intermittent vomiting or "sensitive stomach", diarrhea, lethargy, or weight loss, particularly during times of stress, such as grooming appointments, trips to the veterinarian or kennel, having visitors in the household, or when the owners have left the animal with a pet sitter. Other patients may not have any recognizable clinical signs at all. Physical examination abnormalities can range from mild lethargy and dehydration to complete cardiovascular collapse, with an obtunded patient in severe hypovolemic shock, bradycardia, hypothermia, with diarrhea. Severe muscle tremors have been documented as a clinical sign in two Standard poodles. Critical intervention is necessary in such cases to have a positive outcome.

LABORATORY ABNORMALITIES

Laboratory abnormalities can be found on a complete blood count and biochemistry panel. Normally, glucocorticoids released during times of stress cause a peripheral lymphopenia, neutrophilia, and what is commonly known as a "stress leukogram". The absence of a stress leukogram in a vomiting patient with diarrhea and dehydration or collapse should be a signal to investigate for hypoadrenocorticism, even if characteristic electrolyte abnormalities are not present (secondary hypoadrenocorticism or primary atypical hypoadrenocorticism). In some (but not all) cases, peripheral eosinophilia may be present. In some animals, a normocytic normochromic nonregenerative anemia will be present from GI blood loss and lack of glucocorticoid-induced erythropoiesis. Other changes on a serum biochemistry panel can include hypoglycemia), hypocholesterolemia, hypoalbuminemia, hypercalcemia and azotemia. In many cases, BUN elevations are disproportionate to the observed elevation in creatinine due to GI blood loss. Prerenal azotemia also occurs from dehydration, and decreased renal perfusion secondary to decreased cardiac output. Classic electrolyte abnormalities include hyponatremia, hypochloremia, and hyperkalemia, but are not always present (secondary Addison's with loss of pituitary ACTH release or "primary atypical" Addison's, where aldosterone production is preserved). In dogs, the normal sodium:potassium ratio is

> 27 (> 24 in cats). In patients with lack an aldosterone deficiency, however, the sodium:potassium ratio is less than 27, although this is not pathognomonic for hypoadrenocorticism per se. The gold standard for diagnosis of hypoadrenocorticism remains the ACTH stimulation test. As ACTH stimulation testing has become increasingly more expensive, a baseline cortisol can be performed as an initial screening test for hypoadrenocorticism. Baseline cortisol levels greater than 2 ug/dL help to rule out hypoadrenocorticism without the added expense of using Cosyntropin.

Urinalysis Urinalysis usually reveals a isosthenuric or hyposthenuric urine in the face of dehydration, leading to the differential diagnoses of hypoadrenocorticism or acute renal failure. In primary Addisonian animals, the chronic loss of solute in the urine from hypoaldosteronemia causes a chronic medullary wash-out and loss of renal concentrating abilities, even once serum electrolytes have been replenished. It may take weeks before the urine specific gravity returns to normal. The presence of ongoing azotemia and hyposthenuria in an animal may also be associated with renal damage secondary to hypovolemic shock and hypoxic insult to the kidneys, leading to concurrent acute kidney injury or AKI.

Definitive Diagnosis Definitive diagnosis of hypoadrenocorticism is made using the ACTH stimulation test, considered the gold standard for making this diagnosis. In dogs, a baseline serum cortisol sample is obtained, synthetic ACTH (Cosyntropin 0.25 mg) administered intravenously or intramuscularly, then a second sample obtained 1 hour later to see if there is "stimulation" of the adrenal gland to release cortisol. In cats, the protocol is slightly different, with blood samples obtained at ½ and 1 hour post-ACTH administration. Caution should be exercised before performing an ACTH stimulation test, however, as most glucocorticoids except for Dexamethasone or dexamethasone sodium phosphate will cross-react with cortisol radioimmunoassays. Blood samples should be obtained and the ACTH stimulation test performed before glucocorticoids are administered. Investigations have documented that a single dose of the above compounds will not interfere significantly with the ACTH stimulation test.

Primary atypical hypoadrenocorticism is characterized by low or undetectable pre- and post-ACTH cortisol levels with normal to elevated ACTH levels.

EMERGENCY TREATMENT

Treatment largely consists of treating the clinical signs of hypovolemic shock, correcting electrolyte and acid-base abnormalities, preventing septicemia secondary to bacterial translocation from the GI tract, antiemetics, and treating any secondary conditions such as aspiration pneumonia. Critically ill hypovolemic patients should have rapid correction of intravascular volume deficit using crystalloid fluids. This author usually starts with ¼ of the calculated shock volume and then reassesses perfusion parameters including heart rate, blood pressure, urine output, and capillary refill time. In animals with chronic sodium depletion characterized by severe hyponatremia (Na < 120 mEq/liter), sodium replacement should be corrected slowly and with caution, to avoid rapid increase in serum sodium by no more than 0.5 mEq/liter/hour or 15 mEq/liter in a 24 hour period. Overzealous and too rapid correction of serum sodium to

normal levels has resulted in a syndrome of central pontine myelinolysis, in which idiogenic osmoles and oxidative damage to neurons occurs, and results in cerebral edema. Central pontine myelinolysis is characterized by generalized weakness, ataxia, mental depression and head pressing in animals several days following correction of severe hyponatremia. If serum sodium is less than 120 mmol/liter, a more cautious fluid to administer is Lactated Ringer's (130 mEq/liter sodium) or Normosol-R (140 mEq/liter sodium). Although these two fluids also contain a small amount of potassium, intravascular volume correction and administration of drugs to protect the heart from the effects of hypokalemia (see below) will offset the potential risk of exacerbating hyperkalemia.

Treatment of Hyperkalemia: Hyperkalemia can be treated with regular insulin (0.5 units/kg regular insulin IV) and dextrose (1 gram dextrose per unit insulin administered, followed by 2.5 - 5% dextrose as a constant rate infusion to prevent hypoglycemia), calcium gluconate (0.5 – 1.0 ml/kg 10% calcium gluconate over 20 – 30 minutes) or calcium chloride (1.5 – 3.5 ml total of IV calcium chloride), or sodium bicarbonate (0.25 mEq/kg IV). The effects observed on the patient's ECG rhythm from administration of calcium gluconate and insulin-dextrose are observed within minutes of administration, and typically last for 20 – 30 minutes.

Other ancillary therapies: Broad spectrum antibiotics should be administered in patients with severe melena or hemorrhagic diarrhea. Antiemetics can be administered, too, to combat nausea and vomiting (Metoclopramide 1 – 2 mg/kg/day as a constant rate infusion, Dolasetron 0.6 mg/kg IV once daily, maropitant 1 mg/kg SQ/IV once daily should be administered. Phenothiazine antiemetics such as chlorpromazine should be avoided in hypotensive patients due to their alpha-antagonist effects).

CHRONIC THERAPY

Chronic therapy for hypoadrenocorticism consists of replacing mineralocorticoid and glucocorticoids at physiologic doses. Fludrocortisone acetate (Florinef®) can be administered daily (0.1 – 0.3 mg/kg/day), although this can be quite expensive in large breed or giant dogs. Florinef has both mineralocorticoid and glucocorticoid activity, thus, additional glucocorticoid replacement is not necessary. Some dogs with become polyuric and polydipsic on their maintenance dose of Florinef because of its potent glucocorticoid actions. An alternative therapy is the use of Desoxycorticosterone pivalate (DOCP, 2.2 mg/kg IM; Percorten, Novartis Animal Health) as an intramuscular repository injection every 25 days. Since DOCP has no glucocorticoid activity, additional GC administration in the form of daily Predniso(lo)ne (0.2 mg/kg/day) also must be administered. This form of treatment is less expensive than Florinef, and works quite well, in this author's experience. With DOCP, electrolytes should initially be checked at 12 and 24 days post-injection, as some patients may require up to 0.3 mg/kg every 3 – 4 weeks. In one retrospective study, the mean total dose of both Florinef and DOCP increased over the course of treatment in all dogs with hypoadrenocorticism. The overall long-term prognosis for patient with hypoadrenocorticism after initial treatment for a hypo-adrenal crisis is good, provided that the owners are diligent in maintaining daily and monthly glucocorticoid and mineralocorticoid supplementation.

Índice

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The Ultimate Trauma – Big Dog – Little Dog

INTRODUCTION: *Little dogs presenting with bite wounds inflicted by larger dogs can be exceedingly challenging to manage. Appropriate wound care provided in a timely manner can help prevent morbidity and mortality. The seriousness of wounds should not be underestimated and in many cases aggressive surgical treatment is indicated as soon as the patient is cardiovascularly stable. In some situations the patient may never become cardiovascularly stable without surgery. Secondary problems with wound healing and patient morbidity may relate to inappropriate wound handling during the initial stages. Problems with sepsis secondary to wounds come from inadequate debridement of necrotic tissue, insufficient irrigation, inappropriate choice of antibiotics and inadequate resuscitation.*

FIRST AID STABILIZATION AND RESUSCITATION

Evaluation of the patient should begin always with the ABC's (airway, breathing, circulation). The wound may be the obvious injury but it may be minor compared to an unseen injury such as airway disruption or pneumothorax. Patients with serious wounds should always be provided with supplemental oxygen (flow by, baggie or a non-tight fitting mask) on presentation. If it is determined that the patient is not in shock the oxygen can be discontinued. If the animal cannot breathe an airway will need to be established. In the case of severe oral, pharyngeal or cranial cervical trauma a tracheostomy may be indicated. If the patient is able to breathe adequately but there is a significant amount of hemorrhage present, the clinician may need to make a decision as to whether or not to anesthetize the animal and gain control of the airway since ongoing hemorrhage could potentially lead to an airway obstruction. Once an injured patient is intubated the lungs should be ausculted bilaterally to ensure air is moving though both lung fields. The patient who arrived unconscious and not breathing may have an avulsion of the distal trachea and unless the lungs are ausculted post intubation this may not be detected. Auscultation also will allow the clinician to assess the presence of a possible pleural space abnormalities. If the patient has a sucking chest wound and is critical a chest tube may need to be placed through the wound immediately.

A large bore intravenous catheter should be inserted and fluids started if the patient is in shock. A second catheter may be required. Fluid therapy may consist of a combination of crystalloids, synthetic colloids and blood products depending on the status of the patient. Blood volume and blood pressure should be normalized unless the clinician is providing hypotensive resuscitation due to concerns for ongoing internal abdominal hemorrhage. Blood volume ideally should be estimated through measurement of central venous pressure but since central catheters are rarely placed during resuscitation, the distention of the jugular vein when the vein is held off at the thoracic inlet can be used as a subjective evaluation

of central venous pressure. The hematocrit should be maintained as close to 30% as possible. Autotransfusion may be required in the patient with a significant hemoabdomen. Albumin should be maintained greater than 2.0 g/L (20 g/dL) with fresh frozen plasma. Patients at risk for or confirmed to be coagulopathic may need to receive plasma during the resuscitation phase. Small dogs in particular are prone to hypothermia and fluids should be warmed.

Analgesics are always indicated once the patient has been evaluated and resuscitation has been started. Not only is pain detrimental to the overall well being of the patient but also it is detrimental to the healing process. Catecholamine release can lead to vasoconstriction and poor flow to the wound area. There are two key concepts: the first is that pain kills and the second is that no patient is too critical to receive analgesics. Doses may need to be decreased to 25 to 50% of normal in critical patients but all should receive appropriate analgesia. Patients with chest trauma should always have their pain aggressively controlled, since thoracic pain can interfere significantly with ventilation potentially leading to hypercarbia and hypoxia. Opioids are the primary class of drug used; they can be given intravenously, intramuscularly, subcutaneously, or epidurally. In general pure mu agonists are preferred; however, in very critical patients butorphanol may be preferred initially due to its minimal cardiorespiratory side effects. The intravenous route is preferred over the intramuscular route (painful over time) and subcutaneous route (absorption is unpredictable). If the intramuscular route is used the injection should be given in the epaxial muscles since blood flow to this muscle bed is more consistent even in the face of alterations in tissue perfusion. Constant rate infusions of fentanyl or morphine are indicated in patients with significant pain and are very useful in patients who will require surgery. Local anesthetics (lidocaine, bupivicaine) can be injected as local or regional blocks. Intercostal blocks will help improve ventilation in dogs with fractured ribs. Pain related to the acidic nature of the local anesthetic can be modified by warming the drug to body temperature or by adding 10% of the volume as sodium bicarbonate. Nonsteroidal antiinflammatory drugs generally should be avoided. Some of the COX-2 specific drugs may prove to be safe to use in more critical patients; however, they are not recommended in patients with hypovolemia, compromised gastrointestinal perfusion (related to circulatory disturbances or underlying disease processes), and renal disease.

Active bleeding from wounds will need to be controlled. Capillary oozing and most venous hemorrhage can be controlled with pressure bandages. Pressure should be applied as a temporary measure to control arterial hemorrhage but definitive control using a hemostat followed by ligation or electrosurgery should be achieved as soon as possible.

Gloves should be worn whenever wounds are evaluated since infections often come from the hospital environment as well as the nurses' and doctors' hands. The wound should be kept as clean as possible by covering it with a sterile dressing. Wounds should be kept moist using sterile water-soluble gel or saline-soaked gauze since desiccation interferes with wound healing. If the wound is over the chest wall a patch bandage should be placed over the wound. The ventral aspect of the bandage should not be adhered to the chest wall. This will prevent a tension pneumothorax from developing if the patient has a lung laceration. If the wound is associated with a fracture then a padded bandage or temporary splint should be applied until the patient can be completely evaluated. This

helps prevent further mechanical injury to the tissues from the tearing effects of bone fragments. It also helps to prevent further injury to the bone and provides comfort to the patient. Radiographs can be taken through most bandage materials; therefore, an attempt always should be made to stabilize fractures prior to taking radiographs. Broad spectrum antibiotics should be administered pending cultures.

Aggressive attempts should be made to maintain normothermia, especially in smaller animals since hypothermia can lead to cardiac arrhythmias, hypotension, coagulation problems, and sluggish blood flow. Warming pads, fluid warmers, warm air circulating blankets, oat bags, warmed bubble wrap, and warming the inspired air are measures that can be taken to try and maintain the patient's body temperature.

DIAGNOSTIC TESTS

The type of diagnostic tests required will be dictated by the type of wound. Whenever there are concerns for significant hemorrhage (based on history or physical exam), a packed cell volume and total protein always should be performed to assess for the presence of anemia and/or hypoproteinemia. Blood work should consist of a minimum of a packed cell volume, total solids, electrolytes, glucose, blood urea nitrogen – or preferably creatinine - and a blood gas if the animal has a wound that will require exploration and repair under anesthesia. Albumin and coagulation parameters should be assessed in patients with more severe injuries. Ideally a complete blood cell count and a chemistry panel should be evaluated with older and more critical patients. An electrocardiogram should be performed in any patient with an auscultable arrhythmia or chest trauma. A TFAST scan or thoracentesis to check for air and blood should be performed in every patient that has a wound over the thoracic cavity. Radiographs are indicated in any wound that may be associated with an open fracture, joints, and thoracic or abdominal cavities. Abdominal ultrasound is very useful for detecting free fluid within body cavities and may help diagnose intraabdominal injuries. A FAST scan is indicated in any patient with signs of abdominal trauma. If ultrasound is not available or results of the ultrasound exam are not clear then diagnostic peritoneal lavage should be performed if there is a wound is over the region of the abdominal cavity and it is not certain if the abdomen was penetrated or not.

WOUND ASSESSMENT

Proper assessment of most wounds requires some form of analgesia at a minimum and may require general anesthesia. The injury is painful and even if the patient is stoic attention should be paid to providing adequate analgesia. All wounds should be widely clipped in order to be able to assess them properly. This includes abrasions and bruises. Frequently, animals that are bitten have been impaled by both mandibular and maxillary teeth. If bite marks are seen only on one side of the limb or trunk then the other side should be shaved to search for the wound.

ANESTHESIA

The goal of anesthesia in all patients is to ensure amnesia and analgesia. Analgesic drugs should be continued intraoperatively. If the patient seems to be responding to surgical stimuli additional analgesics may be indicated as opposed to just more inhalant anesthetic to mask the actual problem. This will help ensure a smoother recovery. In critical patients neurolept anesthesia (combination of an opioid and a tranquilizer) may be all that is required to provide adequate anesthesia. While some drugs such as ketamine provide some analgesia it should be kept in mind that many drugs that are commonly used, such

as propofol and isoflurane, have no analgesic properties.

Critical patients should be preoxygenated and intravenous anesthetic agents should be used in all patients to ensure rapid induction. Once the patient is intubated the lungs should be ausculted bilaterally to ensure the air is moving through all lung fields. Drugs that decrease cardiac output or cause vasodilation should be avoided whenever possible. This includes inhalants such as isoflurane and sevoflurane which are two of the most potent vasodilators available to veterinarians. Once the patient is intubated positive pressure ventilation should be instituted. Positive pressure ventilation should be continued until the patient is ventilating well on its own postoperatively based on assessment of capnometry or blood gases. In the author's experience hypoventilation is one of the major causes of instability during surgery as well as postoperative recovery problems.

Close monitoring is essential during anesthesia. Physical exam parameters as well as more advanced monitoring are indicated. Capnometry should be continually assessed intraoperatively and during recovery. Blood pressure monitoring preferably using a Doppler ultrasonic flow detector is indicated. Hypotension (systolic blood pressure less than 100 mm Hg and diastolic pressure less than 60 mm Hg) should be aggressively controlled. Vasoconstrictive agents should be used only if absolutely necessary since they may decrease tissue perfusion and cause hypoxia. Electrocardiography should be assessed continually for evidence of tall T waves (greater than one-quarter the R wave amplitude) indicating myocardial hypoxia and premature ventricular contractions.

It is important to ensure that appropriate venous access is available intraoperatively if drugs need to be administered periodically. T-ports should be placed on at least one catheter since drug volumes are often very small and need to be given close to the catheter to ensure rapid absorption.

OVERVIEW OF SURGICAL MANAGEMENT

All wounds should be widely clipped in order to be able to assess them properly. This includes abrasions and bruises. Frequently, animals that are bitten have been impaled by both upper and lower teeth (especially canine teeth). If bite marks are seen only on one side of the limb or trunk then the other side should be shaved to search for the wound.

All penetrating wounds should be surgically explored. This is especially important in the case of bite wounds since the teeth may only have made a puncture mark in the skin but as the animal was shaken there may have been extensive tearing damage done to underlying tissues. Wounds that penetrate the abdominal cavity may have caused hollow or solid organ damage. The exception to this may be the penetrating thoracic wound. In the author's experience these patients have a high incidence of mortality if they are taken to surgery within the first 6-12 hours. If the patient can be stabilized medically it may be appropriate to delay surgical intervention.

The goal of surgical management of wounds should be to explore and remove any foreign material, control hemorrhage, and remove necrotic tissue. Many wounds will require the use of general anesthesia; however, more superficial wounds that do not require extensive debridement can be managed under sedation and local anesthesia.

The wound should be widely clipped and surgically prepped. This is very important since the extensive nature of the trauma can be easily underestimated based on external visualization. As a general rule at least 10-15 cm (4-6 inches) should be clipped and prepped around the wound to

allow for exploring subcutaneous dissection and placement of drains.

The skin should be incised in order to be able to visualize and assess the entire wound. Most mistakes in wound care are made from a lack of knowledge about the extent of the trauma because the wound was not adequately explored. Foreign material should be removed as it is encountered. Tissues should be debrided back to bleeding edges whenever possible using sharp dissection. This is especially important with fat and muscle. Bone and ligament should be removed if the surgeon is certain it is nonviable but if there is any doubt the tissue should remain since removal may interfere with subsequent function of the affected area. Skin edges of wounds should be debrided using sharp dissection back to bleeding edges unless this might cause problems with wound closure. Scissors generally should not be used for debridement since they may cause crushing of the tissue, which can compromise circulation to the wound edges and cause problems with healing.

Wound and body cavity irrigation form an important part of any surgical procedure. “Dilution is the solution to pollution.” The use of sterile isotonic solutions is preferred. Tap water has been used to irrigate wounds without complication; however, tap water is hypotonic which may negatively impact tissues. Antibiotics should not be added to irrigation fluids since this the concentration is often diluted too much to be effective and extreme care should be exercised when adding disinfectants to irrigation fluids. Body cavities should be irrigated with sterile isotonic fluid only. Irrigation can be provided using mechanical lavage systems designed for wound irrigation or using a 1 litre fluid bag pressurized to 300 mm Hg. The ideal pressure that should be achieved is unknown but most agree on a pressure of 7-8 PSI. Some commercial irrigators generate 15 PSI which appears to be safe. One study demonstrated that use of a 35 ml syringe and needles from 16-22 ga in size routinely generated pressures well above 15 PSI which could lead to barotrauma. Irrigation should not be done blindly or up into holes since this may force infection or foreign material further into the wound or potentially into healthy tissues.

Accurate hemostasis is important since the presence of blood can act as a medium for bacterial growth and the presence of a hematoma can interfere with wound healing. Hemostasis generally is achieved by use of direct pressure, suturing of wounds (compression of vessels), electrosurgery, ligation of vessels, use of vascular clips, omental packing, superglue, hemostatic agents or removal of the organ that may be bleeding. There are numerous products available for hemostasis many of which are based on gelatin, fibrin, bovine thrombin, seaweed, kaolin and a variety of other natural products that have hemostatic qualities.

Wounds should be irrigated again prior to closing the skin to remove any additional foreign material and blood. The presence of blood provides an ideal medium for bacterial proliferation. If there is any doubt about viability of tissues the wound should not be closed initially. Instead wet-to-dry dressings should be placed and the wound should be revaluated on a daily basis. Daily debridement should be performed as necessary until the health of the tissues is assured. Wounds should be cultured prior to closure since multiple species of bacteria have been identified in dog bite wounds and no one antibiotic has been shown to be effective.

The amount of suture left in wounds should be minimized. Skin sutures should not be placed tightly since again this may compromise circulation. Wounds should not be closed under tension since this will

compromise wound healing. Skin can either be mobilized by undermining of healthy tissues or grafting should be used as needed.

Dead space can only be closed effectively using active suction drains or bandages. Both drains and bandages will help enhance wound healing and prevent seroma formation. Sutures can be used to approximate tissues but cannot close dead space. Using sutures to "close dead space" should be avoided since it can create compartmentalization and the amount of foreign material (suture) left in the wound is increased.

Two types of drains exist – passive and active. Passive drains such as Penrose drains allow wound exudate to drain by gravity or overflow. The most serious complication associated with Penrose drains is the risk of ascending infection and ideally Penrose drains ideally should be covered with a sterile dressing. Active drains remove wound fluids by application of negative pressure. Drains are attached to a suction bulb that is primed by removal of air. These are available commercially in various styles and sizes. Drain suction bulbs are emptied as necessary – usually 2-4 times per day and cytology is evaluated as indicated. Drains are left in place until they are no longer functional or until they are no longer needed. Drainage will slow down within 72 hours in most wounds. If a large amount of dead space was created the drains may need to stay in place for up to 5-14 days.

BANDAGES

Bandages are designed to protect the wound and encourage wound healing. They can also provide support to underlying tissues and help improve patient comfort and mobility. Contaminated or infected wounds should have wet-to-dry dressings placed until the wound is clean. When wet-to-dry bandages are removed they will help mechanically debride remaining foreign material and necrotic tissue. These dressings should be compromised of wide-meshed gauze, which will entrap particulate matter, soaked in 0.9% saline. Bandages should be placed over all surgical incisions for at least 24 hours until a fibrin seal has formed.

POSTOPERATIVE CARE

Postoperatively these patients will require close monitoring and treatment with a minimum of fluid therapy, analgesics, and antibiotics. Analgesics should be given on a scheduled basis but should also be given as needed since every patient's injury and tolerance to pain is different. Supplemental oxygen and/or ventilatory support may also be required. Urinary catheters should be placed in patients with difficulties with either ambulation or urination. Other treatment such as chest tube aspiration and care, care of suction drains and bandage changes will vary depending on the type of injury and the surgery performed. Monitoring will be dictated by the underlying trauma and status of the patient; however, a minimum of temperature, heart rate, respiratory rate and effort, and blood pressure should be assessed hourly until the patient is normothermic and stable. Critical patients will require blood work postoperatively; the tests will vary with the patient. Enteral nutritional support should be started within 6 to 12 hours which may require placement of a feeding tube.

Respiratory Emergencies - The Blue Patient

INTRODUCTION: *Respiratory distress most frequently is associated with a condition that is causing hypoxia although these patients may also be hypercarbic. Respiratory distress is a more appropriate term to use in veterinary medicine than dyspnea which is defined as the subjective awareness of altered or uncomfortable respiratory functioning. Patients may be experiencing problems with getting oxygen into the alveoli which can be a result of upper airway obstructive or disruptive abnormalities or chest wall integrity problems such as fractured ribs, sucking chest wounds or a diaphragmatic hernia. It can be caused by difficulties with lung expansion caused by pleural space disease (pneumothorax, pyothorax, chylothorax) or lower airway problems such as pulmonary edema and pneumonia.*

PHYSICAL EXAMINATION

Increased respiratory rate can indicate respiratory distress. This obviously needs to be differentiated from unrelated conditions such as pain or anxiety. Increased respiratory effort should always be taken as a sign of respiratory distress. Open mouth breathing and simply being able to easily observe chest wall movement and auscult lung sounds (unless an electronic stethoscope is being used) should be considered abnormal until proven otherwise.

The patient's posture should be noted. Dogs and cats with increased respiratory effort secondary to injury or disease often are unwilling to lie down although the cat may sit in sternal but refuse to curl up or lay in lateral recumbency. Any cat lying in lateral recumbency with signs of respiratory distress should be assumed to be close to arrest until proven otherwise. Nostril flaring indicates increased respiratory effort but does not necessarily indicate pathology.

Wheezing, crackles, and stridor all indicate abnormalities. The presence of stridor should alert the clinician to the fact that there may be an almost 80% airway occlusion. The trachea should be evaluated in all patients. Once the trachea has been ausculted the neck should be palpated noting tracheal position and tracheal/peritracheal integrity. The presence of subcutaneous emphysema in the cervical or thoracic region in a cat that has a history of a recent anesthetic or trauma to this region often is associated with a ruptured trachea. This can be associated with a pneumomediastinum and pneumopericardium, which can become a tension situation if there is no escape valve to the exterior of the animal. Auscultation of the trachea is often a more direct window into lower airway pathology than transthoracic auscultation

The breathing pattern should be closely observed. Symmetry of chest movement and the presence of any abdominal component to the breathing pattern should be noted. Rapid shallow respiration typically is associated with pain (especially related to chest wall trauma) or pleural space disease where the patient is unable to expand its lungs. Pneumothorax, hemothorax, chylothorax, and pyothorax all

can be associated with a restrictive breathing pattern. If paradoxical chest wall movement is observed a flail chest should be suspected. Increased chest wall expansion often is associated with lower airway disease although can indicate a collapsing trachea. Prolonged or forced expiration is associated with trapping of air in the lower airways such as occurs with allergic bronchitis or other diseases causing bronchospasm. Respiratory muscle abnormalities are associated with a significantly increased effort on inhalation with decreased chest wall expansion. This is most commonly seen with neuromuscular diseases and diaphragmatic hernia. Patients with paradoxical abdominal movement have severe respiratory compromise. These patients also should be assumed to be close to collapse due to exhaustion and ventilatory failure until proven otherwise.

The chest should be ausculted for the presence of breath sounds, areas of dullness, crackles or wheezes in at least 4 quadrants (upper and lower right and left sides). Crackles indicate alveolar exudate – typically pulmonary edema or pneumonia. Crackles may be very difficult to auscult in cats. Wheezes are consistent with obstructive lower airway disease. Foreign bodies in the lower airways also can cause similar sounds. Areas of dullness may indicate severe pulmonary infiltrate, pleural fluid, intrathoracic masses, or the presence of abdominal contents in the thorax. The heart should always be ausculted after ausculting the lungs since once the ear has accustomed itself to louder sounds quieter sounds can be more difficult to hear. Because of the narrow chest wall of the cat, lung sounds can be referred easily across both hemithoraces making it difficult to pick up unilateral abnormalities in this species.

Gastric distention secondary to aerophagia can lead to significant respiratory compromise or even cardiovascular collapse and the abdomen should be examined with this in mind.

Cyanosis is an indication of hypoxemia or a PaO_2 of less than 60 mm Hg. Cyanosis can be difficult to detect if the patient has a hemoglobin less than 5 g/dl or with certain fluorescent overhead lights.

RADIOGRAPHS

Radiographs are an essential component of the evaluation process for the patient with respiratory compromise; however, they should not be a priority in the unstable patient. Stabilizing the patient is always the first priority. Care should be taken to ensure positioning does not compromise the patient's ability to breathe. The radiograph should be evaluated systematically to ensure abnormalities are not missed. The bones, soft tissues surrounding the thorax, pleural space, trachea and large airways, lungs, mediastinum, heart, great vessels and diaphragm all require assessment. In patients with upper airways diseases the cervical trachea and pharynx may require radiographic evaluation. Sedation may be required for diagnostic radiographs but this should be done with extreme caution in the compromised patient. Dynamic studies often provide valuable information.

Ultrasound

Point of care thoracic ultrasound can provide a rapid, efficient means of diagnosing pathology and has been shown to be more sensitive than a stethoscope. Five anatomic locations are interrogated. Pleural and pericardial effusion can easily be diagnosed. Lack of a glide sign indicates a pneumothorax and the presence of hyperechoic lines ("lung rockets") is consistent with pulmonary disease including contusions, pulmonary edema, pneumonia, and neoplasia.

RESPIRATORY SUPPORT

Respiratory support of the critically ill or injured patient can be divided into oxygen support and ventilatory support. The end goal of respiratory support is to ensure adequate oxygen reaches the blood and carbon dioxide is removed from the blood. Oxygen should be considered a first line drug and should be provided to any patient that presents with an increased respiratory rate or effort or evidence of cyanosis.

OXYGEN

Oxygen can be provided in a variety of forms. An oxygen source, baggie, plastic wrap, Elizabethan collar, and red rubber tubes are all that are necessary to provide oxygen to almost any patient. It is recommended that a direct oxygen source be available; however, if an anesthetic machine is used then a "Y"- shaped adapter should be used to bypass the anesthetic circuit. A "Y" connector is placed in the tubing before it enters the circle. A piece of tubing connects the "Y" to the circle and the second arm of the "Y" is connected to the oxygen tubing to the patient. A hemostat or C clamp is used to clamp off the oxygen to the patient or to the circle system depending on what is required. Nasal and tracheal oxygen should always should be humidified, although nasal oxygen may be able to be delivered for up to 24 hours not humidified. Hood, mask and flow-by oxygen should not be humidified.

Oxygen is most easily provided by using oxygen tubing that is connected directly to the oxygen source. The end of the tube is placed in front of the patient's nose or mouth. The flow rate is 1-10 L/min, depending on the size of the patient, but it may need to be decreased based on patient tolerance. A mask also can be used but is often much less well tolerated and may cause increased stress unless the patient is recumbent. If a mask is used the rubber fitting should be removed. Many animals will tolerate having their heads or even most of their bodies placed inside a plastic bag. The oxygen tubing is placed through a small hole in the front of the bag and the back of the bag is left open to allow gas to escape. This is particularly useful in the obtunded patient because high concentrations of oxygen can be provided (75-95%) while allowing other procedures to be performed (blood drawing, placement of catheters, x-rays etc.) An oxygen hood can be made by covering the ventral 75% of an Elizabethan collar with plastic wrap. The Elizabethan collar should be 1 size larger than would normally be used for that size of patient. The oxygen tubing is placed along the inside of the collar and taped in place ventrally. Oxygen concentrations of up to 80% generally can be achieved. Flow rates of approximately 1 L usually provide an adequate FiO_2. Flow rates should be adjusted based on patient comfort, clinical status, pulse oximetry, and blood gases. Oxygen hoods generally are not tolerated by the panting dog as the hood rapidly becomes overheated and over-humidified.

Nasal oxygen is the most effective way to provide oxygen to the patient. For small patients 3.5 to 5 Fr tubes are used. For medium-sized dogs 5-8 Fr tubes are used and for larger dogs 8 to 10 Fr tubes are placed. Cats will usually tolerate 5-8 Fr tubes. The nasal catheter is typically measured from the tip of the nose to the lateral canthus of the eye so that the tip will be in the nasopharynx (nasopharyngeal catheter). Clinically animals tolerate the oxygen better if the tip is at this location as opposed to being in the nostril. A narrow bore red rubber or other pediatric feeding tube is placed in the ventral nasal meatus and sutured or stapled to the patient's nose and on the side of the face or on the bridge of the nose between the eyes. At flow rates of 100 ml/kg/min the FiO_2 will be a

minimum of 0.4 and may reach as high as 0.65. Nasal oxygen should be avoided in the patient with severe nasal or pharyngeal disease and in the patient with severe thrombocytopenia. Sneezing will elevate intracranial pressure and nasal tubes should be avoided if this is a concern.

Oxygen cages also can be used to provide oxygen to patients but have several drawbacks and should be used only if other forms of providing supplemental oxygen are contraindicated. The biggest problem is the inability to evaluate the patient except through observation. Each time the door to the cage is opened the oxygen level drops substantially. This can lead to significant patient anxiety and respiratory compromise. The oxygen flow rates required to operate the units effectively makes this a costly alternative. On occasion, due to the stressed nature of cats with respiratory problems an oxygen care is essential. It would be ideal in these circumstances to use a small volume 'cage' such as a pediatric incubator.

Gastric Decompression

Patients with significant gastric distention that appears to be causing significant respiratory compromise or hemodynamic instability may require immediate gastric decompression. This can be accomplished either by transabdominal trocarization or orogastric intubation. Immediate decompression of a severely distended stomach can lead to cardiovascular collapse and ideally should be avoided until fluid resuscitation has been initiated.

VENTILATORY SUPPORT

If the patient does not respond to supplemental oxygen rapid sequence induction, intubation, and ventilation should be considered. Suction should be readily available. Response to therapy usually can be gauged by monitoring respiratory rate and effort, presence of cyanosis, pulse oximetry readings, and blood gases.

TRACHEOSTOMY

A tracheostomy is indicated in the patient with an upper airway obstructive disorder that cannot be relieved, when airway control is indicated but an endotracheal tube is not possible or not desirable, in patients with severe bronchopneumonia, and in the patient who requires prolonged ventilatory support. If the thought occurs to you that a tracheostomy is indicated then one probably should be placed! Other indications include situations when an endotracheal tube cannot be inserted in a patient with an obstructed or near obstructed airway, when the obstruction is rostral to where the proximal portion of the tracheotomy tube ends, when it is necessary to assess and treat the bronchoalveolar (pulmonary) tree such as delivery of medications and aspiration of exudate, and when it is necessary to decrease the dead space and airway resistance, in order to decrease the work of breathing.

There are no absolute contraindications but there are several relative contraindications. If the tracheostomy is the only breathing route for the patient then the patient must be monitored around the clock since coughing mucus into the tube will cause a complete airway obstruction and suffocation. Appropriate humidification and suction equipment as well as replacement tubes must be pleasant. A tracheostomy may not be ideal when the patient has a coagulopathy, when suction equipment does not exist, and in situations when an endotracheal tube may suffice.

A tracheotomy can be performed most easily on an anesthetized patient. The patient is placed in dorsal recumbency and a towel or IV fluid bag is placed under the neck which pushes the trachea ventrally. An incision (approximately 5-8 cm or 2-3 inches long) is made on the ventral cervical midline about midway between the cricoid cartilage and the thoracic inlet. The "strap" muscles (sternohyoideus) are separated using blunt or sharp dissection and the trachea is exposed. The trachea is elevated into the incision using thumb and fingers. An incision is made between 2 tracheal rings at the level of rings 3 to 6 extending about 40% of the circumference of the trachea and a tube is placed in the incision. Traction sutures are then placed through the 1 ring cranial and 1 ring caudal to the tracheotomy and tied with the knot approximately 8-10 cm or 3-4 inches from the trachea. These sutures are used for opening the trachea when the tube needs to be exchanged. A tube approximately 1-1.5 sizes smaller than what would be used for orotracheal intubation is placed.

Commercial tracheostomy tubes can be used or a clear endotracheal tube can be modified. To modify an endotracheal tube the plastic connector is removed from the end of the tube. Two cuts are then made in the tube 180 degrees apart. The cuts are made long enough so that the tube that remains intact is the right length for the patient (i.e., reaches from the tracheotomy to the thoracic inlet region). Do not cut the cuff inflating mechanism. The 2 wings that are created can be cut short if needed. The tube connector is placed back into the tube. Two holes are created the end of each wing and umbilical tape or IV tubing is placed through the holes and tied around the back of the neck of the patient. The tube is not secured in any other form to the patient. Two or 3 sterile 4x4 squares are placed between the tube and the tracheotomy incision.

Choosing an inappropriately-sized endotracheal tube can lead to a significant problems for a patient if they are breathing spontaneously. One study showed an increase in the work of breathing of 34% and increase in airway resistance of 25% if the diameter of the endotracheal tube was decreased by only 1 mm. When picking an appropriately-sized tube estimation by digital palpation of the trachea was shown to be the most accurate method.

Sterile saline (2-10 ml depending on the size of the patient) should be instilled or the patient should be nebulized (preferred) q2-4 hours to help lubricate respiratory secretions. The tube should be suctioned q6-8 hours after instilling saline and hyperoxygenating, and should be aseptically changed q6-12 hours or as needed. When suctioning larger patients the operator should inhale a normal breath and hold the breath. When the operator comfortably feels the need to breathe suction should be discontinued. For small patients the breath should be exhaled then held. When the operator comfortably feels the need to breathe the suction should be discontinued. Oxygen can be provided via the tracheostomy by placing a sterile red rubber catheter through the tracheostomy tube. Care should be taken to ensure the oxygen tube is not too large and does not obstruct exhalation. When the tube is no longer needed the tracheotomy incision is left to heal by second intention. It should not be bandaged until the tracheotomy incision is healed to avoid developing subcutaneous emphysema.

THORACENTESIS

Pleural space disease (pneumothorax, hemothorax, pyothorax, chylothorax) often can be diagnosed based on the presence of

a rapid shallow respiratory pattern, loss of airway sounds, or hollow sounds on percussion of the thorax. Any patient who is suspected of having pleural space disease should have a thoracentesis performed prior to taking radiographs. The stress of the radiographic procedure in a patient with severe pleural space disease may lead to respiratory arrest. Thoracentesis is performed between the 7th and 9th intercostal spaces. The thoracentesis is performed in whatever position the patient is the most comfortable (sternal, sitting, lateral recumbency). Thoracentesis should always be performed bilaterally unless the patient is known to have unilateral disease. The area is clipped and prepped and if the patient is painful local anesthesia should be instilled in the skin and down to the pleura. The needle is introduced slowly until the pleura is penetrated at which point the needle is angled parallel to the chest wall with the bevel pointed medially. This will prevent injury to the lung as the pleural space is evacuated. If negative suction is not achieved a chest tube will need to be placed.

CHEST TUBES

Chest tubes can be placed under sedation and local anesthesia or under general anesthetic. In most dogs chest tubes can be placed under sedation and local anesthetic. General anesthesia is required in most cats. If general anesthesia is required the patient should be intubated and ventilated. The size of the chest tube should be the approximate diameter of the mainstem bronchus in a patient with a pneumothorax since this is conceivably the largest hole that could exist. It also helps prevent having the tube clog with viscous fluids or blood clots. Smaller diameter tubes may be chosen for patients with a chylothorax or pyothorax.

In the case of a pneumothorax a 3-way stopcock can be placed in the tube and the tube can be aspirated on an intermittent basis; however, this is only advised if it is anticipated that the patient will only accumulate small volumes of air. Ideally continuous underwater suction should be used on chest tubes until it is established that the air leak is resolving.

Analgesia must be provided to every patient with a chest tube. This can be effectively provided using local or regional blocks a mixture of lidocaine and bupivacaine. Intercostal nerve blocks for 1-2 rib spaces either side of the tube can be performed or intrapleural analgesia can be provided by administering the local anesthetics via the chest tube into the pleural space. Local anesthetics should always be either warmed to body temperature or mixed (1:9) with sodium bicarbonate to decrease the sting. Parenteral narcotics should be provided if local anesthetics are not providing sufficient analgesia.

CONTINUOUS POSITIVE PRESSURE AIRWAY SUPPORT

Continuous positive airway pressure helps to decrease the work of breathing and improve gas exchange. It is defined as maintaining the pressure above atmospheric pressure throughout the respiratory cycle. This can be used as a bridge in patients that do not fully respond to oxygen support but positive pressure ventilation is not an option or if it is felt that some assisted ventilation may help avoid the need to positive pressure ventilation. A modified form of CPAP can be fairly easily provided to most awake dogs. A fairly tight fitting mask attached to an anesthetic circuit is placed on the patient. The pop-off valve is tightened down and the oxygen flow rate is increased until the pressure on

the circuit registers at 5 cm H_2O. The patient breathes this oxygen under high pressure.

NEBULIZATION

Nebulization therapy should be used for treating patients with pneumonia and bronchoconstrictive disease (i.e., feline allergic bronchitis). It is provided using a commercial unit or oxygen delivered at high flow rates through a nebulizer. The nebulized fluid can be delivered via face mask, into a baggie placed over the patient's head, or into an enclosed chamber if the patient will not tolerate the flow directed at the face. Saline (0.9%) is an excellent mucolytic if nebulization is being used to loosen secretions. Bronchodilators such as albuterol and terbutaline as well as corticosteroids such as fluticasone can be given by nebulization to asthmatics.

How To Make The Right Decisions In The First 10 Minutes

INTRODUCTION: *The hospital must be maintained in a state of readiness in order to be able to prevent catastrophic consequences from occurring when the severely ill or injured patients are presented to or being treated in hospital. This state of readiness includes not only the physical equipment but also the mental readiness of the doctors, nurses and receptionists. In-hospital training and drill sessions are recommended to practice assessment and resuscitation team skills. Everyone must be trained to deal with typical emergency situations and equipment must be available and ready to be used at a moment's notice. Drill sessions facilitate the practice of psychomotor skills and working as a team, providing for effective and efficient treatment of critical patients. Lack of preparation may make the difference between life and death.*

Trauma protocols for general assessment, management, and treatment for specific catastrophic injuries are recommended. These protocols should be printed and reviewed at staff meetings. They may also be posted in key areas in the hospital or the "ready" area where they can be referred to easily. They act as guidelines and mental reminders for the staff and clinician in-charge - increasing team efficiency and helping to prevent assessment and management mistakes. Each trauma protocol may be organized in a numerical or alphabetical list of steps to follow, or in an algorithm. Protocols should be reviewed and revised as required periodically to insure they remain current, easily understood, and effective in the setting they are used.

GOALS

The fundamental goals of patient care should be to avoid hypoxia and hypercarbia, to ensure the patient has a normal hemodynamic status (normal heart rate, blood pressure, blood volume, urine output), and to keep the patient as pain free and comfortable as possible. Enteral nutrition should be provided as early as possible to help maintain gastrointestinal function and integrity of the gut mucosal barrier.

CRASH CART

A ready area is essential for performing effective CPR and resuscitating critically ill or injured animals since seconds may mean the difference between life and death. Oxygen, fluids ready to be administered, and a crash cart containing all the supplies needed to deal with a life-threatening emergency should be present in the ready area. It is recommended that a multi-drawer tool cart be purchased for use as a crash cart. The crash cart should be mobile (on wheels or transportable) in order to be able to take the equipment and supplies to the patient, no matter where the emergency takes place. The cart should be reserved for emergency use only and material should not be removed unless a code is being run or emergency resuscitation is being performed. Supplies that may be used routinely such as laryngoscopes, catheters or certain drugs such as furosemide should be stocked in a general use area as well as in the crash cart. Emergency resuscitation can be

unsuccessful simply because someone "borrowed" something for another patient and forgot to return it.

An oxygen source must be available and an AMBU bag should be kept connected to the oxygen source. An AMBU bag can be connected using piped-in oxygen or an anesthetic machine. If an anesthetic machine is being used a "Y" connector should be inserted between the oxygen outflow and the anesthetic circuit. One arm of the "Y" is connected to the AMBU bag. A bag of fluids should be connected to a macrodrip set and placed in a pressure infusor bag ("slam bag").

The top of the crash cart should hold an ECG machine, ECG gel, a capnograph, a suction unit, a Doppler blood pressure monitor, ultrasound gel, and at least 3 sizes of blood pressure cuff. The Doppler unit must be charged and a probe left attached at all times. All electronic equipment must be plugged in. Electrocardiogram leads must be attached to the machine. Either a commercial suction unit or a Mityvac (brake line suction unit) should be present and a Yankauer suction tip or some other tip capable or aspirating thick exudate, vomitus and blood from the pharynx, larynx, and trachea should be attached to the suction unit. An endotracheal tube can be attached to a suction unit on an emergency basis and used to suction the oropharynx.

An emergency drug chart should be posted on the cart and ideally on the wall in the ready area so that it can be determined immediately how much medication the patient needs.

The first two drawers of the crash cart should contain airway equipment and drugs respectively. The airway and drug drawer are lined with foam and sections are cut out of the foam to hold the supplies. This serves to keep the drawer organized and also indicates when something is missing from the cart. The airway drawer should contain a laryngoscope with a small and large Miller blade and a variety of sizes of endotracheal tubes. Each tube should have gauze or IV tubing attached to it so once the patient is intubated the tube can be secured immediately in place. Each tube should also have a syringe attached to it primed to inflate the cuff once the patient is intubated. Foerster sponge forceps and Velsellum forceps should be available to remove foreign material from the mouth and oropharynx. A #10 scalpel blade and a pair of sharp Mayo scissors should be present to perform a tracheotomy and open chest CPR if necessary.

The second drawer should contain emergency drugs such as epinephrine, atropine, lidocaine, sodium bicarbonate, dexamethasone sodium phosphate, and furosemide. Other recommended drugs include mannitol, dextrose, dopamine, dobutamine, and calcium gluconate. Both 3 cc and 12 cc syringes with needles attached should be present alongside the drugs. A stiff long 3.5 Fr urinary catheter or red rubber tube should be available for instilling drugs via the endotracheal tube.

The third drawer should contain different sizes hypodermic needles and intravenous catheters. A large syringe with an extension set and 3-way stopcock connected should be present for performing rapid thoracentesis. Tape for securing catheters should be tabbed ready for easy use. Remaining drawers should contain surgical

gloves, gauze squares, intravenous fluids, chest tubes, and surgical instruments and supplies that might be needed for resuscitation. A basic surgical pack should also be present for performing emergency surgical procedures. This should include a scalpel handle, curved Mayo and Metzenbaum scissors, curved mosquito and Kelly hemostats, tissue forceps and a Balfour retractor. Sterile red rubber tubes, gauze and towels can also be very useful.

Intravenous fluids should include a combination of crystalloids and colloids. A buffered crystalloid such as Normasol-R or Plasmalyte-A and 0.9% saline in one litre bags should be available. In addition D5W or 0.9% saline 250 ml bags should be available for mixing up constant rate infusions. A synthetic colloid should be available as well as biologic colloids such as packed red blood cells and fresh frozen plasma. If blood products are not available then a walking donor program where blood can be collected on an emergency basis is vital.

The ready area requires good lighting, similar to that required in the operating room. Dual lights that can be directed at divergent angles are especially important for the care of the seriously injured patient. These patients frequently require emergency surgical procedures that demand this availability of good illumination, e.g., venous cutdown and slash tracheostomy. A focusing high-intensity cool beam light (very useful for close/exacting or deep cavity work) and a wider beam reflecting dish light for general full-body illumination are recommended. A headlamp is very effective for directing light into the appropriate location; inexpensive headlamps are available through camping and hardware stores.

In-house determinations of hematocrit, total serum solids, platelet numbers, white blood cell count and differential cell counts should be standard requirement. An ability to determine blood glucose, activated coagulation time or PT and PTT, creatinine, serum electrolytes, blood pH and blood gases can be life saving. A good microscope must be present for evaluation of blood smears, urine sediments, and body cavity aspirates.

TRIAGE

Triage is defined as the sorting or classification of patients according to the priority of need for treatment. It was designed to help avoid unnecessary death because of inappropriate attention. It starts on the phone when an owner calls into the hospital or when an owner walks in the front door with their pet. The members of the team that have first contact with the pet need to be trained in how to triage - how to take a quick accurate history and to recognize abnormalities.

Certain problems or complaints always warrant immediate attention and immediate evaluation. These include airway or breathing abnormalities, bleeding, a history of trauma, non-productive retching, profuse vomiting or diarrhea, urethral obstruction (“straining to defecate”), seizures, loss of consciousness or collapse, heat prostration, open wounds, shock, anemia or pale mucous membranes, burns, dystocia, prolapsed organs, abdominal distention, and extreme restlessness.

PRIMARY SURVEY

Once the patient has been triaged to the ready area a primary survey is completed within 30 to 60 seconds. The goal of this survey is to determine the presence of emergent conditions that require

immediate treatment. The following steps are recommended.

1. Visually assess the patient from a distance, noting LOC, unusual body or limb posture, the presence of blood or other materials on or around the patient, and any other gross abnormalities. Note breathing effort and pattern and any airway sounds generated.

2. Approach the patient from the rostral direction, noting the level of awareness and its reactions to this movement. Ask questions concerning the patient's temperament. Take appropriate safety precautions in "questionable" animals, (muzzling, head covering, physical restraint).

3. Assess airway and breathing status by closely observing colour of the oral mucus membranes (capillary refill time is also assessed at this time), listening for tracheal breath sounds (first without, then with the aid of a stethoscope), palpating the neck noting tracheal position and tracheal/peritracheal integrity. Injuries to the skin, subcutaneous emphysema, and blood in the nose or mouth are assessed for bilaterally.

4. Continue to assess the patient's breathing status by observing, palpating and then listening to the thorax (first without then with the aid of a stethoscope). Lung sounds should be auscultated bilaterally (heart tones are also assessed following lung sounds). Changes involving the skin over thorax and cranial abdomen such as erythema, bruising or subcutaneous emphysema, should be assessed by visualization and palpation.

5. Cardiovascular assessment is completed by palpating pulses during auscultation of the heart. Pulse strength, vessel tone and rate are easily determined in all but the very smallest animals, unless they are obese or very cold. Assessment of heart tones, mucus membrane colour and capillary refill time, already completed earlier can be repeated, as well as any other part of the primary survey thus far if there are any questions.

6. The primary survey ends with very rapid observation and palpation of the abdominal, flank, and pelvic regions, as well as the spinal column, and limbs, noting anything abnormal.

Any major abnormalities are immediately treated. Unconscious patients should be intubated and ventilated if necessary. Drugs should be given immediately to seizing animals. This may include dextrose or antiepileptic drugs. Dextrose can be given intraosseously in very small patients if immediate vascular access cannot be obtained. Drugs such as diazepam can be given intranasally while an attempt is being made to gain vascular access. If the patient is showing any evidence of an increased respiratory rate or effort then flow-by oxygen should be provided. Transtracheal oxygen may be indicated in cases of upper airway obstructive diseases. A thoracentesis may be required immediately if the patient is cyanotic and has a restrictive breathing pattern. Fluids are infused intravenously if there is evidence of hemodynamic collapse. This may require a vascular cutdown. Temporary sterile dressings are placed over bleeding wounds or herniated organs. Direct pressure will typically control most external hemorrhage. Analgesics should be given to painful patients. Sedatives may be required in combative patients.

COMPLETE HISTORY AND PHYSICAL EXAMINATION (SECONDARY SURVEY)

After the emergency patient has undergone successful resuscitation of the catastrophic

life-threatening injuries or is deemed "stable" following the primary survey, a thorough history and physical examination is performed. The history and initial survey should always be performed in the same manner every time to ensure consistency and that no important details are missed. Vital signs including temperature, heart rate and rhythm, pulse rate and strength, and respiratory rate and effort are recorded during the secondary survey.

In English, the mnemonic AMPLE is one of several mnemonics that can be used for this purpose.

A :Does the pet have any allergies?

M : Is the pet on any medications?

P :Does the pet have any past history of any problem?

L : When was the pet last normal? Last meal? Last urination? Last defecation?

E : What were the events leading up to the problem?

In the severely traumatized patient it should be assumed that fractures are present until proven otherwise and the patient may need to be restrained to prevent further injury. The most effective method of restraint is to tape the animal to a board with duct tape. Backboards made from Plexiglas are useful because not only are they sturdy, but the animal can be visualized on all sides, and radiographs can be taken without having to remove the animal from the board.

The five vital signs are temperature, heart rate, respiratory rate, blood pressure and pain. Each should be assessed. Heart rhythm and pulse rate and strength should also be assessed at this time.

The breathing pattern should be closely observed and evidence of increased effort noted. Symmetry of chest movement and the presence of any abdominal component to the breathing pattern should be noted. The chest should be ausculted for the presence of breath sounds, areas of dullness, crackles or wheezes in at least 4 quadrants (upper and lower right and left sides). Chest percussion in at least 4 quadrants will help detect areas of dullness suggesting pulmonary contusions, hollow sounds indicating a pneumothorax, or a fluid line indicating a hemothorax.

Blood pressure can be recorded directly or indirectly. Direct BP measurement is the most accurate but requires placement of an arterial catheter which is not practical in the emergency setting. Indirect BP measurements can be recorded using a Doppler ultrasonic flow detector or an oscillometric device. Doppler flow detectors are preferred over oscillometric devices since they also allow the clinician to assess flow to the periphery of the limb to which the ultrasonic flow probe is attached. Often irregular heartbeats such as premature ventricular contractions can be detected by the trained ear. Mean BP should be maintained above 65 mm Hg to ensure renal perfusion; however, hypertension in the hemorrhaging patient may worsen the hemorrhage and should be avoided.

Mean arterial pressure (MAP) can be calculated only if both systolic and diastolic BP are measured. Mean BP is calculated using the following formula:

MAP = diastolic BP + (systolic BP - diastolic BP)/ 3

Diastolic BP can be consistently measured using a Doppler flow detector in many patients. Digital palpation of femoral or peripheral arteries may not provide an adequate assessment of BP. The ability to detect pulses depends on the degree of vasoconstriction, pulse pressure (difference

between systolic and diastolic BP), and skill of the clinician.

Jugular veins should be clipped and checked for filling when held off at the thoracic inlet. Flat jugular veins that cannot be raised indicate severe hypovolemia. The presence of jugular distention in trauma patients in shock is most likely an indicator of increased intrathoracic pressure or venous obstruction. In the previously healthy animal this may indicate a pneumothorax or pericardial tamponade. If the animal has underlying heart disease jugular distention can be associated with right heart failure.

Toe web temperature should be taken and compared with the rectal temperature. A difference of greater than 3.5C is strongly suggestive of poor peripheral perfusion. Mucous membrane color and capillary refill time should be recorded.

The abdomen should be auscultated for bowel sounds and percussed for areas of dullness that would suggest fluid in the abdomen or tympany suggesting a torsed hollow viscus. The abdomen should be palpated thoroughly but gently as organs may be bleeding and additional pressure could cause them to rupture. Focal areas of pain should be identified. The caudal abdomen should be carefully palpated for the presence of a urinary bladder. Cranial nerves should be assessed and a fundoscopic exam performed. The ear canals should be evaluated with an otoscope for the presence of fluid (blood, cerebrospinal fluid). Fractures may worsen with movement and if the animal is recumbent only spinal reflexes and the presence of limb sensation should be assessed until radiographs have been taken. The entire body should be palpated gently for fractures, swelling, and wounds. After clipping any wounds a sterile dressing should be applied. Wounds should be covered with sterile saline and a sterile water soluble lubricant prior to being clipped. This prevents further contamination and tissue desiccation, and may help avoid healing complications. Fractures of the distal limbs (below the elbow and stifle) should be stabilized following assessment of the injury. Newspaper splints are placed easily and rapidly and are very effective as temporary splints. Radiographs can be taken through the newspaper.

Gizmos And Gadgets

CRASH CART

A crash cart can be made from a handyman's cart with multiple drawers in it (available from any hardware store) or fishing tool box. Each drawer should be labeled. Foam padding can be used to line each drawer and holes can be cut out of the foam to hold tube and bottles in place. The first drawer should contain airway materials - endotracheal tubes, a long polypropylene 3.5 or 5 Fr catheter for instilling drugs intratracheally during CPR, forceps for removal of foreign material and a scalpel or Mayo scissors for surgical airways. Mechanic's helpers available from any automotive store make very useful grabbers for oral, airway and esophageal foreign bodies. Each endotracheal tube should have a partially inflated syringe attached to the cuff and gauze attached to the tube for securing the airway once it is in place. The second drawer should contain emergency drugs and syringes (1 cc and 12 cc) preloaded with 18ga needles. The third drawer should contain hypodermic needles, peripheral catheters of various sizes, butterfly catheters, and larger 13 cm 14g and 16g catheters for pericardiocentesis and diagnostic peritoneal lavage. Syringes, tape, number 15 scalpel blades for making side holes in catheters and a 35 to 60 cc syringe with an extension set and 3-way stopcock connected should be present. All equipment should be compartmentalized in order to visualize the retrieve the appropriate equipment rapidly. Instead of compartmentalization certain supplies can be placed in labeled zip lock bags. The fourth drawer should contain fluids and administration set, extension sets, t-ports and male catheter plugs. Buretrols are useful for making up smaller volumes of fluids with additives. Blood transfusion sets and filters ideally should be available.

AMBU Bag

AMBU bags are resuscitator bags with one-way valves. A section of corrugated tubing or a rebreathing bag is attached to the end of the AMBU bag to act as a reservoir for oxygen to be stored in while the AMBU bag is squeezed, delivering positive pressure ventilation to the patient. When using an AMBU bag, conventional or high frequency ventilation can be delivered easily without concern for the pop-off valve and worrying about matching flow rates to the rates needed during resuscitation. Because the hands of the ventilator are closer to the patient he/she can "feel" the pressure developing in the lungs much better than if using a rebreathing bag on an anesthetic machine. Also pure oxygen, not oxygen that is scented or containing any anesthetic gases can be delivered via an endotracheal tube or mask if assist ventilation is being attempted. In the former case this is important because even a small amount of anesthetic can have disastrous negative consequences in an animal that is arrested. In the latter case it is important in the conscious patient that frequently objects to the anesthetic.

"Y" CONNECTOR

Oxygen is given by tubing connected to the anesthesia unit by a "Y" connection. The oxygen hosing from the source to the anesthetic machines is removed at the insertion to the anesthetic machine. A "Y" connector is inserted at the tubing. If a "Y"

connector is not available then a "T" connector from the plumbing section of a hardware store is used. One end of the "Y" is connected back into the anesthetic machine via a piece of suction or oxygen hosing. The other arm of the "Y" is connected to suction or oxygen hosing and coiled up for use in emergency situations as oxygen tubing. The "Y" has 2 clamps on it- one going to the anesthetic machine and one going to the oxygen tubing. If oxygen is required the clamp is closed going to the anesthetic leading to the anesthetic circle to prevent oxygen going anywhere than to the oxygen hood, mask, nasal cannula or AMBU bag. The tubing and connectors are all available through medical supply companies but can also be purchased in home hardware stores. A commercially available mare uterine flush system can also be used in place of "Y" or "T" connectors.

OXYGEN HOOD (COLLAR)

These can be made from Elizabethan collars and plastic food wrap or it can be commercially purchased. Homemade collars should have the top ¼ of the collar open to the air. The oxygen tube is placed into the collar from the neck side and taped in place to the inside of the collar and to the outside of the collar to prevent accidental dislodgement. A roll of 1" or 2" tape taped to the outside of the underside of the collar will create a pendulum effect and help prevent the collar from rotating. Oxygen flow rates vary from 1-10l/min depending on the size of the patient. The oxygen should not be humidified to prevent moisture build-up. The patient must be monitored closely for signs of overheating – especially if the patient is panting.

FOERSTER SPONGE FORCEPS

These 10" slightly curved sponge forceps should be always available for the retrieval of oral, pharyngeal and upper airway foreign bodies. A gauze sponge can be placed in the jaws and the oropharynx can be swabbed effectively to help clear an airway obstruction of mucus, blood or vomitus without having to insert fingers into the mouth of the animal. They can also be helpful when used across the hilus of the lung or spleen when these are badly traumatized and hemorrhaging badly.

VELSELLUM FORCEPS

These straight forceps have jaws with 2 sharp prongs on each side. They are very useful for grabbing smooth objects such as rubber balls that become lodged in the oropharynx.

BRAKE LINE SUCTION UNIT

This is a hand-held suction unit used to clear air from brake lines. It is a very effective device for suctioning airways and will generate pressures of up to 760 mm Hg. A fluid trap can be placed between the suction tubing and the suction tip to avoid having fluid accumulate in the suction unit. It can be purchased from hardware and automotive stores.

CLEAR ENDOTRACHEAL TUBE

Endotracheal Tube - The clear low pressure, high volume cuffed tube is the preferred to opaque tubes. This is because of the ability to monitor the inside of these tubes for a vapour trail or the lack of it, blood, vomitus, etc. The cuff is much safer than those in many other types since it is lower pressure than the red tubes. The cuff inflating mechanism has a one-way valve on it,

making it easier to inflate. Red tubes tend to become more brittle with continued use and may create more trauma to the tracheal mucosa.

Tracheotomy Tube - The connector is removed from the end of the tube. Two incisions 180 degrees apart are made in the tube, peeling it down like a banana. Care is taken to keep the cuff inflation mechanism intact. The incisions are made so that the intact section of the endotracheal tube is the right length for the patient (i.e. from the tracheotomy incision to the thoracic inlet). The plastic connector is reattached to the tube and the split pieces are connected to gauze or sections of umbilical tape.

Chest Tube - This can be made out of a clear disposable endotracheal tube and the use of a bone rongeur to make side holes. The cuff inflation tubing needs to be tied off. The chest tubes can be sterilized by ethylene oxide or glutaraldehyde.

Mouth Gag – Sections of 3-4 mm endotracheal tube can be used to make mouth gags.

COPPER WIRE

Copper wire that is sanded on the tip makes a malleable stylet for endotracheal tubes. The size of the cooper wire can be adjusted based on the size of the endotracheal tube.

FLUID BAGS

Closed Collection Systems - Partially or completely empty fluid bags should be kept sterile and saved for use as urine collection bags for closed systems. The drip set can be left attached to the bag and tied off so it won't be used further, or the bag can be emptied, a new drip set is attached and the entire unit is sterilized as a closed collection system.

Irrigation Fluids - Intravenous fluid bags are sterile inside the outer wrap. If the outer wrap is properly opened the bag can be placed on the surgical table and used by the surgeon as sterile lavage fluids.

Dressings - Fluid bags can be emptied and opened to be used as sterile waterproof dressings for open abdominal drainage. This is ideal if there is tension due to abdominal packing, bowel edema, bowel distention, or any other situation when closure may create abdominal compartment syndrome (excessive intraabdominal pressure). The bag can be sutured in place to provide a complete seal. If only a temporary, non-waterproof closure is indicated it can be secured to the wound edges using safety pins.

Fluid bags make strong waterproof coverings to protect foot bandages from getting wet.

Autotransfusion Sets – Empty fluid bags can be sterilized with a blood administration set and kept ready for use as autotransfusion sets. The blood is collected into syringes or sterile suction bottles and then placed into the fluid bag and delivered to the patient.

Enteral Feeding Bags – Empty fluid bags can be filled with liquid enteral feeding formulas and dripped through a regular fluid administration set. The bag should be washed out with very hot water every 24 hours to prevent residue build-up and avoid bacterial contamination.

FLUID ADMINISTRATION SET DRIP CHAMBER

Tracheostomy - The drip chamber is cut in half. The spiked end can be inserted in an emergency into the trachea as a transtracheal cannula. The open end will fit exactly onto an AMBU bag so that the patient can be ventilated. Since the spike is made of hard plastic that can damage the trachea, this device should not be used except in a dire emergency. As packaged these drip sets are sterile. A feeding tube can be placed into the trachea via the drip chamber and the chamber can then be removed. This provides transtracheal access for delivery of oxygen.

Tracheal Prosthesis – The drip chamber is cut into thin sections and sutured in place as a tracheal prosthesis for surgical repair of tracheal collapse.

FLUID ADMINISTRATION SET LINE

Used fluid lines can be recycled and used as ties for endotracheal and tracheostomy tubes. The line is cut into sections of a suitable length and kept in a bag beside the anesthetic machine. The line should be stretched prior to use which will help the knot stability.

SYRINGE CASE

Oropharyngeal Airway - A syringe case with the end cut off makes an effective oropharyngeal airway. This can be used in times of emergency when there is significant oral trauma but the larynx and trachea are functional.

Mouth Gag – The end of the syringe case is cut off and both ends are padded with gauze or tape. The case can then be inserted between the upper and lower canines as a mouth gag.

Tail Protector – Sutured tail wounds tend to rebleed when the dog knocks its tail. The plastic case is placed over the lightly padded bandage on the tip of the tail. When the tail wags the sutures are protected and repeat hemorrhage is minimized.

Mask – The end of a large syringe case is cut off and a hole is made in the tip of the case. This can be attached to oxygen or to a gas anesthetic circuit for birds, pocket pets and other small patients.

SYRINGES

Mouth Gag – The ends of the syringes are cut off and both ends are padded with gauze or tape. The syringe tubing can then be inserted between the upper and lower canines as a mouth gag.

Tubing Connector – The plunger of a 1 cc syringe is withdrawn and discarded. The base of the barrel is cut off. The tip will fit all narrow gauge tubes and the barrel end will fit almost all suction hosing and oxygen hosing.

Suction Drain – The plunger of the syringe is withdrawn approximately two-thirds of the length of the barrel and an 18 ga needle is placed across the base of the barrel to stop the plunger from depressing. The tip of the needle is cut off to prevent injury. The needle is withdrawn and the plunger is depressed fully. The syringe is then

attached to the suction drain and the plunger is withdrawn. The needle is reinserted creating steady negative pressure.

SLAM BAGS

Fluid Infusor - Fluids often need to be given rapidly to patients in hypovolemic shock. This requires that the fluids be delivered under pressure. A pressure infusor bag is an effective way of delivering pressurized fluids. The fluid bag is inserted into the pressure infusor bag and the pressure then can be inflated up to 300 mm Hg.

Pressure Cuff – The bag can be placed proximal to or over the site of a large bleeding wound (with or without padding). The bag can be inflated to a sufficient pressure to control active hemorrhage. In small animals this can be used as an abdominal counterpressure wrap (see below).

BLOOD PRESSURE CUFF

Hemorrhage Control - A Doppler blood pressure cuff can be placed proximal to a bleeding wound and inflated to 20-40 mm Hg above systolic pressure. This will control arterial hemorrhage to the region. This is especially useful for distal limb hemorrhage. Adult human thigh cuffs can be used as abdominal counterpressure wraps in small patients (see below).

FEEDING TUBES

Three and a half, 5 and 8 French feeding tubes can serve multiple purposes - especially if they are made of minimally reactive material such as medical grade silicone.

Nasal Tubes – Feeding tubes can be placed with the tip in the nasopharynx or in the trachea to deliver oxygen into the respective sites. They can be placed into the esophagus or stomach and used as nasoesophageal or nasogastric tubes for decompression and feeding.

Intravenous Catheters - They make effective long central lines in dogs in which the commercial lines are too short. A 14 ga or 16 ga over the needle 2 inch catheter is inserted into the vein. A 5 Fr or 3.5 Fr feeding tube, respectively, is inserted through the catheter to the desired length. A few drops of 50% dextrose placed on the outside of the feeding tube will help the tube slide easier through the catheter. The catheter is withdrawn from the vein and the feeding tube is sutured in place and a sterile dressing is placed.

Vascular Loops - When using as a vascular loop the tube is passed around the vessel or vascular pedicle and the loop is tightened by sliding hemostats down the tubing and tightening on the vessel. This is a modified Rumel tourniquet.

BUBBLE WRAP

Splints - Bubble wrap makes effective lightweight splints for distal limb fractures. Radiographs can be taken through bubble wrap.

Blanket - It also can be heated in a microwave in a bowl of water to create a warm “blanket”. This can be particularly useful in the operating room.

Oxygen Tent - When placed over the front of a cage it creates an effective "oxygen tent".

TOWELS

Abdominal Counterpressure - Towels can be wrapped around the pelvic limbs and abdomen of a patient and anchored with duct tape to serve as external counterpressure wraps. When doing this a towel first should be placed as padding between the pelvic limbs. A second towel is placed around the pelvic limbs wrapping from the toes to the hips in a barber pole fashion. The wrap is continued then around the abdomen to the level of the diaphragm if needed. It is anchored in place with duct tape. Care should be taken not to wrap the towels too tightly. Two fingers should easily be able to be placed under the abdominal counterpressure wrap once it is in place.

SURGICAL PAPER DRAPE

Surgical drape material makes an effective water repellant outer layer for bandages. It can be sutured in place or tied in place using umbilical tape to provide a water repellant outer layer for open abdominal drainage bandages. It can be safety-pinned or taped in place to cover larger wounds.

EMMA®

The EMMA (Masimo, Irvine, CA) is a portable battery run capnograph. Within as short a period of time as 15 seconds the device will provide a respiratory rate and continuous capnogram and an end-tidal carbon dioxide measurement ($ETCO_2$). It is waterproof and has been designed to withstand being dropped. Capnography provides a continuous noninvasive assessment of ventilation and the $ETCO_2$ provides an estimation of the $PaCO_2$. The $ETCO_2$ provides information about pulmonary blood flow in the face of severe hypotension. During anesthesia if the $ETCO_2$ drops below 18 mm Hg the arrest is imminent. During CPR if the $ETCO_2$ rises to 15 mm Hg or higher return of spontaneous circulation is very likely.

RADICAL 7®

The Radical 7 (Masimo, Irvine, CA) is a portable pulse oximeter that continuously calculates a plethysomographic variability index or pleth variability index (PVI). The PVI is an assessment of changes in the amplitude of the pulse oximetry waveforms during different phases of respiration. Patients do need to be mechanically ventilated to maintain consistency in changes in intrathoracic pressure. In the face of hypotension the PVI will indicate whether a patient is likely to be fluid responsive or not.

KITTY KOLLAR®

The Kitty Kollar® (Orange, CA) is a collar designed to replace a standard bandage for an esophagostomy tube. It is made of a soft padded washable fabric. The tube exits the collar through a buttonhole and a Velcro hook and loop fastener secures the tube to the collar. The collar is secured around the patient's neck with a Velcro tab.

Acute Pancreatitis

INTRODUCTION: *Acute pancreatitis is one of the most difficult diseases a clinician can manage. The systemic inflammatory response syndrome can be severe in these animals. Major organ failure – refractory hypotension, liver failure, gastrointestinal failure, ARDS (acute respiratory distress syndrome), and DIC (disseminated intravascular coagulation) may develop. Only through aggressive medical management and sometimes surgical management can the clinician hope to minimize morbidity and mortality. Commonly used diagnostic tests do not necessarily correlate with severity of disease or prognosis, which means that the clinician should treat all pancreatitis patients as having potentially life-threatening disease. The ultimate diagnosis of pancreatitis is a histopathologic one which is rarely achieved. Aggressive fluid therapy, analgesia and nutritional support form the cornerstone of therapy. If patients have necrotic, abscessed or neoplastic pancreatic tissue present, the inflammatory process may not subside until the affected tissue is debrided. Surgery is rarely indicated but may be important in the management of some patients.*

PATHOPHYSIOLOGY

Multiple causes of pancreatitis have been identified but in most dogs and cats it is considered to be idiopathic. Regardless of the cause the pathophysiology is similar and ultimately is a result of activation of the pancreatic enzymes within the pancreas leading to autodigestion as well as digestion of the peripancreatic tissues and subsequent activation of the inflammatory process. If the inflammatory cascades persist unabated the systemic inflammatory response syndrome (SIRS) can result.

The systemic uptake of all of the products that are liberated during the inflammatory process can then lead to systemic inflammation and multisystem involvement. The protective plasma protease inhibitors such as □□2-macroglobulin and □□1-protease inhibitor are consumed as the necrotizing process continues. Alpha macroglobulins change the configuration of the proteases when they bind to them which allows macrophages to clear the enzymes. As the plasma protease inhibitors are depleted death can occur from acute disseminated intravascular coagulation and shock as the circulating proteolysis and cytokines activate the complement, coagulation, and fibrinolytic cascades.

Grossly pancreatitis progresses from that of edema and mild saponification and a few one millimeter sized abscesses to that of severe edema, numerous areas of saponification and many small abscesses. Then it progresses to hemorrhagic pancreatitis, localized peritonitis and edema of the surrounding tissues and advances to necrosis, larger abscesses, and the formation of very firm sections of cellulitis and pancreatitis (a phlegmon). In some cases bacteria are thought to translocate from the duodenal lumen and generalized peritonitis, bacterial abscessation, secondary biliary blockage and necrosis of the ventral aspect of the duodenum may occur. In the most severe cases the entire pancreas becomes involved. In some cases necrosis of fat that normally accumulates in the retroperitoneal space and falciform

ligament may be present. Gastric and duodenal ileus are common.

DIAGNOSIS

Animals with acute pancreatitis are usually presented because of depression, anorexia, vomiting, and in some cases, diarrhea. In severe cases shock and collapse may be present. In other cases the signs are very vague to almost nonexistent. Cats with mild pancreatitis are often presented with a vague history of being inappetent. Some animals with severe pancreatitis will exhibit signs of cranial abdominal pain and even a "praying" position. Pain may or may not be evident. Patients in shock may not show any signs of pain until perfusion is restored with fluid therapy. Occasionally the only clinical signs the patient exhibits are from systemic complications. Physical examination should include careful auscultation, palpation and visual inspection of the animal. Lack of gastrointestinal sounds is consistent with ileus, which may be localized or generalized. The right and left cranial abdominal quadrants should be individually evaluated using palpation underneath the rib cage. Large dogs may need to have their front feet placed on a stool or chair to shift abdominal contents caudally. The umbilicus should be closely inspected since masses involving the umbilicus have been associated with pancreatic neoplasia. A rectal examination should be performed to evaluate for evidence of diarrhea as well as the presence of blood. Vomitus should also be evaluated for blood.

Although a leukocytosis with a left shift is commonly observed in more serious cases there may be no changes in the white cell number or types in milder cases. Red blood cell morphology should be closely examined, especially in cats, for signs of oxidant-induced damage (suggesting depleted glutathione levels). Assays of pancreatic enzymes (amylase, lipase) do not provide any useful information in dogs and cats. Species specific pancreatic lipase immunoreactivity (fPLI and cPLI) are sensitive (85-90%) for pancreatitis but some feel they are not very specific. Both SNAP and Spec tests have been validated. Spec tests are quantitative and repeat tests may allow for trending of the disease process. Liver enzymes and bilirubin may be elevated. If the inflammatory process has progressed then albumin levels may be decreased due to third-spacing. Blood gas abnormalities will reflect the degree of perfusion abnormalities as well as any possible secondary pulmonary involvement (aspiration pneumonia, ARDS). Electrolyte abnormalities typically reflect a combination of dehydration and losses through vomiting and diarrhea. Hypocalcemia may result from calcium soap formation, intracellular shifts due to alterations in membrane function, or altered levels of thyrocalcitonin and parathyroid hormone. Ideally ionized hypocalcemia should be assessed rather than total calcium. Coagulation profiles (PT, PTT, platelet counts or estimates) are indicated in sick pancreatitis patients in order.

Radiographs often reveal increased density, diminished contrast, and granularity in the right cranial quadrant of the abdomen, displacement of the stomach, widening of the "angle" between the antrum and the descending duodenum, and displacement of the descending duodenum to the right with gas patterns in the duodenum. The subjective loss of visceral detail in the cranial abdomen is probably the most common radiographic sign observed. In cats the loss of detail associated with pancreatitis is more commonly seen on the lateral view immediately caudal to the stomach and extreme lateral displacement of the duodenum does not occur.

Ultrasonic interrogation of the cranial abdomen will be helpful but is operator

dependent. The appearance of mixed echogenicity or a mass effect within the pancreas as well as cystic areas, abscesses (complex cystic regions), edema, and free intraabdominal fluid are occasionally observed. Changes in the duodenum consistent with pancreatitis include a fluid and gas-filled descending duodenum, a thick-walled duodenum and atony. Caution should be exercised in ruling out pancreatitis on the basis of a normal ultrasound exam.

MEDICAL MANAGEMENT

Supplemental oxygen should be provided to all patients showing signs of shock, typically using nasopharyngeal catheters. Aggressive fluid support is indicated. This requires a continuous rate intravenous infusion of a crystalloid and often colloids. Use a replacement formula to rehydrate the animal and replace fluids and electrolytes lost secondary to vomiting, diarrhea, and third spacing and plan to rehydrate over 6 to 8 hours. Colloids should be used immediately in more critical patients (hypotensive, evidence of hemorrhagic vomiting or diarrhea, systemically ill patient, hypoproteinemic, evidence of developing coagulopathy) to improve microcirculatory blood flow and help in the prevention of endothelial, interstitial and intracellular edema.

Albumin levels should be maintained above 2 mg/dL using plasma. Not only is plasma an important contributor to oncotic pressure but albumin is important also as a free radical scavenger. Plasma provides a source of ⍰⍰macroglobulin, which binds the activated and liberated proteases. In the author's opinion fresh frozen plasma should be used during resuscitation if there is any concern that a coagulopathy is present or is developing.

To ensure adequate fluids are being administered adequate urine output (at least at 1/2 ml/kg/hr in cats, 1 ml/kg/hour in dogs), central venous pressure (3-7 cm H_20), and normal heart rate and arterial blood pressure should be maintained.

Pain kills. Analgesics should be provided immediately to patients in pain in adequate doses and at frequent enough time intervals to control the pain. Methadone and hydromorphone are effective intermediate acting pure mu agonists. Butorphanol may be indicated in very critical patients (0.05-0.2 mg/kg) and may be effective in cats, but it should be kept in mind that butorphanol may only last 20 to 60 minutes and is not very effective if pain is moderate to severe. A constant rate infusion of butorphanol may be helpful in more painful cats. Patients with severe pancreatitis may require continuous rate infusions of fentanyl. For those with intractable pain peritoneal lavage with lidocaine and bupivacaine is often very effective. Nonsteroidal antiinflammatory drugs (NSAIDs) should be avoided.

Antiemetics are usually indicated, maropitant being the most effective drug in most patients. Serotonin antagonists such as ondansetron hydrochloride or dolasetron can also be used. Metoclopramide may help improve gastrointestinal motility and clinically seems to be more effective given as a constant rate infusion (2 mg/kg/d) than when given as intermittent injections. Nasogastric (NG) tubes should be placed for gastric decompression in patients that have significant gastric distention with fluid or frequent large volume vomiting.

Nutritional support ideally should begin within 12 hours of admission. Partial parenteral formulas can be given by peripheral catheter. ProcalAmine (B. Braun Medical), which is a hyperosmolar solution containing 3% amino acids, 3% glycerol and maintenance concentrations of electrolytes, is an excellent partial parenteral nutritional support product. It is

given at a rate of 0.5 mL/kg/hr as a constant rate infusion. Maintenance fluids to which 3% amino acids and 3-5% dextrose are added can be used instead of commercially prepared solutions.

Enteral feeding is always preferred over parenteral. Jejunal feeding is the ideal route since feeding in this location does not stimulate pancreatic enzyme secretion and is generally well tolerated. Patients that have surgery have an advantage since a jejunostomy or gastrojejunostomy tube can be placed. Evidence also suggests that gastric feeding may be possible in some patients. It is recommended that an NG tube be placed and used for gastric decompression as well as microenteral feeding. This trickle feeding (0.1 – 0.25 mL/kg/hr) of an electrolyte solution containing an isotonic mixture of electrolytes and 3 to 5% glucose is well tolerated. This will help prevent gastric stress ulceration, help prevent the down regulation of the gastrointestinal tract that occurs when the patient is not eating, and help improve the transition to full enteral feeding. This microenteral nutrition is only continued if hourly aspirations of the NG tube reveal no accumulation of this fluid in the stomach and/or no vomiting of the material is detected.

Close monitoring is essential in patients with severe pancreatitis. Monitoring should include regular (q 1 to 4 hr) measurement and documentation of level of consciousness, temperature, heart rate and rhythm, pulse rate and strength, respiratory rate and effort, blood pressure, central venous pressure (if a jugular catheter is in place), pain/analgesia, gastrointestinal sounds, amount and characteristics of vomitus and diarrhea, and volumes of fluid suctioned via the NG tube. Blood tests are indicated at least every 24 hours including packed cell volume, total solids, albumin, glucose, creatinine, electrolytes, blood gas, and blood smear evaluation. Additional tests (complete blood counts, other blood chemistries, radiographs, fluid analysis, etc.) may be indicated based on the status of the patient. All parameters should be kept in as normal a range as possible. More critical patients or those with clinically relevant abnormalities will require more frequent monitoring.

INDICATIONS FOR SURGERY

A decision to perform surgery is made based on history, physical examination findings, laboratory parameters, and diagnostic imaging; however, many of these findings are nonspecific, especially in cats. One study showed that there was no definitive means of determining acute necrotizing pancreatitis from chronic nonsuppurative pancreatitis. The presence of septic peritonitis based on paracentesis or diagnostic peritoneal lavage, or a mass lesion found on ultrasound consistent with an abscess is an absolute indication for surgery. Other indications are more subjective.

Surgical exploration should be considered in patients with a waxing and waning history of recurrent pancreatitis in order to procure an exact diagnosis as well as determine if resolution of the disease is possible. Patients who have been diagnosed with pancreatitis that is not responding to medical management should be explored – again to diagnose the underlying cause, debride or resect necrotic, infected or neoplastic tissue, and place an enteral feeding tube.

Toxicologic Emergencies

INTRODUCTION: *Toxicological emergencies are a common part of veterinary practice. Both dogs and cats have an amazing ability to ingest all sorts of foreign substances. Some of these substances can cause life-threatening problems while some just cause minor problems. In many situations the amount of the toxin ingested will dictate how serious the problem is. Often veterinarians work on assumptions since it is not uncommon that the actual identity of the toxin is never known. Thorough history taking and physical examinations are key in order to avoid missing a diagnosis of a toxin that requires a specific antidote. Aggressive supportive care is indicated for all those patients who ingested an unknown toxin to avoid morbidity and mortality.*

HISTORY AND CLINICAL SIGNS:

History from an owner is essential in the accurate diagnosis and treatment of most toxicities since clinical signs can be extremely variable. If the toxin is suspected or identified it is essential to get accurate and detailed information on the chemical or chemicals involved in order that a poison control center can be contacted for information on expected effects, treatment and prognosis. The type of toxin, the amount ingested, the time since ingestion, the clinical signs the patient is showing, and the previous medical history of the patient are all key. In the case of unknown exposure the owner should be questioned closely as to the type of chemicals, and especially medications that are available in the house that the pet might have access to. Although owners will not uncommonly try to indicate the 'neighbour has poisoned their pet' this is uncommon in the author's experience. It is much more likely that the animal ingested a natural or man-made toxin in the house or on the owner's property.

DIAGNOSIS:

The identification of a specific toxin often requires a high index of suspicion. The clinician should work closely with poison control centers - both local human centers and any veterinary centres that are available. The National Animal Poison Control Center at the University of Illinois has a vast bank of information and is staffed 24 hours a day by veterinarians. Blood, urine and gavage samples may be required for assay to identify suspected toxins and samples of whole blood, serum, urine, and gastric contents or vomitus should be taken on admission whenever possible. If the owner has had the animal vomit at home instructions should be given to have them save the contents in a plastic bag and bring it in with the animal.

TREATMENT OVERVIEW

Treatment will in many cases be symptomatic unless a specific antidote is known. Fluid diuresis may be indicated. Seizure activity, ventilation and oxygenation, blood pressure and perfusion, cardiac rhythms and rates, renal function and coagulation are just some of the parameters that should be assessed and maintained as normal as possible.

INDUCING VOMITING

Vomiting should be induced as soon as possible in the patient ingesting a suspected or an unknown toxin, unless vomiting is known to be specifically contraindicated (strong acids or alkalis, petroleum distillates, etc.). Apomorphine should be used intravenously for induction of vomiting. Hydrogen peroxide and salt can be given by the owner at home and are generally very effective in inducing vomiting. The dose of hydrogen peroxide is 1 to 2 teaspoons of 3% hydrogen peroxide per 10 kg body weight. This can be repeated 3 times at 5 minute intervals. Salt should be avoided whenever possible but can be given at a dose of 1/8 teaspoon per 10 kg. The sooner the toxin is out of the system the less likely toxic effects will be seen... even making the animal vomit in the car on the way to the clinic is a good idea.

Dexmedetomidine or xylazine can be used to induce vomiting in cats; however, in the author's experience neither work very well Both drugs can have serious cardiovascular side effects and the patient should be carefully assessed prior to administration of the drug and monitored for undesirable side effects.

GASTRIC LAVAGE AND ACTIVATED CHARCOAL

Gastric lavage is widely used in small animals poisoned by ingestion of toxins. Experts are beginning to question the value of gastric lavage and it is currently not recommended in human medicine in most situations since studies have failed to confirm its value. Even when gastric lavage can be performed within minutes of ingestion, recovery of the toxin is limited. If the procedure is not completed within an hour of ingestion, recovery of many toxins is less than 15%. In small animal veterinary medicine, it is rare that gastric lavage would be completed within this period. In addition, administration of activated charcoal without lavage has shown very similar outcomes in people with many different types of toxin ingestion.

Activated charcoal should be administered via a gavage or nasogastric tube if it is indicated. Ideally a cathartic should be administered with the charcoal to hasten removal of the toxin. Many activated charcoal compounds are manufactured with cathartic (sorbitol magnesium sulfate) already present. The charcoal may need to be repeated over an extended period (sometimes 3 days) since some toxins undergo enterohepatic cycling. The decision to do this should be on a case-by-case basis. Activated charcoal often seems to stimulate vomiting which should be kept in mind when a decision is being made to administer the compound.

SKIN CONTAMINATION

Skin contaminants should be rinsed thoroughly. Because these compounds also may be toxic to humans gloves should be worn. Sedation may be required with cats and aggressive animals. Make sure if sedatives are used that there is no interaction between the sedative and the toxin that might preclude its use. In many cases large volumes of warm water will suffice. In some situations washing with a mild dish soap or pet shampoo may be indicated. Make certain all soaps are rinsed from the fur and the animal should be actively dried to prevent hypothermia and avoid having the animal lick any residual chemicals from the skin during grooming.

AIRWAY AND BREATHING

On presentation the patient should be checked for the presence of a patent airway and adequate ventilation. If the patient has an obstructed airway an emergency tracheotomy may be required. Patients who do not have a gag reflex should be intubated. Patients who are not ventilating adequately should have positive pressure ventilation instituted immediately. Patients with evidence of anemia, cyanosis, increased respiratory effort, or shock should have supplemental oxygen provided immediately.

If the patient has signs consistent with pulmonary edema then furosemide should be administered intravenously in addition to supplemental oxygen. If the patient will not tolerate an intravenous injection the drug should be given intramuscularly into the epaxial muscles. If the patient is extremely stressed mild sedation with an opioid or acepromazine (if the patient is hemodynamically stable) may be indicated.

If the patient has evidence of bronchospasm then supplemental oxygen should be provided and bronchodilators should be administered. Aminophylline and □□2 agonists can be given parenterally; however, in the author's experience nebulized □□2 agonists tend to be superior to parenterally administered agents. Aminophylline can cause anxiety and tachycardia whereas side effects of □□2 agonists are rare.

CIRCULATION

Patients that are hypotensive may require crystalloids and colloids for resuscitation. Animals that are significantly anemic should receive red cells. Patients with coagulopathies should received fresh whole blood (if also anemic) or fresh frozen plasma. Patients that are hypoalbuminemic may require a combination of synthetic colloid and albumin replacement depending on the serum albumin concentration. Blood pressure and perfusion status should be returned to normal. Some toxins may cause hypotension by depressing cardiac function or by causing excessive vasodilation. In this case positive inotropic drugs, □□blockers, antiarrhythmics, or vasopressors may be indicated depending on the toxin. Patients that are dehydrated should have their fluid deficit calculated and administered over an 8-12 hour period.

Certain toxins can cause hypertension. Systolic blood pressure greater than 200 mm Hg can lead to significant patient morbidity. The underlying cause should be identified if possible in order to treat with the appropriate drug. Nitroprusside at 0.5-10 mcg/kg/min constant rate infusion will lower blood pressure in many patients and can be titrated to effect. Acepromazine will cause hypotension through vasodilation but can be difficult to titrate. If hypertension is associated with tachycardia then a □□blocker (propranolol at 0.02-0.06 mg/kg IV over 5 minutes) should be given. Hydralazine, angiotensin-converting enzyme inhibitors and calcium channel blockers may also be helpful in controlling hypertension depending on the underlying cause. Unfortunately many of these medications are in an oral form only which may limit their usefulness in the acute stages.

Severe bradycardia (heart rates less than 50-60 beats per minute) with concurrent heart blocks, or bradycardia associated with hypotension should be treated with atropine or glycopyrrolate. Bradycardia

associated with normal to high blood pressure should not be treated with anticholinergic drugs.

A urinary catheter should be placed and urine output monitored if the animal was exposed to a nephrotoxin. Alkalinizing the urine by systemic administration of sodium bicarbonate may aid in excretion of certain toxins. The urine pH will need to be monitored in these patients to ensure the goal is being achieved.

SEIZURE MANAGEMENT

Seizures should be controlled using intravenous or intranasal diazepam. If this is unsuccessful intravenous phenobarbital should be given. Both diazepam constant rate infusions and phenobarbital constant rate infusions can be given to help maintain control of seizures. The two drugs are synergistic when given together. Phenobarbital loading may be required to achieve therapeutic phenobarbital levels. If the animal has never received phenobarbital before this generally can be achieved by giving 16 mg/kg divided into 4 doses given every 20 minutes. (A dose of 3 mg/kg will raise the blood level by approximately 5 mcg/ml.) If the patient becomes excessively sedate or loses a gag reflex the clinician may prefer not to give further doses of phenobarbital until the patient is more alert. Muscle activity during recovery from pentobarbital can be easily confused with seizure activity. Levetiracetam is often used instead of phenobarbital due to the high cost of the latter drug.

MANAGEMENT OF STUPOR AND COMA

Patients who do not have a gag reflex should be intubated and positive pressure ventilation should be instituted if the animal is not ventilating adequately. The patient should be placed in a 30 degree body tilt to help minimize the risk for aspiration. Pressure on the jugular veins should be avoided. Patients should be rotated every 2-4 hours to prevent atelectasis and reduce the risk for pneumonia. Pressure points should be padded to minimize the risk of pressure sores developing. The eyes should be kept lubricated with ocular ointments and the tongue may need to be kept moistened. Chlorhexidine rinses may help minimize the colonization of the mouth with potentially pathogenic bacteria.

Mannitol may be useful in helping treat cerebral edema.

A nasogastric tube may be indicated for helping with gastric decompression if regurgitation or vomiting and aspiration. The tube also can be used to provide enteral nutrition. Sneezing can raise intracranial pressure. This is not an issue for comatose patients but if sneezing is not desirable in more aware patients then placement of a nasal tube may not be appropriate.

MANAGEMENT OF TREMORS

Tremors are best controlled by use of intravenous methocarbamol, diazepam or midazolam. Constant rate infusions may be required to control the tremors. Dosing should be adjusted to ensure the patient does not become anesthetized. If general anesthesia is necessary to control the motor movement the patient should be intubated to help protect the airway.

MANAGEMENT OF TEMPERATURE ABNORMALITIES

Hyperthermia may result from excessive seizure activity, muscle rigidity, malignant hyperthermia, or a hypothalamic disorder. The patient should be actively cooled if the temperature is above 104F. While the patient is being cooled appropriate measures to secure the airway, provide oxygen, fluids and control seizures or muscle activity should be taken. Cooling can be done by running the fluids through an ice bath, and placing icepacks around the head and over superficial major vessels such as the femoral and brachial arteries. Spraying the patient with water and then placing a fan on the patient will cause evaporative heat loss. Application of topical alcohol should be avoided since it can be absorbed systemically leading potentially to alcohol intoxication. Cooling should be stopped once the patient's temperature reaches 103F. If the patient's temperature is in an extreme danger zone (greater than 105F) active core cooling may be indicated. This can be done by administering cold water enemas and cold water gastric lavage. These patients frequently develop the systemic inflammatory response syndrome with all of its accompanying complications (hypotension, vasculitis with secondary albumin loss and third-spacing of fluids, coagulopathy, and multiple organ failure).

Hypothermia can be caused by certain toxins that depress the patient's level of consciousness or reset the hypothalamus. Certain medications used to treat toxicities that depress the metabolic rate (opioids, anesthetic agents, etc.) can also lead to hypothermia. Any patient that has a depressed level of consciousness should be kept warm with warm intravenous fluids, blankets, warm water circulating blankets, etc. Patients that require long term ventilation can be cooled significantly from the cold oxygen in the circuit and ideally an air warmer should be placed in the circuit. Spontaneous ventricular fibrillation can occur if the temperature drops to 28C.

ANTICOAGULANT RODENTICIDE

Mechanism of Toxicity: Interferes with production of vitamin K dependent clotting factors (II, VII, IX, X) leading to active hemorrhage.

History and Clinical Signs: Signs relate to hemorrhage which can be external or internally into any body cavity, tissue space, or organ. Clinical signs generally take a minimum of 48 hours to develop and more serious signs usually indicate exposure 4-5 days prior to presentation. Hemorrhage around the larynx can cause an acute upper airway obstruction. Life-threatening hemorrhage can occur into the lungs and mediastinal tissues.

Specific Diagnostic Tests: Prothrombin time, activate partial thromboplastin time, activated clotting time, PIVKA (proteins induced by vitamin K absence or antagonism) test. Prothrombin time will prolong first and return to normal first.

Treatment: Animals who have ingested the toxin should have vomiting induced.

Animals with clinical signs should have supportive care provided (see above). Vitamin K1 should be given subcutaneously at a loading dose of 5 mg/kg followed by 5 mg/kg divided every 12 hours for 2-3 weeks for first generation coumarins and 4-6 weeks for second and third generation coumarins. Once the patient is able to take

oral medications the vitamin K1 can be given orally.

If the owner is uncertain whether or not the pet actually ingested the toxin or ingested sufficient to induce hemorrhage the prothrombin time can be monitored on a daily basis for 3 days. If at 72 hours there is no evidence of a prolonged prothrombin time treatment is not necessary.

PYRETHRIN

Source: Insecticides especially flea products

Mechanism of Toxicity: Neurotoxin (prolongs sodium conductance and antagonizes GABA)

History and Clinical Signs: Pets have usually been exposed to topical or premise spray products. Clinical signs include depression, muscle fasciculations, salivation, vomiting, bronchospasm and ataxia.

Treatment: Skin decontamination should be performed if this was the route of exposure. Vomiting can be induced if the patient ingested the toxin within the previous 1-2 hours and the animal is neurologically stable and able to protect its airway against possible aspiration and the product did not contain petroleum distillates. Atropine can be used to control salivation as long as the patient is not tachycardic. Most patients recover within 24-48 hours with supportive care.

METALDEHYDE

Source: Slug or snail bait

Mechanism of Toxicity: Unknown

History and Clinical Signs: Signs usually appear within 15 minutes to 3 hours of ingestion. Early signs include anxiety, salivation, panting, ataxia and possibly mydriasis and nystagmus. Later signs include muscle fasciculations, hyperthermia, and possible seizures.

Diagnostic Tests: Stomach contents, urine, plasma or tissue can be analyzed for metaldehyde.

Treatment: Emergency treatment to secure an airway, establish intravenous access and control seizures may be required. Gastric lavage should be performed followed by administration of a single dose of activated charcoal. Patients should be placed on a constant rate infusion of methocarbamol or diazepam to control the muscle tremors.

GARBAGE

Mechanism of Toxicity: Bacteria can release endotoxins and exotoxins. Molds can cause gastrointestinal irritation, hepatotoxicity or neurotoxicity.

History and Clinical Signs: Signs usually include vomiting and/or diarrhea. Endotoxemia can lead to the systemic inflammatory response syndrome (SIRS) and multiple organ failure. Certain toxins such as botulism can cause muscle tremors, ascending flaccid paralysis and coma.

Diagnostic Tests: Because garbage intoxication can mimic many other disease processes a full diagnostic workup is indicated.

Treatment: There is no antidote. Appropriate supportive and symptomatic care should be provided. This may need to be very aggressive care if there is evidence of endotoxemia. Supportive care may be indicated for several weeks if flaccid paralysis develops. Broad spectrum antibiotics such as penicillin, ampicillin and/or metronidazole are indicated in all cases of suspected garbage intoxication.

CHOCOLATE

Mechanism of Toxicity: Theobromine is a phosphodiesterase inhibitor that causes an increase in cyclic AMP and a subsequent increase in catecholamines. Unsweetened baking chocolate and cocoa contain very high levels of theobromine. Dark chocolate also contains very high levels. Milk chocolate contains approximately one-tenth the amount found in unsweetened chocolate.

History and Clinical Signs: Vomiting and diarrhea may be present that are not direct causes of the theobromine but are related to the dietary indiscretion. Pancreatitis may be seen depending on the type of chocolate that was eaten. Clinical signs include cardiac abnormalities (tachycardia, arrhythmias), central nervous system excitement (hyperactivity, tremors, seizures), panting, and urinary incontinence.

Treatment: Appropriate symptomatic and supportive care should be provided. Activated charcoal should be administered. Electrocardiographic monitoring is indicated in severe intoxications and arrhythmias should be treated appropriately.

ETHYLENE GLYCOL

Source: Antifreeze, windshield de-icing fluid, solvent in many chemical solutions

Mechanism of Toxicity: Ethylene glycol is oxidized to glycoaldehyde by alcohol dehydrogenase. Glycoaldehyde is oxidized to glycolic acid and then to glycoxylic acid. Glycoxylic acid is metabolized primarily to oxalic acid, which combines with calcium to form calcium oxalate crystals. Other end products include glycine, hippuric acid, formic acid, oxalomalic acid and benzoic acid. Ethylene glycol is an alcohol that can cause central nervous system depression and gastrointestinal irritation. It also inhibits the cytochrome P450 system which leads to increased production of oxygen radicals. The accumulation of acids can lead to a severe metabolic acidosis. The acid metabolites also interfere with oxidative phosphorylation glucose metabolism and protein synthesis and are toxic to renal epithelium. Calcium oxalate crystal deposition occurs in all organs including the brain. The minimum lethal dose is 1.5 mL/kg in cats and 6.6 mL/kg in dogs. Many solutions containing ethylene glycol also contain other toxins.

History and Clinical Signs: An environmental toxin, exposure typically occurs secondary to the animal drinking fluid that has leaked from vehicles and drinking from toilets that have been treated to prevent freezing. Early signs, which can be seen within 30 minutes of exposure and may last 12 hours may include nausea, vomiting, central nervous system depression and signs of “being drunk”. Polyuria and polydipsia may be seen secondary to the osmotic diuresis. Signs consistent with renal failure typically develop within 12-24 hours in cats and within 36-72 hours in dogs.

Diagnostic Tests: Serum ethylene glycol levels can be measured or estimated using a colourimetric test. The colourimetric test is not sensitive enough for cats although if the test is positive the cat definitely ingested a toxic dose.

Treatment: Vomiting should be induced within 30 minutes; after that time it is not likely to be effective due to the raid absorption rate. Activate charcoal is not effective. Treatment includes treatment and monitoring as for any renal failure patient. A central line and a urinary catheter are advised in order to be able to monitor

central venous pressure and urine output respectively. Primary treatment involves administration of an antidote, either ethanol, which acts as a competitive substrate for alcohol dehydrogenase, or 4-methylpyrazole, which is an alcohol dehydrogenase inhibitor. Ethanol has many side effects; therefore; 4-methylpyrazole is preferred. The prognosis is excellent if dogs are treated with 4-methylpyrazole within 5 hours and cats within 3 hours. Dialysis is always advised but is probably unnecessary if 4-methylpyrazole is being administered early. Dialysis is continued until the ethylene glycol test is negative which usually requires 24-32 hours of continuous dialysis.

ETHANOL

Administer 0.6 g/kg 7% ethanol intravenously or 0.6 g/kg 20% ethanol orally as a loading dose. Then begin 100 mg/kg/hr constant rate infusion of 7% ethanol. If intravenous therapy is not an option ethanol can be administered via a nasogastric tube; however, vomiting can be a problem when given by this route. Supplement fluids with multiple B vitamins. Treatment should be continued until the ethylene glycol test is negative (minimum 36 hours).

ACETAMINOPHEN

Source: Prescription and over-the-counter drugs

Mechanism of Toxicity: Acetaminophen is metabolized to non toxic and toxic metabolites. Glucuronidation and sulfation as well as combination of toxic metabolites with glutathione are key to minimizing the toxic effects of acetaminophen. The toxic metabolites cause direct cellular death and methemoglobinemia.

History and Clinical Signs: Dogs will present with signs consistent with liver failure. Cats will present with signs consistent with methemoglobinemia (cyanosis, respiratory distress, brown mucous membranes, brown blood) as well as facial edema. Cats are extremely susceptible to the drug since they cannot efficiently metabolize it.

Treatment: Appropriate supportive care should be provided. Gastric decontamination and activated charcoal administration are warranted. N-acetylcysteine is given at 240 mg/kg loading dose followed by 140 mg/kg every 4 hours for 3 days in dogs or 70 mg/kg every 6 hours for 3 days in cats. This can be given orally or intravenously. Vitamin C at 30 mg/kg orally or subcutaneously or 20 mg/kg intravenously may help convert the methemoglobin to oxyhemoglobin. Because cimetidine interferes with the metabolism of the acetaminophen its administration may be warranted.

STRYCHNINE

Source: Pesticide

Mechanism of Toxicity: Strychnine antagonizes glycine which is an inhibitory neurotransmitter. Most signs relate to inhibition of glycine released by Renshaw cells which are neurons that mediate the activity of antagonistic muscle groups. Inhibition of these neurons leads to uncontrolled muscle contraction. Persistent muscle activity can lead to muscle injury, hyperthermia and rhabdomyolysis.

History and Clinical Signs: Early signs included anxiety and restlessness. Tonic muscle contractions of the extensor muscle groups become evident. A risus sardonicus

is evident from facial muscle contraction. Muscle contractions are worsened by external stimuli. Tetanic contractions of the respiratory muscles can lead to apnea.

Diagnostic Tests: Vomitus, stomach contents, serum, or urine can be analyzed.

Treatment: Appropriate symptomatic and supportive care should be provided. Activated charcoal is indicated. Because of the mechanism of action of the toxin gastric lavage with a protected airway is preferred if clinical signs are evident. Muscle relaxation can be achieved using methocarbamol. Diazepam may be effective. More severe muscle contractions may need to be controlled with pentobarbital. Positive pressure ventilation may be required in serious cases. The patient should be kept sedated in a darkened, quiet room to avoid exacerbation of muscle activity.

Índice

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Clinical Applications Of The Amazing Omentum

INTRODUCTION: *The omentum is extremely versatile in form and function. Omentalization of damaged tissues can be readily performed in the abdomen; in addition, the omentum can be lengthened and passed into the thorax or tunneled subcutaneously to reach just about anywhere on the body! Potent fluid-absorbing, infection-fighting, and wound healing properties make the omentum an asset in a wide variety of conditions.*

The omentum consists of a mesothelial membrane (2 cell layers thick) covering a connective tissue framework that contains scattered fibroblasts, fibrocytes, pericytes, and fat cells. Its rich vascular supply originates from the gastroepiploic and splenic arteries and drains into the portal system. The omentum also has an extensive lymphatic system that drains into cranial abdominal lymph nodes. The mesothelial cells have a glycoprotein-polysaccharide coating that allows the omentum to easily slide over other abdominal organs. The superficial or ventral leaf of the greater omentum attaches to the greater curvature of the stomach and spleen. This leaf extends caudally and then folds dorsally on itself to become the deep or dorsal leaf, which attaches to the left lobe of the pancreas and dorsal body wall. The omental bursa is the potential space between the two leaves of the greater omentum. During an abdominal explore, a hole can be manually torn in an avascular area of the superficial leaf to allow assessment of the left lobe of the pancreas and cranial abdominal lymph nodes and vasculature. The lesser omentum, a single sheet extending between the lesser curvature of the stomach and the liver, is much smaller and more anchored than the greater omentum.

Inflammation activates omental components in a number of ways. Lymphoreticular bodies are glomerular-like capillary structures in the omentum interconnected by lymphatic vessels. When exposed to inflammatory mediators, fenestrations open in the vasculature of the lymphoreticular bodies, allowing entry of fluid and particulate matter from the peritoneal cavity and stimulating the resident white blood cells into action. The extensive network of omental lymphatics provides a very large absorptive surface and effective peritoneal lymphatic drainage. Macrophages in the lymphoreticular bodies migrate to the surface of the omentum and project microvilli into the peritoneal cavity, phagocytosing particulate matter and transferring antigens to omental lymphocytes for antibody production.

Omentum adheres to inflamed or ischemic tissues via activated fibrinogen. This adhesion seals off the diseased area from surrounding tissue, and ensures direct access of omental healing factors to the inflamed region. The omentum aids in hemostasis by speeding the activation of prothrombin and by applying pressure via its adhesion. Angiogenic factors released by the omentum stimulate new vessels to cross from the omentum into the inflamed

tissue within 6 hours of omental attachment, and omental neurotropic factors appear to stimulate reinnervation and may modulate pain.

Omentalization is the process by which the omentum is placed in a specific site by the surgeon. The omentum should always be kept moist and handled gently, with care taken to preserve its blood supply. The moldable nature of the omentum allows it to be placed in or around a variety of organs or defects, although care should be taken to avoid 360 degree wrapping of a luminal organ out of concern for stricture. While the omentum readily adheres to sites of inflammation, it may also be sutured into place with 3-0 or 4-0 absorbable suture. When passing the omentum out of the abdomen, the hole made in the diaphragm or abdominal wall should be large enough to prevent compromise to omental vessels and yet small enough to prevent herniation of abdominal contents. For protection during subcutaneous tunneling to distant sites, the omentum can be temporarily placed in the lumen of a large, moistened Penrose drain.

When needed, the omentum can be unfolded and extended to twice its normal length. One lengthening technique involves dissecting the deep leaf of the omentum away from the left lobe of the pancreas to create an omental pedicle flap based on the left gastroepiploic artery. If still more length is needed, an inverted L-shaped incision is made in the extended omentum, with the base of the L parallel to the greater curvature of the left side of the stomach, and the arm of the L dividing the omentum in half for two-thirds of its extended length. The left side of the omentum is then rotated caudally. When fully extended in this manner, the canine omentum typically reaches beyond the distal extremities and the muzzle.

A common use of omentum in veterinary medicine is placement of the omentum over sutured surgical sites in hollow organs such as the intestine or bladder. The omentum adheres to the incision site, which benefits from the omentum's hemostatic and angiogenic effects. The omentum also prevents leakage of luminal contents through small gaps in the incision and prevents peritonitis by dealing with local bacterial contamination. Overlay of omentum can also prevent adhesions from forming between the surgical site and other organs or the body wall. While the omentum will likely adhere to surgical sites with no help from the surgeon, deliberate omentalization ensures immediate and complete contact between the omentum and operated area.

When placed in an abscess, the omentum's extensive vascular and lymphatic networks absorb fluids (often precluding the need for drain placement) and actively fight infection. The abscess should first be debrided to the degree possible and lavaged, and then omentalized, putting omentum in contact with the inner surface of the abscess. This technique has led to resolution of abscesses in the liver, prostate, uterine stump, and pancreas of dogs and cats without the need for synthetic drains. Similarly, omentalization has been used to resolve intra-abdominal cysts in a variety of locations (liver, prostate, sublumbar lymph nodes, perinephric); the cyst is first deroofed and debulked to the degree possible, and then omentalized.

Omental use is not limited to the abdominal cavity. The omentum can be brought into

the thorax by passing it through a small hole made in the diaphragm. Indications might include the need to provide additional continuous drainage for chylothorax, or to deliver the healing and sealing powers of the omentum to organs like the esophagus, which is normally a poor healer. Chronic axillary wounds in cats resolved after omentalization +/- a thoracodorsal axial pattern flap; the unfolded omentum was passed through a small hole in the abdominal wall and tunneled subcutaneously to reach the axilla, where it was spread out and sutured to the periphery of the wound. Thoracic wall defects in dogs repaired with mesh or porcine submucosal bioscaffold have been reinforced with omentum tunneled subcutaneously to the affected site. The omentum has also been exteriorized to serve as the vascular bed for a full thickness meshed skin graft on the back of a dog. Free pieces of non-vascularized omentum placed around experimental nonunion fractures in dogs significantly enhanced fracture healing.

Reconstructive Surgery: Skin Grafts

INTRODUCTION: *Skin grafts are pieces of skin that are totally removed from a donor site and placed in a recipient site. In veterinary medicine, skin grafts are routinely autografts, i.e. the donor and recipient are the same individual. Partial thickness grafts contain complete epidermis and a portion of dermis, while full thickness grafts contain the complete epidermis and complete dermis. Full thickness grafts are most commonly used in dogs and cats because they provide a more cosmetic repair and because these species have elastic skin that makes it relatively easy to close the donor site. Full thickness grafts contain adnexal structures and can ultimately result in robust, furred skin at the grafted site.*

INDICATIONS FOR SKIN GRAFTS

Closure options for wounds include undermining, walking sutures, releasing incisions, subdermal plexus flaps, and axial pattern flaps. Skin grafts are indicated these options will not work or when second intention healing would take too long, result in a fragile epithelial cover subject to recurrent injury, or lead to scarring that might compromise movement or function.

HEALING OF THE GRAFT

As soon as the graft is removed from the donor site, degeneration of the graft tissue begins. Revascularization of the graft must occur within 7 to 10 days in order for the graft to 'take' (*i.e.* to be established as a viable piece of skin in the recipient site).

Starting immediately after grafting and continuing for several days, the graft survives via plasmatic imbibition, in which fibrinogen-free serum moves from the wound into the open ends of pre-existing blood vessels in the graft via capillary action. The fluid then moves into the interstitium of the graft. Because the newly-placed graft has no draining blood or lymph vessels, the graft becomes edematous. Absorption of heme products and lack of circulation cause the graft to turn blue-tinged.

Inosculation, which begins on 1 or 2 days after grafting, is the process by which pre-existing blood vessels in the granulation bed anastomose with pre-existing vessels in the skin graft. It establishes sluggish blood flow to and from the graft. The graft becomes less edematous and redder in color as these vascular connections are established over the first week.

Neovascularization begins on day 2 to 4 after grafting. New blood vessels and lymphatics grow from the wound into the graft, providing a much more substantial blood supply and allowing the graft to return to normal color during the second week after grafting.

Contact between the graft and wound bed is initially maintained by a fragile meshwork of fibrin, which helps support the vessels involved in inosculation. With time, fibroblasts and endothelial cells migrate into the fibrin and produce granulation tissue that solidifies the connection between wound bed and graft. The graft also becomes stabilized by ingrowth of new vessels and by granulation tissue growing up into the graft's mesh holes. Sutures, bandages, and negative pressure wound therapy provide additional stability.

PREPARATION OF THE WOUND BED AND DONOR SITE

A graft relies on the wound for its survival, so the wound bed must be healthy. Good graft beds include healthy granulation tissue, healthy muscle, healthy periosteum, and healthy peritenon. A freshly-made wound that is capable of developing granulation tissue can support a skin graft as well. Skin grafts have also been successfully used over an omentalized wound. Contamination, infection, exudate, poorly vascular tissue (e.g. tendon, ligament, bone without periosteum), exposed joint, and/or chronic granulation tissue are all contraindications for grafting.

Gentle scraping of the granulation surface with an angled scalpel blade can remove surface biofilm and perhaps help plasmatic imbibition. To decrease the risk of a hematoma forming between the graft and graft bed (which would block inosculation), it may be better to do the scraping a day or two before grafting Trim the skin edges of the wound to provide a fresh edge that will heal to the edge of the graft.

Clip and prep the donor and recipient sites as for any surgical procedure. The ideal donor site is one with redundant skin that can be closed after the graft is harvested and in an area that is accessible when the patient is positioned for surgery on the wound. Skin on the lateral thorax or lateral abdominal wall is commonly used as graft. If multiple donor sites are available, chose the one which best matches the recipient site in color and length of fur.

MESHED SHEET GRAFTS (FIG. 1)

A meshed sheet skin graft is cut to match the size and shape of the wound to be grafted. Make a template of the wound using sterile paper (e.g. from surgery gloves). Place the paper on the wound, let it soak up blood, then cut out the wound shaped from the paper. Lay the cut-out on the donor site, orienting it so the correct side of the paper is up (if you accidentally turn the paper over, your graft will be shaped like a mirror image of the wound!) and so the fur will grow in the desired direction when the graft is moved to the recipient site. Trace the cut-out on the donor skin with a sterile marker.

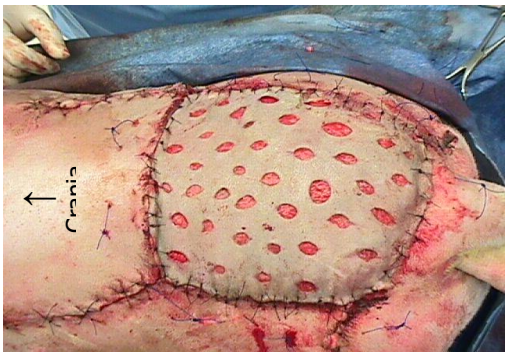


Figure 1. Meshed skin graft sutured to the caudal portion of a wound on the dorsum of a dog. (Dog is in ventral recumbency). The graft was taken from the lateral torso (donor site not in photo). A sliding advancement flap was used to close the cranial part of the wound.

Incise the donor skin along the tracing and excise the skin graft immediately deep to the dermis. Leave as much subcutaneous tissue in the patient as possible because it will interfere with plasmatic imbibition and inosculation. Cover the donor site with moist gauze until you are ready to close it.

Place the skin graft dermis-side up onto a sterilized wooden block, thick cardboard, cork board, or huck towel. Pin the graft in place with hypodermic needles. Scrape off any subcutaneous tissue remaining on the dermis with a scalpel blade held at an acute angle. When all of the subcutaneous tissue is removed, the exposed dermis will look white and 'pebbly' due to the pattern of hair follicles. Keep the dermal surface moist with sterile saline as you work.

Next, mesh the graft with a scalpel while the graft is pinned to the block. Cut staggered rows of 0.5 – 2 cm long holes that are 0.5 – 2 cm apart. Benefits of meshing include: (1) allowing fluid to drain instead of accumulating deep to the graft, where it would interfere with inosculation and revascularization and be a media for bacteria, (2) making the graft more flexible so that it maintains contact with the wound bed in a contoured defect, and (3) expanding the graft so that it covers a wider surface area. The longer the mesh holes, the more the graft will increase in width and decrease in height - take this into account when planning the overall size of the graft.

Place the meshed skin graft in the wound bed and attach it to the wound edges with staples or simple interrupted or cruciate non-absorbable monofilament sutures. Make sure the graft is in good contact with the wound bed, not taut so that it is pulled out of the wound bed. Avoid tacking sutures from the graft to the wound bed, which interfere with graft/wound contact and can induce hematoma which separates the graft and wound further. The mesh holes preclude the need for a drain under the graft.

PUNCH GRAFTS (FIG. 2)

Punch grafts are small (2 -6 mm diameter) plugs of skin that are planted in the wound bed. Multiple rows of punch grafts are typically placed. Unlike sheet grafts, which require a donor site that is approximately the size of the wound, punch grafts can be harvested from multiple smaller areas of available skin. Indications for punch grafts include a wound in a high motion area (e.g. overlying a joint), a wound with a highly contoured surface, or when there is not an adequately sized donor site. Because punch grafts are set down into the granulation tissue, their contact with the wound bed is less likely to be disrupted by movement than a meshed sheet graft lying on the surface of the wound.

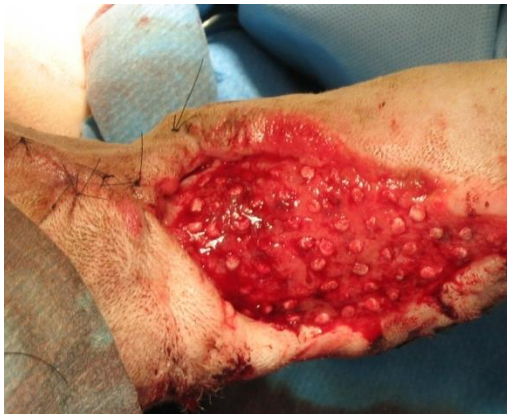


Figure 2. Multiple punch grafts implanted in the granulation bed of a wound on the cranial surface of the elbow in a dog. A splint will be added to the bandage to prevent joint motion during healing.

Punch grafts are easily harvested with a dermal biopsy punch. Angle the punch

parallel to the hair follicles to can increase the number of follicles per punch graft. Punch grafts can also be collected by tenting the skin with forceps or a suture needle and cutting off the elevated skin with a scalpel blade. As for sheet grafts, any subcutaneous tissue still attached to the punch graft must be removed; hold the punch graft on its side against the sterile board with a pair of thumb forceps and cut off the subcutis with a scalpel blade. Make small stab incisions into the granulation bed of the wound, and insert the subcutis-free punch graft; no sutures are used. It is helpful to plant the grafts on the bottom of the wound first, and then move upward; this prevents bleeding from the top row of grafts from running down on the second row as you are working on it. Compared to mesh sheet grafts, the cosmetic result for punch grafts is not as good. Hair growth tends to be sparse and patchy with punch grafts, and since it is difficult to maintain orientation of punch grafts as they are planted, hair is likely to grow in multiple directions. There also tends to be more areas of thin epithelial scar with punch grafts once healing is complete.

The punch graft technique can also be used to harvest grafts from healthy digital paw pads for wounds in the main metacarpal or metatarsal foot pad. Sutures are typically needed to secure paw pads grafts in place. The grafted pad should be protected from weight bearing with an extended splint or bi-valve cast.

POSTOPERATIVE MANAGEMENT OF SKIN GRAFTS

Bandages and activity restriction are absolutely required for skin grafts. The bandage helps maintain moisture in the granulation tissue exposed in the mesh holes, absorbs drainage, and guards against any movement between the graft and the graft bed, which can readily shear off the fragile new vascular connections that are key to survival of the graft. Because even a gentle bandage change can disrupt healing, the bandage is not changed for the first 2 to 3 days unless absolutely necessary (e.g. bandage is badly soiled or there is marked strike-through). The potential for movement between graft and graft bed is further minimized by cage confinement of the patient in the hospital, sedating a patient that is too active, placing a splint or bi-valved cast for grafted wounds on or near joints, and an Elizabethan collar.

A petrolatum-impregnated dressing on the graft is a good choice because it is non-adherent and keeps granulation tissue moist (thus speeding healing) without macerating the skin. The grafted skin typically returns to normal appearance in 3 to 4 weeks. Because of altered sensation that can occur as the graft is reinnervated, it is advisable to keep the site bandaged for ~ 4 weeks to prevent licking. (Splints can be removed after 2 weeks if there are no associated orthopedic injuries).

Negative pressure wound therapy is a good way to pull the graft down against the wound bed and eliminate motion. It also results in faster healing of the graft with less necrosis.

The early blue, edematous appearance of the graft can be disconcerting, but it is normal. The viability of the graft is hard to assess for the first 7 to 10 days. It is not uncommon for some regions of the graft to fail and turn into a black, leathery eschar. In the absence of signs of infection, the eschar can be left in place as a biological bandage. Often, granulation tissue develops deep to

the eschar, and the area can be re-grafted if it is not small enough to heal by second intention. With partial thickness failure of the graft, the epidermis and superficial dermis die, but the deeper dermis is revascularized and survives. In this case, new epithelium will grow from adnexae in the grafted dermis, but hair growth may be spotty.

If the graft develops a superficial infection, it can be cleaned with 0.05% chlorhexidine solution and treated with topical antibiotics with good chance of survival. When an infection develops deep to the graft, bacterial enzymes dissolve the fibrin that helps hold the graft to the wound and stimulate exudate that blocks inosculation. Treatment of deep infection includes lavage and systemic antibiotics, but if the infection is not responding, the graft needs to be removed.

CLIENT COMMUNICATION ON GRAFTING

As always, good client communication is important. Graft success is not guaranteed, and the possibilities of partial or total graft failure and subsequent need for a second surgery should be discussed. The critical importance of minimizing the patient's activity as the graft becomes revascularized should be conveyed. Clients should be prepared for the size and location of the donor site wound. They should also be advised of the expected cosmetic appearance of the graft site once it heals (e.g. spotty hair for punch grafts, anticipated changes in hair color or direction of hair growth).

In summary, appropriate wound selection, proper graft preparation and placement, and diligent protection of the graft postoperatively will maximize grafting success.

Reconstructive Surgery: Subdermal Plexus Flaps

GENERAL PRINCIPLES: *Preservation of blood supply is crucial for success in reconstructive surgery. In dogs and cats, direct cutaneous arteries emerge from deep tissues, turn parallel to the skin, and run in the cutaneous muscle, sending innumerable branches (the subdermal plexus) to the overlying dermis and epidermis (Fig. 1). To preserve blood supply when undermining the edges of a wound or raising a flap, dissect immediately deep to (dashed line) the cutaneous muscle. This preserves the blood supply to the elevated skin. Do not be fooled by the layer of subcutaneous tissue that may be between the cutaneous muscle and the dermis; undermining in this superficial fat cuts off the skin's blood supply. If there is no cutaneous muscle (e.g. limbs), undermine deep to all of the*

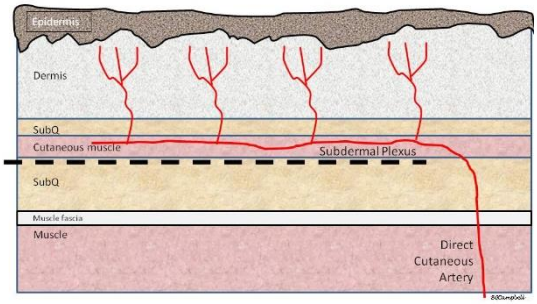


Fig 1.Blood supply to the skin. subcutaneous tissue, just superficial to the muscle fascia, to preserve blood supply.

The viability of subdermal plexus flaps (SPF) depends on the many small vessels of the subdermal plexus; thus, a relatively wide base of attachment is needed to provide adequate blood supply. In order to preserve blood supply, keep the length (longer side) to width (shorter side) ratio of a subdermal plexus flap less than or equal to 2:1. Suture the flap to the wound first, then close the donor site (if undermining of the donor site edges is needed to decrease tension, undermine deep to the cutaneous muscle).

Avoid putting tacking or walking sutures on the underside of a flap, as there is a risk of comprising its blood supply. Walking sutures can be used more safely on the edge of the flap, where it is being sutured to the wound edge. If there is a lot of dead space deep to a flap, a drain (preferably a closed active suction drain) may be placed.

SINGLE PEDICLE SLIDING ADVANCEMENT FLAP AND H-PLASTY

A single pedicle sliding advancement flap (Fig. 2) is rectangular and used when the long axis of available skin is aligned with the long axis of the wound. The flap width (W) is made to equal the width of the wound. The flap length (L) is no more than twice its width. When the flap is advanced into the wound, tissue bunches up and create dog ears at the corners of the flap's base. Burrow's triangles can be cut to remove this excess tissue and allow a smooth closure. The triangles must be made in the skin outside of the flap; if they are made within the base of the flap itself, the blood supply to the flap will be compromised. An H-plasty (Fig. 3) is created with two single pedicle

advancement flaps coming from opposite directions. For the H-plasty, each flap covers a portion of the wound, but the division does not have to be 50:50.

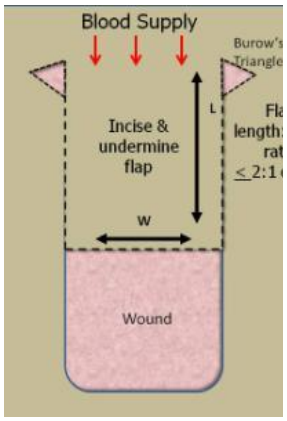


Fig 2. Single pedicle sliding advancement flap.

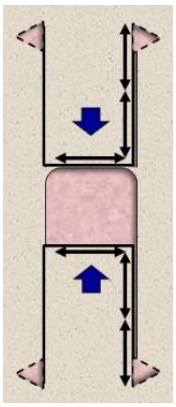


Fig. 3. H-plasty.

TRANSPOSITIONAL FLAP

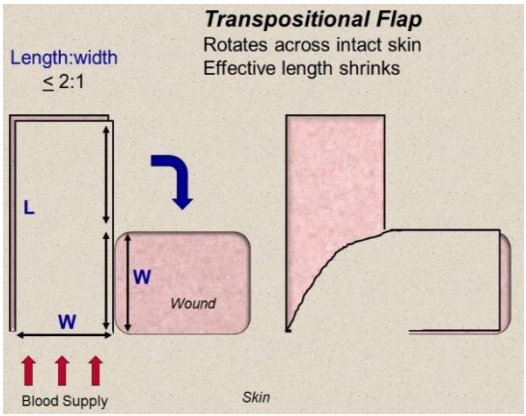


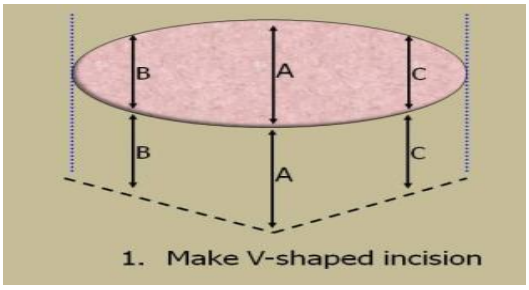
Fig. 4. Transpositional flap.

A transpositional flap is used when the available skin is oriented up to 90 degrees from the long axis of the wound (Fig. 4). The width of the flap is made to match the width of the wound, and the length to width ratio of the flap is no more than 2:1. Once elevated, the transpositional flap is pivoted on its base and into the wound; the flap will cross over intact skin as it is rotated. The closer the transpositional flap is to 90 degrees relative to the wound, the more the flap will effectively shorten when it is rotated. This shortening should be taken into account when assessing the amount of wound coverage the flap will achieve.

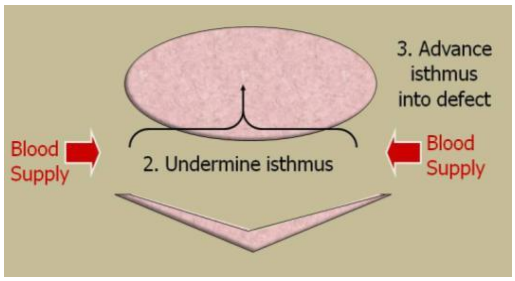
V-to-Y PLASTY

With a V-to-Y plasty, an isthmus of skin is created between the wound and a V-shaped-incision, undermined, and advanced to cover the wound. Step 1: make a V-incision (dashed line) that spans the wound, with the open side of the V facing the wound. The V-incision is placed far enough away from the wound that (1) the isthmus of skin between the wound and the V-incision is large enough to fill in the wound (A=A, B=B, C=C) and (2) the width of each attachment of the isthmus is large enough to support at least half the length of the isthmus, as per the 2:1 rule. Step 2: Undermine the isthmus of skin (staying deep to the cutaneous muscle). Step 3: Move the isthmus of skin into the wound. The original V will expand into a chevron. Step 4: Suture the isthmus to the original wound. Step 5: Undermine the chevron and close it by suturing in from each corner. Step 6: The result is a Y-shaped incision where the V was originally made.

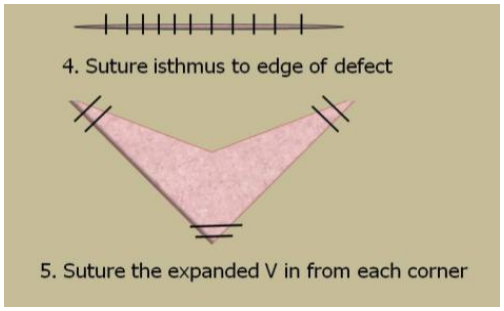
Step 1



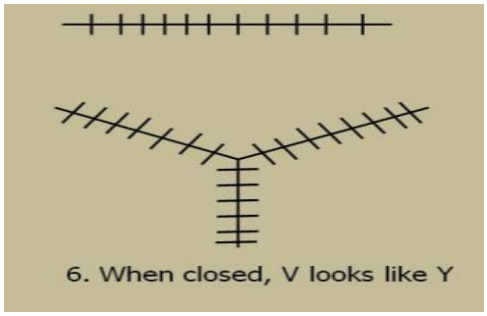
Steps 2 & 3



Steps 4 & 5



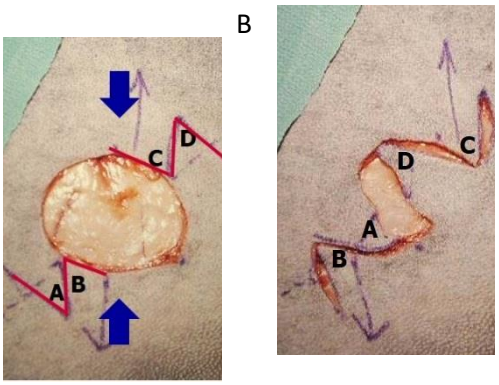
Step 6



Z-PLASTY

A Z-plasty moves skin in the direction of the central axis of the Z by breaking up a line of tension. It is particularly useful for wounds on the limb or perineum where there is skin proximal or distal to the wound that could be used to close the defect if only the tension that would be generated by pulling that skin into the wound was alleviated. While it may not look like much, the Z-plasty can be very useful in tight spots!

The Z has 3 parts: the central limb and two arms. The 3 parts are equal in length and the arms are at ~60° to the central limb. Fig. 5 shows a wound being closed with two opposing Z-plasties made adjacent to a wound. The Zs are oriented to meet two criteria: (1) central arm is parallel to the direction you want the skin to move and (2) one arm is tangent to the defect (Fig. 5A). After incising the Z's the position of the resulting triangles are switched (A with B, C with D), filling the defect with triangles A and D (Fig 5B).



A

Fig. 5. Two Z-plasties used to close a circular defect. (A) The block arrows indicated the direction the skin needs to move. Two Zs are drawn in red on either side of the defect, with their central arms parallel to the direction the skin needs to move. The Zs are positioned so that one arm is tangent to the defect (this arm will not need to be incised as it has already been 'cut' by the wound). Two triangles are formed within each Z (A and B, C and D) (B) After incising the Zs, the triangles were undermined and their positions switched (A with B, C with D) to fill in the defect.

A Z-plasty can also be made at a distance from the wound, creating an isthmus of skin that can be moved into the wound. Just as with the V-to-Y plasty, each isthmus

attachment must be wide enough to supply blood to at least half the isthmus length, as per the 2:1 rule. The central limb of the Z is placed along the line of tension that is palpable as you attempt to pull the desired skin into the wound (Fig. 6).

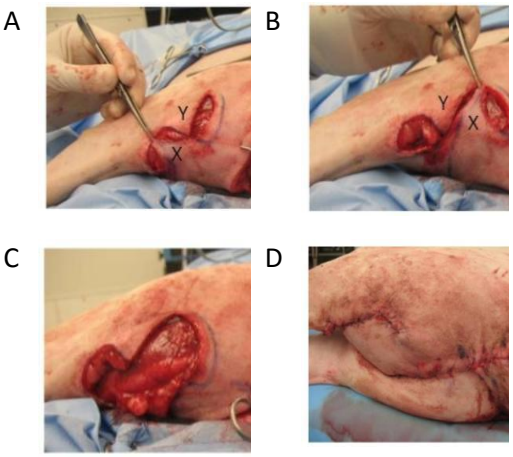
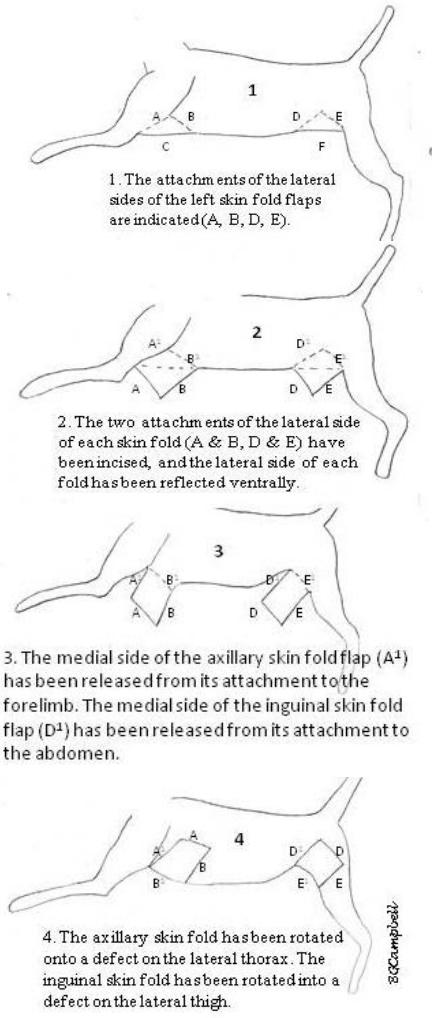


Fig. 6. A Z-plasty was used to break-up a line of tension distant from the wound. (A) A left perineal wound was created by the resection of a soft tissue sarcoma in a dog. (Dog is in right lateral recumbency, dorsal is to the right in all photos). There was too much tension to close the wound directly. (A) A Z-incision was made on the caudal left thigh, with the central arm over the line of tension that kept the skin from the limb from being moved into the wound. This created triangles X and Y, which were undermined. (B, C) By switching the positions of X and Y, the tension distal to the wound was broken up and the limb skin could be moved into the wound. (D) The wound was closed first and the Z-plasty site was closed second.

SKIN FOLD FLAP

Skin fold flaps use the redundant piece of skin between the torso and forelimb (axillary skin fold) or torso and hindlimb (flank or inguinal skin fold) (Fig. 8). When unfolded, the skin fold provides a large, mobile piece of expendable skin that can be moved into wounds on the body wall or proximal limbs. Each skin fold has two sides (lateral and medial); you can slide these two sides relative to each other. Each side has two points of attachment (to the body wall and to the adjacent limb). Cut two or three of the four attachments (often, one attachment lies along the wound and thus is already 'cut'), gently separate the lateral and medial sides, unfold the flap, spread onto the wound, and suture in place. The absence of skin folds does not cause any changes in ambulation. By altering the point of attachment that is preserved, these flap can be rotated into wounds on the lateral or ventral torso or lateral or medial proximal limbs.



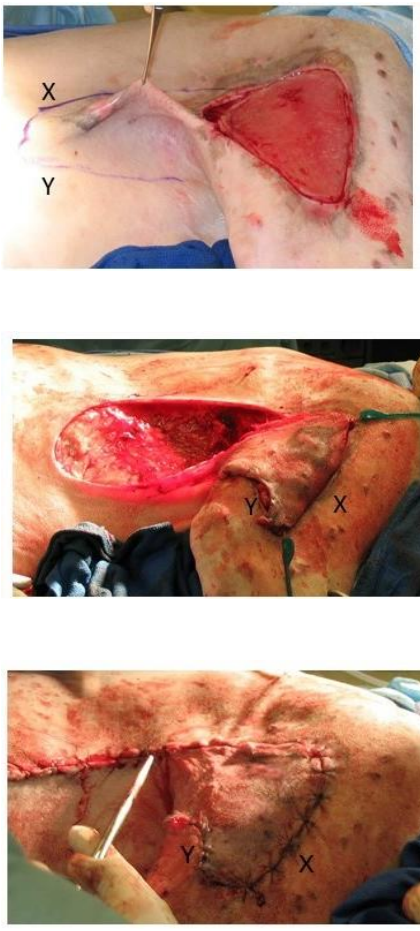


Figure 8. *Skin fold flaps. (A) Four drawings show how an axillary skin fold flap can be used to cover a wound on the lateral thorax by preserving its attachment to the medial body wall, and how an inguinal skin fold flap can be used to cover a wound on the lateral hindlimb by preserving its attachment to the medial limb. (B) For this left lateral thigh wound, the inguinal skin fold flap was cut along the lateral and medial attachments to the body wall (purple ink) and left attached on the medial side of the limb. The attachment to the lateral side of the limb is along the wound, and thus was already 'cut'. X and Y mark the dorsal and ventral sides of the flap before rotating.*

ROTATIONAL FLAP

A rotational flap can be helpful for triangular wounds (Fig. 9). The base of the flap is in line with the base of the wound, and the arc, which is an extension of the shortest side (x) of the defect, is 4x long. Start incising on the wound side and undermine the flap as you go; you may not need to incise the whole flap to get the wound covered. The length (height) to width (base) ratio of the flap should be ≤ 2:1.

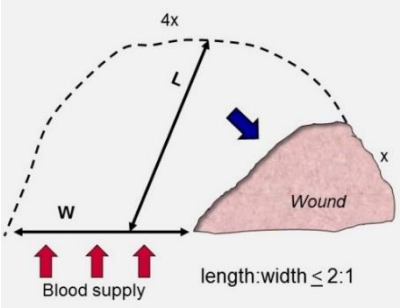


Fig. 9. Rotational flap

INTRA- AND POST-OPERATIVE CONSIDERATIONS FOR SUBDERMAL PLEXUS FLAPS & AXIAL PATTERN FLAPS

Reconstructive surgery patients should be clipped and draped extra-wide because the undermining, stretching, and rotation of skin during flap movement may mobilize more skin than initially anticipated, and because the original surgical plan may need to be changed intraoperatively. Make sure excess skin is free to be pulled into the wound and not trapped under the patient when positioned on the operating table. A sterile skin marker and sterile ruler are used to draw lines before cutting; as in carpentry, you want to "measure twice, cut once". After being elevated, skin flaps will shrink in size, but they can be expanded back to their original dimensions when sutured to the wound.

Penetrating towel clamps can be used to hold skin flaps in place, try different orientations of the flap in the wound to see which works best, and stretch skin intraop. Take into account the patient's normal standing position and the forces that occur during walking to determine the optimal flap position. Be sure to keep the wound bed and the underside of the flap moist

during the procedure. If you are having trouble closing a wound, try releasing a few towel clamps or a restrained limb. Sometimes this is all you need to allow tissue to close without tension.

Pain can be significant with reconstructive surgery, so intra- and post-operative analgesia is a must. An epidural can be very helpful. Postoperatively, a continuous rate IV infusion of fentanyl or an opiod/ketamine/lidocaine combination may be needed. At minimum, systemic analgesics (e.g. opiods) are given for the first 24 to 48 hours. Non-steroidal anti-inflammatory drugs (NSAIDs) may be added to the opiods in patients without renal, hepatic, or gastrointestinal disease. Bupivicaine can be injected into a wound diffusion catheter every 6 hours for topical analgesia. Oral analgesics are given for 1-2 weeks.

Skin flaps may initially look bruised due to the decreased blood supply. The most vulnerable site is the point furthest away from the flap's attachment to the body (and thus its blood supply). Flap failure flap is indicated by progressive transition of the affected skin to black. As long as there is no sign of infection under the dying part of a flap, it can be left in place as a biological bandage. If there is any concern for infection underneath, the necrotic portion is removed and the open wound is managed with moist wound healing techniques until it heals on its own or is ready for revision surgery.

Bandages are not needed for all skin flaps, and in some cases may be contraindicated. Advantages of bandages include keeping the incision and drain sites clean, keeping the pet from chewing/licking, and decreasing dead space (thus preventing seroma formation). The main disadvantage of bandages on skin flaps is the risk of compromising blood supply, which can be very serious. When put on too tight, a bandage can lead to flap failure due to interference with capillary perfusion, venous and lymphatic drainage, or the direct cutaneous artery. Even when a bandage is placed with the proper level of pressure, movement by the animal may cause intermittent vascular compression. Thus, 'less is more' for skin flap bandages. Commonly, a stockinette shirt is all that is needed to protect the surgery site.

Before surgery, clients should be prepped about the importance of seriously restricting the patient's activity, monitoring the surgery site, and keeping follow-up appointments. If client compliance is a concern, suggest hospitalization of the pet for one or two weeks postoperatively, as this is the critical healing period for flaps. Clients should also be made aware of the potential for a portion of a flap to fail, which might necessitate a second surgery. Finally, they should told of expected cosmetic changes: large amounts of hair will be clipped and use of flaps may lead to changes in color pattern, direction of hair growth, and length of hair in a given location.

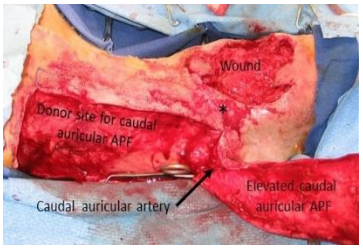
Axial Pattern Flaps

GENERAL PRINCIPLES:

Unlike subdermal plexus flaps [See proceedings on Subdermal Plexus Flaps by Dr. Campbell], which can be made just about anywhere on the skin, axial pattern flaps (APF) are located where a specific direct cutaneous artery will enter their base. The advantage of this placement is a robust blood supply; as a result, an APF can be much longer than a SPF and can be used in wounds that lack a healthy granulation bed and/or that have exposed bone or tendon. The shape and size of the APF is prescribed by that artery's angiosome (i.e. the area of tissue that will survive if that artery is its only source of blood). Most surgery textbooks provide the 'recipe' of where to cut to create each APF. The instructions for commonly used APFs are shown in Table 1. When measuring whether an APF will cover a given wound, take into account the fact that the more the APF is rotated to reach the wound, the more it will shorten in length.

After incising the proscribed borders of an APF, the APF is elevated starting at its most distal location and working back to its base (which contains the entry of the direct cutaneous artery). If the APF is in a region where there is a cutaneous muscle, the flap is elevated by dissecting deep to the cutaneous muscle, so that this muscle (and the subdermal plexus it contains) is kept with the skin. If there is no cutaneous muscle, dissection is done between the subcutaneous tissue and underlying muscle fascia (so that the subcutaneous tissue is kept with the skin). Extreme care is taken to not damage the direct cutaneous artery and vein as the flap is elevated and the base of the flap is reached. The deep side of the flap should be kept moist with sterile saline. Once elevated, the APF is ready to be rotated into the wound.

A)



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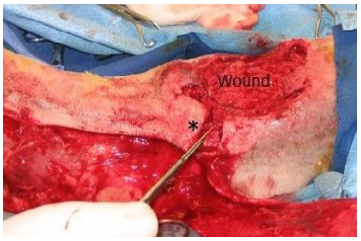

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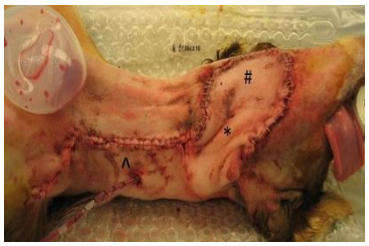

Fig. 1. Caudal auricular axial pattern flap and bridging incision for a wound on the caudal intermandibular region. (Dog is in dorsal recumbency with cranial to the right). (A) A caudal auricular APF has been elevated from its donor site. There is intact skin (*) between the flap base and the wound. (B) A bridging incision (*) is made in the intact skin to create a path for the flap. (C) The flap has been sutured to the wound (#) and bridging incision (*). The donor site (^) has been closed. A Jackson Pratt drain exits dorsal to the donor site.

If there is a section of intact, unwounded skin between the base of the APF and the wound, a ***bridging incision*** is made (Fig. 1). Because the entire underside of the APF needs to be in contact with subcutaneous or deeper tissue (*i.e.* not skin) when it is laid in place, a section of the intervening intact skin is excised to create a non-skinned path from the base of the APF to the wound. The region of intact skin that is removed is called a "bridging incision".

In order to avoid damage to the blood supply, tacking sutures should not be used to secure the flap in the wound bed. Instead, a ***closed active suction drain*** (e.g. Jackson Pratt drain) is used to eliminate dead space deep to the flap, preventing seroma and sucking the flap down against the wound bed to allow the two to heal together. The drain should be placed before the flap is sutured to the wound edges. The drain should not be exited through the any part of the APF (Fig. 2C).

After placing the drain and rotating the APF into the wound, the APF is shifted until the best, tension-free fit is found. Penetrating towel clamps can be used to temporarily clamp the APF to the wound edges to assess different positions. Once the best fit is found, the clamps can be replaced by single sutures to hold the APF spread out in the desired location. The edges of the APF are then sutured to the edges of the wound in a 2 or 3 layer closure (cutaneous muscle, subcutaneous tissue, skin), using absorbable suture in the deeper tissues and non-absorbable suture or staples in the skin.

Sometimes, an APF cannot be rotated all the way to the wound because it is too restrained by the uncut skin at its base. When this occurs, an ***island flap*** can be created by cutting the skin at the base of the APF, freeing the flap from all skin attachments and leaving the direct cutaneous artery & vein as the only connection between the flap and the body. Care is taken not to kink off the vessels as the island APF is rotated into place.

If the recipe for the borders of the APF has been followed, the donor site should be able to be closed routinely after some undermining of the edges.

CAUDAL SUPERFICIAL EPIGASTRIC FLAP

The axial pattern flap based on the caudal superficial epigastric (CSE) artery is the APF most commonly used in veterinary medicine. This flap can be used to cover wounds on the body wall, perineum, and

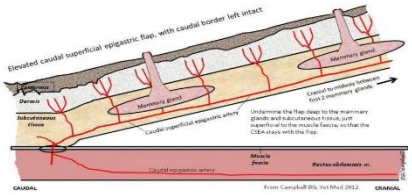

Figure 2. Caudal superficial epigastric flap.

hindlimbs.

The CSE artery) emerges from the deeper tissues in the inguinal area (Fig. 2). The 'recipe' for making a CSE flap starts with an incision along the ventral midline from the level of the CSE artery to a level midway between the first two mammary glands. A parallel incision is made lateral to the nipples, such that the distance of the lateral incision from the nipples equals the distance from the nipples to the ventral midline. The cranial aspects of these two incisions are connected between mammary glands 1 and 2, and the flap is elevated back to the CSE artery. Because there is no cutaneous muscle in this area, the flap is elevated deep to all of the subcutaneous

tissue (and the mammary glands contained therein), immediately superficial to the external rectus fascia of the body wall. This means that all subcutaneous tissue and the mammary glands stay with the skin (Fig. 6). If the patient is an intact female and gives birth, the mammary glands will be functional, but they will be located at the site to which the flap was relocated.

Other common axial pattern flaps include the thoracodorsal APF, omocervical APF, and caudal auricular APF (Table 1).

Table 1. Common axial pattern flaps.

	Borders	
Caudal superficial epigastric The flap is based where the caudal superficial epigastric artery emerges from the inguinal canal. Use for wounds on the abdomen, perineum, hindlimbs.	Medial	Runs craniocaudal along the ventral abdominal midline.
	Lateral	Measure the distance from the ventral midline to nipple, move that distance lateral to the nipple, and draw the lateral border parallel to the medial border.
	Cranial	Connect the lateral and medial borders between the cranial two mammary glands.
Thoracodorsal axial pattern flap Based where the thoracodorsal a. emerges in the depression just caudal to the scapula at the level of the acromion. Use for wounds on the torso (cranial half), forelimbs.	Cranial	Runs dorsoventral along the scapular spine.
	Caudal	Measure the distance from the scapular spine to the caudal edge of the scapula, move that distance caudal to the scapula, and draw the caudal border parallel to cranial border.
	Dorsal	Connect the cranial and caudal borders along the dorsal midline (this flap can also be extended to the opposite shoulder or into an L-shape along the dorsal portion of the opposite body wall).
Omocervical axial pattern flap Based where the omocervical a. emerges in the depression just cranial to the scapula at the level of the acromion. Use for wounds on the neck, thorax, forelimbs.	Caudal	Runs dorsoventral along the scapular spine.
	Cranial	Measure the distance from the scapular spine to the cranial edge of the scapula, move that distance cranial to the scapula, and draw the cranial border parallel to caudal border.
	Dorsal	Connect the cranial and caudal borders along the dorsal midline (this flap can also be extended to the opposite shoulder or into an L-shape along the dorsal portion of the opposite body wall).
Caudal auricular axial pattern flap Based where the caudal auricular a. emerges in the depression between the wing of the atlas and the vertical ear canal. Use for wounds on the head, neck.	Dorsal and ventral	Two parallel craniocaudal lines that outline the central third of the lateral side of the neck; the distance between the dorsal border and the depression between the atlas and vertical ear canal equals the distance between the ventral border and the depression between the atlas and vertical ear canal.
	Caudal	Connect the dorsal and ventral borders at the level of the spine of the scapula.

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Clients should also be made aware of the potential for a portion of a flap to fail, which might necessitate a second surgery. Finally, they should told of expected cosmetic changes: large amounts of hair will be clipped and use of flaps may lead to changes in color pattern, direction of hair growth, and length of hair in a given location.

Bites, Bullets, & Branches

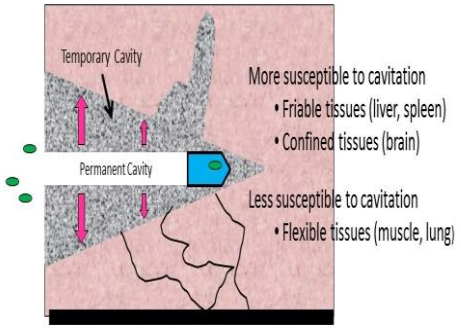
INTRODUCTION: *Penetrating wounds are deceiving! They create an "iceberg effect", where the skin is relatively unharmed except for a few puncture wounds despite severe damage to underlying tissues and blood supply. The patient may appear fine for a few days and then be suddenly overwhelmed by necrosis and infection. Pro-active patient assessment and surgical debridement are important to prevent progression to systemic inflammatory response syndrome (SIRS) and sepsis.*

Cover penetrating wounds to the chest immediately and other wounds as time allows. Even if the patient does not have respiratory signs at that moment, movement of the patient may shift wounded tissues in such a way as to result in pneumothorax. The seal on the chest can be improved by placing sterile ointment on the wound before adding the dressing and keeping pressure on the site with a gloved hand or bandage. Further wound care is undertaken once airway, breathing, and circulatory issues are addressed.

FORCES & TISSUE DAMAGE

A dog bite can exert a force of 450 psi (3.1 N/mm^2) or more. Incisors and canine teeth puncture skin, shear through deeper tissues, and inoculate bacteria and foreign material. When the victim is picked up and shaken, the skin, which is elastic, moves with the teeth while the more anchored underlying muscles and vessels are shredded and torn. Crushing by premolars and molars further compounds deep tissue injury.

A bullet's damage depends on its kinetic energy (KE = mass x $velocity^2$)/2) and the tissue density. The permanent cavity is the tract created as the bullet cuts through tissue. Dense tissues absorb more KE than less dense tissues, which explains why cortical bone hit by a bullet may shatter into multiple pieces (each of which becomes a new projectile) while the same bullet with the same KE may pass cleanly through a lung lobe with little parenchymal damage. Waves of cavitation energy move ahead and perpendicular to the bullet, pushing aside tissue and creating a larger temporary cavity that follows the path of least resistance, separating tissue planes and tearing fixed tissues. The outward movement of tissue also creates a vacuum which draws contaminants into the wound. As the compressed tissues rebound back, they recoil off each other, creating more damage. Because of cavitation, a missile can fracture bones, tear vessels, rupture bowel, and contuse organs without ever directly contacting these tissues.



DIAGNOSTICS

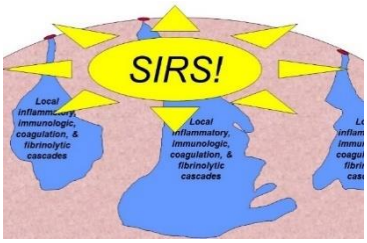
A thorough exam, including shaving fur, is important to find all sites of penetrating injury. Teeth typically leave paired marks, and multiple bite sites are common. Bullets or penetrating foreign material may create entry and exit holes or just an entry hole. Entry and exit holes may not line up because the penetrating object was deviated by dense tissue, fragmentation, or shaking.

Because of the iceberg effect, it is important to assess for deep or internal injuries. Probe wounds with a sterile instrument; it is common to find that multiple bite wounds are all connected beneath the skin due to disruption of subcutaneous tissue. It can be difficult to assess the true depth of a penetrating wound tract with a probe because the path may not be straight. Internal injury may also be revealed via thoracocentesis, abdominocentesis, or imaging. However, because it takes time for tissues to necrose or infection to develop, the absence of abnormalities on these tests does not rule out internal injury. Endoscopy of the esophagus and trachea should be considered when there are deep wounds to the neck.

Imaging is also useful in identifying retained foreign material. The number of intact bullets in the body and the number of bullet holes in the skin should add up to an even number (*i.e.* a bullet that exits the body = 2 holes + 0 bullets on imaging; a retained bullet = 1 hole + 1 bullet on imaging).

TREATMENT

Regardless of the type of penetrating injury, the untreated patient may seem fine for a few days, with just one or more small skin wounds. During that time, however, necrotic tissue, hematomas, compromised vasculature, dead space, inoculated bacteria, and foreign material stimulate local inflammatory, immunologic, coagulation, and fibrinolytic cascades. With insufficient treatment, these cascades may expand into systemic inflammatory response syndrome (SIRS; a severe inflammatory response in which local regulatory control is lost) and sepsis. The body can be ramping up to SIRS even when surface wounds appear mild. Surgical exploration is the only way to definitively assess the extent of trauma and thorough debridement of devitalized tissue is the only effective way to prevent or treat SIRS or sepsis.



After a wide surgical prep, incise entry and exit wounds and assess the underlying tissue. If the damage does not go beyond the skin, lavage and manage as an open wound. If the damage continues, follow the path to its deepest extent; it is common to find increasing amounts of tissue damage as you go deeper. Be prepared to enter the chest or abdomen. Excise clearly necrotic tissue; leaving it perpetuates the inflammatory response, blocks granulation, and increases the risk of infection. If the viability of tissue is uncertain, err on the side of taking it out unless it is essential for function or superficial enough to be debrided during a later bandage change. Lavage extensively. Leave wounds open and manage with moist wound healing techniques +/- serial debridement and lavage until such time as all tissue is healthy and all contaminants are gone. (See author's proceedings "Maximize Healing with Proper Wound Management"). Drains may be indicated if there is a lot of dead space or some uncertainty about the tissue health at the time of closure.

Damage from single, non-tumbling, non-deforming bullets that only pass through skin and muscle before exiting the body may be limited to the permanent cavity since skin and muscle are elastic and able to handle a lot of the energy associated with cavitation. A similar effect may be created by penetration with a sharp, smooth, clean foreign body. Such wounds can often be managed with more conservative debridement and lavage focused on the entry and exit sites.

Because of the high risk of injury to the gastrointestinal tract (which can be life-threatening and may not produce clinical signs for several days), exploratory celiotomy should be performed if there is penetrating trauma to the abdomen. The absence of pathologic findings on imaging or abdominocentesis does not rule out serious intestinal damage and thus cannot preclude the need to explore the abdomen.

REMOVAL OF FOREIGN MATERIAL

Tissue reaction varies with the chemical composition of foreign material. Oils and resins from wood can cause intense inflammation while some plastics slowly release irritating products. Steel shot, which is 99% iron, causes a local inflammatory reaction in dogs that generally subsides after 2 to 8 weeks. Lead bullets embedded in soft tissues are typically walled off by fibrous tissue and do not pose a poisoning risk. Lead in the GI tract or in contact with CSF does cause lead toxicity, and lead in a joint causes severe synovitis, so bullets should be removed from these areas.

A penetrating foreign body (FB) that is still partially exposed outside of the body should be left in place until it can be removed surgically, unless it is clear that the risks of leaving it in are more life-threatening than the risks of pulling it out without surgery. Risks of non-surgical removal include bleeding from holes in major vessels previously plugged by the foreign body, additional tissue damage caused by barb-like projections, and/or leaving behind fragments of foreign material (e.g. pieces of bark off of a stick). Prep the object as you would for a hanging leg prep, cleaning it with surgical scrub and/or wrapping it with sterile material prior to surgery.

One technique for surgical removal of a FB is to slit the tract open to its end and lift out the FB. However, fragments of foreign matter or other contaminants embedded in or around the tract can be missed. The preferred approach is to make an elliptical incision around the tract opening and dissect out the entire tract with a margin of normal tissue around it; this technique is more likely to remove all foreign material.

ARE ANTIBIOTICS INDICATED IN PENETRATING WOUNDS?

All penetrating wounds are contaminated with bacteria and debris from the site of penetration (skin, mucosa) and from the penetrating object. (High temperature in the gun barrel does not sterilize bullets). Cultures taken at the time of injury are typically not helpful because the bacteria that grow at that time do not correlate with bacteria that are cultured if the wound later becomes infected.

The risk of infection increases with the amount of tissue damage and vascular compromise. Debridement and lavage are the keys to minimizing the chance that contamination will turn into infection; antibiotics do not replace the need for local wound care! While prophylactic antibiotics may be given during the actual debridement, ongoing antibiotics are not indicated in immunocompetent patients

with simple wounds that have been effectively debrided. A course of prophylactic antibiotics should be considered in patients with extensive tissue damage and/or SIRS, or in those that are immunocompromised. Avoid fluoroquinolones and aminoglycosides as single agents since they do not affect anaerobes, which are common in devitalized tissues.

Foreign material lowers the concentration of bacteria required for infection. Patients often improve while on antibiotics, but signs return when antibiotics are stopped, and resolution of infection requires removal of the foreign material. *Actinomyces* and *Nocardia* are strong indicators that foreign material is present.

OTHER CONSIDERATIONS

Thorough documentation of wounds and proper storage of foreign material (e.g. bullets) retrieved from the body is particularly important when legal action may be taken. Necropsy by a board certified pathologist should be considered when a patient dies from such an injury.

Refer to local guidelines for proper management of bite wounds in animal not vaccinated for rabies. Cats can be tested for FeLV and FIV 60 days after being bitten by another cat.

Maximize Healing With Proper Wound Management

INTRODUCTION: *Like the white blood cell, our initial goals for wound management are to rid the wound of contamination and necrotic tissue and provide an environment that promotes granulation. These objectives are best achieved by combining knowledge of the body's own wound healing processes with accurate assessment of tissue viability, proper lavage, surgical and/or autolytic debridement, and moist wound management.*

WOUND HEALING BASICS

There are three overlapping phases of wound healing, each of which sets the stage for the phase that follows. The inflammatory/debridement phase typically occurs during the first 3 to 5 days after wounding. White blood cells move into the wound and perform *selective, autolytic debridement* of bacteria, foreign material, and necrotic tissue. This debridement is selective because only cells and matrix that are damaged are removed; healthy tissue is spared. Think of the white blood cells as tiny premier surgeons, cutting away only unhealthy tissues with microscopic accuracy! During the repair phase over the next ~2 to 4 weeks, fibroblasts and endothelial cells moving in from the periphery of the wound fill in the defect with granulation tissue. Epithelial cells from the skin edges migrate across the granulation tissue and build new skin as they move toward the center of the wound. Skin coverage is also achieved via contraction, in which pre-existing skin is pulled over the granulation tissue by myofibroblasts in the center of the granulation bed. The remodeling phase continues for months to years after the wound appears healed. Collagen fibers are realigned and cross-linked along lines of force add to tissue strength.

LAVAGE

Properly done, lavage removes foreign matter, decreases bacterial counts, rehydrates tissues, and speeds healing. Improperly done, lavage can damage tissue, delay healing, and increase the risk of infection. Important components of lavage are pressure, volume, and fluid composition.

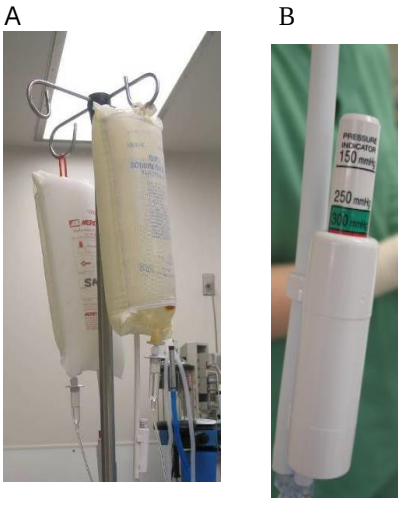


Fig. 1. (A) An emergency pressure sleeve on a one liter bag of fluids is pumped up to 300 mm Hg, as read on the gauge (B).

It takes more pressure to remove microscopic particles and bacteria than it does to remove gross debris. Thus, just because a wound looks clean grossly does not mean it is clean on a microscopic scale. The lavage pressure which maximizes

removal of debris and bacteria while minimizing tissue damage is 7 – 8 psi (48 – 55 kN/m^2). This pressure can be accurately achieved via a needle (16 to 22 g) on an intravenous drip set attached to a bag of fluids pressurized to 300 mm Hg with an emergency pressure sleeve. Sedation and analgesia are likely needed when lavage is done with appropriate pressure.

Bulb syringes, squirt bottles, and syringes without needles do *not* provide enough pressure, and a 35 cc syringe with a 16 to 22 g needle generates too high a pressure (mean = 15-18 psi (103 – 124 kN/m^2)).

As the volume of lavage is increased, contaminants in the wound are decreased, as long as appropriate pressure is used. Tap water is okay for removing surface dirt from the wound to start, but it should be followed by lavage with a sterile solution, such as normal saline. Antiseptics can be added for additional antimicrobial effect; the proper dilutions are 0.05% chlorhexidine solution (e.g. 25 ml of 2% chlorhexidine + 975 ml of diluent) or 0.1% povidone-iodine solution (e.g. 10 ml of 10% P-I + 990 ml diluent). Higher concentrations may delay healing, while lower concentrations may not be antimicrobial. Diluting to a certain color by eye rather than measuring is not accurate. Use the solution, not the scrub, formulation of these antiseptics; the detergent in scrub is harmful to subdermal tissues.

DEBRIDEMENT:

Debridement is the process of removing damaged tissue and foreign material from a wound. It promotes healing by eliminating physical barriers to granulation and epithelialization, removing a media for bacteria, decreasing exudate production, releasing inflammatory mediators that promote healing, and improving the clinician's ability to assess the wound. A combination of debridement methods is typically employed in wound care, and a given wound may require debridement multiple times.

SURGICAL DEBRIDEMENT

While not as precise as debridement by white blood cells, surgical debridement is selective within the surgeon's ability to distinguish between viable and nonviable tissue. Attachment, color, and texture appear to be most reliable means of assessing tissue viability by eye. Tissues that are unattached have clearly lost their blood supply; they should be removed if still lying on the wound. For flaps of tissue that are partially attached, the portion of a flap that exceeds a length to width ratio of 2:1 has a poor chance of surviving. The color of necrotic tissue ranges from black to brown to yellow to grey to white as the moisture content increases. Desiccated necrotic tissue becomes firm and leathery and persists as a dark-colored eschar, while moist necrotic tissue ("slough") is slimy, light-colored, and stringy (like mozzarella cheese on a pizza). Slough impairs healing and should be removed. It should be distinguished from a layer of fibrin, which is also light-colored and moist but more gelatinous (as in grilled cheese). Fibrin is left in place as it does not impair healing (and attempts to remove it may). Inflammatory exudate (pus) is wound fluid containing leukocytes (especially degenerating neutrophils) and dead tissue. Recognize that slough, fibrin, or pus do not mean that the wound is infected.

Tissues that are cold or that do not bleed when cut may be non-viable, but these changes may also be due to hypovolemia and/or hypothermia, common conditions in a trauma patient. Thus, do not make a decision about tissue viability based on temperature or lack of bleeding until

hypovolemia and hypothermia have been treated. When a tissue is cut to check for bleeding, bright red flowing blood is a good sign of viability; dark, oozing blood indicates congestion and poor perfusion.

When warranted by the patient's condition, assess sensation prior to giving analgesics or sedatives. Differentials for a lack of sensation include tissue death, drug effects, or nerve damage. Nerve damage can take weeks to months to resolve, and even if permanent, the tissue can still be valuable to the patient. Thus, do not use lack of sensation alone to decide whether or not to debride a piece of tissue.

Surgical debridement techniques range from the conservative resection of clearly devitalized (and thus insensate) surface tissue in an awake patient in a treatment area to aggressive resection of deeper devitalized tissue in an anesthetized patient in the operating room. Regardless of the approach, the wound should be prepared as for any surgical procedure, with proper clipping and cleaning of the peri-wound area and the use of sterile instruments, gloves, and aseptic technique. Before shaving, place a sterile lubricant (e.g. K-Y jelly) in the wound so that hair clippings do not stick to the wound bed.

Tissues that are clearly not viable must be removed, as their presence only delays healing and increases the risk of infection. When the viability of a given piece of tissue is unclear, use the following guidelines: (1) "When in doubt, cut it out" if there is only one opportunity to access that tissue, there is plenty of residual tissue so it won't be missed, and/or consequences of later necrosis would be severe. Examples include damaged tissue deep in a wound or inside the abdomen or thorax. (2) "When in doubt, if it's superficial or skin, leave it in" if there will be multiple opportunities to assess the tissue, the tissue is needed for later closure, and consequences of later necrosis are not severe because the tissue can be readily removed at that time. An example would be damaged skin on a distal limb, a location where there is limited skin available and which can be easily assessed at the next bandage change.

The wound should not be surgically closed if contamination or non-viable tissue remains or if the closure will be under tension. Treat open wounds with moist wound healing techniques (see below).

MECHANICAL DEBRIDEMENT – WET-TO-DRY NOT RECOMMENDED

For a wet-to-dry bandage, saline-soaked gauze are placed on the wound and bandaged in place. The gauze sticks to the wound as moisture is wicked into the outer bandage layers. When the gauze is removed, the adhered tissue is ripped off with it. Unfortunately, this results in non-selective debridement; *i.e.* healthy cells and new tissue involved in healing are removed along with necrotic tissue, and healing is significantly interrupted. Additional disadvantages of the wet-to-dry technique include loss of cells that migrate into the open-weave gauze, ability of bacteria to penetrate the gauze, aerosolization of bacteria as the dried dressing is removed, pain when worn and when removed, and gauze fibers that stay in the wound and stimulate prolonged inflammation. Wet-to-dry bandages are no longer standard of care in human or veterinary medicine, and have been replaced by moist wound healing practices.

MOIST WOUND HEALING

Wound fluid contains oxygen, water, and a physiological ratio of proteases, protease inhibitors, growth factors, and cytokines appropriate to the current stage of wound healing. Moist wound healing (MWH)

supports the body's own amazing healing mechanisms by keeping wound fluid in contact with the cells involved in healing; these cells need wound fluid to function fully. Thus, during the inflammatory/ debridement phase, MWH supports selective, autolytic debridement by white blood cells 24 hours a day under the bandage. During the repair phase, MWH supports the cells involved in granulation, epithelialization, and contraction. Compared to wet-to-dry or dry dressings, MWH techniques accelerate healing, increase patient comfort, decrease costs (due to longer intervals between bandage changes, faster healing, and less need for sedation), prevent aerosolization of bacteria, do not leave inflammatory components in the wound, and decrease the risk of wound infection.

MWH can be achieved with moisture retentive dressings (MRDs). Four common MRDs (listed from most to least absorptive) are: calcium alginate, polyurethane foam, hydrocolloid, and hydrogel. Choose the dressing that is best able to absorb the amount of expected exudate while still keeping a layer of wound fluid in contact with the wound. Using a highly absorptive dressing on a wound with low exudate will dry out the wound, while using a low absorptive dressing on a wound with high exudate will overhydrate (macerate) the wound; neither is desirable. See Table 1 for guidelines on dressing selection.

Cut the chosen MRD to fit the shape of the wound so that moisture stays in the wound and off of the skin. Cover the MRD with a standard soft-padded bandage (e.g. cast padding, roll gauze or Kling, and Vetwrap).

During early healing when exudate production is highest, a properly absorptive MRD is usually changed every 2 to 3 days. As granulation tissue forms and exudate level subsides, a less absorptive dressing is used, and bandage changes may be 4 to 7 days. (Change sooner if strike-through or soiling occurs).

Many MRDs combine with wound fluid to form a gel. It is normal for this gel to have a slight odor and yellow color ("gel & smell"), which may be misinterpreted as infection. However, diagnosis of infection should be based on examination of the patient (*e.g.* redness, swelling, pain, fever), not the dressing. If infection is present, MWH should still be used to support immune cell function.

Table 1. Characteristics of Common Moisture Retentive Dressings

MRD	Exudate Level	Properties	Indications	Contraindications
Calcium Alginate	High (Absorbs 20-30 times its weight).	Made from seaweed. Felt-like material that turns to gel as absorbs wound fluid.	Especially good for autolytic debridement of contaminated, moderate to highly exudative wounds. Good stimulator of granulation tissue. Hemostatic.	If insufficient exudate, will not gel and can dehydrate wound.
Polyurethane Foam	Moderate to high	Soft foam; does not gel.	Good for autolytic debridement, stimulates granulation and epithelialization. Versatile –pre-moisten foam with saline for use on lower exudate wounds. Place dry foam on macerated skin to wick out moisture.	Insufficient exudate. Foam is too soft to provide protection to boney prominences.
Hydrocolloid	Low to moderate	Sheet, paste, or powdered forms all turn into a gel as absorbs wound fluid. Sheets typically have occlusive backing & adhesive perimeter to attach to peri-wound skin.	Good for autolytic debridement, granulation, and epithelialization in low to moderately exudative wounds. Hydrocolloid sheet with impermeable backing can be used to add occlusive cover over other dressings.	Caution in late repair phase (adhesive edge may slow contraction). Caution if infection, (occlusive backing creates hypoxic environment - may favor anaerobes).
Hydrogel	Low	90-95% water, comes as gel or as sheet that gels in wound. If lacks occlusive cover, can add occlusive film on top to keep hydrogel's moisture in wound.	Dry wounds requiring autolytic debridement, granulation, or epithelialization. Adds moisture back to dry wounds. May enhance contraction on limb wounds.	May inhibit contraction on trunk wounds.

Índice

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Radiographic Evaluation Of The Coughing Dog

INTRODUCTION: *Interpretation of thoracic radiographs can feel intimidating, especially without a systematic approach. The purpose of this brief session is to aid the practitioner in developing an organized approach to the review of thoracic radiographs and specifically to review pulmonary patterns.*

OBJECTIVES

1) Review the 4 part interpretation paradigm
2) Describe the radiographic features of:
   a. the unstructured interstitial pattern
   b. the alveolar pattern
   c. the bronchial pattern
   d. the structured interstitial pattern
3) Review the differentiating features of pulmonary edema vs. pneumonia
4) Review the normal and abnormal appearance of the trachea

INTERPRETATION PARADIGM

In an effort to provide structure to radiographic interpretation of the thorax, a 4 part interpretation paradigm can be helpful.

The thorax can be deconstructed into:

1. the extrathoracic structures (including the osseous structures, body wall, and diaphragm as well as the portions of the abdomen and cervical region in the collimation);
2. the pleural space;
3. the mediastinum (all of it, including the heart);
4. the lungs (pulmonary parenchyma and pulmonary vessels).

These regions can be evaluated separately as long as it is understood that most diseases are multicompartmental, and synthesis of all abnormalities key to developing a succinct, prioritized list of differential diagnoses. We will focus on the pulmonary parenchyma for the purpose of this short communication.

ORGANIZING YOUR INFORMATION

In terms of organizing your information, follow a 4 part process to work through the information you gather from the radiographic image.

1. Describe your findings. Remember to use your Roentgen signs (Location, Number, Size, Shape, Margin, Opacity)
2. Generate a conclusion based on your findings.
3. Generate a succinct, prioritized list of differentials
4. Create a list of next steps

EVALUATING THE PULMONARY PARENCHYMA

The easiest way to evaluate the pulmonary parenchyma is based on peak inspiratory films. There are two questions you must ask as you evaluate the pulmonary parenchyma.

1. Is there an increase or decrease in pulmonary opacity?
2. What is the location of the change in opacity?
3. Is there a change in the position of the mediastinum?
4. How severe are the changes you've identified?

If the lung(s) are generally radiolucent, we need to consider where this change is located. The most common reason for diffuse decreases in opacity is hypovolemia from any cause, followed by pulmonary thrombo-embolism (focal or diffuse). Causes of focal radiolucencies would include: pulmonary bullae, blebs, cavitated lesions (granulomas or tumors), pneumatocoeles and pulmonary thrombo-embolism involving a specific lung lobe.

If the lungs are too radiopaque (white), we must consider the distribution/location of this change. Describe the anatomic location of the abnormality, noting which lung lobes are involved and if there is partial lobar involvement. Is the change peripheral, mid-zone or hilar?

Next, is there a contralateral or ipsilateral mediastinal shift noted on the VD/DV image (assuming the VD/DV image is straight and the sternum is superimposed over the thoracic vertebrae)? In other words, is the mediastinal shift toward or away from the abnormality that you have identified? It is important to understand that this change is an indirect measure of the size of the lung lobes. From this, we can conclude that if the lung is larger than normal, there is too much "stuff" inside the lung (blood, pus, water, cells); if the lung is smaller than normal, there is not enough air inside the lung (atelectasis).

Now we are equipped to approach the pulmonary patterns. We are going to work through the patterns from the easiest to identify to the most difficult.

PULMONARY PATTERNS

Alveolar Pattern

The alveolar pattern is the easiest to find. The components of an alveolar pattern include: uniform increased soft tissue opacity; border effacement with the pulmonary vessels and outer serosal wall of the airways; the presence of air bronchograms; a lobar sign; and border effacement with the heart or diaphragm.

Bronchial Pattern

The next pattern is the bronchial pattern. In general, bronchial patterns are generalized and you are looking for thickened small airways that will create "rings and lines" in the periphery of the lung. The central airways will always be prominent and in older dogs can mineralize. This can be quite striking in appearance but is an incidental finding in an older patient. Try to evaluate for the presence of small airways in the peripheral aspect of the lungs or the thin section of the lungs.

Vascular Pattern

Next is the vascular pattern. There are three options for increased opacity. Increase in size of the pulmonary arteries (heartworm disease), increase in size of the pulmonary veins (left heart failure) or increase in size of both pulmonary arteries

and veins (over circulation from left to right congenital cardiac shunts, arteriovenous fistulas, heart failure in cats, or volume overload in renal failure patients).

Interstitial Pattern

If none of the above fits, you are left with the "dreaded" interstitial pattern. The interstitial pattern can be classified as either a structured (miliary or nodular) or unstructured (diffuse increase in background lung opacity with decreased vessel border definition).

Lastly, assign some degree of severity to the pulmonary pattern (mild, moderate and severe). If you are arguing over a mild unstructured interstitial pulmonary pattern, forget it. You are not going to do anything about it anyway. Ultimately, the interstitial and alveolar patterns form a continuum, with the alveolar pattern being the most severe form of increased lung opacity. Therefore, it is probably redundant to say "severe alveolar pattern".

So a radiographic **description** may sound something like this:

"In the ventral aspect of the right cranial and middle lung lobes, there is an increased soft tissue opacity, with obscured vascular margins and the presence of air bronchograms. A lobar sign is also noted."

Our **conclusion** may be something like this:

"Cranioventrally distributed alveolar pattern."

All of the radiographic features described support an alveolar pattern.

Our **differential diagnoses** would sound something like this:

"Based on the anatomic location, bronchopneumonia or aspiration pneumonia are the top differentials."

If we are imaging a patient with clinical signs of fever, labored breathing or a history of vomiting and/or regurgitation, this radiographic diagnosis and differential list fits our other information (Table 1).

Now we can use this information to confirm **next steps**:

Treat empirically.

Trans-tracheal wash.

The caveats for lung patterns are:

a) The pulmonary pattern <u>will not correlate to a pathognomonic histological diagnoses.</u> Interpret in the context of other findings.

b) Pulmonary patterns are often mixed for a given disease. Decide what pattern is dominant, even if more than one is present.

c) The pulmonary pattern may represent a disease in transition (interstitial to alveolar or vice versa).

Table 1: Pulmonary Patterns

Pattern Name	Radiographic Features	Comments	Disease examples (no way all inclusive)
Alveolar	Lobar sign; Uniform soft tissue opacity; Air bronchograms; Will not see pulmonary vessels or airways; Border effacement of heart or diaphragm.	Location is important for formulating a differential list; Is the easiest pulmonary pattern to recognize.	Aspiration pneumonia; Bronchopneumonia; cardiogenic and non-cardiogenic pulmonary edema; neoplasia; hemorrhage; smoke inhalation; etc.
Bronchial	Rings and lines are noted within the pulmonary parenchyma; look in the periphery and away from the pulmonary hilum.	Usually generalized; be sure to evaluate in the peripheral lung fields and in the thin areas of lung.	Chronic bronchitis; pulmonary eosinophilic pneumonopathy; heartworm disease; allergic lung disease; feline asthma
Vascular	Increased in size of the pulmonary arteries, veins or both (left to right shunting lesions).	Added lung opacity is secondary to enlargement of the pulmonary vessels.	Pulmonary arteries – heartworm disease or cor pulmonale; Pulmonary veins – left heart failure; Both – left to right PDA, VSD, ASD or over circulation secondary to volume overload.
Structured Interstitial (nodules or miliary pattern)	Multiple "millet seeds" or small miliary nodules noted throughout the lung fields; variably sized pulmonary nodules.	Usually needs to be at least 5 mm in size to be seen as a distinct nodule; Fake-outs include nipples, end-on vessels and pulmonary osteomas.	Lymphoma, disseminated neoplasia (carcinoma) and fungal disease. Parasitic, eosinophilic or pyogranulomatous pneumonias. Nodules can be cavitated.
Unstructured Interstitial	Increased opacity to the lung fields with decreased visualization of the pulmonary vessels, aorta and caudal vena cava.	Typically generalized and never mild!	Exposure, exposure, expiration, expiration, lymphoma, fibrosis, fungal infection, edema, hemorrhage, infectious etiologies (viral, bacterial), eosinophilic pneumonopathy.

CONCLUSION

Interpreting lung patterns is less complicated than it seems. Changes in opacity should be identified and localized. The utility of interstitial vs. alveolar patterns is to establish severity and response to therapy. Cats never follow the rules.

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Diagnostic Imaging Of The Spine

INTRODUCTION: *Diagnostic imaging of the spine can be challenging, and multiple modalities are employed. The choice of modality is dependent upon the clinical question to be answered and availability. For many, radiographs serve as a very accurate and efficient screening test for many diseases of the vertebral column. Osseous lesions, including traumatic fractures, subluxations, or aggressive lesions associated with infectious or neoplastic processes can be detected using radiography.*

Evaluation of the soft tissues of the spine, including the spinal cord, intervertebral discs, and direct visualization of soft tissue stabilizing structures require additional imaging techniques. Myelography, or the injection of non-ionic iodinated contrast media into the subarachnoid space, can allow for indirect evaluation of the spinal cord for changes in position, size, and shape. Computed tomography (CT) is a cross-sectional imaging modality that provides contrast resolution compared to radiography. This allows for more detailed assessment of the bones and soft tissue structures of the spine, including the spinal cord. Evaluation of some spinal cord pathology is also possible with CT. Magnetic Resonance Imaging provides even greater contrast resolution of soft tissues and bones. The direct imaging of chemical alterations in anatomy allows visualization of edema, hemorrhage, and alterations in the chemical composition and structure of abnormal anatomy.

Before beginning any discussion of interpretation, it is important to address the concept of image quality. It is nearly impossible to gain any diagnostic information from a poorly performed study in any modality. For radiography, it is important that the patient is properly positioned without rotation; that 2 orthogonal projections are acquired (at minimum); that the x-ray beam is collimated to the area of the spine; and that technical factors are appropriately selected. For CT, appropriate positioning is also critical. The scan must be planned with a small field of view, on a straight spine, and reconstructions should be performed using both a bone sharp and a soft tissue algorithm. Positioning is no less crucial for MRI, and effective neurolocalization will assist in directing the examination to the appropriate anatomy.

RADIOGRAPHY

Radiography is an excellent screening test for many diseases. It is readily available, relatively inexpensive, and rapid. Radiographs are obtained by passing x-rays through a patient, and capturing transmitted x-rays to form a 2 dimensional image of a 3 dimensional object. Images are evaluated using Roentgen signs; lesions are described with respect to their location, size, shape, number, margin and opacity.

Systematic radiographic evaluation of the spine requires evaluation of the osseous structures as well as the spaces occupied by the joint spaces and the spinal cord. Evaluating the dorsal lamina, the pedicles, the vertebral bodies and endplates, the articular facets, and the spinous processes

is part of the osseous evaluation. Additional evaluation of the intervertebral disc spaces, the articular facet joints, and the intervertebral foramina is also required for complete radiographic assessment of the spine.

Evaluation of osseous structures for the presence of degenerative joint disease includes detection of osteophytes and enthesophytes associated with the vertebral end plates and articular facets. Features of aggression should also be noted. Depending on location, these can indicate neoplastic processes (primary bone, such as osteosarcoma) or infectious etiologies (discospondylitis). Finally, in cases of trauma, be sure to note any potential fractures or subluxations. For suspected spinal fractures, care must be taken when positioning the patient, and orthogonal projections are always required.

Narrowing of disc spaces and sclerosis of articular surfaces are features of degenerative joint disease. Intervertebral disc disease, or degeneration of the intervertebral disc, with eventual protrusion or extrusion of disc material, is an extension of these degenerative processes. Protrusion of disc material into the vertebral canal can cause varying degrees of acute or chronic spinal cord compression. This can often not be detected by plain radiography.

Lesions within the spinal cord can cause secondary osseous changes, such as thinning of cortical bone, or widening of foramina. Evaluation of the surrounding soft tissues is also important, and will yield information regarding muscle mass, body condition. Changes in symmetry should be noted.

MYELOGRAPHY

Introduction of iodinated contrast media into the subarachnoid space can allow for evaluation of the spinal cord indirectly. Areas of compression can be identified, as well as regions of spinal cord swelling using myelography. The spinal cord cannot be directly assessed, but this indirect information is useful for diagnosis of extradural lesions, such as intervertebral disc protrusion, extrusion, intradural, extramedullary lesions such as subarachnoid cysts, and intramedullary lesions such as neoplasia or even fibrocartilaginous embolic myelopathy.

Performing myelography requires practice, is invasive, and there are risks. This procedure can be done in general practice, but should not be attempted without proper training and experience. Risks are lower with lumbar injections. With injections into the cerebellomedullary cistern, there is a risk of pithing the patient. The location of intrathecal injection is often said to be dependent on the suspected location of the lesion; however, even myelography of the entire lumbar, thoracic, and cervical spine can be performed via a lumbar injection. Collection of cerebrospinal fluid should be performed prior to injection.

Lumbar myelography requires placing a needle through the interarcuate space typically at the level of L5-6, and into the subarachoid space. The bevel of the needle should be directed cranially with lumbar injection. Often, the spinal cord is penetrated by the needle; this typically does not result in additional morbidity, however there are instances of central canal filling. CSF flow into the needle and hub should be visible, and without blood. Once 1-2 ml of CSF have been collected, and position of the needle in the subarachnoid space confirmed, injection can commence. If the needle is in the epidural space, contrast will be delivered into the epidural space, which will result in a non-diagnostic study. Injection at the cerebellomedullary cistern involves inserting a needle at the

junction of the skull and C1, into the dorsal subarachnoid space. Penetration of the medulla/cranial spinal cord can result in the death of the patient. The needle should be directed caudally. Epidural injection does not typically occur at this level, as the dura is adherent to the bony strucxtures of the skull and C1.

Complete evaluation of the subarachnoid space and indirect evaluation of the spinal cord requires complete filling of the subarachoid space. The volume of non-ionic iodinated contrast medium is typically 0.3 ml/kg, not to exceed 10 ml, even for large dogs. The volume of any tubing should also be added.

Injection of non-ionic iodinated contrast should be performed slowly, with only mild pressure. In cases of spinal cord swelling or severe compression, injection can be challenging as the flow of CSF and contrast can be restricted. Adding too much pressure can also result in an epidural injection and a non-diagnostic study.

Deviation of the contrast columns is indicative of a lesion. Axial deviation suggests an extradural lesion such as intervertebral disc protrusion/extrusion. Thinning of the contrast column typically is also present. Widening of the contrast column, with what is often referred to as a "golf-tee" sign is suggestive of an intradural-extramedullary lesion, such as a subarachnoid cyst. Abaxial displacement of the contrast column is suggestive of an intramedullary lesion, such as a tumor, inflammatory lesion, or potentially an infarction.

COMPUTED TOMOGRAPHY

Computed Tomography, or CT, also uses x-rays to generate an image. The physics of CT is beyond the scope of this discussion, but it is important to note that CT is a cross-sectional imaging modality that creates "slices" of anatomy, eliminating the superimposition and distortion that hampers radiographic examination. Depending on scanner technology, speeds can be relatively fast. For some cases, heavy sedation will allow the patient to remain still for the study. If contrast is required, general anesthesia may increase patient safety and reduce motion artifact that can occur during injection.

The cross-sectional nature of the modality and superior contrast resolution compared to radiography, results in more accurate, direct assessment of the vertebrae, the epidural space, and the spinal cord while eliminating superimposition. Faint mineralization of intervertebral discs can be detected, making non-contrast CT a relatively accurate test for intervertebral disc disease, especially in chondrodystrophic breeds.

Assessment of the osseous and soft tissue structures is routine, as outlined for radiography, but more of the anatomy can be evaluated. While the spinal cord can be evaluated directly, pathologic features may not result in changes in attenuation, and can be missed. For diseases such as fibrocartilaginous embolic myelopathy, lesions will not be evident.

Myelography can also be coupled with CT (CT Myelography) to increase the sensitivity, specificity and accuracy for detection of spinal cord disease, including intervertebral disc protrusion/extrusion/sequestration as well as diseases such as fibrocartilaginous embolic myelopathy.

MAGNETIC RESONANCE IMAGING

Magnetic Resonance Imaging or MRI utilizes strong magnetic fields to manipulate hydrogen atoms, creating an image that is dependent on the magnetic properties of protons and their immediate environments, including their chemical bonds. As with CT,

the physics of MRI are beyond the scope of this lecture.

MRI is not a screening test for most disease processes. Although the technology continues to advance at an incredible rate, MR units are typically not as readily available due to their expense, the need for specialized power and room shielding, and maintenance. Sequences are getting shorter and new image sequences are being developed, but most spin-echo sequences require several minutes to perform. In addition, the patient must remain still for the entire sequence as movement will ruin the acquisition, therefore general anesthesia is necessary.

MRI has contrast resolution that is superior to radiography and computed tomography. Pathology creates alterations in the local magnetic field, which has a direct effect on the signals generated by the protons in that region. Further, MR sequences have been designed to maximize our ability to see imaging features that can allow detection of hemorrhage, edema, mineralization, contrast enhancement, and infarction. MRI has become the standard for the diagnosis of central nervous system lesions.

CONCLUSIONS

Multiple imaging modalities are available for the evaluation of the spine. Choosing the appropriate test will be based upon clinical signs, availability, and cost. While MRI is superior to radiography and computed tomography for diagnosis of neurologic diseases, it is not always the most practice imaging test to choose, and much can be learned from a properly performed radiographic series or CT examination.

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The Limping Dog: Imaging Of The Lame Patient

INTRODUCTION: *The interpretation of orthopedic radiographs can be a daunting task. Investigation of lameness is a common presenting complaint, and radiography is still the primary screening test for lameness diagnosis in dogs. As with any radiographic assessment, having a systematic approach to radiographic interpretation is the cornerstone of accurate evaluation. Differentials primarily revolve around degenerative joint disease from developmental orthopedic diseases; joint injury, such a cranial cruciate ligament rupture; or aggressive changes associated with infection or neoplasia.*

ROENTGEN SIGNS

Roentgen signs (location, opacity, size, shape and number) may seem simplistic, but are fundamental in interpreting radiography. Of these, the location of a lesion may best serve to narrow your list of differentials. Determining whether the geographic center of a lesion is bone, joint or soft tissue significantly affects the list of differentials and focuses the diagnostic plan.

DISEASES OF THE IMMATURE SKELETON[4]

There is a concise list of musculoskeletal diseases that are associated with large breed, rapidly growing dogs. These diseases often have characteristic radiographic appearances, and can be easily recognized. The first step in this process is isolating the lameness to determine the radiographic area of interest. For example, it is important to determine if the lameness is joint related or bone related. Remember, it is NOT acceptable to open up the collimation and radiograph the entire thoracic limb. Imaging studies should be targeted to an area of interest to best maximize contrast and spatial resolution.

Specific diseases often occur is certain breeds, or in breeds of certain sizes (Table 1).While it is important to consider diseases of the immature skeleton when presented with a young dog that had a history of lameness, it is important not to become limited in the scope of your considerations. Cruciate injury/rupture can occur in young dogs as well as mature dogs. In addition, osteosarcoma is bimodal in its age distribution, and may be seen in dogs that are 2 years of age. Other neoplasms, such as lymphoma and histiocytic sarcoma, may also occur in younger patients. Osteomyelitis, either fungal or bacterial, can also occur in young patients, and should not be excluded as a consideration for lameness in young dogs. Therefore, while it may be tempting to exclude neoplasia or other aggressive diseases from your differential list in young patients, it is important that you continue to include all differentials prior to radiography.

DEGENERATIVE JOINT DISEASE[3]

Degenerative joint disease (DJD) is a common radiographic diagnosis. Radiographic features of DJD include subchondral bone sclerosis, periarticular new bone formation (osteophytes), and joint effusion/capsular thickening. Severe degenerative changes with marked periarticular new bone proliferation and

subchondral bone cysts may mimic aggressive disease. Be diligent in your assessment of aggressive radiographic features.

DJD can be classified as primary (idiopathic) or secondary. Most commonly DJD is a secondary process, occurring as a result of joint instability. Once signs of DJD are noted, the task then becomes to identify the cause of instability. The cause may be evident radiographically, as in a case of osteochondritis dissecans; or it may not be visible radiographically, as in a case of cranial cruciate ligament injury or rupture. Therefore, it is important to understand the common pathophysiologic mechanisms of instability in specific joints.

Table 1: Disease processes of the immature skeleton

Disease	Signalment	History and Physical Exam Findings	Radiographic Findings
Osteochondrosis/ Osteochondiritis Dissecans	6-9 month old Large breeds Rapidly growing	Lameness Joint pain +/- Effusion	Subchondral bone defect (flattening) affecting articular cartilage of afflicted joint, often with sclerotic margins Secondary DJD Locations include: • Caudal head of humerus • Distomedial humeral condyle • Lateral femoral condyle • Medial trochlear ridge of the talus • Lateral trochlear ridge of the talus
Elbow Dysplasia	6-12 month old Large breeds Rapidly growing	Lameness Joint pain +/- Effusion	Umbrella term that includes: • Ununited anconeal process • Fragmented medial coronoid process • Osteochondrosis/ Osteochondritis dissecans • Incongruity
Hip Dysplasia	6 months old - Adult	Pelvic limb lameness	Subluxation of coxofemoral joint Secondary DJD
Avascular Necrosis of the Femoral Head	1-2 year old Miniature breeds	Pelvic limb lameness	Appearance depends on stage of disease. Early: May be normal or may see lucencies in subchondral bone of proximal femoral epiphysis and metaphysis Late: Flattening and irregularity with remodeling of the femoral head and neck Secondary DJD
Hypertrophic Osteodystrophy	2-7 month old Giant Breeds	Lameness Depression Inappetance Pyrexia Hyperkeratosis Leukocytosis	Typically located at distal radial and ulnar physis Double Physis Periosteal new bone cuff along physis Soft tissue swelling
Panosteitis	5-12 months old Large breeds	Lameness Long bone pain Pyrexia	Solitary or multiple Medullary opacities with blurring and accentuation of trabecular bone Centered on the nutrient foramina of long bones Smooth, well-defined periosteal new bone

AGGRESSIVE VERSUS NON-AGGRESSIVE BONE LESIONS[3]

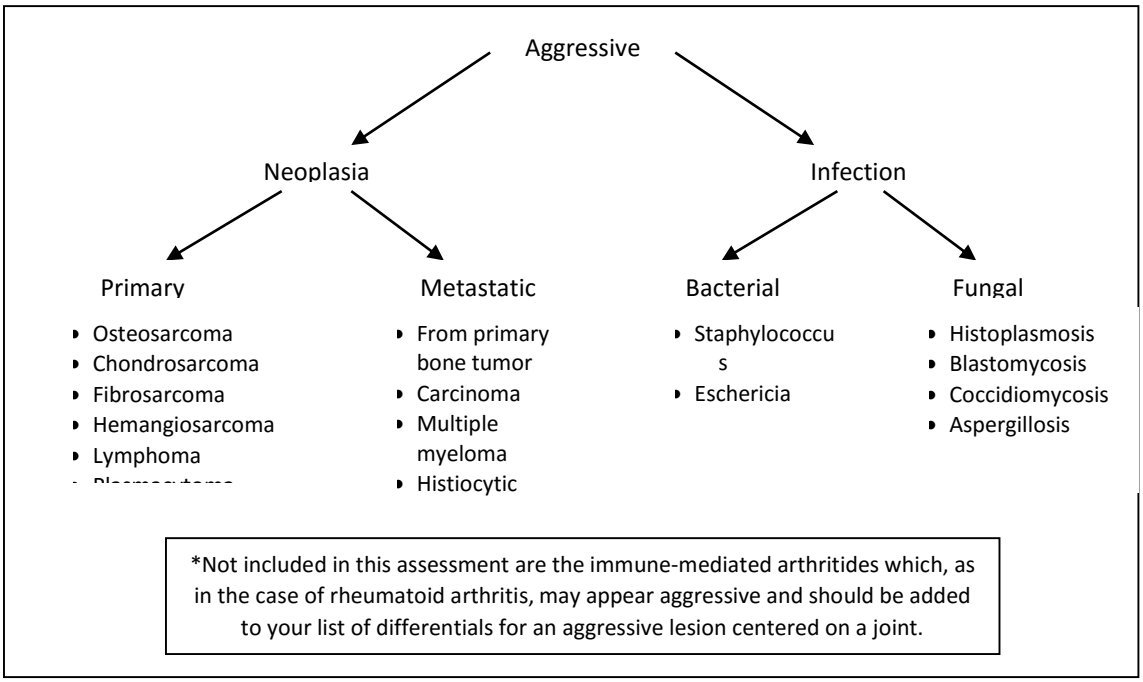
The first step in evaluating bone lesions is to assess for features of aggression. This is one of the most important determinations to be made early on in the interpretation process as it effectively narrows the differential list. The primary focus of this assessment is the characterization of the features of lysis and periosteal new bone formation; however other features also help in differentiating aggressive lesions from non-aggressive lesions (Table 1). It is important to remember that the presence of one aggressive feature from the list is compatible with an aggressive lesion.

Table 1: Radiographic Features of Aggressive Bone Lesions

	Non-Aggressive	**Aggressive**
Lysis	Geographic	Moth-eaten Permeative
Periosteal Reaction	Smooth Well-defined Continuous	Irregular Ill-defined Interrupted
Zone of Transition	Narrow Well-defined	Broad Ill-defined
Rate of Change	Slow	Fast
Number of Sites	Monostotic	Polyostotic

Once a lesion is determined to be aggressive, there is a logical decision tree that can allow you to arrive at a short, reasonable list of differentials (Table 2). This map can be useful, and one can navigate down the appropriate path in light of the patient's signalment, history and clinical findings. An appropriate list of differentials will assist in generating a plan for additional tests such as biopsies and thoracic radiography, or help to institute a therapeutic plan.

Table 2: Tree Diagram of Aggressive Bone Lesion Differentials



WHAT DO I DO NEXT?

Once an aggressive lesion is identified, it is important to proceed with obtaining a cellular diagnosis. This can be attained via fine needle aspiration or biopsy techniques. Fine needle aspirates have been shown to have reasonable diagnostic accuracy in cases of osteosarcoma. For other neoplasms, biopsies using a jamshidi needle may be required.

In cases where fungal disease may be possible, fungal titers may also provide additional information on a final diagnosis.

Finally, it is important to stage disease. This typically includes radiography or computed tomography of the thorax, and possibly ultrasonography of the abdomen. In cases of infection, a urinalysis may also be performed. In some instances, other screening tests for metastases, including nuclear scintigraphy and/or whole body CT may be indicated.

SUMMARY

While many clinicians find the interpretation of musculoskeletal radiography a challenge, a systematic approach to the information included in an image results in a maximal benefit. Assessing the aggression of an osseous lesion is the first step. The best use of radiography is often not to attain a definitive diagnosis, but rather to narrow your list of differentials and help plan the next diagnostic test.

Radiographic Evaluation Of The Heart

INTRODUCTION: *Radiographic evaluation of the cardiac silhouette can be a daunting task. There are several semi-objective measures of cardiac size, and a structured method of evaluating the cardiac shape and contour. These include rules about the number of intercostal spaces a normal heart should cover in the thorax; the height and width of a normal heart as a percentage of the total thoracic diameter; the vertebral heart score; and in the case of assessment of the cardiac shape, the "clock face" analogy.*

The diagnosis of heart disease involves further assessment of the cardiopulmonary structures, including the size and shape of the pulmonary vasculature (arteries and veins), the presence of pulmonary edema or pleura effusion, and the presence of ascites.

OBJECTIVES:

1. *Review and understand the different methods of cardiac size and shape assessment in the dog*
2. *Review and understand the utility of the clock face analogy in assessing cardiac shape*
3. *Understand the utility and the limitations of such tests*
4. *Discuss the methodology of successful detection and characterization of heart disease in dogs*

KEY POINTS:

1. *The vertebral heart scale, and other assessments of cardiac size, are useful tests but should not be used in isolation*
2. *The value of radiographic interpretation of cardiovascular disease is in the complete, summed assessment of all structures rather than in an individual finding*
3. *Significant breed variation exists, and a range of normal appearances is possible*

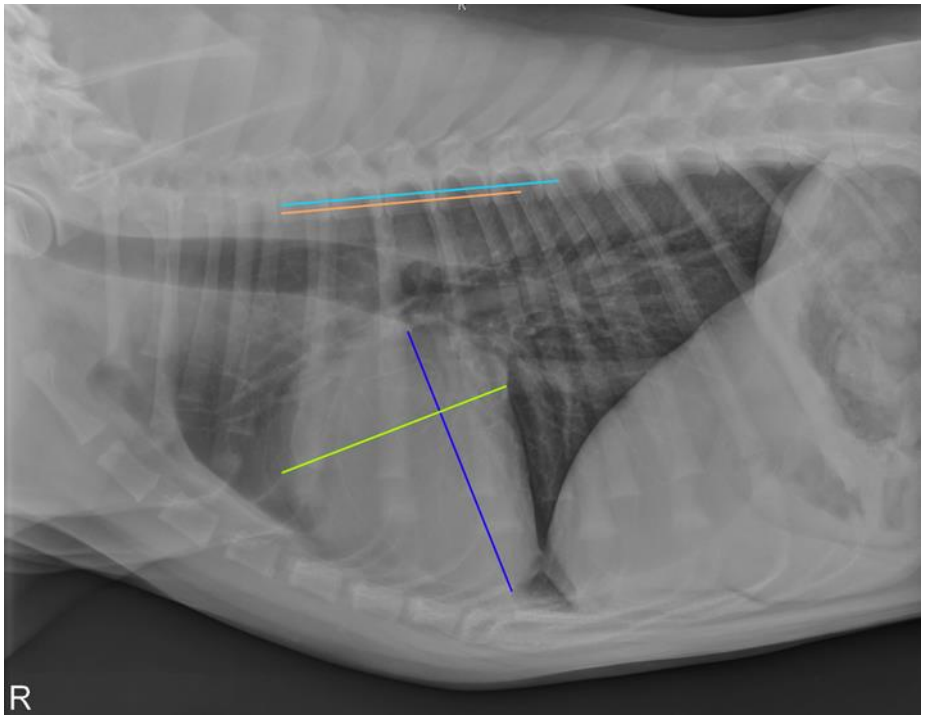
CARDIAC SILHOUETTE EVALUATION

The radiographic evaluation of the cardiac silhouette requires the use of all Roentgen signs. It seems strange to think that all Roentgen signs (location, size, shape, number, margin and opacity) can apply here. For example, there is only 1 cardiac silhouette. But if we consider the *number* of chambers that might be involved in cardiac disease (1, 2, 3 or 4), then *number* becomes an important part of our cardiac assessment, and along with the evaluation of the *location* of enlargement, helps us determines the *distribution* of disease (right-sided, left-sided, generalized). In addition, while we expect that the heart should be soft tissue opaque, we need to recognize that components of the cardiac silhouette contain fat (pericardial fat) and may influence our assessment of cardiac size if we are not careful. Also, there are some instances where we may see abnormal mineralization of the aortic valve or coronary arteries.

Heart Size

The vertebral heart score was reported by Buchanan *et al* in 1995 as a method for objectively assessing cardiac silhouette size in dogs. This score should typically range from 8.7 to 10.7 in normal canine patients and 6.9 to 8.1 in feline patients. Since that time, there have been adaptations of this

score to cats, and modifications to increase its accuracy in specific breeds, and in puppies. This is a useful score, but it should be noted that, on lateral projections, this test has a sensitivity and specificity of 86% and 80% respectively, meaning that some animals measured normal that had disease, and some animals measured enlarged, with no disease. In addition, the ranges of the VHS can vary significantly with breed, with some normal dogs of certain breeds having hearts that are much larger than the reference range. This further supports the idea that complete assessment of thoracic radiographs is important in reaching an accurate diagnosis of cardiac disease, and familiarity with breed variations is of utmost importance.



FIG(1)*Example of how the vertebral heart score is measured. Note the placement of the calipers on both the heart and the vertebrae. On this same image, note the fact that the heart occupies less then 3 intercostal spaces.*

Another valuable tool for evaluation of cardiac size is assessment of the number of intercostal spaces (ICS) that the heart covers on a lateral projection. In general, the cardiac silhouette should not cover more than 3-3.5 ICS on a lateral projection.

Finally, an increased cardiac height and/or width can correlate with increased cardiac size and, potentially, cardiac disease. Typically, the heart should be ½ to 2/3 of the height of the thorax on a lateral projection, and ½ to 2/3 the width of the cardiac silhouette on the VD projection at the level of the 5th ICS.

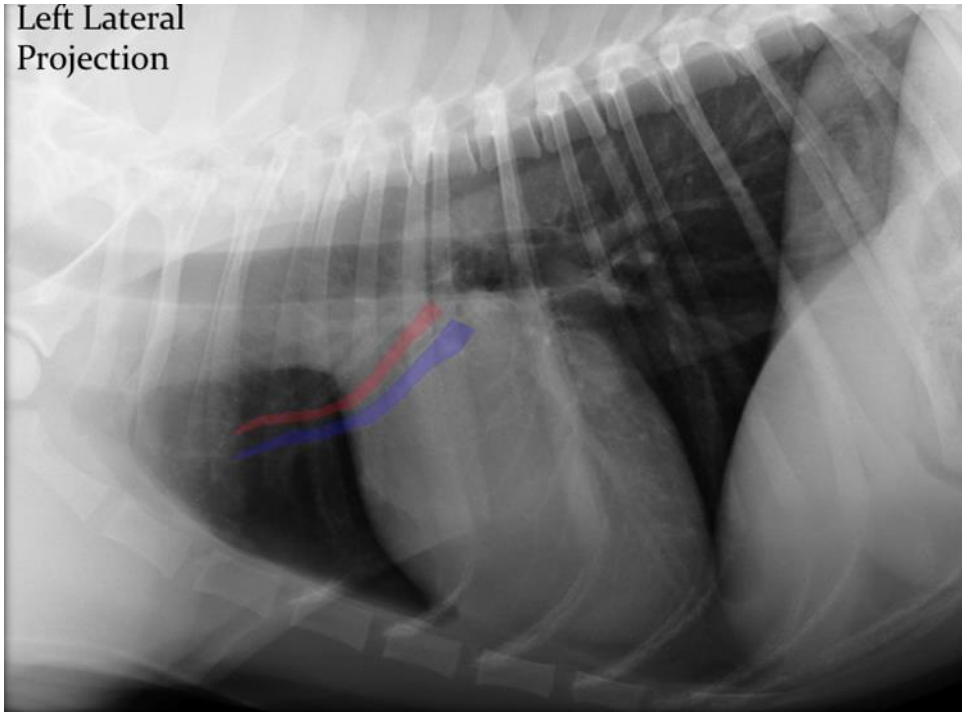
THE CLOCK FACE ANALOGY

The “clock face” analogy is a tool used to assess changes in cardiac shape that can be attributed to enlargements of specific chambers or great vessels. In combination with assessment of cardiac size, the accuracy of cardiac disease diagnosis will be increased.

OTHER CARDIOPULMONARY FINDINGS

Pulmonary Vasculature

The pulmonary vessels are a window into the "plumbing" of the cardiovascular system, and thorough evaluation of the pulmonary vessels will allow for determination of congestion (left – sided heart failure, fluid overload), pulmonary overcirculation (left to right shunts, fluid overload), pulmonary undercirculation (pulmonic stenosis, dehydration, cardiovascular shock), or pulmonary thromboembolism (oligemia).



FIG(2) *Left lateral projection showing the right cranial lobar pulmonary artery (red) and pulmonary vein (blue). Paired arteries should have similar size and taper as they move out to the periphery of the lung.*

Fluid Accumulation

Fluid in the lungs (pulmonary edema), in the pleural space (pleural effusion) or in the peritoneal space (peritoneal effusion) may also indicate an increase in pressures that relate to cardiac dysfunction. While other differentials for the accumulation of fluid are possible, when these findings occur in conjunction with cardiac enlargements, and in specific regions, they should be included in a succinct conclusion and differential list that involves cardiac dysfunction.

CONCLUSION

The radiographic evaluation of the cardiac silhouette in the dog should be done in the context of all Roentgen signs, including size, shape, margination, location, number and opacity should be used

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Radiographic Evaluation Of The Vomiting Patient

INTRODUCTION: *There are many indications for abdominal imaging in the veterinary patient. In the vomiting patient, abdominal radiography and abdominal ultrasound are commonly performed, especially in cases of potential gastrointestinal obstruction where surgery may be indicated. Abdominal radiography is an excellent screening test, readily available, inexpensive and rapid to perform. However, many radiographic findings can be non-specific, and the presence of moderate to severe peritoneal and retroperitoneal effusion can decrease the diagnostic value of abdominal radiography. It can sometimes been difficult to reach a diagnosis of mechanical obstruction and decide on surgical intervention based on radiography alone.*

PERITONEAL AND RETROPERITONEAL SEROSAL MARGIN DETAIL

Serosal margin detail, or the ability to define the margins of abdominal organs on the radiograph, is dependent on the amount of fat in the peritoneal and retroperitonal spaces. On initial radiographic evaluation, serosal margin detail should be assessed. Specifically, evaluate the ability to visualize the serosal margins of the abdominal organs, and the amount of fat in the abdomen. Each patient should be treated individually, as there is significant variation in the amount of peritoneal and retroperitoneal fat, and brown fat in young patients does not provide the same degree of contrast as yellow fat in more mature patients. In patients with moderate peritoneal and retroperitoneal fat, the margins of abdominal organs should be easy to delineate. If not, consider the presence of fluid. Free peritoneal fluid could be blood, pus, water, cellular (malignant), urine, chyle or bile. Thin or emaciated patients will have poor serosal margin detail; detecting free peritoneal fluid in these patients can be challenging. Be sure to also consider the contour of the abdominal body wall in these patients. If an emaciated patient with poor abdominal serosal detail has a rounded, distended abdominal body wall, consider the possibility of free peritoneal fluid.

Radiography can be an excellent test for the detection of free peritoneal gas. The identification of gas lucencies that cannot be reliably localized to the lumen of an intestinal segment is highly suggestive of free peritoneal gas. Additionally, the ability to visualize the peritoneal surface of the diaphragm, or the impression that serosal margin detail is increased in some areas can further support a conclusion of free peritoneal gas. Barring recent abdominal surgery or full-thickness body wall trauma, the primary differential diagnosis for free peritoneal gas is a rupture in the gastrointestinal tract. This can be a complication of a chronic foreign body or mural lesion.

MECHANICAL OBSTRUCTION

Radiography has long been the mainstay of diagnostic imaging. With the introduction and rapid advancement of digital imaging, radiography has become even more efficient, and allows a clinician to obtain opinions from radiologists, internists and surgeons with a few mouse clicks. Fundamentally, image interpretation is

based on thorough evaluation of Roentgen signs: ***location***, ***size***, ***shape***, ***number*** and ***opacity***. Evaluation of *location* refers not only to changes in organ location but also *lesion location and distribution* (*focal, multifocal, diffuse*). Determination of *size* is, in some cases, subjective, but objective measurements for some organs have been published. *Shape, contour and margination* of an organ or structure are also important in evaluation of pathology (*rounded, irregular, smooth*). While *number* of organs is relatively constant, the number of lesions (one, two, multiple) along with their *distribution* can be used in concert to arrive at a more narrow list of differentials. Most tissues in the abdomen will have *soft tissue opacity*. Relative differences in the soft tissue opacity of organs are often related to physical density, or thickness. *Fat* opacity is responsible for the contrast available in the abdominal cavity. The basis for interpretation is recognizing when an organ deviates from its expected normal appearance. Roentgen signs provide an organized, systematic method to evaluate an organ for normalcy, and to decide exactly how it has become abnormal.

Evaluation of the gastrointestinal tract for evidence of mechanical obstruction has been extensively studied. The primary radiographic finding associated with GI obstruction is small intestinal dilation, which manifests as in increase in small intestinal diameter, and is commonly referred to as ileus. In the context of Roentgen signs, it is also important to evaluate the extent and distribution of small intestinal distention. The presence of focal or segmental ileus is most commonly associated with mechanical obstruction. Diffuse or generalized ileus is most commonly associated with functional disease.

The most commonly used tool for measurement of small intestinal diameter in dogs is the comparison of intestinal diameter to the height of the mid body of L5 on the lateral projection. Normal intestinal diameter should be no greater than 1.6 times the height of the mid body of L5. Intestinal diameter that is greater than this suggests the presence of ileus.[1] However, this imaging test can be associated with a significant number of false positive diagnoses of mechanical obstruction, and care must be used to interpret this finding in the context of other radiographic features that suggest mechanical obstruction. Additional radiographic features that should be assessed include the presence of sharp, hairpin turns (contour, shape); a gravel sign (opacity); and the distinct presence of two, discrete populations of bowel – normal diameter and abnormal diameter (distribution). In cats, intestinal diameter greater than 12 mm is considered a sign of pathologic dilation. This measurement is made from serosal to serosa, as determination of the serosal margin is difficult due to fluid contents.

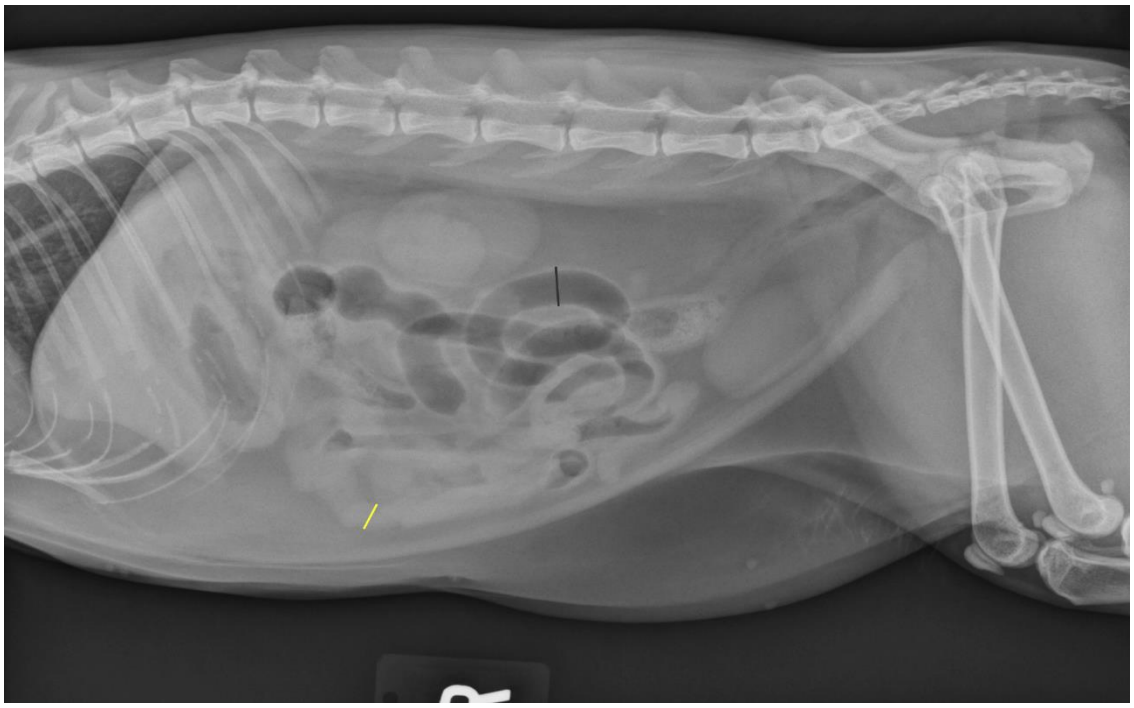

FIG.1 *Lateral projection of the abdomen of a cat showing the measurements of the bowel segments. The gas filled segment is mildly larger than the fluid filled sements, but still measures within normal limits.*

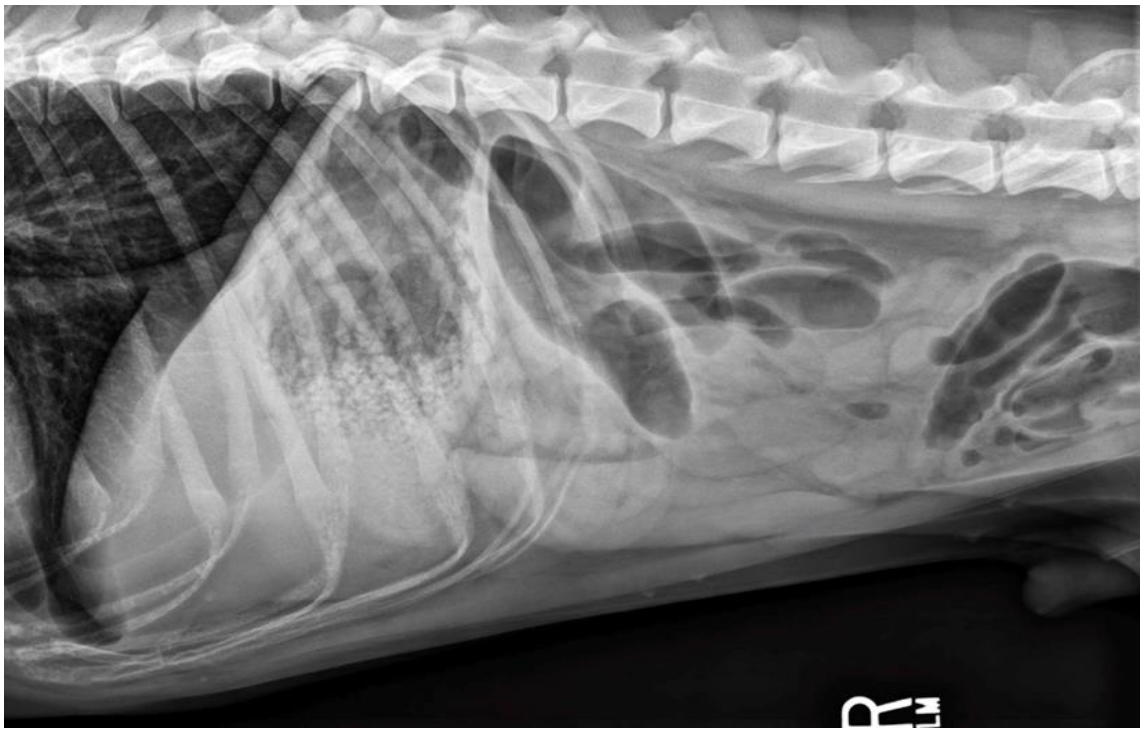

FIG.2 *Right lateral projection of a dog that is vomiting. Note that there are two distinct populations of bowel.*

More recently, it has been suggested that the ratio of largest small intestinal diameter to smallest small intestinal diameter should be used to more accurately assess for the presence of mechanical obstruction. A ratio of 2.4 or less is considered normal, while a ratio of 3.4 or greater is highly suggestive of mechanical obstruction. Clearly this results in a wide grey zone that may be difficult to interpret. Therefore, it is still important to assess additional radiographic features that can assist in making a diagnosis of mechanical obstruction. Finally, when present, a well-visualized foreign body is always helpful! However, without evidence of obstruction (dilation/ileus), a foreign body may pass, and surgery may not be required. Linear foreign bodies can be a challenging radiographic diagnosis. The position, location and distribution of small intestinal segments that contain a linear foreign body are frequently altered, classically resulting in GI plication. However, GI plication can be difficult to identify radiographically, resulting in many false negative diagnoses based solely on radiography. The addition of positive contrast media or the use of alternative imaging such as ultrasound may help confirm this diagnosis.

Intestinal wall thickening is not reliably assessed with plain radiography. GI contents are soft tissue opaque, and border effaces the mucosal surface, leading to an erroneous observation that the GI wall is thickened. The administration of positive contrast media is required to identify the mucosal surface and to accurately diagnose mural thickening. Larger mural lesions can be detected as a mass, but it may not be possible to identify an intestinal mass lesion definitively, especially if there is concomitant peritoneal effusion.

PANCREATITIS

The canine pancreas is normally not visible. Abnormalities in pancreatic size may cause displacement of surrounding organs, specifically the descending duodenum, the pylorus and the transverse colon. In obese cats, the left limb of the pancreas may be visible in the triangle of fat surrounded by the gastric fundus, the spleen and the left kidney.

In canine pancreatitis, the peritoneal serosal margin detail may be reduced focally, in the right cranial abdominal region, due to regional peritonitis and/or steatitis. If the pancreas is enlarged, displacement of the nearby organs may also be seen. Enlargement of the right pancreatic limb may result in lateral displacement of the duodenum, with medial displacement of the cecum and ascending colon. Enlargment of the pancreatic body may result in widening of the pyloroduodenal angle. Enlargement of the left pancreatic limb may result in caudal displacement of the transverse colon.

In feline pancreatitis, these radiographic changes occur less commonly. For both dogs and cats, pancreatitis can be present in the absence of radiographic or ultrsasonographic findings.

CONCLUSION

Radiography is an excellent screening test for gastrointestinal obstruction. Specifically, it is fast, and allows for a multitude of diseases to be assessed. It is often used in conjunction with abdominal ultrasonography, which is appropriate. These two imaging tests are complimentary; ultrasound should not be used as the sole imaging study in a vomiting patient.

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Sonographic Evaluation Of The Vomiting Patient

INTRODUCTION: *Abdominal ultrasound (AUS) is a readily available, non-invasive cross-sectional imaging modality that complements radiography. AUS has become more readily available and less expensive in veterinary medicine. With the increased availability of AUS, the role of abdominal radiography often comes into question. However, it is important to remember that there is a very direct relationship between the accuracy and value of AUS and operator experience. It requires years of experience to obtain and accurately interpret diagnostic AUS. There are multiple pitfalls and artifacts, therefore the advantages and disadvantages of this imaging modality when compared to abdominal radiography need to be completely understood. Prior to performing AUS, specific questions should be asked of the abdominal ultrasound examination based on the physical examination, clinical history and other laboratory data.*

Computed Tomography (CT) is another cross-sectional imaging modality that is steadily becoming more available in veterinary practice. CT uses x-rays to create thin slice images that, like AUS, eliminates superimposition. With ever increasing speed, spatial resolution, and the ability to create muliplanar reformatted images, CT can replace abdominal radiography and ultrasonography in some applications.

For the purposes of this session, we will focus on the utility of radiography, ultrasound and computed tomography for the diagnosis of gastrointestinal obstruction in the vomiting patient.

OBSTRUCTIVE DISEASES

Abdominal ultrasound (AUS) has long been used to evaluate patients in which radiography is inconclusive or in cases where additional information regarding the possibility of neoplasia may be desired. Where abdominal radiography provides contrast resolution that is limited to gas, fat, soft tissue, bone and metal, AUS provides a greater degree of contrast resolution that is based on acoustic impedance, which determines echogenicity. This increase in contrast resolution allows for discrimination of GI layers, and changes in acoustic impedance/echogenicity often accompany a multitude of disease processes. Many of these changes can be very non-specific, but when interpreted in conjunction with abdominal radiography and in the context of clinical history and suspicion, diagnostic accuracy increases.

The mucosal and the serosal margins of GI segments can be reliably identified and characterized using AUS. This allows resolution of both the wall and the lumen as separate, distinct regions. Luminal dilation of small intestinal segments <1.5 cm is a useful discriminatory finding for the diagnosis of moderate to severe distention. When this degree of intestinal distention is observed, a search for a cause of mechanical obstruction should be initiated.[3] Foreign bodies have a relatively characteristic appearance sonographically, showing a significant degree of distal acoustic shadowing and surface reflection that hints at their shape.

Due to the aforementioned superior contrast resolution, mural lesions are identified better with AUS than with radiography. Assessment of intestinal wall thickness as well as the presence, alteration or absence of intestinal layering can be accurately performed with ultrasound, and can provide insight into underlying etiology of mural lesions. In dogs, the presence of intestinal wall thickening and loss of layering is 50.1 times more likely to result from neoplasia than from enteritis. Intestinal wall thickening and loss of layering can also be seen with oomycoses such as Pythium and Lagenidium. The presence of gastric wall thickening along with pseudolayering is highly correlated with gastric epithelial neoplasia. And gastric wall thickening in conjunction with concave mural defects and gas dissection is correlated with gastric ulceration. Alterations in individual layer thickness and/or echogenicity can also be evaluated with AUS. The presence of linear striations in the intestinal mucosa has been correlated with lacteal dilation in the dog. The presence of a mucosal stripe in cats has been associated with fibrosis, possibily related to inflammatory bowel disease. And the identification of muscularis layer thickening in cats has been linked to the diagnosis of intestinal lymphoma.

In patients with peritoneal effusion, AUS will allow visualization of structures that cannot be seen with radiography. In fact, AUS can be indispensable in guiding sampling of effusion, and of abnormal organs. In addition, AUS has been reported to be accurate in identifying free peritoneal gas, and some consider it to be more sensitive than radiography.

COMPUTED TOMOGRAPHY

Recently, the utility of computed tomography (CT) in the evaluation of acute abdomen in dogs and cats has been reported with increasing frequency. While still in relative infancy for the diagnosis of intestinal obstruction, CT has been shown to be more accurate, faster and better for surgical planning compared to US in a population of dogs with suspected intestinal obstruction. CT was as accurate as US in the identification of intestinal foreign bodies, with equal sensitivity and slightly lower specificity. In addition, CT has been show to more accurately identify the location of an intestinal lesion, and can be used to identify plication associated with linear foreign bodies. Further, depending on equipment, CT can be far more rapid, especially if performed under sedation. Due to the tremendous number of images created in a CT dataset, interpretation time can be increased, and when considered together, study and interpretation time for CT and US are similar.

PANCREATITIS

Ultrasonography can also be useful in the diagnosis of pancreatitis. Ultrasound often allows direct visualization of all or part of the pancreas; occasionally gas in the stomach and/or transverse colon obscures portions of the left pancreatic limb and pancreatic body. In addition, gas in the ascending colon and/or duodenum can obscure the right pancreatic limb.

In acute forms pancreatitis, the affected region of the pancreas is hypoechoic, reflecting edema associated with inflammation. Fluid may also accumulate around the pancreas as well as within the peripancreatic fat. The surrounding fat is typically hyperechoic, and hyperattenuating, which can interfere with the ability to evaluate the regional structures, including the pancreas. Due to the local inflammation, the duodenum may be gas filled, or may appear corrugated.

This corrugation reflects the irritation of the of duodenum due to regional peritonitis.

The sensitivity, specificity and accuracy of ultrasound in the evaluation of pancreatitis has not been recently assessed. Earlier studies suggest that ultrasound has a sensitivity of approximately 66% for the detection of pancreatitis. While advances in ultrasound technology may have increased the sensitivity of ultrasound detection of pancreatitis, it must be noted that some cases of pancreatitis will not have sonographic abnormalities, and other tests should be used to confirm this diagnosis.

CONCLUSION

The arsenal of imaging modalities available for imaging gastrointestinal disease is expanding. With advancements in technology, the cost of these modalities has decreased, and the frequency of their use in veterinary practice is increasing. Understanding the strengths and limitations of each in the context of clinical diagnosis is important to inform proper decision-making for your patient.

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Índice

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1.5.1. Update on estrus synchronization 126

Update on estrus synchronization

INTRODUCTION*: Various estrus synchronization protocols began to be developed and proposed in the 1990's and since then a variety of protocols have been devised for beef and dairy cattle. The reason for so many different protocols lies on the intent to maximize their efficiency according to their specific use. For example, some are designed for beef heifers; others are designed for breeding by appointment or breeding that require estrus observation. The intent of this discussion is not to present all existing synchronization protocols for cattle but to list a few basic protocols and their rationale. Once we understand the basic pharmacology of commercially available drugs and the concepts of reproductive physiology, it becomes easier to understand them and even to propose some modifications depending of the availability of pharmaceutical hormones in your region and the needs of your clients. The protocols discussed below are example of those that preclude the need for estrus detection.*

OVSYNCH

Although PGF2α and GnRH (gonadotropin-releasing hormone) had already been used for estrus synchronization for several years, it was not until 1995 when a publication (Synchronization of ovulation in dairy cows using PGF2α and GnRH. Pursley JR, Mee MO, Wiltbank MC. Theriogenology 1995 44(7):915-23) described the first synchronization protocol for timed AI (breeding by appointment). That event sparked a series of investigations and publications describing a number of protocol modifications. Until 1995, PGF2α was the main hormone used to synchronize cows but PGF2α only affects the luteal function and not the development of follicle waves. The ability to induce or manipulate follicle waves is one of key events in ensuring success of synchronization protocols and fertility outcome.

OvSynch basically consisted of:

- An initial administration of an intramuscular injection of GnRH (100µg) at a random stage of the estrous cycle. This GnRH treatment will trigger ovulation of a dominant follicle and subsequently induce a new follicular wave. The very first follicle wave begins ~ 1 day after ovulation in cattle.
- Exactly a week later, an injection of PGF2α is administered to induce the regression of any corpus luteum that might be present.
  - Some studies have shown that some cows may benefit from a second PGF2α given 24 hours after the first PGF2α injection.
- Forty-eight hours following the PGF2α administration, another injection of GnRH is given to trigger ovulation of preovulatory size follicles that are expected to be present after the previous PGF2α injection.
  - Greater conception rates have been show by postponing this GnRH until 56 hours

- Artificial insemination is then performed at 12 to 24 hours following this last GnRH injection

  - Artificial inseminations are done without heat detection

PROTOCOL MODIFICATIONS

CIDR (controlled internal drug release)

- CIDRs are an intravaginal device made of nylon coated with a silicone coat that is impregnated with progesterone. Opposed to the action of PGF2α, a CIDR device mimics the action of a corpus luteum and its withdrawal resembles a rapid luteolysis.

- A CIDR may be inserted in cows at the beginning of a OvSynch protocol and removed a week later at the time of the PGF2α injection

PRE-SYNCH

- Because merely exposing cows or heifers to the OvSynch protocol may not guarantee that a new follicle will be initiated, it has been shown that adding a pre-synchronization scheme will improve ovulation synchronization and subsequent fertility (pregnancy per AI).
- Pre-sycnhronization is typically done with 2 injections of PGF2α 14 days apart.
  - Then, OvSynch can start 14 days following the second PGF2α
  - To maximize conception rates, OvSynch may be started 11 or 12 days following the last pre-synchronization PGF2α injection

Double-OvSynch

- It has been proposed that cows with inactive ovaries may benefit from using receiving a double OvSynch protocol, with a week interval between the two OvSynch.
- Timed AI is done at the second OvSynch.
- Cows with already cycling ovaries are not affected by this protocol.

MODIFICATIONS FOR BEEF CATTLE

Several modifications have been proposed for beef cattle, especially to decrease costs associated with labor. For example, when using the OvSynch protocol with CIDR, instead having the last GnRH injection given before timed AI, GnRH is given at the time of AI, typically 60-66 hours following the last PGF2α injection.

- When GnRH is given at the time of AI, the modified protocol is termed CO-Synch
- When CIDR is added to the CO-Synch protocol, the modified protocol is termed CO-Synch + CIDR
- A 5-day CO-Synch + CIDR has been proposed for beef cattle
  - At CIDR removal, a first injection is given followed by another PGF2α injection ~ 8 hours later
  - AI is performed at 72 hours after CIDR removal and first PGF2α injection
    - GnRH is still given at AI
- For ***Bos indicus*** cows, another PGF2α injection has been recommended to be given along with CIDR i*nsertion* and the first GnRH injection
  - AI is to be performed at 66 hours post CIDR removal and not at 72 hours as it is done for ***Bos Taurus*** breeds

Clinical Management Of Accidents Of Gestation In Cattle

FETAL MUMMIFICATION

Background

The overall incidence of fetal mummification varies from 0.43 to 1.8% of pregnancies but it can be as high as 3-4% in feedlot heifers due to incomplete regression of corpus luteum following induced abortion. Mummification of the fetus can only occur after calcification of the fetal skeleton, therefore, fetal death followed by mummification may occur anywhere from 3rd to 8th month of gestation; most cases occur during 4-6 months. Causes may include torsion of the umbilical cord, Campylobacter fetus, fungal infection, leptospirosis, Infectious Bovine Rhinotracheitis and Bovine Viral Diarrhea, etc. These infectious agents have in common the fact they do not induce placentitis and endometrial release of prostaglandin F2alpha; and are not pyogenic. Fetal mummification may be seen in ewes that become infected with toxoplasmosis during early gestation.

Pathogenesis and Clinical Signs

In mummification, fetal death occurs without luteolysis and adequate cervical dilation. It results in autolysis and fluid resorption in a non-pyogenic environment; fetus & membranes become dehydrated, resulting in a dark brown, leathery fetus with shrunken dried skin and bones. There is no odor or exudate present. Cows affected with mummified fetuses do not show signs of estrus because there is a persistent corpus luteum that not only keeps the cervix closed but also prevents signs of estrus, thus, mummified fetuses may be retained indefinitely. Spontaneous expulsion of mummified fetuses seldom occurs; affected cows typically have a history of failing to calve on time.

Treatment

The treatment of choice is PGF2alpha (25 mg dinoprost i.m); expulsion of mummified fetus is expected in 2 to 4 days. The breeding prognosis good; cows typically conceive in 1 to 3 months following expulsion of fetus. Surgical removal is indicated if fetus is not expelled after therapy with PGF2alpha.

FETAL MACERATION

Background and Pathogenesis

The incidence of fetal maceration is 0.09% of pregnancies. Fetal death followed by maceration may occur at any stage of gestation. If it occurs prior to calcification, the fetus will decompose in fetal tissue debris contained in a purulent liquid material. If fetal demise occurs after fetal calcification, and bacteria from the caudal reproductive tract gain access to the uterus, it will result in bacterial decomposition of autolyzing fetus and membranes. Clinically, there will be a compact mass of bones in a collection of purulent material. For these reasons, fetal maceration with a closed cervix and a functional corpus luteum is rare.

Clinical Signs

Usually a chronic, fetid reddish-gray watery or mucopurulent discharge from the vulva is seen over a period of several weeks to months. In some cases, there may be toxic metritis early but systemic illness is typically absent in later stages. Cows with macerated fetus may experience gradual weight loss and decline in milk production.

Treatment

No satisfactory treatment is available. Poor breeding prognosis due to severe endometrial damage. Surgical removal can be attempted if warranted by the animal's value but it is often a frustratingly unrewarding effort.

FETAL EMPHYSEMA

Similar to fetal maceration in that putrefactive bacteria invade the uterus through an open cervix.It is often detected in later term pregnant animals. Fetal death may be associated with dystocia or incomplete abortion in late gestation. Gross fetal changes include putrefaction, distension with fetid gases, crepitation, dry hair and coat secondary to extensive fluid loss and fetal dehydration. Dystocia involving a fetal emphysema is a complicated and grave condition that is commonly fatal to the dam. In ewes, Clostridium chauvoei may be involved; usually poor prognosis.

HYDRAMNIOS (HYDROPS OF THE AMNION)

Background and Pathogenesis

Normal amnion: The amniotic fluid is clear, colorless, and mucoid in nature. Under normal conditions, the volume of the amniotic compartment is regulated by the fetal swallowing. In early to mid-gestation the amniotic fluid is watery; in late gestation, the fetal bladder sphincter prevents urine outflow and the amniotic fluid becomes more viscid. The accentuated mucoid nature is owing to saliva and secretions of the nasopharynx from fetus.

Hydramnios is a relatively uncommon condition caused by autosomal recessive genes characterized by an abnormal accumulation of amniotic fluid. Pathologically the amount of amniotic fluid greatly increases up to 8-10 times (25-150 liters in cows).Its incidence is only 5-10% of uterine dropsy cases. The condition is associated with a genetic or congenitally defective fetus that has impaired swallowing. The increase of amniotic fluid is gradual. Hydramnios is seen most commonly in cattle, and occasionally in sheep.

Clinical Signs and Prognosis

Hydramnios is characterized by a gradual enlargement or filling of the amniotic cavity over several months during latter half of gestation. The gradual abdominal enlargement lasts leads to a pear-shaped abdomen when the cow is seen from her rear. It is often not noticed until parturition, when a large volume syrupy, viscous, meconium stained fluid is released during calving. In examining cows suspected to having hydrops, it would be important to differentiate hydrops of the amnion or the allantois compartment. In cows affected with hydramnions, the placentomes, and often fetus can be palpated because the chorioallantois, amnion and placentomes are normal. Dystocia is common due to uterine inertia and defective/abnormal

fetus. Retention of placenta is a common sequela; milk production in subsequent lactation is generally poor.

- The prognosis for future breeding life of the dam is good, but the fetus is invariably defective and nonviable.

HYDRALLANTOIS (HYDROPS OF THE ALLANTOIS)

Background and Pathogenesis

Normal allantois: The allantois fluid is clear, watery, and amber colored. There is only a small amount of allantois fluid produced from the allantois epithelium prior to functional fetal kidneys. The allantois cavity stores fetal urine delivered through the umbilical cord via urachus. In late gestation and under normal conditions, the volume of fluid may reach 8 to 15 liters.

Hydrallantois is a much more common hydrops condition than hydramnios (85-90% of hydrops conditions); both beef and dairy cattle are affected. In hydrallantois, fluid accumulation may reach 50 to 200 liters. Excessive fluid has specific gravity and characteristics of a transudate due to vascular disturbance occurring in allantois. It is generally characterized by a diseased uterus with many non-functional caruncles and some placentomes greatly enlarged. Adventitial placentation is common, with portions that are necrotic and edematous. Cystic kidneys, hydronephrosis and dysfunction of the fetal tubules with resultant polyuria are seldom involved in pathogenesis of hydrallantois. It generally affects cows > 3 years, unless heifers are affected with congenital caruncle deficit. In older cows caruncle deficit may reflect prior uterine infection or injudicious removal of fetus and/or retained membranes leading to a defective endometrium and caruncles.

Clinical Signs and Prognosis

Occasionally develops as early as 5th month of gestation in severe cases. It usually develops rapidly over 1 to 3 weeks during late gestation; the distended uterus fills a tense, barrel-shaped abdomen (bilateral abdominal distension). The resulting marked abdominal enlargement leads farmers to question breeding dates or suspicion of triplets! Eventually, the abdominal distension leads to digestive symptoms such as anorexia, decreased ruminations and ultimately constipation. Hydrallantois can be misdiagnosed as indigestion, bloat, or traumatic gastritis. The pulse is elevated (90-140/min), accompanied by expiratory grunt.

Reproductive examination: the excessive fluid cause the uterus to palpate greatly distended and tense; the distension in the allantois compartment precludes the palpation of placentomes or fetus. Complications include uterine rupture, rupture of pre-pubic tendon, and ventral hernia. In mild cases, hydrallantois may not be diagnosed until term, when an excessive clear, watery, amber fluid with characteristics of transudate is passed during calving. A greatly enlarged and atonic uterus may cause dystocia. Fetuses are usually slightly smaller than normal and present with edema and ascites. Fetal membranes may be tough and difficult to rupture. The fetus is generally dead at birth or dies soon after. Fetal membranes may be heavy and edematous, and retained fetal membranes and septic metritis are common sequelae. These complications account for the relative high morbidity and mortality of hydrallantois. The prognosis is therefore guarded for life and fertility. Salvage by slaughter is the best option for most producers. Milk production in subsequent lactation is generally poor.

TREATMENT OF HYDRAMNIOS AND HYDRALLANTOIS

Varies with duration and severity of condition. In severe cases, an early decision to salvage is best while affected cows are still in good physical condition. Alternately, prompt termination of pregnancy is desirable and the best approach. Induction of parturition/abortion in affected cows can be achieved by administering 20 mg dexamethasone and 30 mg of PGF2alpha that result in cervical dilation and abortion within 24 to 48hrs. Inducing abortion is more successful with hydramnios. Dystocia can occur in association with defective fetus (hydramnios) and uterine inertia secondary to uterine distension (hydrallantois). Weak abdominal muscles and absence of strong abdominal pressure are common; cervical dilation are often incomplete. A trochar or plastic tube can be used to draw fluid off slowly over 24 hours prior to Cesarean section. Rapid removal of large volume of fluid may induce shock. Appropriate fluid therapy in large volumes indicated before, during, and after surgery. When terminating hydrallantois by Cesarean section, the uterus may continue to fill with transudate for about 48 hours and it may require further draining. Retention of fetal membranes and secondary metritis is common; treat early with local and parenteral antibiotics and oxytocin to aid in continuing evacuation of the uterus.

Table 1. Hydrallantois and hydramnios

Characteristic	Hydrallantois	Hydramnios
Prevalence	85–95%	5–15%
Rate of development	Rapid (within 1 month)	Slow over several months
Shape of abdomen	(Bilaterally) round and tense	(unilaterally) pear-shaped, not tense
Palpation (per rectum) of placentomes and fetus	Nonpalpable (tense uterus)	Palpable
Gross characteristics of fluid	Watery, clear, amber-colored transudate	Viscid, may contain meconium
Fetus	Small, normal	Grossly abnormal
Placenta	Adventitious	Normal
Refilling after trocharization	Rapid	Does not occur
Occurrence of complications	Common	Uncommon
Outcome	Abortion or maternal death common	Parturition at approximately full term

Adapted from: M. Drost. Complications during gestation in the cow. Theriogenology 2007;68:487-491;
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UTERINE TORSION

Background and Pathogenesis

Uterine torsion is most common in dairy cows, but occasionally seen in beef cows, sheep and goats. The etiology involves anatomy, manner of lying down, and maybe sudden falls or rolling. The lesser curvature of uterus in late gestation is supported dorso-laterally by the broad ligament. The greater curvature lies free in abdominal cavity resting on abdominal floor, supported by rumen, the viscera, and abdominal walls. In ruminants the gravid horn is in the shape of an arc or a U-shaped loop with the vagina and ovary at the respective ends of the arc. The ovarian end of the gravid horn forms a narrow base upon which the uterus rests. Torsion involves the rotation of this arc on its transverse axis, and involves both gravid and non-gravid uterine horns. Each time cow lies down or rises, the gravid uterus is suspended in the abdominal cavity, and a sudden slip or fall could cause torsion (down front first; up back first). An increased incidence is seen in cows subjected to stall confinement in the winter. Torsion of the gravid uterus occurs more frequent in pluriparous than primiparous animals; it generally occurs in advanced pregnancy. Most torsions occur in late first stage or early second stage labor and may be associated with strong movements of the fetus. Uterine torsions <180º may be present for days or weeks without clinical symptoms until labor begins and dystocia results.

Clinical Signs and Prognosis

Usually history of prolonged 1st stage of labor (i.e. restless, colic behavior); abdominal straining is absent, or mild and intermittent as fetus cannot enter into birth canal and initiate the cervical dilation and contractions (Ferguson Reflex). Torsions or rotations of the uterus at 45 to 90º are often found during pregnancy and generally correct themselves. Unusual cases involve 180 to 360º torsions that leads to obstruction of blood supply to uterus → congestion, edema, shock, and may be gangrene of uterus. Under these conditions, fetal death is unavoidable.

Reproductive Examination: Diagnosis of uterine torsion and its direction is done via palpation per rectum. The amount of tension on the broad ligaments and arteries indicates severity of torsion. Vaginal walls spiral and a stenosis of the vagina is present but in pre-cervical torsions there may be no vaginal involvement. In ~ 75% of the cases, the cranial portion of the vagina will show a characteristic twisting of the vaginal folds. Torsions may be clockwise or counter-clockwise. Left torsions are more common than right. A left torsion means that the right uterine (gravid) horn moved to the left side. In most cases, the position of the fetus will be dorso-pubic in 180º torsions. Prognosis in torsions prior to term depends upon duration and degree of torsion, and severity of symptoms. If torsions > 180º are diagnosed and treated early, prognosis for dam and fetus are good. Prior to term, best methods of correction are rolling or via laparotomy. Complications include uterine rupture and hemorrhage from ruptured vessels. Only rarely does torsion recur in the subsequent pregnancy.

TREATMENT OF UTERINE TORSION

A. Rolling the Dam
- Disadvantage is manpower (3-6 needed).

- Caste cow in lateral recumbency onto side of torsion.
- Tie both hind limbs and both forelimbs together, leaving 8-10 foot of rope free for pulling.
- Hold head extended with halter.
- Rotate cow rapidly onto opposite side → body overtakes the more slowly rotating gravid uterus.
- If successful, the spiral folds and stenosis of birth canal will have disappeared. If cervix dilated, fetus may be palpated with ease and there may be a rush of fetal fluids.
- May require 2 or 3 rapid rotations to succeed.
- Schafer's method requires less assistance as the cow is rolled slowly with a plank holding uterus stationary.
- Cow caste on side of torsion.
- One end of a 3-4 meters plank (20-30 cm wide) placed over cow's para-lumbar fossa and assistant stands on plank.

B. Rotation of fetus and uterus per vagina

- Only possible if cow at term & has a partially dilatedcervix.
- Must be able to introduce hand into uterus to grasp fetus – access depends on severity of twist.
- Rupture membranes first to release fluids → reduce size and weight of the uterus.
- Rock back and forth → momentum → vigorous twist in opposite direction to torsion.
- Detorsion rod – 1 cm steel rod, 80-100 cm long with eye at either end.
- Loop passed over one fetal limb and loop on other side of rod passed over 2nd limb.
- Aim to have loops just above fetlocks.
- Use short broom handle or large screw driver through eye in rod and wrap chain tightly around it.

C. Laparotomy

- Useful earlier in gestation, or when cervix closed.
- Try rolling first if assistance available.
- Open on side of torsion and pass hand down between uterus and abdominal wall and grasp fetal limb. Rock up and down → momentum → lift in direction opposite to torsion.

D. Cesarean Section

- Indicated when other methods fail or when the cervix not adequately dilated.
- In dystocia, cervix may have undergone constriction and emphysematous fetus now present.
- Generally cervix only partially dilated due to atony of cervix and uterus(circulatory disturbance).

E. Complications/Sequelae

- Uterine rupture with peritonitis - Internal hemorrhage - Retained fetal membranes - Septic metritis.

Índice

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The 5 Most Important Things You Must Do During Every Appointment

USING COMMUNICATION TO DRIVE COMPLIANCE AND ADHERENCE

We see our patients once or twice a year on average. We must develop systems to maximize our time with clients to ensure that pet owners understand what services and products their pet needs to optimize health and well-being. The foundation of communication is the physical examination. It is important that we develop a step-by-step, systematic approach to the physical examination to ensure consistency and effectiveness. This session will review the critical touchstones that drive compliance during the physical examination.

Performing a physical examination is a part of nearly every patient interaction in a veterinary hospital. Too often we fail to recognize the vital role this service plays in creating value and confidence in our services. Further, how we perform and communicate our physical examination plays a large role in our compliance with recommended diagnostic and treatment offerings. While every doctor will have his or her own style and routine, it is important that the examination be methodical, efficient and conveys thoroughness and compassion to the client while keeping the pet as comfortable as possible.

Your Entrance

I believe the ideal physical examination begins with a warm entrance and greeting of the client and patient by names. I have all of our staff greet by shaking palms and paws. We then sit down and the technician reviews the pertinent medical history she has previously obtained. The doctor then asks any additional questions and we begin the examination.

THE 5 CHIEF COMPONENTS OF THE PHYSICAL EXAMINATION

1. General Condition, Ears, Eyes, and Nares

The technician gently lifts the pet onto the exam table. If the pet is large (over 30-40 pounds), we typically perform the examination on the floor where a larger dog is often more comfortable.

I start by performing a body condition scoring (BCS) and assessing the pet's general appearance. I make comments regarding the coat, stature, weight and overall state of appearance. I then move to examining the ears, eyes and nares and work my way back, articulating each part of the exam. It is very important that you explain each step and point out any abnormalities as you discover them. I start the examination with the pet facing me to gain better access to the head and neck. It is important that you have a logical examination flow that incorporates natural transition points. Transition points are opportunities to change orientation naturally so you appear coordinated and rehearsed. Without a well-thought plan of execution, your examination may appear haphazard and incomplete. Further, by training a coordinating with your staff, you avoid embarrassing gaffes such as bumping into each other or having to ask them to turn the pet back to you because you failed

to complete an aspect of your examination. Whatever your desired flow, you should discuss and role-play it with your staff so that everyone is on-board and understands your methods.

I like to dim the lights as we start the exam by looking into the ears and eyes. This helps me in my examination and reinforces the seriousness of the examination to the client. Dimming the lights is a natural transition point in that it takes the client from the previous ten or twelve minutes to talking and discussing their pet's medical history to the action of performing the examination. We then create another natural transition point when we turn the room lights on again.

I recommend detailing your findings verbally whenever a client cannot see what you're examining. This serves not only the client but the staff member assisting you as well. When your staff is involved in the examination, they can help you complete the physical examination report. While we use a video-otoscope in diseased ears and nares, for most routine examinations we use traditional otoscopes. Describe the retina, lens, conjunctiva, tympanum, nares and other pertinent structures. This reinforces to the client that you are performing a thorough and complete examination and are capable and competent. I use an ophthalmoscope followed by a halogen penlight to test pupillary light responses. After describing my findings, I use the penlight to illuminate the nares to assess patency, architecture, and the presence of any discharge or other abnormalities.

2. Oral Exam

I then turn the room lights on as I begin the oral exam. Because turning the lights on is a visually dramatic step, it also helps change the level of involvement from the client. I ask the client to stand up (out of their chair) and come close to the exam table and pet to show them any problem areas. I want them to see and smell any periodontal disease. Many clients with pets that have advanced periodontal disease will not routinely look into their pet's mouth. Many will actually avoid the oral cavity altogether due to malodor or unsightly appearance. I believe it is important to confront periodontal disease directly by having the client interact and see the issue(s). This doesn't mean we should make pour clients feel guilty; rather, it means we should be thorough and demonstrate any areas of improvement whenever possible. Point out gingivitis, calculus, bleeding or inflamed gums, loose teeth and recessed gums. Have the client smell the breath and discuss the cause of the offensive odor (pathogenic bacteria). If the teeth and gums look healthy, take this opportunity to congratulate the client on their efforts. By actively engaging and interacting with clients, you're more likely to impress upon them the seriousness of the condition. If a client remains physically distant and uninvolved from the oral cavity, it is much easier for them to ignore our recommendations for dentistry. If a veterinarian remains distant and uninterested, the client will assume that his or her own ignorance is justified. Make sure you lift the lip and take a whiff with each patient and client.

It is imperative to make the connection between oral health and systemic diseases such as endocarditis. Too many clients view dentistry as merely a cosmetic procedure and see no value in keeping their dog's teeth "Hollywood-white." It is our professional obligation to make sure clients understand the relationship between the mouth and general health and well-being. If you note loose teeth, discuss pain. If you see gingivitis, remark about gum recession, tooth root exposure and the potential to

adversely affect adjacent teeth. It is important to keep in mind that few clients thoroughly examine their pet's oral cavity at home. Take this opportunity to lift the lip and educate your clients on the dangers of periodontal disease.

One final note: I recommend that you point out and briefly touch on an abnormal finding during the course of the examination. I recommend that you reserve the more detailed discussion for the time after your examination when you review pertinent findings. Additionally, you can also have a trained staff member give more detailed information on conditions such as periodontal disease after you exit the appointment.

3. Lymph nodes and Skin

With the pet still facing me, I then feel all the primary accessible lymph nodes, while explaining what I'm doing and why. I start with the submandibular lymph nodes and work my way caudally. I create a natural transition point for turning the pet away from me as I palpate the inguinal and popliteal lymph nodes. The assistant turns the pet and I palpate these caudal lymph nodes. I next verify if the client has observed any new lumps or bumps as I run my hands along the pet's body. I make sure to emphasize to the client to closely monitor the areas of the lymph nodes for any swellings as I feel the body. Any masses are noted and measured, and we generally take a digital photograph of any sizable or suspicious masses to be included in the pet's electronic medical record. If there are any new masses, we strongly advise the client to have a fine needle aspiration performed and submitted to the laboratory for histopathological review.

4. Thoracic Cavity, Abdomen and Hindquarters

I next auscultate the chest. I prefer to close my eyes to demonstrate my attentiveness and also as a signal to the client that it's time for quiet. Besides, closing my eyes allows me to concentrate better. I usually say to the pet and client that I'm going to feel their pulse as I listen to the heart. Again, this provides me with valuable information regarding pulse strength and quality and further enforces to the client that their pet is receiving a complete and thorough exam. After I auscultate each quadrant of the thorax, I open my eyes and briefly describe what I heard and then move to palpating the abdomen. I start in the cranial abdomen palpating the kidney(s), liver, spleen and bladder. I pay close attention the any structures that I feel are enlarged or abnormal in any respect. It is very important that you explain what you're doing and why, especially when you are engaging in seemingly incomprehensible actions. I make it a point to describe what I'm feeling as I probe the abdomen. I also comment to the patient and client if there is any tenseness or apprehension as I palpate their pet's abdomen. Some pets are more accepting of this procedure than others. If a pet is nervous or fearful, I must take great strides to calm both the patient as well as the client. If a client perceives that I am being rough or in any way less than gentle with their pet, I risk forever damaging my reputation and kind and compassionate. Often all that is required to allay even the most challenging fears is communication.

Once I've completed palpating the abdomen, I lift the tail to evaluate the rectum and hindquarters. While this is arguably the least glamorous part of the physical examination, it is critical that we are thorough. This includes the anal region. There may be an emerging perianal

adenocarcinoma that will go unnoticed until the groomer sees it in two months.

5. Musculoskeletal System

The final step in my physical examination is the evaluation of the joints, limbs and paws. With the pet still facing away from me, I lift up and examine the rear paws and begin flexing and extending the joints as I progress toward the hips. Any withdrawal or tenseness is noted. I repeat the same for the front limbs. I end by putting the head through a normal range of motion by extending and flexing the neck up, down and side-to-side.

REVIEWING THE EXAMINATION WITH THE PET OWNER

Once I've completed my exam, we return the pet to the floor, the client's lap, or in the carrier and I resume sitting on my stool. I pull close to the client, generally about 36 to 42 inches away and at a 45-dgree angle to the client. This is the ideal position for seated communication. I review my findings and outline a course of action. This is the time to discuss more fully the importance of weight reduction and oral care. I have found that by introducing a problem area during the examination and then reviewing it more formally after the procedure is complete, the client is much more receptive and attentive. It sends a clear message to the client when you take additional time and effort to revisit a particular area of interest concerning their pet's health. It also improves your communication when you're properly seated and without the distraction that a pet on the exam table may create. This discussion also reminds the assistant of topics you want them to enforce in the patient discharge. During this time the technician is filling out the physical examination report and taking notes or entering charges.

Once I've completed my review and outlined a plan, I ask the client if they have any questions and tell them we'll be right back after we read any lab tests such as fecal parasite evaluation or heartworm tests, finish their paperwork or obtain any recommended items. It's important to maintain an open body posture at this time, facing the client as you prepare to leave. If you turn to the side or begin approaching the door, you're sending a signal that you're ready to leave. Before I exit the room, I make sure to shake "palms and paws." I exit the room leaving the assistant or technician to tell the client what we're doing and then they also leave the exam room.

It is important to keep the amount of time the client spends waiting alone at this point to a minimum. One to three minutes to finalize reports, read basic lab tests, and get any products is about all it takes for most uncomplicated, routine examinations. In sick pets, the time spent performing and evaluating laboratory tests will be longer. Be sure to have a plan for dealing with patients that require radiographs, blood tests or more involved discussions.

TECHNICIAN REVIEW OF EXAMINATION FINDINGS

Up until this point, the client has had a staff member with them for the entire appointment. I like having a staff member 'velcroed' to the client because I find it is a great way to build trust, ensuring that you thoroughly educate and answer a client's questions and creating continuity of care within this appointment. It also helps reduce missed charges and increase compliance and follow-up care.

The only time we leave the client alone is when the technician and doctor leave the room to get any additional paperwork or products and complete the written discharge instructions. It is critical that you are aware of the time you leave the client alone in the room. This generally takes us one to five minutes, depending on the complexity of the case (see above).

Once I've finalized my examination report, the technician will return to the exam room and begin the discharge. We are now approximately twenty to twenty-three minutes into the appointment with seven to ten minutes to review the reports and recommendations and bill out the client.

The technician returns to the client and begins reviewing the doctor's report with the client. My strategy is to repeat my recommendations three to five times during the appointment. This offers the most repetition and increases the likelihood a client will take our recommendations seriously and act upon them. Reinforcing a message through a variety of team members will help you improve your compliance with any service or product.

The first opportunity to offer medical advice comes when the technician is obtaining the medical history. For example, if she notes that a pet needs to lose weight, this is a great time to start the discussion of diet and exercise. The next opportunity occurs when I am performing the physical examination. If I observe stage two or three periodontal disease during the exam, I will recommend a dental scaling and polishing. The third chance to offer recommendations happens when I sit down and review my examination findings. The fourth and fifth opportunities take place when the technician goes over our written discharge report and when the receptionist reviews the appointment at billing.

By strategically reinforcing the care a pet needs with the client throughout the appointment, you'll gain increased compliance and that means pets live longer, healthier lives.

Before the assistant enters the room to review the examination report, the first step is to double check the doctor's report. This will help the staff member remember to get all your charges in and make sure that there aren't any typos or other errors. If there's a problem, your staff should correct it before it reaches the client and causes embarrassment and diminished credibility. Even simple mistakes such as the wrong gender or age on an otherwise flawless report can be enough to create doubt in a client's mind. Everyone, including doctors, makes mistakes and the team can help the doctors look good to clients. Doctors may forget that the patient was prescribed a drug or follow-up care and the technician will catch that mistake and that double-check helps reduce gaps in follow-up care.

The staff member then takes a seat and starts to summarize the written report. The assistant or technician should review the physical examination and comment on the normal findings as well as the abnormal ones. It's important to point out where clients are doing well so any areas that need work don't come across as a reprimand. The staff member takes a highlighter and highlights any test results or abnormal findings and highlights any treatment or follow-up care instructions. The highlighter helps personalize the report and allows the assistant to focus the client's attention on what the client really needs to do for their pet.

TIME

In the real world, there is no set time for the amount of time obtaining a medical history, performing a physical examination, reviewing the exam report and billing will take. In general terms, we allocate thirty minutes for the entire appointment with a third of the total time allocated to 1) Welcome, medical history and lab samples, 2) Physical examination, and 3) Reviewing the exam and billing. Some pets will require a longer history while others will require fifteen minutes to perform the physical examination. The key is to know your schedule and where are you in the appointment both from a time as well as procedural perspective. Five minutes is a long time if you're staying on topic and discussing what the pet needs. Five minutes is a relatively short period if you're discussing the weather or church and then realize you've neglected the pet's obesity.

While there is no right or wrong way to perform a physical examination, I encourage you to develop a systematic approach that allows you to be as thorough as possible in as short amount of time as practical. Your clients will thank you for being through and compassionate and our patients deserve it.

5 IMPORTANT TOPICS TO ADDRESS DURING EACH EXAM

While there are literally hundreds of issues and areas we strive to address during each exam, here are five universal topics you should consider for every patient during every appointment.

1. **Preventive Care** – Vaccinations, heartworm and flea/tick preventive(s), basic hygiene
2. **Diet and Body Condition** – Thin, normal, or overweight? What food should they feed, how much, how often, and why?
3. **Behavior** – "Is there anything Scooter does that bugs you?"
4. **Life Stage and Lifestyle** – What should they expect from a 2-year old Lab? 5-year old? 10-year old? Why? What changes should they make?
5. **When will they see you again?** – Follow-up care scheduled

The 5 Ways To Make Your Clinic Happier, Healthier, And More Productive

INTRODUCTION: *Our habits are the secret of our success or foundation of our failure. Much has been written about what behaviors and customs help make you a success but little time is spent on those troublesome thoughts and actions we repeat each day that sabotage our happiness. Our routines define us. Repeating certain actions each day allows us to seek refuge from the chaos and unpredictability of the real world. We carve out a space that is uniquely ours and we control. For the most part, this is healthy. But what about habits we retreat to that are negative? They're more prevalent than you might think. Take the time to reflect on these five bad habits and seek ways to minimize their influence on your life. Who knows, you just might find happiness and success along the way.*

Bad Habit #1 – "Why you're wrong" - The Change Killer

I meet negative people all the time. Rarely do I conclude a lecture before someone points out why what I've been doing for the past twenty-one years in my practice simply won't work for them. And you know what? They're absolutely right. Whenever we fill our minds with "why it won't work," we release ourselves from the possibility that it might. This simple mental trick instantly removes any chance of what we really don't enjoy – change. Change requires work and action and lots of other potentially cumbersome things we'd rather avoid.

The problem with "why you're wrong" and "why it won't work" is that they don't add any value to the proposition. I realize that people who tell me this sincerely believe they're helping me better understand their unique situation or are pointing out a hidden flaw in my idea. They're also being polite in their disagreement as they hide behind a thin veil of agreement.

What they really mean to say is they know better. They're the authority or superior to someone else. The net result is it stops cold any possibility of change in the person's life. And if you're not careful, in the lives of those around you.

I was on a flight recently sitting next to a man and his teenage daughter. I couldn't help but overhear their conversation about choosing a college. The young lady was excitedly showing him a brochure from a quite famous Ivy League school. The father remarked with a laugh that they'd have to sell their home and cars to be able to afford the college tuition. Bad habit appears ("why it won't work"). The girl replied that the school offered several scholarships and two looked promising for her. The father scoffed that "he'd believe it when he sees it." Bad habit resurfaces ("why you're wrong"). I noticed the young girl's shoulders slumped as she slumped back into her chair. Not another word was spoken regarding the Ivy League college for the remainder of the flight. In fact, she began talking about a large state school not far from their

hometown. I had just witnessed a dream shattering.

"Why you're wrong" rears its ugly head too often in our lives. We think we're being helpful, wise, or experienced while in reality we're simply an impediment to progress, hope, and change. Listen carefully to yourself and consider carefully before killing change. Open yourself up to the possibility of new ideas, new ways of doing things, of a different future. After all, we're not nearly as clever alone as we are together.

Healthy Habit # 1 – "That might work" try it for yourself.

Bad Habit #2 – The Answer Ranking Game

"Do these jeans make me look fat?"

"No, you look amazing!"

"You're just saying that. Of course, I look fat."

Sound familiar? This and billions of other intimate conversations occur by each day. The problem is, this conversation was destructive. The subtle context was that the opinion of the trusted person was meaningless. The person asking about the jeans ranked the answer given as wrong, insincere, and misleading. Was it? We can't help but compare the other person's answers with our. Perhaps we secretly feel the jeans make us look fat so when someone tells us otherwise, we reject it. I think we must assume a position that people are truthful until proven otherwise. My interpretation is that the person looked great in those jeans.

Ranking or judging answers or responses, especially from people we're close to, is a common bad habit that serves no positive purpose. There's nothing wrong with someone giving you their opinion, good or bad, after you've asked them for it. In fact, it's vital that people agree and disagree.

What's not warranted is to pass judgment on an answer when we specifically request feedback about us. If you ask for someone's opinion about you, what you're doing, or what you intend to do, accept it. Respect it. Assume the trusted person is being truthful. This isn't naiveté; it's good interpersonal relations. After all, you asked.

When you regularly rank people's answers (good or bad, accurate or inaccurate, agree or disagree) after you've requested their opinion, they stop giving you accurate advice. Why bother? After all, all you do is dismiss their answer.

For the next week, I want you to shift into a neutral mindset. That is, you don't pass judgment on any advice or answers you receive when you ask someone. Accept them for what they are. Respond with a simple, "Thank you."

At the end of the week I guarantee you'll find you had fewer fights, pointless arguments with family and co-workers, and less contentious interactions. After three weeks, you'll find "Thank you" becomes an automatic response, people will remark how much easier you are to get along with, and people will view you as an open-minded, welcoming person (all good traits). This doesn't mean you agree with everyone or everything, of course you won't, it simply is a change in how you react to peoples' answers about you.

Healthy Habit #2 – "Stop judging answers to your questions." Try accepting other opinions and answers without ranking or judgment

Bad Habit #3 – Avoiding Apologies

Have you ever felt the sense of liberation, cleansing, or relief after uttering two simple words, "I'm sorry"? You felt better, didn't you? If this is true, why is it that so few of us are willing to apologize?

Maybe we think our life is some sort of contest with winners and losers. Maybe we equate apologizing with losing or failure. Maybe its' too painful to admit we're wrong. Maybe we feel humiliated when we ask forgiveness. Maybe we believe that if we apologize we appear weak or less powerful.

Whatever the reason, refusing to apologize creates more problems in our relationships at home and work. If you look back at the broken relationships in your life, I bet you'll find that many fell apart due to someone's refusal to say, "I'm sorry." And that's a shame.

When you make a mistake, own it. Apologize for it. Don't let your pride wreak havoc in your life. You don't earn a prize when you die for winning the most. The only thing left behind are the memories of you and the impact you had on the world.

By apologizing we release the past. We're saying, "I can't change what happened. I'm sorry for what I did wrong. I want to make it better moving forward." That's hard to resist, even for the most cold-hearted among us.

Truly successful, powerful, and significant people understand the power of the apology. We must work with others to succeed and admitting our mistakes is an essential element of effective collaboration. You can't get it right every time; when you don't, be brave enough to say you're sorry.

Healthy Habit #3 – "I'm sorry." Apologies make you stronger.

Bad Habit #4 – Not Thanking People Enough

Perhaps the two most powerful words in the English language are "Thank you." That phrase is so powerful that whenever I visit a foreign country I always learn how to say "thank you" in the native tongue (I also learn how to say "I'm sorry"). For some reason, we don't tell the people that matter most "thank you" nearly often enough.

Why are we so cheap with gratitude? Maybe we view it similarly to apologizing; we somehow feel less powerful or important when we thank others. I shouldn't have to go around thanking people all the time. Of course you should be doing your job excellently. Of course you should provide excellent service to me. Of course you should love and cherish me. For the next week, I want you to tell people how grateful you are for them whenever possible. Not insincerely, you've got to really mean it. People do amazing things for us each day. Take the time to thank them. This is the easiest bad habit to break and holds almost unlimited potential. You're welcome.

Healthy Habit #4 – "Be grateful for everything you have." Practice gratitude each and every day. It's better than an apple for keeping you healthy.

Bad Habit #5 – Waiting for the Heart Attack

"It won't happen to me" is something few people ever say out loud yet this is exactly how they live their lives. Sixty-eight percent of US adults are overweight or obese. The top four causes of death are caused, linked to, or exacerbated by excess weight: 1) Heart disease, 2) Cancer, 3) Chronic lower respiratory diseases, 4) Stroke (cerebrovascular diseases) (latest CDC data from preliminary 2011 report). Most highly successful people realize the importance of taking care of themselves. So why doesn't everyone live healthier?

The reasons for being unhealthy are numerous and complex. I don't have the answers; however, I do have solutions. The most important decision you make today is whether or not to pursue health. Every single person, regardless of genetics, socioeconomics, or even current state of health decides to do things each day that encourage or discourage better health. The multitude of tiny, seemingly insignificant decisions you'll make during the next 24 hours add up to promoting health or destroying it. The choice is yours. And it is a choice.

Try going for a brisk walk or run each day. Join a yoga class or a gym. Stop eating a bag of chips or soda with lunch each day. Put down the candy bar or doughnut. Drink more water. Small changes can create tremendous positive health impact. Don't think a heart attack won't happen to you. You can't wish yourself to good health.

What I don't wish for anyone is to wake up with chest pains at age 48, 52, or 68. What I don't want is for you to feel breathless after ascending a single flight of stairs. I don't want you to be forced to sit down relegated to watching your children or grandchildren play soccer. I want you to play soccer with them – maybe even win. I want you to be vital, energetic, and independent well into your eighties. That is my wish for your life.

Healthy Habit #5 – "Good health requires daily effort." What are you going to do with your one amazing life? The answer begins now.

Life is a precious gift. A gift we choose to enjoy to the fullest or one we waste, cut short in both duration and quality. Our decision to pursue (or not) personal health extends well beyond ourselves; ultimately our loved ones must pay any health debt we incur. We owe it to the people that matter most to take care of ourselves the very best we can.

Don't wait for the heart attack; avoid it. Start a journey of change that makes life more enjoyable, fulfilling, and lasting. That's what I'm trying to do: remaining "Fit to Practice" for as long as possible. Longer, even.

Adding New Services And Products Into Your Clinic

INTRODUCTION: *I'm always searching for ways to improve my patient care and client service. Over the past 25 years, I've helped pioneer senior care programs, long-term drug monitoring protocols, and weight management and nutritional counseling alongside regimented staff training, communication role-playing, and innovations in veterinary hospital design. If you're considering adding a new product or different therapy or service in your clinic, try these simple and straightforward steps to improve compliance and success.*

Step 1: Believe It

Successfully adding a new service or product into your daily practice begins with personal experience and firm belief. Whenever veterinarians struggling to gain acceptance of a new therapy confront me, I ask if they've tried it themselves and believe in it? It's nearly impossible to recommend a medical procedure to your clients you haven't personally experienced and sincerely trust. I wouldn't offer a product or perform a treatment on my personal pets I didn't wholeheartedly believe was safe and effective and the same goes for my patients. Nothing speaks louder than personal testimony. To testify convincingly, you first must believe.

I'd like to illustrate this concept with the story of how I added class 4 laser into my practice and why I say the first step to adding anything new into your clinic is to believe in it.

Class 4 laser therapy is an FDA-cleared treatment. That's important to me as a veterinarian because FDA clearance indicates a therapy is safe for my patients. Numerous research studies conclude class 4 laser therapy helps reduce pain and inflammation while facilitating tissue regeneration. In simplest terms, class 4 laser therapy can boost circulation to damaged tissues, creating an optimal healing environment. Scientific studies aside, I know firsthand the power of class 4 laser therapy.

Many of you may know my love of endurance sports, especially the Ironman triathlon. The Ironman is an event combining a 2.4-mile swim, 112-mile bike, and 26.2-mile marathon in a single day. Years of this sort of fun have caused a few scars and stresses on my joints. In 2011 as I was preparing for an Ironman, I developed debilitating bursitis in a previously injured shoulder. I was in real danger of not making it to the starting line. Friends recommended a variety of remedies, none of which helped. Finally, an exercise physiologist buddy suggested class 4 laser. He'd had success and I had nothing to lose. After two treatments I could move pain-free. Within two weeks I was back in the ocean swimming miles. I was a believer and bought a class 4 laser unit for my practice. If

it aided me so dramatically, I was eager to share this new modality with my pet patients.

My best advice for anyone considering adding anything new to his or her practice is to first prove it to yourself before offering to your patients. Don't believe the hype; believe in your interpretation of the data and results. Evaluate the science, examine the safety record, and establish if the service or product aligns with your core values. If it passes those three tests, try it. If you're satisfied with the results, ask your team to review it and give their opinion. If your team loves it, you've got a winner.

What you've done is achieve belief and buy-in the old-fashioned way: you earned it. Lukewarm acceptance won't cut it; your team needs to be excited and enthusiastic if they're going to effectively promote the innovation. If you and your staff genuinely believe in what you're recommending, your clients are much more likely to act on your advice.

Step 2: Frequently Use It

I have a friend who worked in marketing at Coca-Cola for many years. One evening we were discussing the "cola wars" and he revealed the secret to winning: Accessibility. The goal of Coke, in his opinion, was to be as close as possible to every human being on the planet. If Coke was within arm's reach, an individual was more likely to reach for it. I've taken those words to heart and applied it to my practices. If you want a recent addition to gain acceptance in your daily practice, it's got to be within arm's reach, literally.

I recently visited a practice owner friend who had completed a new facility. He had bragged about all the latest gadgets he'd acquired, and I was eager to see his dream clinic. As he escorted me through his sparkling high-tech temple, I didn't see his boasted high-dollar ultrasound machine anywhere. He led me to me a back room where he kept it until needed. He told me it was simply too expensive to risk leaving out in his treatment area. I asked how often he used the gleaming gizmo. At least a couple of times a month, he replied. Needless to say, I wasn't impressed, and his investment certainly wasn't as profitable as it should've been. Out of sight, out of mind, out of use, out of revenue. Use it or lose it, as the saying goes.

Back to my story of successfully adding class 4 laser and the importance of frequent use. Part of our daily clinic routine is to place our class 4 laser device in the treatment area, turn it on, and confirm it's ready to go on a moment's notice. Because our teams understand how class 4 laser can help heal inflammation and mitigate pain, I want its presence to serve as a constant reminder we can help. The more my teams remember to use it, the better they'll become using it.

The better we become at something, the more likely we are to do it. Frequent use accelerates learning and understanding of a new product or service. If you only use or recommend something a couple of times per month. You're never going to gain the expertise our clients and patients need and deserve. Have your class 4 laser unit easily accessible and prepared for action; you'll be surprised at the number of opportunities you have each day to use it.

Step 3: Share It

As your confidence in a new product or service flourishes and your experience grows, you'll want to share it with your clients and community. Begin by creating a "Success Book" detailing complex cases with outstanding results. Take pictures of your "new thing" (in my example, successful class 4 laser therapy cases) at diagnosis, throughout treatment, and at completion of healing. Ask pet owners for a brief quote about their experience and if they'd recommend the new service or product to others? Combining personal testimony with client's recommendations is powerfully compelling and can aid pet owners in making more informed veterinary care decisions.

In today's fast-paced and constantly changing medical environment, it's more important than ever to keep your clinic welcome brochures and website updated with new services. Consider crafting personalized client brochures explaining what the "new thing" is, how it works, and why you're excited to offer it. Take this content and add it to your website and repurpose for social media postings.

After you're comfortable with the new product (accessory, drug, OTC, etc.) or new therapy, it's time to contact your local media. Newspapers, radio, and television are always looking for local stories to highlight and pet news is always welcomed. Other good outlets for sharing your news are weekly or monthly community or business magazines. I also recommend working with rescue groups and animal bloggers to distribute clinic news and updates. Don't make the mistake of delaying a new service until you've published a press release and gained news media. Start with yourself, extend to your team, and then share with your clients and community.

KEEP AN OPEN MIND WITH NEW PRODUCTS AND SERVICES TO SUCCEED

There's one final element you need to achieve sustainable success in practice and in life: Keep an open mind. Don't discount new ideas, products or therapies simply because you don't initially understand them. We live in an amazing time full of rapid changes, astonishing advancements, and wholesale upheavals. I'm not suggesting you accept every new thing; I'm advocating you investigate innovations that connect with your beliefs and practice values. Approach new services and products impartially and unbiased and you may discover incredible improvements in your patient care and client service.

Communicating About Pet Food: Top Pet Owner Nutritional Myths

INTRODUCTION: *Nutrition is perhaps the most confusing, complex, and contentious medical discipline. Innumerable approaches, overwhelming opinions, and conflicting conclusions obfuscate consensus. In other words, there's a whole lot of debate going on about nutrition and clear answers are scarce. Veterinary medicine isn't immune to this food-diet-lifestyle confusion. Strong opinions abound and an increasing number of untrained, uncredentialed, and unlicensed individuals are offering therapeutic recommendations based on personal experiences and unsubstantiated claims. This is leading to confused pet owners and a surge of myths and misinformation. Here are some of the top pet nutritional myths and how your team can politely bust them.*

(Presentation Note: Specific and current pet food and diet myths will be reviewed during presentation. Below is a communication strategy you can use to create your own tactics to help shape your client's behavior and beliefs toward healthier and evidence-based approaches. EW)

MYTH BUSTING 101 FOR VETERINARY TEAMS

Busting myths is hard. Whenever people believe in something, regardless of how illogical or unproven, it's a real challenge to change their minds. Instead of attempting to change someone's beliefs or behaviors, I recommend trying to gently shape them. Regardless of the myth, misperception, or potentially wrong behavior, here is a strategy I've found helpful when politely busting untruths:

1. **Start with Thank You** – No matter the situation, I advise always leading with compassion when interacting with others. Your alternative is to be emotionally neutral or negative; neither encourage collaboration and trust between two people. Start by acknowledging your sincere appreciation that the pet owner is seeking information to help their pet. "I'm really glad you're interested in learning more about the best way to feed Buster." or "I really appreciate the effort you've taken to find information about the best way to feed your cat." Begin each conversation tilted toward positivity by thanking the client for their efforts, however potentially misguided.
2. **Find the Why** – The next step in myth-busting and shaping behavior is to determine why the owner is interested in or made a dietary change. "What made you interested in the BARF diet for Buster?" or "Was there something about your cat's condition or health that made you switch foods?"
3. **Find the What** – Myths may take many shapes and forms. It's essential you clarify exactly what the client means by "raw," "BARF," "no-grain," "organic," and other poorly defined terms. "Tell me more about Buster's typical breakfast or dinner." or "Walk me

through your preparation of a raw meal for Kitty." or "When you say grain-free dog food, is there a specific brand you're feeding or what do you look for on the label?" Try to solicit as much detail and specifics as possible: types of proteins and where they're sourced, quantity fed or prepared, how the food is handled and stored, and so on. Be sure to document this information in the pet's medical record.

4. **How does this make their pet feel?** – Many pet owners are using new dietary strategies for a specific reason (see above). The next step is to connect that change with how they perceive their pet's feelings and emotions. You'll need to incorporate how the pet "feels" in your advice. "How do you think Buster feels on the new diet?" After all, helping a pet "feel better" is the whole point of diet. Improving a pet's quality of life is often sought in the food bowl.

5. **Thank You Again and a Nudge –** After you've obtained the basic diet history, thank the client again for sharing. Keep in mind it can be intimidating for a pet owner to disclose these details, especially if they fear judgment or disagreement from the veterinary healthcare team. "Thank you again for sharing this with me. The more details I have, the better medical advice I can provide to help you and Buster." By adding "medical advice," you've taken the first step in subtly shaping and reframing the conversation. Use your authority as a medical professional to politely move the message away from marketing and unsubstantiated claims and toward an evidence-based nutrition discussion.

6. **Do you really, truly object?** – Is it harmful? Sometimes a client's diet change, despite being unconventional or a strategy you're unfamiliar with, is within the realm of possibility and safety. I've had clients feed a diet they drive 100 miles across state lines to purchase, the label makes some outlandish claims, yet appears complete and balanced. That's not a diet I'm likely to dismiss or even attempt to change unless I have evidence it's harmful or the pet has a medical condition that needs another approach. Be careful not to dismiss a diet or brand unknown to you; there are plenty of safe diets out there that aren't my choice and others I don't know much about. Before trying to change a behavior or diet, be sure you really, truly object to it.

7. **Facts Don't Matter Most of the Time** – Unfortunately, it's rare that a medical professional can prompt sustainable change based on facts alone (see: smoking, heart disease, obesity, substance abuse, etc., etc.). people can cling to some pretty outrageous beliefs, despite an abundance of hard scientific evidence to the contrary. Be prepared with plenty of facts supporting your claims and preferences, but be aware that you'll rarely shape or change behavior solely based on the evidence.

8. **It's Not a Debate** – The natural tendency in these situations is to immediately counter inaccuracies, dispute claims, and correct wrongs. That's probably not the best choice

when dispelling food myths with your clients. Avoid interrupting, listen intently, and demonstrate interest and concern regardless of what the pet owner is telling you (in other words, no eye rolling). "I feed him XYZ food because, as you know, dogs are really just wolves." Instead of pointing out that dogs are not, in fact, wolves, try a softer approach when confronted with inaccuracies: "I'm really glad you're interested in what the best diet for dogs is; so many dog owners simply buy the cheapest food. In general terms, dogs need… (insert your nutritional philosophy here)." Don't use their preferred terms; frame the topic in your terms. In this example, repeating "dog," not "wolf" is best. Even saying, "No, dogs aren't really wolves." removes emphasis from the topic at hand – finding the best nutritional approach for their dog – and places it on whether dogs are taxonomically "wolves." It's not a debate; don't make it one.

9. **What's the alternative?** – The most important motivator for shaping or changing behavior is offering a better alternative. The client originally made a change because they believed or were persuaded that there was a better option than their current situation. Your next alternative must be even better (and easier, cheaper, something…). This is the time to make sure you have a deep roster of alternative diets and dietary strategies to accommodate many clients. If not, don't be surprised that every client doesn't fit neatly into your limited offerings. Long gone are the days when carrying a couple of brands or only believing in a single nutritional philosophy will work with today's well-educated clients who are exposed to countless diets and ideas.

10. **Why is your option better?** – Sharing a few facts can help support your case, but if you've uncovered the real reasons for change (see above), this is where you put that information to work. "I totally understand that you were looking for a more ancestral, healthy diet for Buster. I also understand your concerns about highly-processed dog foods. My primary concern is that the raw diet you're feeding, which you said is mainly chicken necks, liver, and hearts, is not nutritionally complete, leaving Buster deficient in many key nutrients essential for a healthy immune system and preventing disease. Of course, I'm also worried about your family contracting a contagious food-borne disease like E. coli or salmonella, but I want to focus on Buster's long-term health. With that in mind, and your interest in whole or less-processed foods, do you know about … (insert your option here: i.e., high-pressure pasteurized raw diets)? That may be an excellent alternative that helps reduce some of my nutritional worries while giving you piece of mind."

11. **Repetition is Key** – You're usually not going to change people's behavior after a single interaction. Shaping behavior often requires patience, strategy, and persistence. Many times, a client will politely listen, not change, but begin considering what you said. When they return, whether in a week or a year, you should re-engage and reinforce your message(s). It may

take months to years before change occurs; don't take it personally if at first you don't succeed. Many clients are also observing if you're sincere by whether you bring up the topic in the future. Repetition often signals credibility; it's so important you talk about every time you see them. If you're following your passion and scientific evidence, you'll never waver, despite rejection. In fact, your passion will compel you to try and try again!

These techniques work on busting diet and nutrition myths, promoting weight loss and dental programs, and any other medical service or product you feel important to tell pet owners about. Role play with your team and explore the optimal ways you can effectively communicate your key messages to clients and good luck busting diet and pet food myths!

Innovating The First Year: The 9-Month Pet Visit

The first year in a pet's life is arguably the most important in terms of veterinary care. Healthy habits, immunizations, preventive care, client education, and training occur within those first 12 to 18 months. The reality is veterinarians have traditionally been left out of much of this critical developmental period. After spay and neuter around 6 to 8 months of age, we typically don't see our pet patients again until 16 to 18 months of age. This creates a "gap year" and an "advice void" increasingly filled by providers outside the veterinary profession. There is an elegant solution to this dilemma: the 9-month visit. This innovative program will improve patient care and promote veterinary services and products at a critical developmental stage. The 9-month visit

1) creates an additional veterinary intervention point for behavioral, nutritional, life stage, and parasite problems

2) Further establishes routine veterinary visit habits, especially of impressionable Millennial pet owners

3) A unique opportunity to introduce additional veterinary services and products and increase revenue

4) Ensures proper and accurate dosing of parasiticides, heartworm preventives, and food.

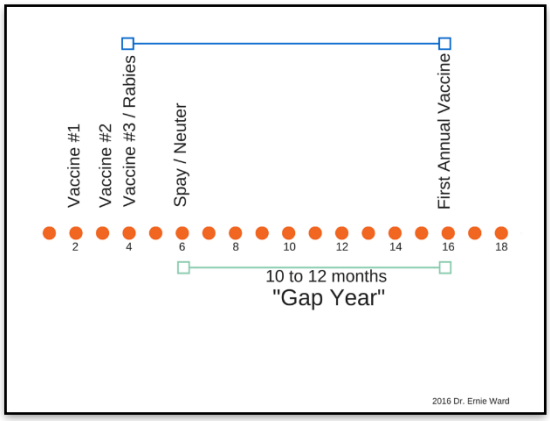
I created our clinics' 9-month visit concept in 2008. At that time, we were witnessing a decrease in pet visits coinciding with the U.S. Great Recession of 2008 to 2010. In 2006 and 2007, I had initiated several feline-friendly programs within our clinics that were beginning to see positive results, but I knew we had to do more to continue to grow during such challenging economic times. I began to focus on the first year of life for puppies and kittens to search for opportunities to innovate. 2007 and 2008 were also the years we began seriously developing novel ways to reduce stress, improve handling and restraint, and reinvent the veterinary experience for our patients and clients.

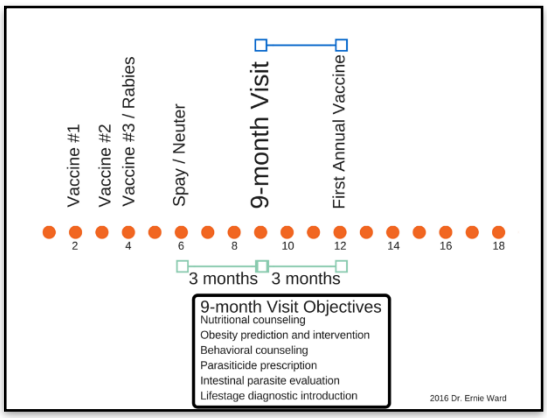
As I reviewed our clinics' existing puppy and kitten appointment protocols, I struggled to uncover hidden opportunities. It would take innovation and creativity to solve this problem. I vividly recall the stream-of-consciousness brainstorming exercise in which I made the breakthrough. This particular creativity method is one I use frequently when attempting to solve a vexing problem. The exercise simply entails taking a blank sheet of paper and writing or drawing everything that comes into my mind for five minutes. Ideas can be ideas about the issue or, more often, completely random thoughts or images. And so I began.

I drew a line and labelled the left endpoint "Birth" and the right "Death." I wrote "a bunch of stuff" in between the two (told you it could be random). I then divided the line at about the left-quarter mark and labelled the two partitions "Puppy and Kitten" and "Adult.

Next, I drew a new line that started at "Birth" and ended at "Adult." I marked a spot labelled "6 to 8 weeks," and additional marks at 3, 4, 6, and 8 months. I jotted "First

Core Vaccines" at the first mark and repeated those vaccines at 3 and 4 months. At the 4-month mark, I wrote "Rabies" and at 6 months "Spay/Neuter." Above "Rabies," I extended a line toward "Adult" and labelled it "12 months later = First Adult Core Vaccine Visit." And then it hit me. I had been doing everything wrong for the past 20 years of clinical practice.

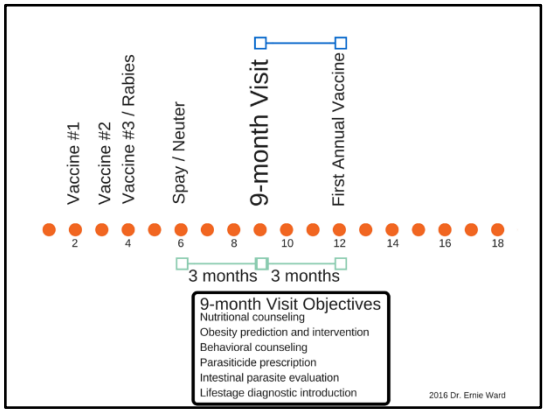




As I stared at scribbling, I began to realize we had largely abandoned our patients and clients during what I believe is a pet's most critical developmental stage: Between 6 months of age and nearly a year-and-a-half. During this "gap year," behavioral problems, nutritional questions, and heartworm, flea and tick control were being left to the pet owner. Not knowing the best source to turn to for pet care advice, concerned owners were asking providers they encountered: Pet store personnel, groomers, trainers, and the internet. By the time they returned to the veterinarian, if they returned at all, many important health and hygiene habits were established. When confronted with scientifically sound advice from a licensed veterinary professional, many pet owners perceived they were being "sold" something by their veterinarian and viewed them skeptically.

In short, we had created an "advice void" that was being filled by non-veterinary providers. Even worse, we had failed to create real value in our services beyond vaccinations and sterilization during the first year. I realized at that moment why pet owners equated veterinarians with "shots" and our primary surgical expertise was spay and neuter. Clients hadn't been exposed to our expertise in nutrition, behavior, internal medicine, surgery, and more. There experience with us during the formative first months was limited to a quick jab with a vaccine and a dose of heartworm preventive. Armed with that insight, I set out to innovate the first year of veterinary care.

I studied my sketch and looked for an appointment opportunity that was physiologically and medically appropriate for my patients and convenient and economical for clients. I also wanted the timing of a new visit to be strategic to maximize the chances of identifying and intervening in emerging problems to optimize positive outcomes. It didn't take me long to find the perfect visit: 9-months of age.



The 9-month Visit was created. I knew from my work in pet obesity that a veterinarian could accurately predict adult cat obesity by comparing the first obtained weight with what the kitten weighed at about 8 to 10 months of age. The weight gain during that period served as a precise predictor of adult obesity in cats and evidence was mounting for similar prediction in dogs. I also knew from behavior research that many behavioral issues began between about 6 and 12 months of age. Weight gain after the last visit during sterilization could change rapidly, jeopardizing accurate and effective heartworm, flea, and tick preventive dosing.

Perhaps most importantly, I knew we needed a visit to talk about important health issues other than vaccines. The focus of our 9-month Visit would be

1) Behavior

2) Nutrition

3) Parasite Prevention

4)Future Veterinary Care Needs. If additional or overlooked booster immunizations, fecal parasite testing, or other services were needed, the 9-month Visit would serve as an important safety backstop.

If you're interested in adding the 9-month visit to your practice, here are some tips for a successful transition. First, write a brief explanation of the concept and share it with your managers, key team leaders, and senior associate veterinarians. Be open to suggestions, especially in messaging and pricing, and gain consensus form your leaders before presenting to the full team. Next, schedule a staff training session to review the new plan and solicit general feedback. Continue to refine the new service and discuss the implementation. Train your staff on the topics and information you believe are most important during this rapid developmental period of a pet's life. This initial process will typically take about a month to complete. Don't rush; get comfortable with a new service or product before you begin offering it to patients and clients. That advice goes for the 9-month visit or a new flea and tick preventive.

My advice on launching the 9-month visit is don't make it a big deal to your clients. Pet owners are used to coming in frequently during the first year and few question or object when you schedule it. Many new pet parents are young Millennials coming to the veterinarian with their "first real pet." I've found the 9-month Visit helps establish good pet care habits and reinforces the importance of regular veterinary visits. The 9-month Visit also positions veterinarians as the primary pet healthcare information provider, both online and in-person.

Be sure to change your current reminder protocols. Adjust the current 12-month appointment reminder generated by the Rabies vaccine (usually given at 16 to 20 weeks of age which generates a reminder 12 months later) to create a reminder 4 months later (at 8 to 9 months of age). Our reminder text simply read, "9-month Appointment."

Nutrition, behavior, and ecto- and endoparasite prevention are my focus for the 9-month Visit. Other clinics will be excited to emphasize certain vaccinations, additional diagnostic and parasite testing, dentistry, and training. Whatever topics you choose to discuss, the real emphasis is on reinserting the veterinary profession into the "Gap Year" and allowing us to fill the "Advice Void" with sound, reliable, and accurate information and guidance. The 9-month Visit creates additional opportunities and exposure to the diverse services and expertise veterinarians provide. It encourages regular routine veterinary visits and corrects the "veterinarian equals shots" falsehood. It exposes young pet parents to proper veterinary care and expertise. Ultimately, the 9-month Visit allows us to connect with new pet parents in a more meaningful, impactful manner.

Social Media Professional: Winning Social Media For Practice Success

INTRODUCTION: *Have you ever played the telephone game? One person whispers a message to an adjacent person in a group. The communiqué is passed along until the last player reveals the message to everyone. Inevitably, and this has apparently been studied by academic types, the final version of the message varies significantly, sometimes almost unrecognizably, from the start. "Mary is wearing a green dress to the dance with Bill." morphs into "Mary wore a green dress while dancing with Phil." I fear veterinarians have literally been playing the telephone game with our clients. We've been relying on outdated and outmoded telephone conversations, faxes, and mail while the rest of the world whisks away at the speed of electrons. We pass information slowly by word-of-mouth, often creating confused conversations and mixed messages while politicians move millions with 140 characters and Millennials modify their life by memes. It's time veterinarians get serious about winning social media for practice success. It's time to hang up the phone and tap that app.*

HOW DID WE GET HERE?

Over the past decade, it's become increasingly apparent to me that many of my colleagues aren't keeping up with communication technology. For most of our profession's existence, we thrived simply by sending postcards. A pet owner received a notice in the mail it was time for her pet's vaccinations, and, voila, she booked an appointment. No more. For starters, few check their mail and when they do, tend to toss anything suggestive of marketing. Secondly, young pet owners may not have a mailbox, at least not a physical one. Finally, who has the time to read a postcard, dial a number, talk to someone and haggle over a date and time that works with everyone's schedule? And we wonder why visits and revenues are plunging and pet owners are increasingly skeptical and unimpressed with our services? Check your mailbox.

It's not that veterinarians are incapable of changing or taking action; it's more often our inability or stubbornness to take appropriate action. I gave my first "social media and text message" lecture at the 2007 North American Veterinary Conference. I may have been the first veterinarian to advise clinics to join Facebook and monitor this new thing I thought would be important to the profession. I also touted text as the next "postcard" and urged owners to pressure software developers to implement these features in their reminder systems. I was roundly laughed at and ignored for several years. It's okay; I'm over it.

When our profession finally began approaching social media, email and text, we were hampered by our postcard postscript. Emails weren't much more than mailers mated to a screen. Text messages became intrusions screaming "Discount day!" or "How's it going, client!" instead of brief, personal interactions. Social media became a mess for the few daring to dip into its murky waters. Without a meaningful vision, strategy, or comprehension, social media and digital communications became haranguing hinterlands to be avoided or minimally appeased. Thankfully, we're

emerging from those dark days, and, although the electronic glare can be overwhelming at first, progressive clinics are seeing growth in revenue, patient care, and client satisfaction by embracing Client Communication 2.0.

WHERE ARE CLIENTS LISTENING?

The first step toward transitioning to Client Communication 2.0 is understanding where your clients are talking. This is important because a recent McKinsey Report concluded that businesses who utilize social media and electronic communication experienced 20 percent more revenue and 60 percent higher profit growth (1). Over half of all U.K. adults use Facebook on a regular basis with an estimated 78 percent of over 18's checking their status routinely. 20 to 29-year-olds comprise the largest group of Facebook users followed by 30 to 39's and 40 to 49's. Over 14 million Britons Tweet, 36 million watch YouTube, and 40 percent of the nearly 20 million Instagram addicts log in daily. About 15 percent of the estimated 10 million U.K. Pinterest participants eyeball their boards every day. (2,3,4)

For most veterinary clinics, this means focusing your social media efforts on Facebook and dabble in YouTube, Twitter, Pinterest, and Instagram. I suggest securing clinic profiles on all social media platforms (I'm talking to you, Snapchat, WeChat and the like), but concentrate on connecting with your Facebook family.

HOW ARE CLIENTS LISTENING?

Facebook, Twitter, YouTube, Pinterest, Instagram, texting and email each attract a unique audience and establish specific expectations. You can think of it in terms of planning to attend a concert; the musical genre and venue will largely determine how you dress and behave. *Iron Maiden* fans will typically look a bit different than those attending an *Adele* performance. It's fair to say there are general expectations and tendencies worth noting to prevent you from showing up at the *Spandau Ballet* reunion clad in leather and safety pins. The lesson is you need to dress your online content appropriately for the show.

DRESSING YOUR PRESENCE BASED ON PLATFORM

A common 'dress code' mistake I see is applying the same branding, messaging, and strategy across all social media platforms. Because each platform operates in a distinctive manner, here are a few tips when creating content on the major social media outlets:

FACEBOOK

Facebook is the modern pub crawl. People check their Facebook feed to get the latest gossip, trending news, and entertainment. Facebook is where most funny cat videos are viewed. A simple rule of thumb I follow is about 80 percent of content should build your brand, educate, and add value to the veterinary profession and 20 percent can promote a service, product, or promotion. Creating a weekly or monthly schedule can help balance your strategy. If you post daily, consider four or five posts per week consist of: breaking pet news, reposts of feel-good animal stories, and advances in veterinary medicine. One or two posts each week can highlight your senior pet care program, a weight loss promotion, or seasonal emphasis on flea and tick products. You can further build your brand when sharing other posts by adding, "ABC Veterinary loves

research demonstrating the powerful human-animal bond! Check this out!" Show your personality and passion in your posts.

An easy way to ignite engagement and educate on Facebook is by sharing pictures. After obtaining permission, nothing sparks a smile and a conversation more than a picture or video of cuddly puppy or a pet combating a challenging condition. "We were all hugs today with these cuties in for an intestinal parasite check and immunizations!" or "Mabel is a 15-year old kitty beating the odds. Diagnosed with kidney failure six months ago, her owners are proof that love, compassion, and commitment can make a difference. That's the face of a fighter! If your older cat is drinking or urinating more, losing weight or acting tired, let us check them out. Way to go, Mabel!" Entertain, inspire, and educate.

TWITTER

If Facebook is the neighborhood pub, Twitter is a cruise ship. Loads of anonymous people climb aboard hashtags and hurl clever quips and offensive oratory over cyberspace cocktails. For most clinics, Twitter isn't incredibly helpful. Use it to share hospital blog posts, a special event, or breaking news. I discourage tweeting discount codes, product sales, and other blatant promotions. Social media backlash can be brutal, particularly around perceived "advertising." Tweet compassionately, cleverly, and carefully.

YOUTUBE

YouTube is a search engine run by Google. That's critical to remember when creating YouTube content for your clinic. Users subscribe to channels to learn from or they find entertaining and interesting. Most veterinarians should use YouTube to provide virtual hospital tours, how-to videos, and information their clients are searching for. Video production is rapidly improving on YouTube; shaky smartphone video with faint audio is a no-no. be sure to link to your clinic's website and other social in each video's description and optimize end screens and cards.

INSTAGRAM

Pictures. Beautiful pictures. That's it. I consider Instagram for clinics as a platform to reveal 'behind the scenes,' 'wow,' and 'gorgeous' sides of practice. Messages that pop on other platforms can fall flat unless fabulously framed for Instagram. What's in it for us? Showing your softer side and lots of heroic pictures.

PINTEREST

A widely-publicized Pinterest stat is that 80 percent of its users are female. That looks great on paper, but I've found using Pinterest as a standalone or primary social media marketing platform to underperform. Besides, Pinterest growth has plateaued and appears to be on the decline. Share original blog posts and infographics along with how-to's and YouTube videos. Showing up seems to be half the battle for Pinterest.

SNAPCHAT

Snapchat Stories brought business potential to Snapchat but current demographics skew awfully young for veterinarians. About a quarter of Snapchat users are under 18 years old and 60 percent under 25. (5) My advice is get onboard and monitor for now. Big changes are promised that should help small businesses connect with the next generation of pet owners.

LIVE STREAMING

Facebook and YouTube have evolved into excellent live streaming services. While these are the early days of live video, watch this space closely. Try hosting a 30-minute live Q-and-A, offer a five-minute highlight of a new product or service, or announce an event. The live events are automatically archived for later viewing. With a little planning and promotion, you could reach scores of clients and potential new clients with little effort.

Today's texting and email are like yesterday's phone call and postcards in many ways. Each represents a different communication opportunity than social media. Understanding how people use, and want to use, text and email is critical for Client Communication 2.0.

TEXT MESSAGES

This is my preferred way to remind clients and check on patients. Five texting caveats: 1) If you're requesting to schedule an appointment, the mechanism to make that appointment needs to be embedded in the text. No dialing or texting back-and-forth. Click here or reply to book. No more. 2) If confirming an existing appointment, same rules. 3) If checking on a patient, make it personal and be prepared to discuss. Texting creates a sense of urgency and when a client responds; they expect you to be available to reply. If the client responds after hours, have an autoresponder with what to do in an emergency in place. 4) Text checkups are best for minor medical conditions and routine visits. Call after surgery, anesthesia, and major diagnoses. 5) Limit text messages to only when necessary. You don't want your number blocked because you sent a cat owner a generic sales pitch for a dog product.

EMAILS

Emails continue to serve as both the primary mode of connection as well as a backstop to text messages. Our client admission forms ask in what order they prefer to be contacted: phone, email, or text. Use emails to remind about appointments (see text rules about incorporating 'single-click solutions'), new blog posts, announcements, seasonal educational messages, and surveys. I've found occasionally asking clients for their opinion on adding new products or services to be an effective way to gauge interest and build awareness. Monthly clinic update emails are ideal for most accompanied by personalized reminders. Embedding a quick video summary is bonus.

WHO'S IN CHARGE OF YOUR PRACTICE'S SOCIAL MEDIA?

Nearly everyone on your team should be a part of creating social media content, snapping photos, writing blogs. Creating isn't the same as posting. Before you press publish, an administrator should verify, clarify, and proofread every message bearing your brand. This is another reason I encourage you to use a calendar to guide your outreach and solidify your strategy. Simply posting cute kitten pics, lost dog posters, and homeless pets isn't a plan and won't grow your business.

WHAT'S THE ROI ON ALL THIS?

Does social media make business sense for your clinic? I'll repeat what I've been saying since 2007: Return on investment (ROI) on social media is hard to measure and perhaps the traditional ways to calculate it don't apply. Internet conversations about you are happening with or without you. It's

far better to insert yourself in these discussions than pretending they aren't real. It's even better to influence the conversations and control your image and protect your reputation. Many veterinarians get interested in social media after discovering a poor review or negative post. That's great, but it's always better to be proactive with communications than reactive.

Social media and electronic communications are also important to elevate the bond you share with clients and patients. We often mistake client's desire for increased access with extending office hours. What many want is a richer, more frequent method to interact with us. Social media, texts, and apps provide a contemporary way to connect with clients that an increasing number of other professionals offer. My own physician has an app and online portal through which I can access my medical information, test results, and chat with a medical professional around the clock. Systems for veterinarians are just beginning to appear and I expect them to be universal within two to three years. Apps and websites won't replace social media and texts; they'll augment each other.

Finally, determining ROI is a challenge because social media allows you to expand your reach farther and more focused than traditional marketing. Sure, you need to boost a Facebook post to get it in front of your audience, but boosting allows you to precisely target pet lovers within your professional perimeter. Even better, it's possible to showcase your personality, passion, and expertise in ways we could only imagine a decade ago. Go ahead, turn up the volume on your social media and be prepared for the celebration of the century!

BOOSTING YOUR CLINIC MANAGEMENT SOFTWARE

Veterinary practice management systems continue to serve as the mixing board and amplifier for our client communications. To get the most out of your clinic software, make sure you're dialing up the volume by following these simple tips:

OPTIMIZE EMAIL REMINDERS: Link services and products to a specific email reminder that reminds pet owners not only that something is due, but why it's important to do it. For example, an immunization or preventive reminder should be coupled with a few sentences explaining why fleas are problem in your geography, any prevalence data, and consequences of flea bites. Vaccinations should include a short statement detailing why their pet is at risk of a specific infectious disease (what I call "individualized immunizations" based on a "Lifestyle risk assessment"), why the immunization is given at a certain frequency, and disease dangers. For the past decade, we've been sending out at least two email reminders scheduled one to two weeks prior to due date, a week following deadline, and then a final email ten to fourteen days later before resorting to mail. Use pet name, age, gender, and any other pertinent information to make your outreach as personal as possible.

TEXT MESSAGES: In addition to email reminders, ask clients if they prefer SMS reminders and updates. I've had success with monthly medication refills and preventives, weekly weight and progress updates, and daily critical care check-in's over text. Be respectful of your client's preferred communication platform and

crank up your software's text features to be heard above the email crowds.

SOCIAL MEDIA: Your management software can also help grow your social media by adding Facebook or Twitter links to all correspondence, generating survey contacts to conduct client satisfaction research, and connecting with your blogs and breaking news. I recommend you're your management system to send a monthly electronic newsletter highlighting hot social media posts or stories, embedded with direct signup and sharable links.

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Moquillo Canino

RESUMEN: *El virus de Moquillo canino/Distemper parece haberse generado en el Perú y de allí se expandió a España en el Siglo 17 y de allí a todo el mundo eventualmente.*

Este virus no solo afecta a perros, sino a todos los cánidos y a muchas otras especies de mamíferos, desde félidos salvajes, a mustélidos y prociónidos.

Hoy en día la enfermedad produce alta mortalidad en poblaciones de canidaes no expuestas previamente al virus o libres de vacunación: 1999 brote de DC en la Isla Santa Catalina, California USA afectando la población de zorros nativos pasando de 1.330 individuos a menos de 100. En el año 2001 en la Isla de Pascua, Ecuador se registraron 596 casos de DC en perros silvestres; 275 murieron por la enfermedad y 294 fueron sacrificados ya que estaban con sintomatología.

Todas las vacunas disponibles tienen la capacidad de proteger perros contra VDC, lo de la "nueva" tipificación quiere decir que gracias a nuevas técnicas moleculares se está aprendiendo más del virus y de las cepas circulantes en el mundo.

Hay que comprobar el diagnostico con la detección del virus, por Kits comerciales o por PCR en laboratorios disponibles (los cuales han validado la técnica de detección).

Las vacunas a virus vivo modificado contra distemper, pueden producir depresión del sistema linfoide por unos días después de la inoculación, lo que es altamente riesgoso en ciertas poblaciones de perros débiles, desnutridos, y/o estresados. Esto es un factor de consideración en ese tipo de animales, donde la respuesta a la inmunización no solo contra distemper, sino también contra otros antígenos presentes también en la vacuna aplicada [adenovirus (hepatitis), parvovirus] puede afectarse. Algunas cepas de vacunas a virus modificado de distemper tienen la capacidad de producir encefalitis post-vacunal, y aunque la incidencia de este problema es muy baja, es importante analizar esto y evitar utilizar vacunas a virus vivo modificado de distemper en animales débiles, desnutridos, y/o con problemas inmunitarios. Al diseñar el protocolo de inmunización de cada paciente, tener en cuenta que solo la vacuna de distemper recombinante vectorizada cruza la barrera de anticuerpos maternales (como se hizo hace 50 años con la vacuna de sarampión humana aplicada a cachorritos). Por su inocuidad y alta protección la vacuna recombinante de distemper ha estado ayudando desde 1998 a que poblaciones de especies salvajes susceptibles a esta enfermedad estén prosperando; Leopardos de las nubes, pandas gigantes, pandas rojos, y hurones de patas negras.

REFERENCIAS: D*isponibles por petición a la Conferencista.*

Parvovirosis Canina

INTRODUCCION: *El virus de parvovirus canino se propaga a través del contacto oral con heces infectadas o por superficies contaminadas (el suelo, pasto, zapatos, juguetes), en lugares donde se concentran cachorros/perros jóvenes; "socialización temprana", criaderos y perreras, tiendas de mascotas, parques, entrenamiento: clases de agilidad/obediencia, etc.*

Produce una muy buena inmunidad en perros que sobreviven la enfermedad.

Se le denomino PVC-2, para distinguirlo de otro parvovirus ya presente; el "minute virus of canines; (MVC)". Fue identificado y aislado por primera vez hace 40 años, en 1978 por los Dr. Max Appel & Dr. Leland Carmichael del James A. Baker Institute for Animal Health, parte de la Facultad de Medicina Veterinaria de la Universidad de Cornell, Ithaca NY. USA. Con pruebas de laboratorio se empezaron a hacer muestreos y se detectó que el virus original (CPV-2) no era el único circulante en el campo, se detectaron dos variantes adicionales de campo a las que se les denomino respectivamente CPV-2a en 1979 y CPV-2b en 1983. Gracias a la nefasta moda de "Vacunación y Socialización temprana" empezando a vacunar cachorros a las 4 semanas de vida, acabando el ciclo a las 8-10 semanas de vida, se empezaron a tener brotes de parvovirus en Europa y USA en el año 2000-2002, cuando desde hacían muchos años no había brotes de esta enfermedad. Esta situación intereso a investigadores en Italia, Dr. Nicola Decaro y Dr. Canio Buonavoglia los que identificaron una tercera variante de campo por esa época llamándola "Glu-426", la cual fue definida después como CPV-2c en 2002. Subsecuentemente se encontró en muestras congeladas de heces que CPV-2c ya estaba presente en el mundo desde 1996…solo que nadie la había detectado ya que las técnicas de laboratorio antes de los años 2000 no la podían detectar.

REFERENCIAS: *Disponibles por petición a la Conferencista.*

INTERROGANTES COMUNES EN LA CLINICA:

- *Cual vacuna debo utilizar en mi clínica? Hay CPV-2c en Colombia, Perú, Venezuela, Ecuador?, tiene que tener la vacuna de Parvo que yo uso la cepa CPV-2c?*

Respuesta: No es necesario tener el antígeno CPV-2c en la vacuna para proteger contra esta variante, ya que todas las vacunas de parvovirus canino protegen contra todas las variantes; CPV-2a, CPV-2b y CPV-2c. Eesto se ha demostrado bajo estudios de vacunación / desafío con perros SPF [libres de patógenos específicos = sin ninguna inmunidad previa a CPV].

-Brunet, Toulemonde, Minke: Presentado Públicamente International Parvovirus Meeting, Bari, Sept 2007. Lineas de vacuna Eurican / Hexadog / Recombitek del laboratorio Merial *(ahora BI)*

-Spibey N, Greenwood NM, Sutton D, et al. Canine parvovirus type 2 vaccine protects against virulent challenge with type 2c virus.Vet Microbiol. P*resentado en el WSAVA Praga* 2006. Linea de vacuna Nobivac del laboratorio Intervet *(ahora MSD*).

-Dr. Ron Schultz et al. Wisconsin University. Presentado en 2007 CRWAD, Chicago, USA,, demostró que todas sirven!.

- *Si todas las vacunas de parvovirus canino protegen contra todas las cepas y variantes de campo porque cachorros con 1 o hasta 3 dosis de vacuna contraen parvovirosis?*

Respuesta: Primero que todo, hubo diagnóstico de parvovirus excretado en sus heces (Kits), o solo se sospecha de parvovirus por tener el cachorro diarrea con/sin hemorragia? Si, sí hubo diagnóstico definitivo el problema muy probablemente no es la vacuna en sí, sino el animal; ¿se tuvo en cuenta la interferencia de los anticuerpos maternales al diseñar el programa de vacunación para ese perro?, A qué edad se le administró la ultima dosis de vacuna?, Estaba el cachorro libre de stress, de fiebre, parásitos, de tratamiento con corticoides en los días inmediatamente previos o posteriores a la aplicación de la vacuna?. Se ha considerado si antes de recibir el ciclo de al menos las primeras 2 dosis de vacunas ese cachorrito estuvo aislado en casa, sin contacto con ningún otro perro, sin salir nunca de casa, y con la absoluta certeza que nosotros no introducimos el virus de parvovirus a nuestros hogares?.. No podemos descartar que se hubiese contagiado antes de recibir su curso de vacunaciones. Hay que recordar que el antígeno de parvovirus es de los más resistentes a desinfección y es altamente contagioso.

Leptospirosis, Enfermedad Zoonotica: Diagnostico Y Prevencion

INTRODUCCION: *La enfermedad de Leptospirosis es una zoonosis grave y una de las más comunes en todo el mundo. Ha resurgido considerablemente en perros & humanos en las últimas dos décadas en humanos ya fuese en zonas urbanas por sobre población de roedores, al practicar deportes acuáticos en zonas endémicas, y en perros por el aumento de viviendas en áreas en las que habitan portadores infectados (roedores, capibaras, mapaches etc.) aumentando el riesgo al contacto e infección y también porque en algunos países como USA, desde hace unos años se asumió que la vacunación canina contra esta enfermedad no era "core" o vital. Estudios de epidemiologia con reislamiento de espiroquetas y relacionados con serología correspondiente con niveles de más de 1:1600 MAT han sido demostrado con serovares de L. grippotyphosa en USA, Alemania y Brazil & Alemania, L. hebdomadis en Japón.*

REFERENCIAS: *Disponibles por petición a la Conferencista.*

POBLEMAS Y RIESGOS AL NO HACER DIAGNOSTICO ADECUADO

En la clínica humana, así como en la clínica veterinaria esta grave enfermedad muchas veces no es diagnosticada a tiempo por varias razones:

- Falta de sintomatología patognomónica, (la descrita ictericia no se vé en casos con infección por serovares como *L. grippotyphosa*, *L. pomona* o *L. canícola*, sino más que todo se presenta con la infección con la serovar *L. icterohaemorrhagiae*.
- Interpretación de serología MAT: Si se mandan sueros al laboratorio para hacer serología MAT, muy pocas veces se submiten muestras pareadas, pero sobre todo muy pocas veces el Clínico comprende los resultados; solo el título ≥1:1600 es indicativo de infección activa y acerca de cuál es la serovar involucrada, solo hay que tener en cuenta el resultado de los anticuerpos más elevados a la serovar correspondiente (no a todos los que muestren resultado "positivo" de 1:40, de 1:100, 1:200). Ya que no es inusual al tener infección activa por una serovar y generar respuesta elevada a esta, el tener también reacciones cruzadas (falsos positivos MAT) para otras serovares.
- A muchas personas y perros aun así sin tener diagnóstico definitivo, se les administra antibióticos, pudiendo tener una mejoría; pero si el tratamiento no es adecuado (a largo plazo y con doxiciclina) el animal puede seguir eliminando espiroquetas por la orina y sigue siendo portador intermitente.
- Cuando los niveles de BUN y la creatinina están elevados, ya hay un daño grave del riñón, la enfermedad es crónica, el animal ha estado excretando espiroquetas por mucho tiempo, aumentando el riesgo de infección a otros y la probabilidad de salvar la vida del animal es muy baja.

NOVEDADES EN DIAGNOSTICO PRACTICO

Existen kits muy buenos que detectan antígeno en la orina diagnosticando leptospiuria (sin determinar por cual serovar), junto con la correcta interpretación de serología MAT, y el cuadro clínico se puede hacer un buen diagnóstico de esta zoonosis (tendremos ejemplos de casos a discutir).

PERCEPCIONES NO ADECUADAS ACERCA DE INMUNIZACION CONTRA ESTA ENFERMEDAD:

Muchas bacterinas en el mercado no generan el mismo nivel de protección, pero algunas han demostrado experimentalmente mejor nivel de protección, con fracción preventiva mayor a 90% contra leptospiuria bajo condiciones experimentales de desafíos altamente virulentos (es decir perros en el campo nunca estarían expuestos al nivel tan elevado de infección) contra serovares *L. icterohaemorrhagiae*, *L. grippotyphosa*, *L. canicola* y *L.pomona* comparado al índice de leptospiuria en perros no vacunados. Bacterinas contra esta enfermedad han sido relacionadas en el pasado con reacciones post-vacunales, pero el tipo de reacción generalizada como urticaria con estas se veía en los años 80, pero desde entonces no se ve con la misma incidencia ya que muchos laboratorios hoy en día ofrecen vacunas con muchísimo menos contenido de suero albúmina bovino (BSA) y otras proteínas (remanentes del medio de cultivo) las que producían las reacciones adversas. El causante de reacciones locales (nódulos) que aún se ven con las bacterinas de leptospira es el adyuvante que aún está presente en algunas presentaciones, es por esto importante indagar su contenido y el nivel de protección contra leptospiuria en las vacunas que usa en su clínica.

RECOMENDACIÓN DE INMUNIZACION:

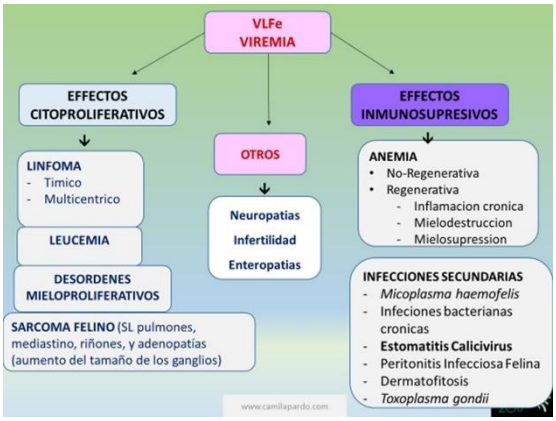
Para perros que salen a la calle, que están en su jardín, que están en contacto con cualquier otro animal están en contacto con suelo, Es por todo esto, además de ser zoonosis, que para mí la bacterina de leptospiras es una vacuna CORE anual global.

Leucemia Felina Subclinica Y Repercusiones En Salud Felina

INTRODUCCION: *En los Estados Unidos, Canadá y muchos países europeos hoy en día esta enfermedad retroviral es menos común ya que desde por lo menos dos décadas los animales infectados se pueden diagnosticar con kits que detectan el antígeno p27 del virus circulante en la sangre, y estos animales se sacrificaron o vivieron en aislamiento sin salir nunca de casa y sin ningún contacto con otro gato en la casa. Este es el manejo apropiado para evitar la difusión y reducir la incidencia de esta enfermedad. Por el efecto destructor en el sistema inmune, muchos gatos con leucemia felina sufren de una gran variedad de enfermedades y patologías felinas; es importante recordar esto durante el examen clínico sospechando así infección inicial por este virus. Ninguna vacuna en el mercado complica el diagnóstico con los kits disponibles, esto es muy importante que este claro y discutiremos la razón de esto.*

PATOLOGIAS Y MORBILIDAD POR LEUCEMIA FELINA:

La mayoría de gatos infectados morirán de enfermedades degenerativas, una minoría desarrollará enfermedades neoplásticas y proliferativas. Los gatos persistentemente virémicos sucumben a enfermedades relacionadas a VLFe dentro pocos años (2-4) después de la infección inicial. Es muy importante enfatizar que gatos con estomatitis para los que se sospecha infección por calicivirus, muy probablemente tuvieron infección inicial con VleF. El virus de FeLV se inserta en el genoma celular del gato, resultando en activación del oncogén induciendo actividad neoplásica en cualquier célula afectada. Grafico adjunto de patologías felinas por este virus.



DIAGNOSTICO:

Previamente, se creía que los gatos adultos con infecciones "abortivas" eran los que tenían una viremia transitoria seguida por el "despeje" de infección viral. Sin embargo, mejoras en la sensibilidad de la prueba de Reacción de Polimerasa en Cadena (RCP) revelo que gatos que "solo tuvieron 1 resultado positivo" y luego fueron antígeno negativo (p27 negativos), todavía tienen provirus de VLFe en sus tejidos resultando en infección regresiva. Los gatos con infecciones regresivas generalmente son avirémicos, no excretan el virus infeccioso. Así que muy probablemente pasan desapercibidos en los estudios epidemiológicos en los que solo se usa

como criterio el p27 y tampoco desarrollan enfermedades asociadas a VLFe, sin embargo, son *portadores con el potencial de reactivación y futura excreción*.

RESPUESTA INMUNE:

Después de la infección con el virus de campo, altos niveles de linfocitos T citotóxicos- CTLs específicos de VLeF, aparecen antes que los anticuerpos se formen en los gatos que se recuperaran de la exposición al VLeF. En contraste, la viremia persistente se asocia con un silenciamiento de los mecanismos inmunes del huésped (respuesta virus-específica humoral y mediada por células efectoras VLeF CTL's). Es por esto que pruebas de serología contra el virus de leucemia felina no son utilizadas para el diagnóstico de la enfermedad.

PREVENCION:

Varias vacunas están disponibles, preferiblemente usar las que generen respuesta celular y que no contengan adyuvante.

REFERENCIAS:

Disponibles por petición a la Conferencista.

Inmunizacion O Administracion De Vacunas... No Es Lo Mismo

RESUMEN: *Inmunidad celular y humoral, que tipos de vacunas generan estas. Revisaremos conceptos prácticos básicos, que son útiles para comprender la respuesta inmune a los productos biológicos.*

Para que haya Inmunización, no solo hay que analizar el tipo de producto biológico… sino el ESTADO del paciente que recibe el producto.

¿Está Sano?

¿Está estresado? Hace cuando fue destetado, separado de su camada, ¿y en un nuevo hogar?

¿Está parasitado, desnutrido?

¿Está con dermatitis?

¿Está bajo tratamiento contra la corticoides y por hace cuanto tiempo? ¿Que dosis de prednisolona puede afectar la respuesta vacunal?

¿Es muy joven y aún podría tener inmunidad pasiva maternal?

TODO puede afectar la respuesta a la vacuna.

Es por esto que antes de aplicar un producto bilógico, hay que preguntar y analizar

- *De donde proviene la mascota?,*
- *Cual es su historia vacunal?,*
- *Historia de reacciones post-vacunales previas?,*
- *Cual es la epidemiologia de la zona?*
- *Cual es el estilo de vida del paciente*

De acuerdo a su concepto médico se PRESCRIBIRA (o no) vacunar y que vacuna es la apropiada para cada paciente.

REFERENCIAS: *Disponibles por petición a la conferencista.*

Programas De Vacunacion: Puntos Clave

RESUMEN: *Hoy en día el Médico Veterinario tiene a su disposición una amplia gama de antígenos, ya sea en presentaciones, monovalentes, bivalentes, con o sin adyuvante, o de grandes combinaciones, de pequeño volumen etc., para diseñar el producto que mejor cumpla con las necesidades individuales de sus pacientes.*

Recordando un poco la epidemiologia y patología de los antígenos que producen la enfermedad se pueden seleccionar las vacunas que son necesarias solo en animales joven (por ejemplo parvovirus canina y leucemia felina) y las que siempre hay que usar (rabia, leptospira), sin importar la edad del animal y así sea que salen de casa "casi nunca" o "muy pocas veces al mes" porque estas son enfermedades zoonóticas. También discutiremos la importancia de reconocer el entorno epidemiológico, la frecuencia de vacunación en perras adultas y la influencia de la presencia de la inmunidad pasiva /la inmunidad maternal conferida por la ingestión del calostro al diseñar programas de inmunización en animales de menos de 4 meses / 16 semanas de vida. Explicare que significa tener en una vacuna un titulo alto y bajo, o nivel de pasajes alto o bajos, y si esto realmente influencia o no el diseño del plan de vacunación individual de cada paciente. Algo que tiene mucha importancia indagar, es el grado/ nivel de protección que confiere cada producto; disminuye la sintomatología?, ayuda a la reducción de enfermedad?, o previene la enfermedad?.

Ya hay "Guías de Inmunización" para perros y gatos enfocados en América Latina; COLAVAC pero hay que recordar que estas son guías, sugerencias disponibles y prácticas, pero es el Médico Veterinario la única persona que decide PRESCRIBIR O NO la administración de una vacuna, y de que tipo (inactivada, con o sin adyuvante, a virus vivo modificado, o recombinante); de acuerdo al estado de salud del animal, a su historia previa de inmunizaciones y la de su madre (en caso de cachorros), al riesgo de infección por su localidad y al estilo de vida del paciente.

REFERENCIAS: *Disponibles por petición a la conferencista.*

Índice

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Common Diagnostic Techniques and Procedures in Dogs and Cats

OBJECTIVES –

1 – Learn how to perform a myringotomy, nasal biopsy, Transtracheal wash and Bronchoalveolar Lavage
2 – Understand the potential complications of each of the procedures
3 – Discuss the potential results of these tests

Myringotomy –

Indications –

Patients with middle ear disease (otitis media).

Myringotomy allows access to the inside of the middle ear for diagnostic (obtain samples for diagnostic evaluation) and therapeutic (lavage the tympanic cavity) purposes.

Anatomy –

The ear has three portions: Outer canal (external acoustic meatus), Middle ear and Inner ear.

The Middle is a laterally compressed cavity in the petrous portion of the temporal bone. It is defined laterally from the bony external acoustic auditory meatus by the Tympanic Membrane (the ear drum) and medially from the internal ear by a bony wall in which are found two small windows, each covered by a membrane: the fenestra vestibuli above (oval window) and the fenestra cochleae (round window) below. The posterior wall of the of the middle ear has a large opening which leads into an air space (mastoid antrum). Anteriorly, the auditory (Eustacean) tube connects the middle ear to the nasopharynx. The continuity of the mucous membranes from the nasal passageways and pharynx is a natural pathway for the spread of pathogens from the nose and throat to the middle ear and mastoid antrum.

Supplies –

- Otoscope with sterilized speculum (a video otoscope is a bonus, but not required)
- 3" spinal needles (22 g) or sterile catheters (e.g. 3.5 Fr Tom Cat catheter, 3.5 Fr Feeding tube)
- Syringes for collection of samples and flushing (3, 6, 20 ml)
- 3 way stop cock
- Sterile, non-bacteriostatic saline
- Ceruminolytic agent for cleaning the ears
- Culture swabs
- EDTA tubes
- Clean microscope slides
- At least one assistant

Patient preparation –

• General anesthesia is required (use appropriate pre- and anesthetic protocols, including necessary laboratory analysis, lubricating the eyes to protect the corneas)

• In these cases inflammation of the external canal is often a hallmark of disease. NSAIDS or a short course of gluccocorticoids may be needed to help visualize the TM

• The patient should be intubated with an inflated cuff to prevent any possible aspiration of material that may emerge from the Eustacean tube during the procedure

• Position the animal in lateral recumbency (healthy ear down) or sternal (if interrogating both ears)

• Sterile drapes with a small fenestration to fit over the pinna

• Surgeon should wear proper protective gear and sterile gloves

Procedure –

• Clean and dry the external ear canal

- sterile saline or ceruminolytics (not in cats due to the risk of neurological complications or if there is a ruptured TM)
- Always flush any ceruminolytic after application
- Suction and dry the canal prior to the myringotomy

• Visualize the TM with the otoscope

• Using one of the catheters flush any remaining debris from the deep recesses of the canal using only saline.

• Using either a 22g, 3" spinal needle, or the sharply angled cut tip of the 3.5F Tom cat catheter, perform the mytingotomy by puncturing the TM at the ventral margin between the 6-7:00

• While holding this position, an assistant flushes 1 cc of the sterile saline into the middle ear. Immediately afterwards, the saline is aspirated. This can be utilized for microbial culture and cytology

• This process is then repeated several times until the fluid aspirated after flushing returns clear

• No treatment is needed to close the small hole in the TM

• NSAIDS, systemic antibiotics and topicals based on the results of the culture and cytology

Complications –

• Neurological signs (head shake, head tilt, Horner's) may result from overly aggressive lavaging, accidental over-deep penetration of the middle ear, reaction to the ceruminolytics

Nasal Biopsy –

Indications –

• Patients with evidence of nasal pathology: epistaxis, nasal discharge, sneezing, stertorous breathing, radiographic changes and surface masses or disfigurements of the nasal profile or hard palate

Caution –

• A coagulation profile is encouraged prior to obtaining nasal biopsies

• Proper dental assessments should be made to ensure that changes in the nasal cavity are not from dental disease

• Disease of the paranasal sinuses can be difficult to evaluate without invasive surgery

• Small dogs, brachcehpalic dogs and some cats are nearly impossible to adequately scope the entire nasal cavity

Anatomy –

The caudal portion of the nasal cavity is separated by a bony septum. A cartilaginous septum divides the rostral portion of the nasal cavity into left and right chambers.

The rostral chambers are each divided further by dorsal and ventral turbinates (endoturbinates). The caudal nasal chamber is filled with ethmoturbinates which extend into the frontal sinus. The frontal sinus and nasal cavity are interconnected by narrow ostia.

The caudal nasal cavity is separated from the cranial cavity by the cribiform plate, a porus bony partition that extends dorsally to the frontal bone and ventrally to the presphenoid bone.

The blood supply to the nasal cavity originates from the external carotid artery via branches of the maxillary artery.

The inaccessible paranasal sinuses include the frontal sinus, which extends approximately from the medial canthus of the eyes to the temporal line. The maxillary sinus is dorsal to the roots of the third and fourth premolars and medial to the infraorbital canal. Cats, but not dogs, have a small sphenoid sinus.

Supplies –

• Otoscope with speculum or various sized cones (a video otoscope is a bonus, but not required)

• Physiologic saline for flushing, irrigating. Pre-warmed to 37 degrees

• Gauze for packing the pharynx during the procedure

• Oral speculum or support for the head during the procedure

• Sterile, water soluble lubricant

• Biopsy instrument (flexible cup style or small rigid)

• 35-60 ml syringes for flushing

• Pathology specimen jars

• Pre-cleaned microscope slides

• Epinephrine or hemostatic agent

• 2% lidocaine

• At least one assistant

• Surgeon should wear proper protective gear and sterile gloves

Patient preparation –

• General anesthesia is required (use appropriate pre- and anesthetic protocols, including necessary laboratory analysis, lubricating the eyes to protect the corneas)

• The patient is anesthestized, then intubated and the cuff properly inflated.

• The caudal oropharynx is packed with saline soaked gauze

• The patient is placed in sternal recumbency with the head propped up on rolled towels or suspended from a cross bar to allow straight, comfortable access by the operator.

• 1 – 2 ml of 2% lidocaine is dripped into each nostril prior to inserting the otoscope or biopsy forceps

•After several minutes, gently irrigate both nasal cavities with the warm saline using the

large syringe. It may take a few tries, but generally you can flush through to the nasopharynx. Material that is flushed through the nares can be saved in EDTA tubes for cytological analysis

• If necessary, place a suction apparatus in the caudal pharynx to minimize flooding of the work space resulting from the irrigation process

Procedure –

• Always begin with the less affected side

• Coat the otoscope/endoscope with the lubricant. If a lidocaine lubricant is available, that is even better

• Start by aiming the scope dorsally under the nasal planum, then immediately redirecting ventromedially (toward the base of the opposite ear) into the ventral meatus

• At this point it may be necessary to perform additional saline irrigation

• NOTE – Even in the healthy nose the mucosa is normally friable and bleeds easily. It is not uncommon to lose visualization of the region due to hemorrhage

• With large patients and appropriately sized equipment, it is possible to interrogate all the way to the posterior nares and the nasopharynx

• After evaluating this region, retract the scope and interrogate the dorsal meatus and ethmoid turbinates

Biopsy techniques –

• ALWAYS take biopsies, brushings or swabs from multiple sites unless an obvious foreign body is found

• Flexible biopsy instruments can be inserted through working chanels or through the otoscope if large enough. If too small, then the flexible or rigid biopsy instrument can be passed alongside the scope to the region of interest.

• Always mark the instrument to the level of the medial canthus of the eyes. This line should never be passed. Have your assistant watch this mark while you are concentrating on the procedure. This will prevent inadvertent damage to the cribiform plate

• In small patients it may not be possible to pass the biopsy instrument either through or alongside the scope. In these cases it is possible to perform "blind" nasal biopsies. This is fondly referred to as the "Ram and jam" technique

• GENTLY insert a pre-marked biopsy instrument, with the jaws closed, to the level of the suspected lesion. The jaws are opened and then it is GENTLY rotated on its long axis to seat the mucosal tissue in the cutting surface. The jaws are closed, the instrument is GENTLY twisted, and then retracted.

• Patient's may sneeze even while anesthetized – so protective gear is highly recommended

• Sample 3 – 6 sites if possible

• The samples are transferred to fixative for processing

• In some cases, it may be prudent to make impression smears of the samples prior to immersion into formalin

Complications –

• Aggressive technique could damage the cribiform plate, especially if there is severe disease and existing bony damage

• Epistaxis (direct pressure, cold packs or epinephrine sprayed into the nostril will help control this)

Transtracheal wash –

Indications –

• To obtain samples from the airways (trachea, primary and secondary bronchi) for cytology and bacteriology sampling in medium to large dogs and the upper airways in small dogs and cats

• Can be performed in awake (+/- sedation) patients

Caution –

• Refractory or difficult to handle patients may need heavy sedation – caution not to oversedate so that the patient loses its cough reflex

• Patients with severe respiratory compromise may have difficulty with the procedure

Supplies –

• Supplies needed for aseptic preparation of the skin (clippers, scrub)

• Through-the-needle jugular catheter (19 – 22 g, 20 cm for dogs < 10 kg; 19 g, 30 – 60 cm for dogs > 10 kg)

• Alternate option is a 3 – 6 Fr polypropylene male urinary catheter and a 12 – 14 g over-the-needle catheter

• Lidocaine or bupivicaine

• Variety of syringes, 1, 3, 6 10 and 20 ml

• Non-bacteriostatic physiologic saline (pre-warmed)

• 3 way stop cock

• #11 scalpel blades

• Sterile plain Red top and EDTA tubes

• Pre-cleaned microscope slides

• Sterile gloves for surgeon

• Sterile dressing for the skin post procedure

• One assistant

Patient preparation –

• General anesthesia is not required

• I sedation is used, keep it light enough that the patient maintains the cough reflex

• The patient can be sitting or sternal with the head and neck pulled back and up

• The fur should be clipped from the larynx to the mid cervical region

• Sterile scrub

Procedure –

• Using the local anesthetic, block the skin and SQ at a site directly over the cricothyroid ligament or anywhere between the rings from 3 – 5 ventral to the larynx

• Stabilize the trachea with one hand and using the #11 blade, made a small stab incision though the skin in the desired location

• When using the **through-the-needle catheter**, insert the needle though the desired location into the trachea, angled down the lumen

• Advance the entire catheter through the needle then withdraw the needle

• Attach the 3-way stop cock to the end of the catheter

• Inject the warm 0.9% saline into the catheter (0.5 ml/kg)

• It is not uncommon for the patient to cough at this point

• As soon as you are done injecting, immediately aspirate back, pulling the full length of the syringe

• Repeat this process up to three times to maximize return of aspirate

• Repositioning the patient, tipping the patient into a head down position and coupaging may help increase yield

• When using the **over-the-needle catheter**, place the catheter into the lumen of the trachea as previously described, then remove the needle

• Thread the polypropylene urinary catheter through the large bore IV catheter to the approximate level of the carina (4th intercostal space)

• Attach the 3-way stop cock to the end of the catheter

• Inject the warm 0.9% saline into the catheter (0.5 ml/kg)

• It is not uncommon for the patient to cough at this point

• As soon as you are done injecting, immediately aspirate back, pulling the full length of the syringe

• Repeat this process up to three times to maximize return of aspirate

• Repositioning the patient, tipping the patient into a head down position and coupaging may help increase yield

• For either technique, when finished aspirating, remove the catheter and apply gentle pressure to the site for a few minutes

• Apply a light dressing over the region

Laboratory analysis –

• Cytology – fluid in EDTA tube and freshly made smears

• Culture of the fluid from the syringe (not from the EDTA tube)

Complications –

• If the patient becomes compromised, abort the procedure and start it on high flow oxygen immediately

• If the patient has difficulty post procedure, consider a bronchodilator

• Laryngeal or airway spasm (especially feline patients)

• Compromise to the patient from the added pulmonary fluid

• Accidental aspiration of the catheter into the trachea

• Trauma to the lower airways from the tip of the catheter

• SC emphysema

• Pneumomediastinum

Bronchoalveolar Lavage (BAL) –

Indications –

• To obtain samples from the airways (trachea, primary and secondary bronchi) for cytology and bacteriology sampling

Caution –

• Minimal sampling from the bronchioles and lower airways

• Patients with severe respiratory compromise may have difficulty with the procedure

Supplies –

• Sterile endotracheal tube

• 4 – 6 Fr. Male dog urinary catheter (polypropylene or red rubber)

• Non-bacteriostatic physiologic saline (pre-warmed)

• 3 way stop cock

• 10 and 20 ml syringes

• Sterile plain Red top and EDTA tubes

• Pre-cleaned microscope slides

• Sterile gloves for surgeon

• One assistant

Patient preparation –

• General anesthesia is required (use appropriate pre- and anesthetic protocols, including necessary laboratory analysis, lubricating the eyes to protect the corneas)

• The patient should be intubated with an inflated cuff

• Position the animal in either lateral or sternal recumbency (NOTE – if radiographs suggest unilateral disease, place the affected side DOWN)

• If there is excessive mucus or discharge in the oral cavity, wipe with gauze and flush with saline

• Prior to intubating the patient, measure the length of the catheter compared to the length of the ET. Mark the packaging of the catheter so that when inserted, the end protrudes through and past the end of the ET by at least 4 – 6 cm

Procedure –

• Insert the sterile ET after induction – be careful to avoid oropharyngeal contamination of the end of the tube by touching the oral cavity

• Carefully cut the end of the packaging from the sheath of the catheter

• Advance the catheter through the ET by pushing the outside of the packaging – thus avoiding surface contamination of the catheter as it passes through the ET

• Attach the 3-way stop cock to the end of the catheter

• Inject the warm 0.9% saline into the catheter (0.5 ml/kg)

• As soon as you are done injecting, immediately aspirate back, pulling the full length of the syringe

• Repeat this process up to three times to maximize return of aspirate

• Repositioning the patient, tipping the patient into a head down position and coupaging may help increase yield

• If you can add supplemental oxygen during the procedure it would benefit the patient

• If the patient becomes compromised, abort the procedure and start it on high flow oxygen immediately

• When the aspiration is completed withdraw the catheter and place the patient on oxygen until recovery

• If the patient has difficulty post procedure, consider a bronchodilator

Laboratory analysis –

• Cytology – fluid in EDTA tube and freshly made smears

• Culture of the fluid from the syringe (not from the EDTA tube)

Complications –

• Laryngeal or airway spasm (especially feline patients)

• Compromise to the patient from the added pulmonary fluid

• Accidental aspiration of the catheter into the ET

• Trauma to the lower airways from the tip of the catheter

Fracture Repair in Reptiles

INTRODUCTION: *Fractures in captive reptiles are common, usually being secondary to primary nutritional deficiencies. Specifically, pathological fractures frequently occur as a result of Nutritional Secondary Hyperparathyroidism (NSHP), which is a general lack of dietary calcium, excessive phosphorus or deficiency in exposure to ultraviolet light/vitamin D3. Even traumatic fractures, which under normal conditions with healthy bones would not occur, are more likely due the generalized osteopenia associated with NSHP.*

Extremity fractures are rarely compound or comminuted. As a result, most fractures are readily treated with external coaptation. In addition, since most fractures are often associated with demineralization and softening of the bones, internal fixation is usually not indicated. In the unlikely event of a traumatic fracture involving normal bone, internal fixation can be utilized.

Regardless of the etiology, nutrition and diet should be thoroughly evaluated in all fracture cases. Before attempting any repair calcium homeostasis should be established. The medical management in these cases is equally as important as the surgical attention.

OBJECTIVES –

1 – Understand the most common causes of fractures in reptiles
2 – Learn the best techniques on how to repair fractures
3 – Discuss expected healing timelines for fractures in reptiles.

GENERAL CONSIDERATIONS

Frye states that most fractures occur as a result of low impact forces, thus making the incidence of comminuted fractures uncommon. In addition, due to their relatively inelastic skin, open or compound fractures are infrequent.

Little information is available on fracture healing in reptiles. No controlled studies have been conducted. Most of the information that is known comes from anecdotal reports relating treatment successes/failures in cases of NSHP. It is generally accepted that reptilian bone heals slower that either mammalian or avian bone, requiring from two to eighteen months to completely heal.

When planning fracture repair in reptiles general principles of orthopedic management apply. Proper alignment, rigid stabilization, minimal disruption of soft tissue and conservation of the blood supply is paramount. The forces acting on the fracture (bending, rotation, compression and shear) must be evaluated and neutralized to promote rapid healing. In general, the more forces that must be neutralized by the type of fixation, the higher the incidence of complications and failures.

Additional considerations when deciding upon type of fracture repair include the patient's functional requirements (pet lizard in a terrarium vs. a Komodo dragon being returned to the wild), cost limitations set forth by the client, the cost and availability of the required materials and the experience of the veterinarian.

Most long bone fractures will heal in time with nothing more than strict cage rest. Although there may be some severe

malunions, these complications do not seem to affect captive reptiles in an adverse manner.

The size of the patient and its nutritional state may have a direct impact on the type of fixation required. Large, heavy bodied lizards and turtles may require internal fixation, whereas small, delicate lizards may do well with a light splint.

The general condition of the patient often plays a major factor in the selection of fixation methods. In many of these NSHP animals it is physically impossible to utilize any type of internal fixator, as the bones just are not physically strong enough for the implant to gain purchase.

As in anything in veterinary medicine, the dollar is often the deciding factor in final determination of fixation technique. Internal fixation carries a higher price tag due to the cost of the materials, the time necessary for application and the training of the surgeon. Although internal fixation may be the best for the patient, it is not always an option.

EXTERNAL COAPTATION

External coaptation involves the use of splints, slings, casts and any other technique needed to immobilize a fracture. This is by far the most commonly utilized technique in reptilian fracture repair. In general, the best splints/casts are those that are lightweight and comfortable for the patient. If the patient's activity is restricted lightweight splints/casts are effective.

When treating pathological fractures secondary to nutritional disease external fixation is the treatment of choice. NSHP is the most common disease presenting to reptilian veterinarians, and most frequently seen in the Green iguana.

Bone is a dynamic organ, undergoing constant remodeling. During prolonged hypocalcemia/hypovitaminosis D, the mineralization process lags behind the formation of organic bone matrix, resulting in the formation of hypomineralized bone. When this occurs in young, growing animals it is called rickets, and in adults, it is known as osteomalacia. Pathological fractures occur when the calcium content decreases to approximately one-third of its baseline. Aside from pathological fractures of the long bones and appendicular skeleton, soft, swollen mandibles and long bones (fibrous osteodystrophy), stunted growth, deformed heads and abnormalities in ambulation are common.

These bones are too soft to provide support to the implants used in internal fixation techniques. IM pins, cerclage wires and bone screws all penetrate, crush and pull out when used in these wax-like bones. An IM pin may be utilized for alignment in long bone fractures, but when used, it should be in conjunction with external coaptation.

Once the calcium homeostasis is corrected the healing progresses rapidly, with a bony callous forming in about three to four weeks. Correcting management and husbandry deficiencies and providing proper dietary and supplemental calcium is needed. In addition, treating the patient with synthetic salmon derived calcitonin helps speed recovery by inhibiting the actions of parathyroid hormone, blocking the actions of the osteoclasts, stimulating the osteoblasts and providing bone analgesia. 50 IU/kg of calcitonin, IM in the triceps, administered q 1 week for two treatments is the recommended dosage. It is important that the patient is eucalcemic prior to the administration of the calcitonin.

There have been numerous methods reported in the literature for external coaptation in reptiles. There is no one right way. Whatever technique works best in

your practice situation is the best method to use. The most important thing to remember is that the best splints/casts are the lightest and most comfortable to the patient.

When applying external coaptation remember that the patient is most likely in pain. Anesthesia or sedation is recommended for patients that struggle or if extensive manipulation of the fracture(s) is required.

The initial padding around the limb can be performed with many different types of bandage material (Specialist Cast Padding, Johnson & Johnson, New Brunswick, NJ; Conform, Kendall Co., Boston, MA). Make sure that the padding is cut to the appropriate width to prevent bunching of the padding around the joints.

Tape stirrups should be incorporated into the padding when applying the splint/cast to prevent slippage. It is not uncommon for the splint/cast to slide down the leg after the cast padding compresses.

This padded limb can now be reinforced by adding aluminum rods, tongue depressors and light weight casting material. It is important to conform the shape of the splint/cast to the natural angles of the limb. This will prevent the development of fracture disease, or periarticular fibrosis, in the immobilized joints.

Veterinary Thermoplastic (IMEX Veterinary, Inc., Longview, TX), Hexcelite (Hexcelite Medical, Dublin, CA) and Orthoplast (Johnson & Johnson, New Brunswick, NJ) are rigid at room temperature, but malleable when heated in a water bath. The Veterinary Thermoplastic is easy to apply when heated and cools to make a rigid splint. It comes in different sizes and thicknesses, making it convenient for different size patients.

Splints/casts can be easily applied to any of the long bones in lizards. When applying splints/casts it is important to follow general principles of fracture stabilization. The joints both proximal and distal to the fracture should be immobilized.

For both humeral and femoral fractures a modified spica-type splint must be used. The splint should incorporate the distal joint, and then have a portion that crosses over the body. For the femur, the band should cross cranial to the vent so that it does not interfere with elimination. In humeral fractures, the band can cross diagonally across the chest, passing between and under the front legs.

Chelonians can also be splinted, but modifications in technique are required. It is usually not possible to apply a splint to a proximal long bone (humerus/femur). These bones can be reduced (with sedation/anesthesia as needed) and then taped into the leg opening in the shell. I recommend covering the limb with cast padding to add stability to the "set limb" before taping over the opening. I also recommend taking a radiograph of the leg folded up within the shell to make sure that fracture alignment is appropriate.

Splints/casts do not provide rigid fracture fixation. As a consequence, fracture healing is not as rapid as it would be with a plate or external fixation device. However, the bone will heal.

I recommend re-checking the fit of any splint/cast within one week of the initial application. You should always check for slippage, swelling of the distal extremities and pressure sores. Splints/casts are usually left on for a minimum of four, and usually six to eight weeks. Follow-up radiographs should be taken at four weeks, and again when the cast is removed.

INTERNAL FIXATION

Internal fixation is warranted for long bone fractures in reptiles where external coaptation is not a practical option. Large, heavy, active and otherwise healthy reptiles all do well with internal fixation. Internal fixation techniques utilized in mammals and approaches to the long bones are similar to those employed in reptiles.

Steinmann pins, Kirschner wires, spinal needles and stylets can all be used as IM pins in reptiles. In addition, these devices can all be used as parts for External Skeletal Fixation (ESF). ESF can be used in a variety of fracture types in reptiles of all sizes.

When using these delicate implants as a part of the ESF, the external connecting bar and clamps are replaced by a methylmethacrylate polymer. This is inexpensive, easy to use and light.

Pin loosening is a common problem with ESF. Whenever possible it is recommended to use threaded pins. The threads should be applied to the outside of the pin, not cut into it.

Bone plating can be utilized, but in general requires a larger patient. Cuttable plates (Synthes, Paoli, PA) with 1.5 mm diameter screws can be applied to bones as small as 3 mm diameter. Finger plates are also applicable in certain situations.

In general, plates do not need to be removed. IM pins and ESF should be removed when there is radiographic evidence of bone healing. In some cases a fibrous union may be all that is needed to ensure eventual healing, thus allowing the removal of loose pins as needed.

AMPUTATION

When there is severe tissue trauma, loss of blood supply or granulomatous infection in the limb, fracture repair may not be a viable option. Amputation of either the fore- or hind limbs is a viable option in reptiles, as they do quite well with three limbs. Amputation of digits or limbs can be accomplished with excellent cosmetic and functional results.

When amputating limbs it is best to remove the entire appendage. Disarticulation at either the scapulohumeral or coxofemoral joints is recommended. Limb muscles are transected distally and then elevated proximally. The joint is exposed and the limb removed. The muscle bellies are then sutured over the joint space to provide soft-tissue padding. Nerves can be transected with a scalpel and injected with bupivicaine to provide local analgesia post-operatively.

In chelonians after a limb amputation it may be necessary to provide some sort of prosthesis. A block of wood, a plastic skid or a furniture roller can be glued to the plastron to aid in locomotion.

ANALGESIA

As healthcare providers we have to assume that any patient suffering a fracture must be experiencing some type of pain or discomfort. A thorough discussion of analgesia is beyond the scope of this presentation, but, those treating reptiles with such injuries should address these concerns. NSAIDs and narcotics should be considered.

Local Analgesia in Dogs and Cats

INTRODUCTION: *It used to be believed that "Pain keeps them quiet!" in reference to our veterinary patients. If you provided pain relief, the patient would be active and potentially cause itself harm. Fortunately, that old school thinking is not longer acceptable. Analgesia, or the "absence of pain" is mandatory for all procedures in veterinary medicine, whether it is something minor like a small mass removal, or for major surgery.*

Local analgesic blocks render complete anesthesia to the surgical site – meaning that there is no sensation of pain. The sensation of pain is alleviated or even eliminated for the duration of the block. Local anesthetic drugs work by blocking sodium channels in nerve membranes. Decreased permeability to sodium slows the rate of depolarization so that the threshold potential is not achieved and an action potential is not propagated, thus the pain impulse is not propagated.

It is always best to prevent pain – that is – prevent the phenomena called "wind up." This is done by utilizing local analgesia prior to or before a surgical procedure, so that when the patient recovers, it immediately feels less pain and discomfort. The clinician does not have to "chase" the pain that the patient experiences when it awakens, thus, providing for a more comfortable post operative period.

Of significance, when utilizing pre-operative pain medications, especially local analgesics, less general anesthesia is needed. This is referred to as "multimodal analgesia/anesthesia."

OBJECTIVES –

1 – Understand the need and benefits of Local Analgesia
2 – Learn about the different drugs commonly used
3 – Learn the most common procedures used

ADVANTAGES OF LOCAL ANALGESIA

- Lower cost
- Minimal systemic side effects
- No or minimal recovery period
- Safe in high-risk patients
- Decrease MAC of inhalant anesthetics
- Prevents "wind up"
- Overall better patient pain scores

COMMONLY USED LOCAL ANESTHETIC DRUGS IN VETERINARY MEDICINE INCLUDE

- Lidocaine

- Onset of action: rapid (less than 5 minutes)
- Duration of action: 60-120 minutes
- Dose 2-6 mg/kg (use the lower end of the dose in cats)
- Convulsive dose in dogs: 11-20 mg/kg
- Lethal dose in dogs: 16-28 mg/kg
- 'Toxic dose' in cats reported as 6-10 mg/kg
- The general recommendation for clinical use is ≤ 6 mg/kg in the dog and ≤ 3-4 mg/kg in the cat.

- Bupivacaine

  - Onset of action: approximately 5-10 minutes after injection (up to 20 minutes)
  - - Duration of action: 4 to 6 hours
  - Dose 1-2 –(4) mg/kg (use the lower end of the dose in cats)
  - Toxic dose in dogs: 5-11 mg/kg or potentially any amount given IV
  - Data is mostly anecdotal in the cat but the general

feeling is that 3 mg/kg is the toxic dose.

- The general recommendation for clinical use is ≤ 2 mg/kg in the dog and ≤ 1 mg/kg in the cat.
- • Augmentation
- - Additives such as epinephrine, hyaluronidase, bicarbonate

ADVERSE EVENTS CAUSED BY LOCAL ANESTHETIC DRUGS:

Extremely rare but can include any of the following:

Main disadvantages are:

- Does NOT provide restraint or sedation
- Can cause nerve damage
- Overdose possible

- Local tissue effects – swelling, bleeding, inflammation, 'paresthesia? (unknown if this occurs in animals).
- Anaphylaxis – rare
- Central nervous system – muscle tremors, seizure, coma
    - At lower concentrations, depression of inhibitory neurons occurs and can cause cerebral excitation, which may lead to seizures. At higher concentrations, profound CNS depression with subsequent coma, respiratory arrest and death can occur. The latter is more likely following IV boluses of large doses.
- Cardiovascular system – the myocardial conduction system is sensitive to local anesthetics and IV boluses can result in cardiovascular collapse.
- ONLY LIDOCAINE CAN BE ADMINISTERED IV (and never with epinephrine).
- Methemoglobinemia – rare, but can occur in cats.
- Motor and autonomic nerves are also blocked by local anesthetics, and so motor weakness and vasodilation may occur with certain techniques.

ALWAYS CHECK DOSE – LOCATION – ASPIRATE PRIOR TO ADMINISTRATION!

Three common uses for local analgesia

- Topical analgesia (densensitization and analgesia to the skin)
- Local tissue analgesia (infiltration)
- Regional analgesia (IV or Epidural)

Examples of Topical Analgesics

- Analgesic creams
- 5% lidocaine patch
- Topical Sprays
- Proparacaine HCl drops
- Tetracaine HCl drops
- Physical coolants – local HYPOTHERMIA (Ethyl Chloride)

LOCAL TISSUE (INFILTRATION) ANALGESIA

• Field Blocks

- Non specific

- Skin, Nerves, Periosteum, M. membranes >> SQ, Fat, Muscles, Bone
- Debriding abscesses, removing skin tags, small masses

• Line Blocks (Ring Blocks)

- Precise
- Administered between site of invasion and spinal cord
- Post operative incisional blocks

• Nerve Blocks (e.g. Dental Blocks)

- Most Precise
- Small amount of drug needed
- Nerve locators available

Regional Analgesia

• IV ((Bier block)
• Plexus block
• Intrathecal
- morphine
- lidocaine

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Radiology in Reptiles

OBJECTIVES –

1 – Learn the best techniques to produce diagnostic images
2 – Learn radiographic anatomy
3 – Evaluate radiographs from clinical cases

Some form of imaging is mandatory in reptilian diagnostics. While certain clinical skills such as auscultation and palpation may be useful in some species like snakes and lizards, others, like most tortoises, are a literal black box when it comes to physical examination.

Radiology is the most common diagnostic tool in reptilian imaging, followed closely by ultrasound, and more frequently, computed tomography and magnetic resonance imaging. Nuclear scans and other contrast procedures are also gaining use. Unfortunately, where in mammalian medicine we have years of experience and "normals" to rely on for comparison, in the field of reptilian imaging there are still a lot of "seat-of-the-pants" interpretations. There is the added difficulty of obtaining high-resolution, diagnostic-quality films through the thick, often ossified dermis. With the advent of digital imaging, this obstacle has been less of an issue.

Fortunately, many of these problems can be overcome with practice and patience. Patience is probably the most important ingredient in successful reptile medicine, and in reptilian imaging, it is absolutely essential.

RESTRAINT

As with small mammals, many reptiles can be easily radiographed without sedation. Turtles and tortoises will often withdraw into their shells in novel surroundings, thus making positioning easy. However, some will walk endlessly regardless of their placement. These animals can be placed in a radiolucent container such as a lucite restraint box, or in a simple alternative such as a rigid cardboard box with the bottom cut out.

If an animal is restless, it can often be calmed by placing an inverted bucket, small trash can, or cardboard box over the pet for a few minutes. By leaving the animal in a dark, quiet place, it will often calm down long enough to take the radiograph. The plate should be marked, the beam centered, and the control unit set. When everything is ready, the rotor is started and the cover is removed. The radiograph is exposed before the animal has a chance to move.

A simple vagal response can be elicited by taping cotton or gauze over the patient's eyes. Many times this can be used in lieu of chemical restraint.

I personally like telazol (tiletamine/zolazepam) for brief restraint in my reptile patients. Some with less experience using the drug prefer other medications such as dexmedetomidine, morphine, ketamine, propofol, and others. Regardless, make sure that patient's condition is stable and that it is able to handle any chemical immobilization. Remember, it is more important to have a

live patient than a dead one with quality radiogaphs.

POSITIONING

Many of the same radiographic positions used in mammal medicine apply to reptiles. Perhaps the one big difference is the preference of the dorsoventral (DV) view in herps over the ventrodorsal view commonly taken in mammals.

CHELONIANS

The DV view in turtles and tortoises is useful in evaluating osseous integrity, overall conformation, gastrointestinal disorders, and urinary tract (specifically, bladder) abnormalities. It is of little use in evaluating the respiratory system.

To properly evaluate the lung fields and air sacs, horizontal beam radiographs must be taken. Some references advocate using a vertical beam and just spatially positioning the animals as is performed with mammals. However, in the experience of this author better visualization of the pulmonary spaces can be obtained with the use of horizontal beams. Cardboard boxes, lucite pedestals, and foot stools all work well to place the animal in a position for a horizontal beam.

Unfortunately, with some of the newer digital radiographic units, it is not possible to capture horizontal beams because the imaging plates are in a fixed position. In these cases, patient positioning becomes paramount.

Measurements for establishing radiographic techniques in turtles and tortoises can be a challenge. Technique charts vary with the machine used, but, in general, settings that are adequate for skull settings for dogs provide proper penetration of the osseous shell in chelonians. The shell should be measured at its widest part (just cranial to the rear legs), its highest point (at the crown of the carapace), and its maximum length (from the caudal most portion of the carapace to the gular notch).

The craniocaudal and lateral view horizontal beams permit good visualization or the air spaces. Evaluation of the “fullness” of the gastrointestinal system is possible, but detail of the gastrointestinal structures are lost in these views. Animals in good flesh will have filled intestinal loops. In anorectic or cachectic patients, the air space seems uncharacteristically large.

Extremity views (including the head and neck) of chelonians are usually overexposed when using techniques for evaluating the shell or internal structures. Additionally, many of these animals are so confined within their shells, the scutes from either the carapace or the plastron obscure the limbs from view. It is necessary to extend these body parts for proper measurements and exposures. Light gauze tied to the extremity can be used to extend the body part for a brief time to allow exposure. Gentle traction is more effective than brute force.

However, in some chelonians, it is not even possible to gain access to the limbs. In these patients, it may be necessary to use sedation to get the appropriate views.

SNAKES

Snake radiology is technically much easier than in chelonians. The DV and lateral views are necessary for proper assessment. Avoid the pitfall of coiling the snake in the bottom of a plastic bucket and making a quick exposure of a coiled patient. The DV can be obtained much the same as with a tortoise. Place the patient in a radiolucent, rigid restraint tube (these come in various sizes for various size snakes). The inside diameter

of the tube should approximate the circumference of the patient. This will prevent the snake from turning back on itself within the tube, or "kinking" during the radiograph. When the snake is safely restrained within the tube, it can then be positioned for DV, lateral, and oblique radiographs. This technique is invaluable when working with venomous or dangerous animals.

Radiographs of small snakes in a straight line (as when they are placed in a restraint tube) can often be performed on a single plate. DV views of an entire animal can be accomplished by allowing the patient to curl up in the bottom of a box or bucket, providing that the animal doesn't overlap on itself.

Larger patients need to be radiographed sequentially. This is especially important with animals many feet in length, because many sequential body sections are nearly identical. Each film should be marked with a sequencing number on each side of the plate (eg. 1–2, 2–3, 3–4, etc.) A small piece of labeling tape, placed directly on the patients scales, works well for this. A small area of overlap should be included on each film. If possible, the lateral views should be included in the same orientation and position on the same plate next to the DV views.

LIZARDS

Radiology in lizards presents the greatest challenge to the reptilian practitioner. Not only can these animals be very skittish, many of them have such diverse body shapes that normal positioning is nearly impossible.

Many of the tricks for restraint mentioned for tortoises and snakes can be employed for lizards. Patience and gentle handling will allow positioning for most DV views. Some animals must be covered with lucite boxes to prevent them from darting off the radiology table. Sedation and tranquilization may be warranted if proper positioning cannot be accomplished by standard techniques.The body conformation of many of the large body lizards allow for basic DV and lateral views. However, some of the lizards, such as the monitors, are dorsoventrally flattened, thus making consistent lateral radiography difficult. Even with this technical difficulty, a lateral view should always be a part of a radiographic survey.

CONTRAST STUDIES

Contrast studies are often indicated in the same way as in small animal medicine. Thirty percent weight to volume barium sulfate suspension is the most commonly used contrast medium for gastrointestinal tract studies. Approximately 25 ml/kg given by gavage tube into the crop is the suggested guideline. Radiographs are taken when contrast is given and 30 minutes, 1, 2, 4, and 24 hours later. In some of the larger tortoises, gastrointestinal transit time may be as long as 1 week, so daily exposures may be indicated. Double contrast studies, use of organic iodine mediums in GI studies, intravenous excretory studies, and urography have been also described.

SUMMARY

A working knowledge of reptilian anatomy is essential for proper evaluation of radiographs. Fortunately, reptilian anatomy is fairly basic compared to mammalian anatomy. Written descriptions of pathology are no substitute for actually visualizing radiographs of normal and clinical conditions. Once a basic level of competency and confidence has been attained when performing radiology in

these animals has been attained, the practitioner can then utilize the various special techniques commonly employed in mammalian radiology to enhance visualization of the various organ systems. Gastrointestinal barium contrast studies, venography, bronchography, etc can all be performed just as is done in dogs and cats.

Radiography can greatly augment the reptilian practitioner's diagnostic capabilities. As with anything in non-domestic pet medicine, it is just a function of taking our knowledge of domestic animal internal medicine and adapting it to the peculiarities in the species of our special interests.

RECOMMENDED READING

1. McArthur S, Wilkinson R, Meyer J, eds. *Medicine and Surgery of Tortoises and Turtles*. Ames, IA: Blackwell; 2004.

2. Rübel GA. *Atlas of Diagnostic Radiology of Exotic Pets. Small Mammals, Birds, Reptiles and Amphibians.* Prescott, AZ: Wolfe; 1991.

3. Silverman S. Diagnostic imaging. In: Mader DR, ed. *Reptile Medicine and Surgery*. 2nd ed. St. Louis, MO: Elsevier; 2006:471–489.

Table 1. Recommended radiographic exposures for reptiles.

	Dorsoventral*	Craniocaudal+	Lateral+
Chelonians			
Respiratory tract		✓	✓
Digestive tract	✓		
Genitourinary systems	✓		
Carapace and plastron	✓	✓	✓
Skeleton	✓		
Snakes			
Respiratory tract			✓
Digestive tract	✓		✓
Genitourinary systems	✓		✓
Skeleton	✓		✓
Lizards			
Respiratory tract			✓
Digestive tract	✓		✓
Genitourinary systems	✓		✓
Spine	✓		✓
Extremities	✓		

**Vertical radiograph beam.*

+Horizontal radiograph beam.

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Table 2. Species of monitors (*Varanus* species) in which mineralized hemibacula are present within hemipenes.

Mineralization present	Mineralization absent
V acanthurus (spiny-tailed)	*V bengalensis* (Bengal)
V beccarii (black tree)	*V dumereli* (Dumeril's)
V cVaudolineatus (stripe-tailed)	*V exanthematicus* (savannah)
V eremias (desert pygmy)	*V griseus* (desert)
V giganteus (Perenty)	*V mertensi* (Merten's water)
V gilleni (Gillen's pygmy)	*V niloticus* (Nile)
V gouldii (Gould's)	*V rudicollis* (roughneck)
V indicus (mangrove)	*V salvator* (water)
V karlschmidti (peach-throat)	*V timorensis* (Timor)
V komodensis (Komodo)	
V olivaceus (Gray's)	
V panoptes (Argus)	
V prasinus (green tree)	
V salvadori (crocodile)	
V storri (Storr's)	
V tristis (freckled)	
V varius (lace)	

Structures are easily visualized on radiographs and can be used for sex determination. Reproduced with permission: Mader DR. Reptile Medicine and Surgery. St. Louis, MO: Elsevier; 2006.

Why won't my Reptile Eat ?

OBJECTIVES –

1 – Understand the feeding response in reptiles
2 – Learn the most common causes of anorexia
3 – Discuss treatment options

There is no question that the most common health problems associated with captive reptiles are diet related. These can be from nutritional deficiencies, such as is so commonly seen in the Green Iguana, excessive calorie consumption, as in overweight animals, or the most common problem, anorexia, or a lack of appetite.

There are numerous causes for a reptile to lose its appetite. These underlying causes must be identified and corrected before the problem will be resolved.

When an animal is "off-food" there has to be a reason for it. It may be psychological, or it may be medical. Although this may seem elementary to most readers, it bears mentioning. It is essential that the veterinarian understands the natural history and biology of the particular type of reptile that they are treating. If they do not know, they must have a ready reference source. If one is not available, then, ethically, they should refer it to someone that has the proper training.

In order to more easily arrive at a proper diagnosis, a brief discussion of the normal feeding response is in order.

An animal normally eats to satiety. The cessation of the feeding response prior to the satisfaction of caloric needs is termed anorexia. Clinically, this is referred to as an "absence of hunger."

The body is naturally, continuously in a state of hunger. Eating is the process that satisfies or controls that hunger state. The hunger and satiety centers are located in the brain.

Stimulation of the lateral hypothalamus initiates the feeling of hunger. Activation of the ventromedial hypothalamus results in satiety. Several neuroendocrine and metabolic factors affect these feeding control centers. Of clinical importance, the senses of taste and smell play major roles in the triggering of these responses.

There are several factors that play a role in anorexia. The major task of the small mammal clinician when faced with an anorectic reptile is to determine whether the condition is caused by pathologic, physiologic or psychologic embarrassment.

There are many diseases that can disrupt the normal neurologic, endocrine or mechanical processes involved in the feeding response. A pure division into categories is not possible, as many diseases have components that overlap. In some situations, the cause is obvious (such as gross malocclusion in turtles), and in others, elusive (cancer cachexia).

Categorizing signs and laboratory data may help in assessing the problem. In general, anorexia may be classified as either primary or secondary with respect to disease. In addition, there is a third, more category, called pseudoanorexia, which is not directly related to suppression of the feeding centers in the brain.

Primary anorexia should be considered in any case where the inciting factor directly involves the feeding centers of the hypothalamus, or from psychological

disorders that have a direct impact of neural control of the feeding response.

Any cranial injury or insult, such as trauma, cerebral hemorrhage, cerebral edema or hydrocephalus (acquired or congenital) may cause anorexia. In humans, severe headaches, such as migraines, may be directly responsible for appetite suppression. Diseases or pathology within the cranial vault, such as encephalitis/meningitis or neoplasia, can have a direct or indirect effect on the hypothalamus.

Any such condition, whether primary to the hypothalamus or merely affecting the hypothalamus, may also have other neurological manifestations in addition to anorexia. Thus, a thorough neurological examination, as part of a complete physical evaluation of the patient, is imperative.

Psychological disorders are more easily characterized in people, as our veterinary patients are less likely to articulate their emotional state. As a result, at the risk of anthropomorphizing, it is often necessary to attempt to interpret what the anorectic patient may be "feeling" in a given situation. For instance, although an owner may enjoy taking their pet reptile to the movies, the reptile may respond differently to the darkened, air conditioned interior of the theater, the bright flashing lights of the projector, the laud responses of the audience and the artificially buttered popcorn.

Anorexia nervosa, a disease common to young human females, has not been documented in animals. "Maladaptation Syndrome," a condition common to recently captive animals, may have some psychological or physiological similarities, but, this is not a likely problem in the captive reptile.

Any external influence that incites stress or anxiety (identified as fear or depression in people), such as changes in the environment (temperature, caging, air exchanges, noise etc.) can result in anorexia. Two common psychological influences include the alteration of social structure within the animal's environment (addition of a new animal to an established group) and the offering of a new food type.

Secondary anorexia includes diseases or influences from outside the brain and have a direct effect on the neuroendocrine control of hunger. Some conditions or diseases may produce signs associated with anorexia such as nausea and vomiting (although the latter is not seen in reptiles). It is believed that the stimuli associated with these conditions are similar and the controlling centers within the brain are most likely neuronally interconnected.

Abdominal pain is a common cause of anorexia in reptiles. Constipation may contribute.

Inflammatory conditions, such as coelomitis, hepatic, renal, pancreatic or visceral inflammation can all lead to anorexia by directly or indirectly stimulating the appetite centers.

Exogenously or endogenously produced toxins can affect the appetite by either directly affecting the feeding centers, or indirectly by affecting other areas of the body, such as the abdominal organs. Drugs and toxins can also affect the chemoreceptor trigger zone which produces nausea and anorexia, or can act directly on the hypothalamus.

Endogenously produced toxins, such as azotemia or hyperammonemia, as seen in renal or hepatic failure, respectively, have serious consequences on appetite.

Hypercalcemia, by yet an unidentified factor, also leads to anorexia.

Neoplasia and cachexia are frequently associated together. However, oftentimes, the cachectic patient still has an appetite. Cancer patients may not always desire food, as the peptides and nucleotides associated with certain neoplastic diseases are known to cause anorexia. Neoplasia should be on the differential list for anorectic patients.

Miscellaneous causes of anorexia should include any systemic illness. Cardiac, pulmonary or pancreatic disease (eg. diabetes - although not well documented in reptiles) can be contributing factors.

Lastly, pseudoanorexia, which is a physical inability to eat, rather than the lack of desire to eat, must always be considered with the clinically anorectic patient. Dental disease, or malocclusion as seen in many turtles, is a frequent contributing factor in reptiles.

A thorough physical examination is warranted for every case, including cases with apparent obvious explanations for the anorexia. In addition, a proper cranial nerve examination must be conducted. An open mouth oral examination (using sedation as needed) must be performed.

Equally important as a the physical findings is the collection of a thorough history. Discern if there have been any changes to the animal's environment, including caging, food, conspecifics, ambient temperatures etc.

Radiographs, laboratory analysis (including complete blood counts, serum chemistry analysis and urinalysis) should be a part of every minimum data base.

Anorexia and food deprivation has serious consequences on a patient, especially to those that are convalescing.

Tissues such as the brain, the red blood cells, renal medullary cells and neural tissue has an obligate glucose requirement. To maintain blood glucose, body protein is rapidly affected. The ramifications of food deprivation are far beyond the scope of this discussion, but it is obvious that it will have serious consequences.

Anorectic reptiles need to be supported with appropriate diets. Appropriate gruels can be administered via syringe feeding, and when necessary, nasogastric tubes. Attention must be given to the animal's fluid balance and other medical needs, such as the administration of antimicrobials and analgesics, as required.

Índice

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Avian Pediatrics

INTRODUCTION: *Veterinary continuing education is filled with discussions surrounding an infinite variety of illnesses and appropriate medical approaches for such. Less time appears to be spent on basic husbandry and veterinary participation in the care of "normal" animals. Increasing numbers of exotic pets presented to veterinarians have illustrated the tremendous need for information regarding the basic husbandry of exotic animals being kept as pets.*

Pet birds probably comprise the largest group of exotic pets. Years ago most parrots, macaws, cockatoos, etc. were imported as adults or juveniles. With these birds came problems associated with the stress and crowding experienced by the birds during quarantine. Now that importation no longer exists most birds supplied to the pet trade are bred within the U.S. and sold at the consumer level as babies. A large proportion of individuals acquiring these babies is grossly under-educated and inexperienced in caring for them. To add to the complexity, little information has been disseminated to owners or to veterinarians on proper handfeeding and weaning practices.

BASIC HUSBANDRY

The vast majority of avian pediatric cases presented at Avian & Exotic Animal Medical Center are the result of incorrect feeding practices. Many novice owners are told "the crop should be filled each time it empties until the baby becomes self-sufficient". Problems arise from the lack of a definition for "full crop" and a failure to recognize what constitutes reasonably "empty". Sometimes owners are simply directed to feed a certain volume a certain number of times each day. Novice owners will force babies to eat specified volumes at regular intervals regardless of the signals displayed. The weaning process exacerbates this scenario because weaning age babies have completed their log phase of growth and require substantially less nutrition than a younger bird. If babies are over-fed at this age they may not become hungry enough to desire other food. In some cases a baby will attempt to resist a feeding only to have the formula forced upon him. A struggle such as this often ends in tracheal aspiration of the formula with resultant aspiration pneumonia or asphyxiation.

Thirty years of personal experience has resulted in a feeding and weaning protocol which drastically reduces the number of feeding related problems. The maximum volume of formula that should be fed to any baby prior to actual weaning should approximate 10% of his body weight. The feeding interval (the length of time between feedings) is determined by the amount of time that it takes the crop to empty. An empty crop is defined as one in which little or no food can be palpated in the crop, although the crop may remain slightly pendulous. Once a day (preferably at night) the crop is allowed to remain empty for an additional 33-50% of the calculated interval, providing one longer period for complete emptying. For example, if the crop empties every 4 hours, the feeding interval would be 4-5 hours from 6 A.M. until midnight, with at least a six-hour period of time for extended crop emptying. This allows residual food (with increased numbers of bacteria) to be eliminated (and provides the

feeder with often-needed rest.) As the baby grows, the absolute (not relative!) volume will increase while the frequency decreases. Recent experience suggests that in some species it may be better to maintain frequency and decrease volume as babies grow. Most importantly, ***never should the volume per feeding exceed 10% of the baby's weight*.** As the baby matures a time will come where a feeding will be resisted. The baby may initially be responsive, but it will then resist and retreat. At that point, all feedings are permanently reduced in volume to the quantity consumed eagerly. When feedings are being administered three times a day, and the quantity is being reduced, solid food in the form of softened pellets or table food is introduced. Solid food will usually be consumed over the next 2-3 weeks allowing cessation hand feeding.

Failure of a baby to thrive with the above protocol often suggests illness. When a baby refuses food, it is critical to note the manner in which it resists. It is perfectly normal for a juvenile to act eager to eat then refuse food. He may accept some formula then spit it out and refuse any more. He may even run when approached. He simply has reached a plateau of growth where his nutritional demands are drastically reduced. This must be contrasted with the baby who is depressed, inactive, and shows no interest in food. Crop stasis is often accompanies illness in babies and may be the first indicator of a real problem. *A depressed baby bird demonstrating crop stasis is a medical emergency*.

Crop stasis is one of the most common legitimate reasons for the presentation of juvenile psittacines to a veterinary practice. While "sour crop" is the term most often used to describe the condition, rarely is the crop the problematic organ. The vast majority of babies presented for "sour crop" are actually experiencing illness unrelated to the crop. Lower gastrointestinal disturbances, chlamydiosis, bacterial septicemia, or metabolic diseases such as hepatic lipidosis are all examples of conditions that may present with crop slowing or stasis as a part of the clinical picture.

FIRST AID

The presentation of a sick psittacine baby is often featured by the presence of a large pendulous crop full of spoiled hand-feeding formula. The term "sour crop" is descriptive of the condition of the crop contents at presentation but rarely is it a disease in itself. Food that has stagnated in the crop spoils similarly to food which has remained sitting unrefrigerated in a warm environment for several hours. The bacterial density of this formula becomes excessive while bacterial toxins accumulate. Regardless of the reason for the stagnation the spoiled food becomes a significant source of pathogens and the toxins produced by them. In order to stabilize the patient this material must be removed.

In most patients the spoiled formula can be removed through a feeding tube passed orally. Depending on the particle size of the formula, either a standard red rubber or a ball-tipped metal feeding tube can be introduced into the crop. The crop contents can than be aspirated by direct suction. It is sometimes necessary prior to aspiration to thin the spoiled material by introducing warm water or electrolyte solutions into the crop. The contents can then be mixed by palpation and aspirated. It is important to palpate the tube in the crop during aspiration to prevent the crop wall from being suctioned against the end of the feeding tube. Once the crop has been reasonably emptied it should be lavaged by

repeatedly filling it with a warm balanced electrolyte solution, massaging the crop and mixing its contents, and aspirating the fluid until clear.

The vast majority of babies presented for crop stasis will be moderately to severely dehydrated. The lack of fluid intake from the static crop combined with the continued high fluid losses that accompany much pediatric illness results in fluid deficits that can be life threatening. Once the crop contents have been removed it is necessary to tend to the fluid needs of the patient. Handling a baby with a full crop to administer I.V. or I.O. fluids can easily precipitate regurgitation with subsequent tracheal aspiration. Subcutaneous fluids may be beneficial prior to crop washing, but once the crop is empty I.V. or I.O. fluids are preferred.

Psittacine babies that have not fully feathered often require environmental temperatures of 29°-32°C (85°-90°F). Those that are ill are even less able to thermoregulate. Care should be taken to provide hospitalized pediatric patients with adequate environmental heat. High humidity should be maintained to avoid contributing to dehydration.

Pharmaceuticals other than broad-spectrum antibiotics are not usually indicated in the initial care of pediatric illnesses. Regardless of the primary etiology, the bacterial overgrowth in the crop and the remainder of the gastrointestinal system (GI) must be addressed. While antifungals may ultimately be useful, antibacterials are far more urgently needed in acute pediatric illness. Occasionally antifungals may prove to be more appropriate, but rarely is fungal pediatric disease acutely fatal, whereas bacterial illnesses are often rapidly fatal if not quickly addressed.

Avian Restraint and Physical Exam

During the initial encounter between veterinarian and patient, a number of very important dynamics occur. The patient is approached; eventually it is captured and consequently restrained. Ultimately, the patient is examined by the veterinarian so its condition may be assessed. Various diagnostic and therapeutic procedures may be rendered in the process. While the veterinarian is usually focusing on the physical exam, it is the handling of the patient by the doctor that the client is most aware of.

For the veterinarian, the physical exam is one of the most important aspects of his initial visit with a patient. This is when the veterinarian develops a first impression of the patient's condition, and much depends on his or her ability to detect even the subtlest of details. This first encounter between veterinarian and patient is extremely important for the client as well, but for very different reasons. This is where the client develops a first impression of the veterinarian. Everything the veterinarian does with the patient suggests to the client how thorough, perceptive, knowledgeable, etc. the veterinarian is. For this reason the veterinarian is judged not only by how well he relates information, but also for how well he relates to the animal. Nothing can destroy the confidence an owner has in a veterinarian faster than incompetent handling of the patient during an exam. Therefore, before the physical exam may ensue, the patient must be captured and restrained with minimal distress to both the patient *and the owner*.

Capture of the patient may be effected through a few basic techniques. Rarely does a pet bird surrender willingly to the entrapment of the handler. Almost always, some form of mechanical assistance, in the form of a towel, net, etc. must be utilized.

The most obvious means of handling large psittacines (capable of biting off a digit of the handler) is a pair of thick gloves, such as welder's gloves. This is also probably the single worst method of handling a pet psittacine. The gloves can be traumatic, they prevent the handler from adequately monitoring the patient's movements and resistance, and they teach the bird to be afraid of hands. There really is no place for gloves in pet bird medicine. Gloves are a standard part of falconry, but never are they used to restrain a raptor. Instead, they are used to protect the falconer's hand as it functions as a perch for the bird.

A net is occasionally useful in an avian practice. There comes a time when a flighted bird is able to evade capture by even the most skilled handler. High ceilings or large rooms allow an escapee to stay just beyond reach of those in pursuit. A long-handled fine-mesh net allows the extension of reach necessary to corner the bird. A short-handled net also may be useful to corner and trap an especially quick escapee. The fine mesh is essential to prevent tangling of the bird's legs, wings, or head.

The single most useful tool in a bird capture arsenal is a towel. A regular terrycloth

towel can be tossed lightly over an unsuspecting patient temporarily disorienting him and allowing his capture. As the towel is quickly but smoothly draped over the bird the handler takes hold of the patient's neck from behind. The handler's hand encircles the neck somewhat stretching apart the head and shoulders. The bird's neck is held by one hand while its torso and legs are encircled through the towel by the other hand. The wings are usually naturally kept at the bird's side by the weight of the towel. Once the bird is under control the legs are held from the *anterior* aspect while the head and neck continues to be held from the *posterior* aspect, and the bird is stretched as much as possible without it being injured. A snugly restrained bird resists much less than one held timidly.

A small bird or a cooperative large bird may be captured with bare hands. Always, the bird should first be captured from the rear and behind the neck. When the handler's hand acts as a wide snug collar around the bird's neck, it is practically impossible for the bird to bite the hand that holds him. Even the bird's feet will have difficulty reaching up to the restraining hand when the bird is properly held.

An aid to the capture of quick patients is darkness. Turning off the lights in a closed exam room may temporarily freeze the bird's attempts to escape. The handler can position himself near the patient, an assistant can turn off the lights when prompted, and the bird can be captured in darkness. This can be quite effective whether a net, towel, or bare hands are employed.

Once the patient is restrained the physical exam should be completed as quickly and atraumatically as possible. All details of all visible aspects of the patient should be noted. The best way to ensure completeness in a physical exam is to adopt a step-by-step routine in which the assessment of every feature of the exam is planned. When a checklist-type approach is used, oversight is less likely to occur.

The best approach is one in which a logical progression is followed through the patient. It may be best to begin with general observations such as respiratory rate and character, cardiac auscultation, etc. in order to ensure that the patient is in no danger from the exam itself. Once vital signs are noted the exam may proceed in whatever sequence the examiner chooses, provided the sequence is complete and logical. Randomly examining the oral cavity, then the legs, then the ears, etc. will inevitably cause omissions of anything from small details to large body parts. Regardless of the presenting clinical signs, omissions should not be allowed. More than one bird has been presented for a nasal discharge only to have a cloacal problem discovered. Once the exam is complete and the bird is released, his response to handling should be noted. Often, poor stress tolerance will be a major indicator that illness exists.

The physical exam should always take into account physical features of the bird's visible environment. The bird's droppings, feathers lost in the cage, spots of blood on the floor, etc. all may reveal important information about the patient. Every detail is a significant detail, even if only to document normalcy.

Clinical Appearance of Avian Viral Disease

INTRODUCTION: *Viral disease in avian species is much more significant than once believed. In the early days of avian medicine, bacteria were cited as the primary causes of avian illness. It was eventually recognized that bacterial disease in birds is often secondary to malnutrition, stress, or other predisposing factors. In recent years, it has become obvious that viruses are extremely prevalent and significantly pathogenic in certain avian populations.*

A practitioner is constantly faced with the challenge of trying to determine the etiology of a clinical presentation. While almost no illness can be diagnosed based on clinical signs, certain diseases provide visual cues that should lead a practitioner to suspect a viral etiology. Never should the possibility of a viral etiology be dismissed; too much is at stake in a facility where others could be exposed to potentially fatal disease. Advance warning of the presence of a certain viral disease however allow action to be taken which more directly and efficiently allows an illness to be characterized and appropriately treated.

PAPOVAVIRUSES: POLYOMA

Polyomavirus is currently one of the most threatening of all avian pediatric diseases. It appears in two primary forms based on affected species: Budgerigar Fledgling Disease and non-budgerigar Psittacine polyoma infections. Both presentations affect neonates most severely and are characterized by peracute to acute death in pre-weaning babies. Prominent feather signs and carrier states that commonly occur in budgies are rare in other psittacines.

Budgerigar Fledgling Disease may appear as sudden death or death following a brief illness with depression, cutaneous hemorrhage, feather abnormalities, and abdominal distention. If babies are infected later than a few weeks of age, they may exhibit feather dystrophy. "French Molt" is a mild to fatal condition of budgies in which the majority of flight and contour feathers are markedly dystrophic. Polyomavirus is one cause of this condition. While individual birds may clear themselves of the infection, the virus may circulate through the flock causing the flock itself to act as a carrier.

In all other psittacines Polyomavirus generally produces either rapid terminal illness or transient inapparent infection. Again, the age at which the bird is infected determines how it is affected. The younger the host, the more serious and rapid the disease.

In non-budgerigar psittacines less than 16 weeks of age the infection is usually fatal. Birds 3 to 6 weeks old may die without clinical signs. Those 5 to 16 weeks old often display sudden widespread ecchymosis visible in random patterns throughout subcutaneous regions. Most often the hemorrhage is seen along the ventral aspect of the neck where normal feeding reflexes cause rupture of the fragile vasculature. Bleeding may be observed in the absence of other clinical signs although some degree of depression, anorexia, crop slowing, regurgitation, etc. usually occurs. The vast

majority of babies that die of polyoma do so at fledging, the period when flight feathers have matured and contours are emerging.

Young birds between the ages of 16 and 21 weeks of age demonstrate variable response to polyoma infection. Anything from subtle feather dystrophy to fatalities with characteristic signs may be observed. The maturity and condition of the immune system probably determines the severity. Birds which are malnourished or weakened by other ailments are more likely to fall victim to the serious effects of the virus. Once birds exceed five months of age most will experience a brief viremia with or without obvious signs and fully recover from the infection. In fact, evidence indicates that there are far more subclinical adult infections than fatal neonatal ones. Unlike budgies, the carrier state in other psittacines is undetermined. No doubt, non-budgie psittacines can transmit the virus, but it is unclear whether these are transient versus latent infections.

Antemortem diagnosis of polyoma infection involves DNA probes of cloacal/choanal swabs or whole blood.

Necropsy of deceased babies usually reveals random areas of profound hemorrhage, usually in the subcutaneous spaces. Musculature and internal organs may be extremely pale due to exsanguination. Other findings include hepatomegaly, pericardial effusion, splenomegaly, and ascites. Diagnosis is confirmed through a DNA probe of affected tissues. Histopathology may reveal hepatic necrosis with karyomegaly and intranuclear inclusion bodies in the liver and spleen. The bursa of Fabricius may be depleted of lymphocytes. Vascular necrosis in many areas explains the hemorrhage and transudation.

Treatment of affected patients is purely supportive. Survival once hemorrhage is visible is unknown. Prevention depends on minimizing exposure and vaccination. Babies older than 3 weeks of age may be vaccinated every two weeks until they are 9 weeks old. Protection is significant after 7 weeks of age. Those vaccinated after that period require one initial vaccination and one booster at 2 weeks. Vaccination of adult birds is controversial but undoubtedly would help prevent circulation of the virus through a susceptible flock.

PAPOVAVIRUSES: PAPILLOMA

Papillomaviruses have been identified in many mammalian species as causes of isolated epidermal masses. The common wart in humans is a typical example of the well defined pedunculated growth that occurs. These growths appear as small fleshy pedunculated masses originating primarily from featherless areas such as the feet of face. Their clinical significance depends on mechanical effects.

PAPILLOMATOSIS

The most common association of papillomavirus with disease in pet birds involves “papillomatosis”, granulamatous masses that develop in the cloaca, choana, oropharynx and to a lessor degree other areas of the gastrointestinal tract. Fact is, papillomavirus has never been identified in these lesions. In fact, no virus or any other infectious agent has ever been identified as the cause of this syndrome. Although some

evidence supports an infectious etiology, other situations produce conflicting evidence. For example, the introduction of an affected bird in certain populations has resulted in increased prevalence of the disease in that population, while in other situations the mates of affected birds have remained unaffected.

The clinical significance of papillomatosis depends in part on the location of the lesion. Large granulomas in the cloaca may reduce breeding potential although in one aviary the highest production came from the isolated papilloma colony. Secondary bacterial infections are common in protruding inflamed tissue. Aside from the mechanical effects, papillomatosis has been suspected to be related to the development of bile duct carcinomas. Also, some association has been suggested with the Herpes virus of Pacheco's Parrot Disease.

In recent years, evidence strongly suggests that the etiologic agent may be a Herpes virus. Birds exhibiting papillomatosis have demonstrated antibodies to parrot Herpes. Also, many birds suffering from cloacal papillomatosis have eventually developed bile duct carcinoma, a condition thought to be related to Herpes virus infection.

Because the agent has not been confirmed, means of control are not clearly established. Isolation and hygiene are probably effective. Flock closure has not prevented the disease from occurring. Pronounced lesions may be excised but recurrence is likely.

PSITTACINE CIRCOVIRUS

For many years the cause of Psittacine Beak and Feather Disease (PBFD) was unknown. The suspected etiologies included autoimmune disease, endocrine disorders, infectious agents, etc. Ultimately a virus was discovered representing the smallest class of viruses known to infect animals. These viruses are currently classified as Circoviridae.

The primary site of viral replication in the avian host is epithelial cells. As with polyoma, the severity of disease depends on the species of bird involved and the age at which he is infected. Birds more than a few months old do not develop clinical disease but rather experience a transient viremia, then clear the infection. In some species, especially juvenile African Gray Parrots, the virus may cause fatal peracute disease attacking primarily the thymus and cloacal bursa with no epithelial component. Typically however the epithelium of growing feathers and to a lessor degree the epithelium of the feather follicle, beak, and nails is affected. Clinical signs are entirely related to the age of exposure and the extent of epithelial damage. The hallmark of PBFD is the occurrence of deformed, stunted feathers many of which are strangulated at the base and fall out prematurely. The percent of plumage affected depends on what stage of molt the bird is in at the time of infection. Baby birds producing their first growth of plumage may show no normal feathering while an older bird already beyond the juvenile molt may demonstrate scattered feather dystrophy. Evidence indicates that birds of any age showing clinical signs were in fact infected a very young age. Incubation is minimally 4 weeks but may be as long as months to years. Onset of clinical signs correlates to the onset of significant molting.

A variation of PBFD is the peracute illness seen frequently in African Gray Parrots, among others. It is characterized by sudden depression and anorexia with death occurring within one to five days after onset. These birds demonstrate profound anemia with variable leukopenia. Although birds have been known to survive this form of the disease, it is usually fatal. One survivor confirmed by DNA probe demonstrated a PCV of 4 at the peak of illness.

Diagnosis of PBFD is accomplished with a DNA probe of blood or epithelium. Histopathology of developing feather shafts or follicles demonstrates both intranuclear and intracellular inclusion bodies. It is imperative that diagnosis be confirmed; birds have been euthanized which ultimately proved to have disease not related to psittacine circovirus. Also, asymptomatic adults testing positive to the DNA probe but showing no clinical signs may mount an effective immune response to the virus and entirely clear the infection. Euthanasia of these patients is not warranted, but strict quarantine is.

Control of PBFD centers around eliminating clinically affected individuals which are the sources of infection for susceptible individuals. A vaccine does not currently exist so preventing spread of the disease is the only means of control. Clinically normal individuals which test positive should be isolated until a subsequent test is negative. Those testing positive and demonstrating typical feather signs are unlikely to recover and euthanasia may be warranted. No successful therapy exists and these individuals shed inconceivably high numbers of viral particles posing tremendous threats to susceptible babies.

POXVIRUSES

Poxviruses are the largest and most diverse group of viruses known to infect avian species. Unlike some of the other viruses, poxviruses are highly host specific and severity of infection is highly dependent on the species of both host and virus involved.

Pox infections occur in three forms which are represented by particular species in the pet bird population. The cutaneous form consists of discreet 2-4mm crusts that appear on the eyelids and feet and is frequently seen in lovebirds. The diphtheritic form is characterized by ulceration and the formation of pseudomembranes in the oral cavity and upper airways, a common finding in Amazon Parrots especially when importation was practiced. Canaries commonly suffer from the worst form, a fatal septicemia.

Transmission of avian poxviruses is highly dependent on precipitating factors. The virus can be destructive once introduced into the host but it is unable to penetrate intact epithelium. In order for the virus to gain entry there must occur a break in the epithelium such as a wound or insect bite. As such, mosquitoes pose a serious threat in transmitting the disease.

The severity of illness depends on the manifestation of disease and the degree of secondary problems. The cutaneous form is rarely fatal unless it produces a viremia. The diphtheritic form may result in fatalities due to oropharyngeal discomfort and inability to eat leading to starvation. Viremia produces hepatic necrosis, myocarditis, pneumonia, air sacculitis and peritonitis.

Diagnosis is accomplished by visualizing the pathognomonic "Bollinger" inclusion bodies via histopathology. Vaccination is available for some species but is not commonly practiced. Minimizing exposure to mosquitoes and avoiding other causes of epidermal disruption greatly reduces incidence. Treatment is directed at secondary problems.

HERPESVIRUSES

The viral infection most frightening to many aviculturists is "Pacheco's Parrot Disease", or avian herpesvirus. Pacheco's disease has been known to kill as many as 7000 psittacines in one outbreak. Its ability to strike quickly and with little or no warning and its relative disregard for age or species has caused many an aviculturist to awake to large numbers of dead specimens.

Herpesviruses are one of the most ubiquitous viruses in nature. In most cases in the animal kingdom, the viruses exist in a latent stage shedding periodically with few or no clinical signs. In psittacines it is believed that there are pathogenic and non-pathogenic strains. At the very least, the virus may remain dormant for an extended period of time surfacing during periods of stress. Historically, Patagonian and Nanday conures are accused of being the primary carriers of this disease, but almost any psittacine is capable of surviving an outbreak and becoming a permanent carrier.

Clinical signs of a Pacheco's outbreak often don't exist. When they do they are extremely brief, vague, and consist simply of listlessness, depression, anorexia, and yellowing of the urate portion of the droppings. The sudden appearance of yellow urates with death occurring in less than 24 hours should send chills down the spine of any aviculturist. The vast majority of outbreaks witnessed by the author have begun this way. In contrast to traditional beliefs, most of these outbreaks have lasted 3 - 7 days and resulted in no more than 20% of the flock being lost. Reports of outbreaks support the variability of expression. Flock losses range from one bird to 100% of the colony.

Transmission of herpesviruses is typically through close contact. Husbandry and hygiene may have an influence on spread of the disease. Outbreaks may follow the introduction of a carrier into a flock although most arise somewhat spontaneously or after periods of increased stress. Virulent strains of the virus produce death in 3 - 10 days after introduction. Any bird that survives infection is believed to become a carrier. The virus which infects psittacines does not affect non-psittacines. Not all psittacines develop disease when infected. Some acquire the infection in the absence of clinical signs and become carriers.

Diagnosis is based on clinical features and necropsy samples. Deceased birds display an enlarged yellowish-brown liver, splenomegaly, and vascular congestion in almost any organ. Histopathology demonstrates necrosis, congestion, and hemorrhage within the liver, spleen, and kidney. Intranuclear inclusions may be demonstrated in the above organs as well as the pancreas and esophagus. Although the inclusions are suggestive they are not pathognomonic for Pacheco's disease. Confirmation is achieved with electron microscopy, cell culture, Antigen detection, or DNA probe.

Of all the viral diseases, Pacheco's is the one which responds to some degree to antiviral therapy. Acyclovir has been used effectively in outbreaks to reduce the duration and severity of the disease in the flock. The greatest benefit is obtained in birds not yet showing clinical signs. Those already showing clinical are not likely to survive even with treatment.

Prevention depends on vaccination and avoiding exposure, but neither is foolproof. There is no way to guarantee that a carrier does not exist within an aviary. Colonies which have been closed for four years have broken with Pacheco's. A vaccine is available which affords some protection, but it is likely that serotypes exist apart from that included in the vaccine and therefore not affected by it. The best means of preventing large losses from Pacheco's is to practice good avicultural hygiene. Birds in a collection should always be handled in a manner which minimizes the spread of any agent from bird to bird.

PARAMYXOVIRUS

In the early 1970's The USDA imposed restrictions on the importation of exotic birds. Paramyxoviruses are a large and very diverse group of viruses, one of which causes spectacular losses in domestic poultry flocks. This virus, paromyxovirus type 1 (PMV-1), is not endemic in the United States but was known to have been introduced via birds imported for the pet trade. Even though legal importation is essentially non-existent, smuggling still creates the possibility of an epidemic in domestic poultry populations. Other paramyxoviruses exist in avian populations in this country, but none present the threat that PMV-1 does. PMV-2 and PMV-3 cause variable illness, if any, in some passerines and psittacines.

Paramyxovirus type 1 is more easily recognized by the familiar name of "Newcastle Disease". Four classifications of disease exist based severity of illness. In order of increasing severity they are lentogenic, mesogenic, velogenic, and viscerotrophic velogenic. It is the latter which destroys poultry flocks. Poultry display acute diarrhea, respiratory distress, and neurologic signs with death occurring within a few hours. Psittacines infections are usually less severe and appear as conjunctivitis, rhinitis, diarrhea, depression, torticolis, tremors, paralysis, and seizures. Often the clinical signs may escape detection or be inapparent.

Transmission occurs via virus laden secretions which can be passed directly or indirectly. The virus survives well outside the host making insects, pests, and man possible vectors. Incubation period is 3 to 28 days. Birds with inapparent infections or those recovered from illness may shed virus for as long as one year. The virus is zoonotic and can cause vague illness with conjunctivitis in man.

Gross lesions vary from none to cardiomegaly, splenomegaly, hemorrhage, pulmonary and tracheal congestion, and edema of the respiratory and gastrointestinal systems. Microscopic lesions reflect hemorrhage, edema and necrosis of the described systems as well as the brain. Intranuclear or intracytoplasmic inclusion bodies are rare and found in the brain. Diagnosis is confirmed with virus isolation, paired serum samples, or electron microscopy.

Control of paromyxovirus in poultry is through vaccination, but this vaccine may be fatal in psittacines.

PMV-2 and especially PMV-3 are the paramyxoviruses endemic to avicultural populations in the U.S. PMV-2 causes mild to no illness in passerines but more serious disease in psittacines. Illness is nonspecific and includes tracheitis, pneumonia and enteritis. PMV-3 causes vague illness in psittacines and is common in grass parakeets. Diagnosis is as for PMV-1.

AVIAN BORNAVIRUS

Blue and Gold Macaws were the first species reported to suffer from a disease in which the proventriculus became paralyzed and dilated resulting in wasting away and death of the bird. Thus "Blue and Gold Wasting Disease" eventually acquired the names Psittacine Wasting Syndrome, Proventricular Dilatation Syndrome, Neuropathic Gastric Dilatation, Splanchnic Neuropathy, and others. "Proventricular Dilatation Disease" (PDD) is the term currently employed, the etiology of which is avian Bornavirus.

Birds with terminal PDD usually present with three characteristic signs: vomiting, weight loss, and passage of undigested food in the droppings. Another form of the disease which often goes undiagnosed is a peripheral weakness manifested by decreased leg strength when perching or unsteadiness when ambulating. Weakness may occur with or without proventricular involvement. Finally, a large percentage of birds may harbor the virus while never showing clinical evidence of its presence.

Necropsy in gastrointestinal cases may reveal a proventriculus enlarged to the capacity of the abdomen and thin-walled enough for ingesta to be seen through the proventricular wall. Pathology of deceased individuals demonstrates an accumulation of lymphocytes and plasma cells in the gastrointestinal tract, spinal cord, and brain. Necropsy of other Borna infections may show no gross lesions at necropsy. Suspect submissions should include brain and spinal cord.

Because the epidemiology and pathogenesis of PDD has not been completely proven, hygiene and careful management is the only means of prevention. Most birds showing gastrointestinal signs of this disease die. Rare cases have demonstrated classic signs and survived. Treatment has been directed at feeding highly digestible, low bulk foods, and controlling secondary infections, dehydration, etc. NSAIDS such as Celebrex may improve the prognosis of affected individuals.

CONCLUSION

The most important characteristic for a serious avian practitioner to posses is open-mindedness. Most of the viral diseases described here were defined only after years of clinical encounters in which the etiology remained unidentified. It is likely that in the future new viruses will be discovered amidst problems being experienced today.

Therapeutic Avian Techniques

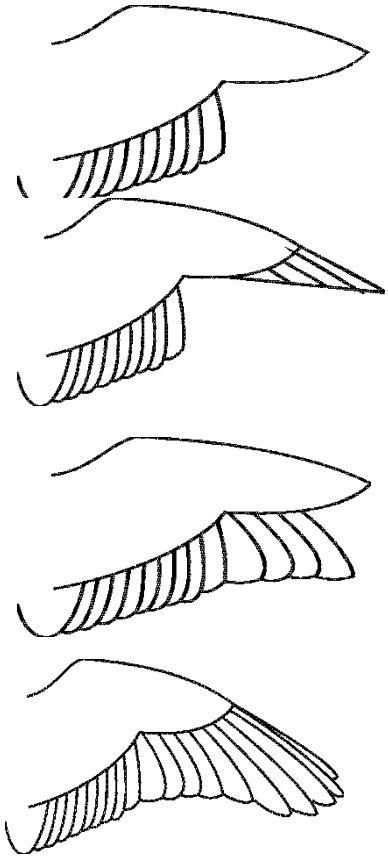
INTRODUCTION: *The practice of avian medicine is highly mechanical. While it may be obvious to anyone that a bird is dehydrated and in need of intravenous fluids, not everyone will be prepared to install a catheter and fulfill that need. Administering fluids intravenously to a patient weighing as little as 15 grams is challenging. Knowing what to do in a given situation is not enough; being able to accomplish various tasks involving avian patients often determines success or failure.*

GROOMING

While maintenance of the avian patient's cosmetic features may not always be therapeutic, it can in some cases constitute preventative maintenance. Wing clipping may deter escape and prevent injury from ceiling fans, windows, and other potential dangers. Overgrown nails may require trimming to prevent avulsion from entrapment of the nail in caging or fabric.

Wings

As simple as wing clipping appears, it has been the subject of more controversy than any other mechanical technique in avian medicine. Everyone who uses a particular method seems to believe that his or her technique is the only correct one. In reality, only one thing must be accomplished: the bird's wings must be trimmed in a manner that prevents flight but does not allow injury. No one technique is appropriate for all birds. Clipping more than a few primary flight feathers from an African Gray's wings may cause it to fall too rapidly resulting in injury. Cockatiels may practically require wing amputation to prevent flight. Trimming one wing versus two is a matter of personal preference, except possibly in cases where asymmetric landing (to avoid keel impact) is desired. Birds which traumatize the fleshy wingtips should probably not have the first primaries clipped too closely. The one rule which may be consistent in most (not all!) cases is that prevention of flight requires clipping of a variable number of ***primary*** flight feathers only. Rarely is it necessary to trim secondary flight feathers, and ***never*** should secondaries be trimmed ***instead*** of primaries. Figure 1 illustrates commonly employed patterns of wing trimming.



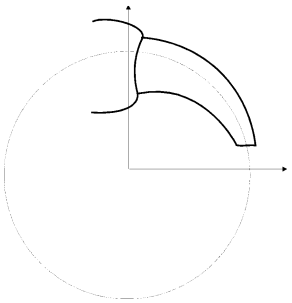


Figure 1

Nail trimming is necessary to minimize direct (i.e. entrapment, tangling) or indirect (i.e. arthritis, decubitus ulcers) injury to the bird or to minimize owner discomfort during handling of the bird. Three methods are routinely employed: clipping with standard human clippers, cutting with a cautery tool, and grinding with a handheld grinding tool. The length to which the nail should be trimmed is usually at about that level where the nail forms a quarter-circle (figure 2).

Clipping with human clippers is preferred for smaller species (canaries to cockatiels) because the human clippers trim the nail much more cleanly than conventional cat or dog trimmers. If the nail's vasculature is encountered it can be controlled with chemical cautery such as ferric subsulfate or silver nitrate. Human clippers can be useful in larger birds for removing the sharp tip of the nail, especially in juveniles.

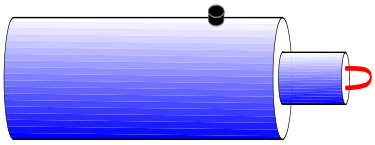


Figure 2

The cautery tool sometimes used consists of a fine U-shaped wire attached to a battery handle (figure 3). The wire becomes red hot and melts through the nail cauterizing as it cuts.

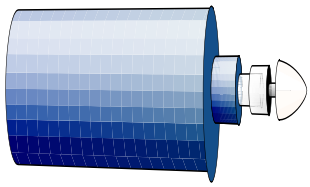


Figure 3

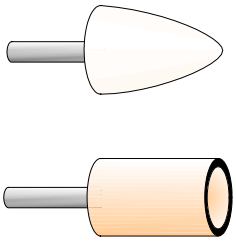


Figure 4

The electric hand grinding tool (figure 4) is extremely useful in larger birds. It quickly cuts, cauterizes, and smoothes nails as they are trimmed. A conical sanding bit has traditionally been used, but the

author prefers a cylindrical bit with a concave end (figure 5). The latter offers the advantages of a defined edge for cutting and a concave end into which the tip of the nail can be inserted for smoothing. When using either of these bits, bleeding is usually controlled by cauterization from heat generated in grinding. If bleeding still occurs it can be controlled by applying ***light*** pressure with the bit, ***heavy*** pressure with the grinder's collet, or simply using chemical cautery.

Beak

A normal beak never needs trimming. It is a misconception that pet birds' beaks require routine trimming. The length of a bird's beak is self regulating through normal occlusion, and the thickness is controlled through surface wear. Two conditions require corrective action: malocclusion and dorsal hyperkeratosis.

Malocclusion occurs as lateral deviation (scissors beak), prognathism, or brachygnathism. Causes include genetics, injury, infection, and malnutrition. In some cases, especially younger birds, shaping the beak with the grinding tool may permanently correct the deformity. Care must be taken not to enter vital areas when shaping. More often than not, malocclusion requires surgical intervention or repeated trimming at regular intervals.

Dorsal hyperkeratosis is the frequent and direct result of a "concrete perch deficiency". A bird rubs the dorsal surface of the beak on the perch to remove accumulating keratin, which accompanies normal beak growth. Many an owner has commented on the pet bird constantly "cleaning" his beak on the perch. Standard wooden dowels supplied in cages lack the abrasiveness necessary to wear down the normal keratin buildup. Mineral blocks and lava rocks sold for such purposes do not suffice. Use of the concrete perch available in most retail pet shops has tremendously reduced the incidence of this problem. When the condition is observed in a clinical setting, the excess keratin can be scratched away with a thumbnail or sanded off using the grinding tool with a conical bit.

Respiratory Support

Oxygen can be extremely beneficial in the early stages of critical care. Respiratory emergencies certainly require oxygen administration, and since many critically ill patients are acidotic their conditions can improve with oxygen supplementation. The method of administration depends on the primary problem.

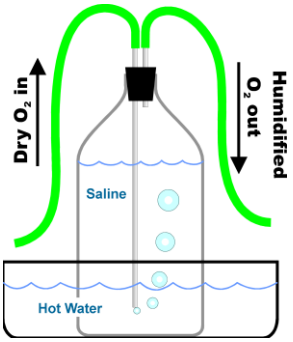


Figure 5

When possible, it is best to humidify and warm the oxygen prior to delivery to the patient. This is best accomplished by bubbling the gas through warmed isotonic or half-strength saline solution. A canister can be devised by using rigid tubing and an empty I.V. fluids bottle immersed in warm water (figure 6).

Chamber

Any patient that can benefit from oxygen administration can initially be rested in an enclosed container into which oxygen is delivered. Although commercial chambers are available for this purpose, even a cardboard box will suffice. All that is

required is that the oxygen be somewhat contained so as to increase its atmospheric concentration.

Commercially manufactured intensive care units offer the advantage of being able to supply heat and humidity in addition to oxygen. The unit can be kept in a "ready" configuration so the oxygen, heat, and humidity are immediately available in an emergency. In situations where extreme hypothermia is part of the presentation, increased humidity minimizes the risk of rebound hypovolemia caused by warming the periphery of the patient before the body core. Effective warming humidity can be provided by placing the bird on a grid over a pan of moderately hot (not scalding) water in the ICU.

Mask

Oxygen can be supplied via a typical anesthesia mask. The cone can be placed over the heads of large birds while small birds can be placed completely into the cone as though it were a chamber. Care should be taken that the patient does not struggle and aggravate its already fragile condition.

Air sac cannula

Tracheal obstructions from foreign bodies, neoplasia, fungal granulomas, etc. initially require the creation of an alternate breathing passage. The existence of the air sac system in birds provides a means of ventilation not possible in mammals. Effective respiration can be achieved by intubating the caudal thoracic air sac.

Anesthesia is helpful in birds that are capable of resisting restraint. Those which are severely dyspnic may offer little resistance and the urgency of establishing effective respiration may preclude anesthesia. It is critical that the patient be evaluated for the cause of the dyspnea if possible; air sac cannulation is life saving for tracheal causes of dyspnea but it is contraindicated for pulmonary causes.

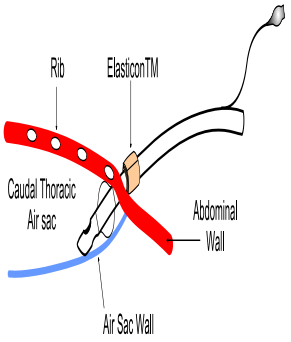


Figure 6

The type of tube utilized depends on the size of the patient and the urgency of the situation. In small birds a 2-3 cm section of I.V. tubing will suffice. In larger birds a standard 3.0 mm I.D. cuffed endotracheal tube can be modified for abdominal installation. The tube is trimmed just above the air line thereby preserving the integrity of the cuff. A 1 X 3 cm strip of Elasticon™ is wrapped around the endotracheal tube 2-3 mm above the cuff. Inflation of the cuff after placement in the bird offers the advantage of securing the tube in place and more importantly expanding the air sac thereby improving the patency and effectiveness of the tube (figure 7).

The breathing tube can be installed into the caudal thoracic air sac either between the last two ribs or just behind the last rib, just dorsal to the dorsal edge of the pectoral muscle. The patient is secured in lateral recumbency and the area is surgically prepped. The leg is ***flexed and abducted*** (not pulled cranially or caudally) to expose the last rib. A stab incision is made through the skin with the point of a #15 scalpel blade. A fine mosquito hemostat is used to bluntly dissect through the intracostal or abdominal muscles forming a hole barely large enough to insert the breathing tube. The tube is inserted and secured either by suturing or inflating the cuff, or both. Patency can be tested by

holding a microscope slide at the opening to observe for breathing-induced fogging. Once secured, the bird can breathe freely through the tube or an air line can be connected for oxygen administration or anesthesia.

Nutritional Support

Maintenance of nutrient intake is critically important in avian patients. Due to their high metabolic rates, negative effects of starvation occur quickly. When a bird fails to eat due to illness or injury, it must be nutritionally supported by force-feeding directly per os, via a gavage tube, or through an indwelling alimentary catheter.

Composition of supportive formulas is a matter of individual preference. Commercial products are available that provide calories and other nutrients for ill patients. Pelleted diets can be fine-ground and mixed with water or electrolyte solutions. Hand-feeding baby formulas can also be utilized. When determining a feeding schedule it is extremely important to consider and meet the patient's fluid needs.

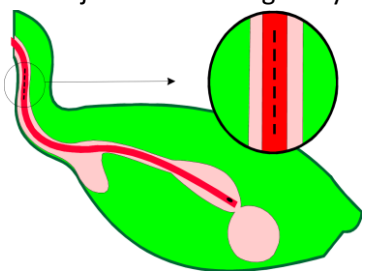
Birds that have been domestically hand raised often accept warmed liquid foods orally. Even untamed birds will sometimes voluntarily accept oral feeding. Often however force-feeding is necessary and use of a gavage tube or indwelling feeding device becomes inevitable.

Tube feeding

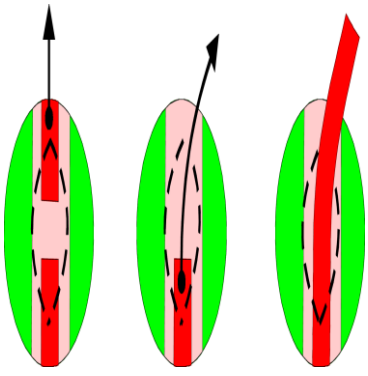
The simplest way to force feed is to employ a ball-tipped metal or rubber tube to deposit liquid food into the crop. Various commercial sources are available for tubes of both types specifically designed for this purpose. The size14 French red rubber feeding tube is an ideal size for most birds over 100 grams because it can be cut at any length at it will still fit snugly on a standard syringe tip. Birds are fed by sliding the tube over the tongue toward the right side of the bird's neck (where the esophagus proceeds) and depositing formula in the crop. An oral speculum is used when the bird has enough strength to bite and damage the tube. A safe volume of formula for feeding directly into the crop is roughly 3-5% of the bird's normal body weight. Feeding should always proceed slowly to avoid overfilling and reflux. Force-feeding should never be performed on birds that are recumbent as regurgitation may occur leading to pulmonary aspiration and its consequences.

Esophagostomy

Certain situations mandate bypassing the crop and depositing food directly into the proventriculus or beyond. Babies suffering from crop burns or those with refractory crop dysfunction, and birds with severe beak injuries benefit greatly from an indwelling



proventricular feeding tube installed via an esophagostomy. A 14 French red rubber feeding tube is passed down the esophagus of an anesthetized bird, manipulated through the crop and into the thoracic esophagus, and continued to the



proventriculus until resistance is felt. At that time a 1 cm longitudinal incision is made on the right side of the neck over the feeding tube identified within the esophagus (figure 8). The tube is isolated and transected beneath the

Fluid Administration

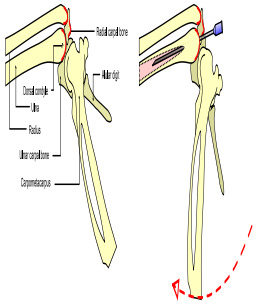


Figure 10

Parenteral fluid administration is one of the most important aspects of avian critical care. Principles of fluid replacement follow those in other fields of veterinary medicine. Methods of replacement are not so traditional.

Subcutaneous

Subcutaneous administration of fluids is an acceptable but not ideal practice. Generally, when a critical avian patient is in need of fluid therapy, the subcutaneous route will not restore circulatory fluid volume as effectively as intravenous or intraosseous techniques. Subcutaneous fluids can be administered in the prepatagium (wing web), dorsally between the wings, or in the inguinal fold.

Intraosseous

Because of the difficulty in stabilizing a standard I.V. catheter and the small size of many patients, the intraosseous catheter has become popular among avian veterinarians. Two primary sites are used: the distal ulna and the proximal tibiatarsus. The technique simply involves installing a spinal needle through the end of the bone and into the marrow cavity. The femur and humerus are not used because of their pneumatic properties.

When utilizing the ulna, the most important landmark to identify is the dorsal condyle of the distal ulna (figure 10). The carpus is flexed and the ulna is identified by palpation. Once the insertion site is located a surgical prep of the area is performed. The needle is directed under the dorsal ulnar condyle and proximally into the shaft of the ulna. Once the needle is placed the stylet is withdrawn and the needle is capped and secured with tape or stay sutures..

The approach to the tibiatarsus is similar to that for a normograde pinning of the bone. The cranial cnemial crest is identified on the anterior aspect of the proximal tibiatarsus between and just distal to the femoral condyles (figure 11). The area is prepared for sterile technique and the spinal needle is directed into the tibial plateau just posterior to the cnemial crest and distally into the marrow cavity of the tibiatarsus. The stylet is withdrawn and the hub is then taped or sutured in place.

With either technique fluids should be administered especially slowly to avoid leakage (minimal with careful technique) and pain (significant with high pressure).

Intravenous

Fluids can also be administered directly intravenously as in other species. Birds as small as 15 grams can be administered fluids with single boluses given via the jugular. The basilic and medial metatarsal veins may be used in larger birds. In some cases one or two boluses provide such improvement that further I.V. administration is unnecessary.

Use of the basilic vein usually results in the formation of a large hematoma at the administration site. This can be minimized by removing the needle from the vein but not the skin following fluid administration

and injecting a large volume of fluids subcutaneously. The fluid compression then lessens vascular leakage.

Jugular

Installation of a jugular intravenous catheter is easily accomplished in many species of birds. Jugular catheters have routinely been installed by the author in the jugulars of patients as small as 75 grams. The more prominent right jugular and often the left jugular are readily accessible on the ventrolateral sides of the neck. A standard I.V. catheter of appropriate size and length is installed into the jugular in a cardiac direction. It is important to place the catheter as near the thoracic inlet as possible to avoid kinking when the neck is in normal flexion. Use of a more rigid polypropylene catheter also minimizes the possibility of kinking. The catheter can be sutured in place or enclosed in a tape collar following installation. The tape collar actually increases the catheter's stability but should be applied loosely to avoid constricting the crop and esophagus.

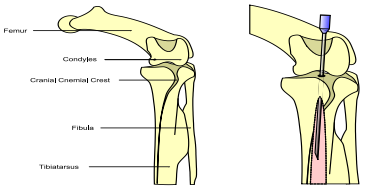


Figure 11

Basilic

A small diameter Teflon catheter can be placed in the basilic vein of larger birds as it crosses the medial aspect of the elbow. A section of tongue depressor may be taped alongside the catheter to provide stability. After the catheter is installed the wing should be placed in a figure "8" bandage to prevent dislodging.

Maintaining Proficiency of Technique

Regular and frequent performance of routine mechanical techniques on avian patients establishes skill and competence in the avian practitioner. Two tremendous benefits are subsequently realized: increased clinical success and improved client confidence. In many situations the owner of a sick bird will be more impressed by the clinician's mechanical skills than by his knowledge; the mechanical skills are more visible. A competent avian clinician has both the knowledge of what to provide and the skill to provide it. The avian practitioner owes it to the patient, the client, and himself to develop his skills in handling avian patients.

Avian Diagnostic Testing

INTRODUCTION: *Clinical pathology holds the key to unravelling much of the mystery surrounding the avian patient. Although many avian diseases present with identical clinical signs, laboratory data can often distinguish between infectious and metabolic disease, bacterial versus fungal, renal versus hepatic, etc. In a clinical situation, an appropriately broad selection of laboratory tests offers the best odds of quickly determining the nature of the patient's problem.*

COMPLETE BLOOD COUNT (CBC)

The complete blood count (CBC) is one of the most important components of an avian diagnostic panel. For analytical purposes the CBC may be divided into components describing: the volume and character of the red blood cells; the numbers, percentages, and characteristics of the white blood cells; the concentration of solids in the plasma; the relative number of thrombocytes; and the presence or absence of blood-borne parasites. While many other tests provide information not demonstrated by the CBC, no other single test provides such a broad range of information. Differences exist between avian and mammalian blood but, once these differences are recognized, the similarity in functions of the various components becomes evident.

The primary differences between mammalian and avian blood are that:

- normal mature avian red blood cells are nucleated, and regenerative anemia is therefore demonstrated by polychromasia among the red cells in a stained smear; (basophilia of cytoplasm, increased nucleus to cytoplasmic ratio, and more spherical shape to the nucleus and cell).
- white blood cell types parallel mammalian cells except that the heterophil is present in avian blood instead of the neutrophil; and
- thrombocytes are present in avian blood instead of platelets.

Unlike dogs and cats, cell counts of birds may vary widely among members of a given species. To determine normal cell count for an individual, baseline data must be collected during periods of apparent good health. Reference values for various species have been published, but these tables should only be used as rough guidelines. Published ranges will typically be wide, therefore subtle patient variations may not be apparent.

Beyond these differences, the functions of the various cellular components in avian blood are roughly comparable to those of mammals. Infections, non-infectious inflammation, necrosis, neoplasia, etc may cause a leucocytosis. A moderate heterophilia often indicates the presence of bacterial infections or cellular necrosis, and extremely high heterophil counts often accompany pansystemic illness such as chlamydiosis, aspergillosis, or tuberculosis. These changes are usually characterized by

varying degrees of toxic changes in the white cells. Subtle-to-moderate heterophilias, without toxic changes in the white cells, may reflect stress leucograms.

An overwhelming bacterial infection, sepsis, or a severe viral infection may result in a leucopenia with a heteropenia or occasionally a lymphopenia. The leucopenia may be due to decreased production or increased consumption of the cell line. Increased consumption is evidenced by the presence of immature and toxic cells, findings not present with decreased production.

In some avian species, the relative lymphocyte count may be higher than in others. An absolute lymphocytosis may suggest a viral infection or certain stages of chlamydiosis.

A lymphopenia may occur in severe viral infections, such as avian circovirus in young African grey parrots. (This presentation, in fact, usually demonstrates a pancytopenia.)

A monocytosis implies the presence of chronic infection, granulomatous disease, or extensive necrosis in which a large amount of phagocytosis is occurring. Classic examples of this include chronic forms of aspergillosis, tuberculosis, and chlamydiosis.

Eosinophil functions have not been clearly defined. Intestinal parasitism may produce an eosinophilia, but not consistently. Peripheral eosinophilia does not appear to occur in allergic conditions.

Basophils are uncommon findings in normal avian haemograms. Conditions that cause their appearance include respiratory infections, resolution of tissue damage, parasitism, and some chlamydial infections.

Electrophoresis (EPH, SPE)

The fractionation of plasma proteins via protein electrophoresis is analogous to the separation and identification of white blood cells in the differential cell count. Just as different families of white cells are separately quantitated, so the relative percentages of the plasma proteins are measured. It should be realized that the technique of performing an electrophoresis does not produce absolute values of each protein fraction; rather, it reveals the percentage of each as part of the premeasured total protein. The absolute values must be calculated after the total protein has been determined through another method. The electrophoresis then yields information regarding a variety of physiologic and immunologic states of the patient. The primary categorization of avian plasma proteins includes pre-albumin, albumin and globulin components. Globulins are then divided (and sometimes subdivided) into alpha, beta, and gamma fractions.

One aspect of protein determinations that should always be observed is the albumin:globulin (A:G) ratio. More important than the patient's total plasma protein are the relative quantities of pre-albumin, albumin, and globulin. The ratio is calculated using the formula (pre-albumin + albumin)/globulins, and the normal A:G ratio ranges from 1.6–4.5. The importance of this ratio is illustrated by the following example. Snowflake and Peaches each have

total plasma proteins of 4.0. At first glance, according to published data, each patient's proteins appear to be normal. Snowflake's albumin is 3.0 and his globulin is 1.0, resulting in an A:G ratio of 3.0, normal. Peaches, however, has an albumin of 1.0 with a globulin of 3.0. Peaches' A:G ratio is 0.33, grossly abnormal. Peaches' albumin is very low and her globulins are very high–an indication of a potentially serious condition. Peaches is losing or failing to produce albumin, while at the same time some portion of the globulins is being produced at an accelerated rate.

Pre-albumin and albumin fractions

The significance of the pre-albumin fraction of the serum protein in birds is uncertain. It may function as a transport protein, similar to albumin. There does not appear to be a comparable component in mammalian blood. In avian samples, it may comprise as much as 40 per cent of the total serum protein. In some species, it appears that low pre-albumin values may have the same significance as low albumin.

The albumin fraction typically comprises 45%–70% of avian serum protein in species that have high pre-albumin values, and tends to be lower in species with low pre-albumin values. Albumin functions primarily as an osmotic pressure regulator and a transport protein, as it does in mammalian species.

GLOBULIN FRACTION

The globulin fraction has alpha (), beta (), and gamma () components, and high-resolution electrophoresis will divide the globulins into the protein components listed under the , , and subgroups discussed below. Each of the 3 primary globulin fractions contains proteins active in different physiologic and pathophysiological conditions.

Alpha globulins: Alpha globulins consist of 2 principal fractions: 1 and 2. Contained within this group of globulins are acute phase inflammatory proteins such as -lipoprotein, 1-antitrypsin, 2-macroglobin, and haptoglobin. The 2-macroglobin sometimes migrates into the range. One condition associated with elevated globulin levels in birds is parasitism. Other consistent correlations have yet to be identified. Elevations in alpha globulins are somewhat uncommon.

Beta globulins: Beta globulins constitute other acute phase inflammatory proteins, including 2-macro-globulin, fibronectin, transferrin, and -lipoprotein. In some species, namely the African grey parrot, the component of the EPH consists of 2 primary components: 1 and 2. Elevated beta globulin levels may be indicative of chronic liver or kidney disease, or chronic inflammatory diseases such as aspergillosis or chlamydiosis. The most common reason for elevated beta globulins in birds, which is attributable to the transferrin component, is egg production. A significantly elevated beta globulin level, combined with a 1.5- to 2-fold increase in the blood calcium level, in birds of unknown sex, is almost 100% suggestive that the bird is an ovulating female.

Gamma globulins: In mammals, gamma globulins appear as 2 primary fractions: 1 and 2. In avian species, only one fraction is demonstrated. The primary components of the gamma globulins are antibodies, complement, and complement degradation products. Elevated gamma globulins are a

common finding in birds suffering from acute Chlamydia infection.

Serum Biochemistries

Alanine aminotransferase (ALT, SGPT)

Alanine aminotransferase is an enzyme found in the cells of many avian tissues. In other animals, elevations have been shown to be associated with hepatocellular disruption, but no such association has been consistently demonstrated in birds. Little clinical significance can therefore be applied to ALT values in avian patients.

Albumin

The function of albumin is discussed above. It should be noted here that an accurate albumin determination is best calculated through electrophoresis. Because of the linear range of most laboratory instrumentation, currently utilized chemistry assays often do not provide accurate avian albumin measurements.

Amylase

In birds, the pancreas, liver, and small intestine produce amylase. Elevations have been associated with acute pancreatitis and enteritis. Because more than one source of amylase exists, an elevation is not in itself diagnostic.

Alkaline phosphatase (AP, SAP)

Alkaline phosphatase is found in bone, kidneys, intestine, and liver. The hepatic fraction composes only a very small proportion of the total reported in routine testing. Because changes in the hepatic fraction have little and inconsistent influence on the overall value, no correlation can be made between liver disease and AP levels. The inconsistent elevations from variable sources cause AP to be of almost no value in avian diagnostics. Disruption of bone probably causes elevations of AP more than other sources.

Aspartate aminotransferase (AST, SGOT)

The intracellular enzyme most useful for diagnosing hepatocellular disruption in avian species is aspartate aminotransferase. Although present in liver, skeletal muscle, kidney, heart, and brain, elevations are frequently associated with liver disease or muscle damage. Whenever an elevation in AST is detected, the creatine kinase (CK) level should be reviewed. An elevated AST without a concurrent elevation in CK is highly suggestive of hepatocellular disruption. It should be emphasized that this does not confirm liver disease; nor does a normal AST positively rule out liver disease. As with all diagnostics, the AST provides evidence towards a diagnosis but does not in itself determine the diagnosis. Also, the AST in no way indicates the functional capacity of the liver. The bile acids test, discussed shortly, is more appropriately used to evaluate hepatic function.

Bile acids

Bile acids are produced by the liver to aid in the digestion of fats. After excretion into the intestinal tract, bile acids are reabsorbed and returned to the liver via the portal circulation. The liver then extracts the bile acids from the blood for recycling. Elevation of bile acids in the general circulation implies decreased ability of the liver to extract the bile acids from the portal circulation, and therefore suggests impaired liver function.

Confusion arises when it is noted that the liver is the organ of bile acid synthesis. It would seem logical that hepatic insufficiency would result in decreased production of bile acids, and therefore in decreased circulating levels. However, hepatic extraction of bile acids from the portal circulation is apparently more dependent on efficient liver function than is the synthesis of bile acids. It is reasonable to presume (and it does appear to happen) that at some point the production of bile acids does diminish and values fall. As with aspartate aminotransferase, a normal bile acid level does not absolutely rule out hepatic disease.

It is important to distinguish between the information provided by the AST and bile acids. Aspartate aminotransferase is a leakage enzyme, and therefore an inverse indicator of hepatocellular integrity, while the bile acids is an inverse indicator of liver function. One is not necessarily dependent on the other. For example, a patient may exhibit a normal AST but elevated bile acids. This would imply impaired liver function, even though the cells are intact; such conditions include hepatic lipidosis, chronic fibrosis, etc. Conversely, many diseases such as salmonellosis or acute chlamydiosis may cause hepatocellular damage without being extensive enough to impaire overall liver function. Marked elevations in the AST may be observed without concurrent elevations in bile acids. Again, normal values of either or both do not rule out hepatic disease. A totally fibrotic end-stage liver lacks enough functional hepatocytes to produce measurable AST or bile acids. Finally, a significantly elevated AST along with a significantly elevated bile acids presents a worse-case scenario in terms of liver disease.

Bilirubin

Since biliverdin is the major avian bile pigment, bilirubin is uncommonly observed in avian serum samples. In occasional cases of severe liver disease, significant bilirubin levels are present; therefore, hepatic pathology may be suspected in patients demonstrating elevated bilirubin levels.

Calcium

Calcium levels are profoundly influenced by a number of normal as well as pathological conditions, and great care should be exercised in the interpretation of abnormal findings. Almost all pathological changes are secondary to conditions not associated with dietary levels. Because of the effectiveness of the parathyroid gland, dietary deficiencies of calcium will rarely cause obvious subnormal blood levels. Blood calcium levels are also directly linked to albumin levels. Hypoalbuminaemia will result in artifactual depression of measured calcium levels. Other causes of lowered blood calcium levels include hypoparathyroidism in African grey parrots, glucocorticoid administration, and insufficient exposure to full spectrum lighting.

Dehydration will sometimes elevate albumin, and therefore blood calcium. Two-fold or greater elevations typically occur with ovulation. Elevated levels of calcium have been associated with vitamin D3 toxicity, osteolytic bone tumours, renal adenocarcinoma, and dehydration.

Cholesterol

Cholesterol levels in birds may accompany various physiological or pathological conditions, but there is inconsistency and a lack of specificity associated with abnormal

findings. Generally, elevations are associated with liver disease, hypothyroidism, high fat diets, and starvation, especially in obese birds. Subnormal levels are rarely significant but may be observed with endotoxaemia, aflatoxicosis, spirochaetosis, and low dietary fat. Unfortunately, there are no clear indicators to determine whether or not an abnormal cholesterol level is associated with a particular condition. For example, if an obese bird were to display elevated cholesterol, it would be unclear whether the elevation was a result of hypothyroidism, excessive dietary fat, hepatic lipidosis, or mobilization of body stores during anorexia. Other tests and observations usually provide evidence of these conditions, with or without the support of the cholesterol level. Again, normal values do not rule out the aforementioned conditions.

Creatinine

Creatinine levels in avian serum samples typically fall below a measurable range, and are rarely useful in avian clinical pathology. Also, certain technical factors contribute to a high incidence of artifactual changes. Elevations have been associated with kidney disease, but creatine is not considered to be a reliable indicator of renal function. Many commercial labs include creatine in an avian profile, but its significance should be regarded with suspicion.

Creatine kinase (CK)

The primary sources of creatine kinase include skeletal muscle, cardiac muscle, and nervous tissue, and elevations are associated with significant disruptions of these tissues. The primary usefulness of this enzyme is in distinguishing between hepatic and non-hepatic causes of elevated aspartate aminotransferase (AST). Any elevation in AST should be compared with the patient's CK level. If the CK is normal, it is relatively safe to conclude that the liver is the source of the elevated AST. If the CK is elevated along with the AST, muscle should be considered a possible source of the elevated AST. Other possibilities for the dual elevations would, of course, be concurrent liver and muscle or liver and neurological disease. Lastly, an elevated CK in the absence of other biochemical pathology is often the result of significant neurologic disease.

Glucose

Pathological changes in avian blood glucose levels principally involve elevations. Hypoglycemia is extremely rare in birds and, when present, is almost never associated with starvation. The primary cause of hypoglycemia in pet birds is septicemia.

Hyperglycemia occurs commonly due to stress or recent intake and, occasionally, from diabetes mellitus. Because of the frequency with which hyperglycemia is caused by stress, a diagnosis of diabetes mellitus should be considered carefully and only if other evidence supports it. A visibly normal hyperglycemic patient displaying no polydipsia, no polyuria, and no weight loss, etc should not automatically be considered diabetic. Repeat testing, other investigative tests and observation are necessary to confirm a diagnosis.

Glutamate dehydrogenase (GLDH)

Sources of GLDH in birds include the liver and, to a lessor degree, the kidney. Although not widely available, the GLDH level can provide significant information in

the investigation of hepatic disease. GLDH is a leakage enzyme, so elevations are observed when significant cellular destruction occurs. If the assay for GLDH could be refined, the GLDH could replace the paired AST and CK as an indicator of hepatocelluladisrption.

Lactate dehydrogenase (LDH)

LDH is found in skeletal and cardiac muscle, liver, kidney, bone, and erythrocytes. Elevations can be observed with disruption of any of these tissues or in haemolysis, and are therefore extremely non-specific. One benefit of measuring LDH levels may be in following the progress of liver disease, in which LDH levels apparently change more quickly than AST levels; lowering LDH values may imply improvement even though AST levels remain elevated.

Lipase

Serum lipase levels may be elevated in cases of acute pancreatitis. Currently, the only reliable antemortem confirmation of pancreatitis is by pancreatic biopsy.

Phosphorus

Elevated serum phosphorus is frequently observed in advanced renal failure. An elevated phosphorus level resulting from renal disease suggests chronicity and presents a guarded prognosis. Elevations are also observed in hypoparathyroidism and nutritional secondary hyperparathyroidism. Hemolysis may artifactually elevate serum levels. Malabsorption and vitamin D deficiencies may cause lowered blood phosphorus levels.

Potassium

As with sodium, pathological changes in potassium levels indicate a serious and usually life-threatening clinical situation. Hyperkalemia develops with advanced kidney disease, adrenal disease, muscle damage, and during episodes of acidosis. Hypokalemia may result from loss through diarrhea, and during states of alkalosis.

Sodium

Changes in sodium values usually reflect serious conditions. Elevated levels occur with salt poisoning, water deprivation, and dehydration. Decreased levels occur due to sodium loss in kidney disease or diarrhea.

Total protein (TP)

A total serum protein level must be evaluated in light of its components, albumin, and globulin. The total value is influenced by various factors but, as discussed previously, a normal value does not rule out abnormalities of the individual protein components. Overall, dehydration and immune stimulation may cause a hyperproteinemia. Hypoproteinemia may be caused by overhydration, protein loss in kidney disease, starvation, liver disease, or intestinal disease. As stated earlier, the protein should never be considered normal until the A:G ratio is known to be normal.

Urea

Because of the low level of urea (blood urea nitrogen—BUN) in avian blood, its usefulness is limited. Also, the avian kidney appears able to excrete most urea as long as the patient's hydration is adequate. Therefore, blood urea may be a better indicator of hydration than renal function.

Uric acid

The blood uric acid level is the primary indicator of renal function in birds. An elevated uric acid (UA) level is a reliable indicator that kidney function is impaired. With many tests, substantial elevations are necessary before there is reason for

concern; however, even a subtle elevation in the uric acid warrants suspicion of renal disease. Conversely, serial determinations should be made after adequately hydrating the patient before concluding a diagnosis of renal disease. While it has been argued that dehydration has little effect on uric acid levels in birds, this is not the author's experience. Many patients with profoundly elevated UA levels have reverted to complete normalcy after fundamental rehydration. An artifactual elevation of UA often occurs if blood is collected via nail trim due to contamination of the nail with the patient's droppings.

Fecal examinations for parasites

Gastrointestinal parasites, while rare in pet birds, are occasionally significant causes of avian illness and sometimes even death. Suspect cases should be examined through the use of direct saline smears, flotation techniques, and certain specialized assays.

Giardia species are parasites for which there are a variety of diagnostic techniques available. Direct saline smears are occasionally revealing, although the organisms may be more clearly visible when stained with Lugol's iodine. Trichrome staining will sometimes display the organisms more readily. An ELISA test is now available that shows great promise in identifying difficult cases. Molecular diagnostic assays are also readily available to the avian practitioner.

Gram staining

At one time, the Gram stain was the most commonly utilized test in avian medicine. Much controversy now surrounds its significance in assessing avian health.

The Gram sain itself will always be a useful test; the problem lies not in the test but in its interpretation. Many birds are erroneously diagnosed with gram-negative bacterial infections due to artifact from poor staining techniques. The findings of a correctly performed test are often misinterpreted as abnormal when in fact, they may be acceptable. An understanding of avian bacteriology is a prerequisite for accurate interpretation of avian Gram stains. The Gram stain may then be used to suggest the major flora in a given fecal sample. Usually, abnormal findings should be validated through bacterial/fungal cultures.

Summary

Laboratory testing is an essential component of avian medicine. When examining avian patients, both routinely and in the face of illness, it is necessary to utilize an appropriate assortment of tests in order to obtain a reasonably complete profile of the patient. Knowing how to interpret the findings is as important as knowing which tests to utilize. By employing the right tests, and correctly interpreting the meaning behind the findings, avian patients can be diagnosed and treated with maximal accuracy and effectiveness.

MEDICINA DE RUMIANTES

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Maximizing Health Of Calves

INTRODUCTION: *Maximizing calf survivability is crucial to economic success of beef producers. Having healthy calves starts many months before calving season. Proper herd nutrition impacts calf survivability more than any other factor. Proper environment/facilities are also important. If these two factors are under control, herd outbreaks of calf diseases will be minimized.*

Calf losses are greatest in the first week of life, and most of these are a direct result of dystocia. Some causes of dystocia, such as fetal malpositioning, are impossible to control. However, other causes of dystocia, such as poor nutrition (under or overfeeding), and poor heifer and bull selection, can be minimized with proper management.

PREVENTION

Nutrition

Underfeeding late gestation cows can have a major impact on calf survivability. First, stillbirths will increase, probably due to failure of the cow to go into labor, or due to prolonged labor. Second, birth weights of calves may decrease, as will calf vigor. The producer may not notice this unless records are maintained of cow body condition scores and calf birth weights and survival. This slight decrease in calf birth weight and vigor increases failure of passive transfer, increases cold stress and hypoglycemia, and decreases disease resistance, all of which decrease calf survivability.

Although overfeeding is less common, it can be as damaging as underfeeding. Excess fat in the vaginal cavity can cause dystocia. Overfeeding of heifers can increase fat in the udder, and impact milk production later in life.

Nutrition also impacts vaccine response due to its impact on the immune system (both humoral and cell-mediated immunity). Cows can only respond to vaccines if they have proper energy, protein and mineral levels in the diet. For example, if a cow isn't taking in enough protein to maintain her body condition, she can't make antibodies. Therefore, vaccinating cows to protect calves through colostral transfer of immunity will only work with proper cow nutrition.

Historically, focus has been placed on the influence of nutrition in the third trimester on calf health. A newer focus is the influence of nutrition in early gestation and its impact on placental weight (and subsequent fetal growth), neonatal weight and conformation, and body fat makeup and metabolism. Subsequent growth later in life and long term reproductive health of calves may be impacted by nutrition in these early stages of gestation in their dams. More recently, evidence suggests that supplementation of protein in beef brood cows effects the growth and reproductive efficiency of the female offspring of these cows.

Failure of Passive Transfer

Many immune system defense mechanisms are lacking or deficient in the neonatal calf. Therefore, intake of high quality colostrum to provide adequate passive transfer of

immunity is an important factor in protecting calves from disease. Besides providing circulating immunoglobulins, colostrum provides local immunity in the gut, WBC's that also contribute to local immunity and stimulate cell-mediated immunity (if fresh), and nutritional elements. Calves that receive colostrum have higher growth rates than calves that don't receive colostrum, even if those calves don't become ill. This increase in growth rate carries over even into the feedlot.

Several factors can contribute to failure of passive transfer (FPT). Low immunoglobulin concentrations in colostrum of beef cows is usually a result of poor nutrition. Weak calves and poor udder conformation or a poor environment can all interfere with the calf's ability to ingest colostrum. And, even if calves ingest adequate amounts of good quality colostrum at the appropriate time, sometimes they do not absorb the proper amounts of immunoglobulins. Dystocia leading to hypoxia and acidosis is probably most commonly associated with poor absorption. Other causes that are implicated but difficult to prove are placental insufficiency due to fetal oversize and/or poor nutrition in early gestation.

Prevention of FPT in an individual calf involves ensuring at least 100 grams of immunoglobulins is ingested. Two liters of beef or four liters of high quality dairy colostrum is recommended. Many times two liters is hard to get from a beef cow. Feed whatever can be safely obtained. I usually only try to milk beef cows if I have had to intervene to deliver a calf and I am worried the calf will not nurse in time or at all.

High quality dairy colostrum is hard to find, and biosecurity issues should be considered. Fresh colostrum is better than frozen. But frozen colostrum is far superior to colostral supplements or replacers. The colostral *supplements* available at this time are not great substitutes for real colostrum, and do not warrant the purchase cost. Colostrum *replacers* are better, but should not be considered a substitute for good hygiene and management. They may be a better option if the source of outside colostrum is of unknown disease status.

Prevention of FPT on a herd basis involves providing adequate nutrition, providing an environment that allows the calves to stand and nurse without difficulty, minimizing dystocia, and culling cows with poor udder and teat conformation. It's important to remember that FPT is not a death sentence. On any one farm, there are going to be a few calves that have FPT, even if these farms are well managed. Disease outbreaks in the calf herd start to arise when the numbers of FPT calves increases (usually poor nutrition). The more FPT calves on a farm, the more likely a calf will become sick with a contagious disease (ex. infectious calf diarrhea). Calves are amplifiers of disease because they shed more organisms than the adults, even if not clinically ill. So one sick calf can be the start of a vicious cycle of disease transmission that spreads throughout the herd.

If a specific infectious organism is identified in a calf disease outbreak, vaccination of the cow a month prepartum to passively protect the calf can be considered. But, since outbreaks are more common at the end of the calving season, vaccination rarely helps prevent disease in the current year, but may help prevent cases the following year.

Sanitation

It’s very important to remember that an adequate passive transfer status of the herd can be overwhelmed by a dirty, contaminated environment. Feed troughs and hay racks should be moved periodically, and placed away from waterers and shelter

to discourage congregation of cattle in one area, and subsequently concentration of pathogens. If a disease outbreak occurs in the calves, move pregnant animals to a clean pen and leave sick animals in the already contaminated pen. Healthy calves should not move with pregnant cows, since they may be incubating the disease and risk contaminating another pen. If possible move healthy calves to a third pen. On large operations, cattle should be segregated in groups according to calf age, so that young calves are not exposed to older calves.

Other Preventive Strategies

Umbilical infections can be a problem in some herds, especially during wet years. Dipping navels with 7% tincture of iodine at birth, as well as controlling cattle movement and congregation as discussed earlier will help decrease the number of umbilical infections in a herd. Umbilical infections, even mild ones that go unnoticed, can lead to weakening of the body wall and umbilical hernias later. Sometimes “outbreak” of umbilical hernias suspected to be genetic in origin are actually from previous umbilical infections.

Proper heifer development and selection, and bull selection are beyond the scope of this article, but are extremely important in decreasing dystocia, weak calves, and FPT. Heifers should be bred to calve early in the calving season so they can be observed more closely for dystocia. In some years, early weaning of calves born to heifers (and cows if necessary) may make maintaining proper body condition in these heifers easier as they continue to grow.

If embryo transfer is employed, selection of good embryo recipients is crucial. Heifers should be avoided, as should cull diary cattle (mastitis, Johne's disease, poor udder conformation, etc.). Producers should be encouraged to select proven dams from their own herd as recipients. An alternative is to lease proven cows from other producers. Cows should be leased from farms with BVD and Johne’s control programs in place, as well as other biosecurity measures. Embryo recipients are more likely to have large, overdue calves, so close observation during calving is very important.

Feeding at night, or even in the afternoon increases the likelihood of daytime calving. On farm lay help and veterinary services are more likely to be available during the day, so dystocia problems and weak calves are more easily handled and treated more promptly.

TREATMENT

First hours of Life

The earlier "at risk" neonates are identified and treated, the better the prognosis for a healthy and productive life. Any calf that is born following a dystocia, even if it appears normal, should be considered at risk. Many of these calves will look normal for a few hours, but deteriorate quickly, so will need to be watched closely. Immediately following the dystocia, while the cow is still restrained, the cow should be milked (if possible) and the calf bottle or tube fed. This ensures colostrum intake and precludes having to restrain the pair later if the calf has not nursed. It also helps prevent hypothermia and hypoglycemia.

Calves born following dystocia can be depressed due to hypoxia, metabolic acidosis, and/or hypothermia. Calves with mild depression can be warmed and given intravenous sodium bicarbonate inexpensively, and this can greatly improve the chances of survival of these calves. If

only mildly hypothermic, heat lamps and hot water bottles may work. However, if severely hypothermic, peripheral perfusion is poor and external warming is not very effective. Therefore these calves need to be warmed from the inside. Warm oral and intravenous fluids (balanced electrolyte solution with 1.5% dextrose) are best, along with external sources of heat. Correcting for a base deficit of 10 is usually safe. Concentrated sodium bicarbonate (5-8%) can be administered undiluted at a rapid rate through a needle if a catheter for other fluids is not needed. Calves with more severe depression may require oxygen therapy, which will increase the cost of treatment. However, many times a small amount of supportive care early prevents having to do more extensive, prolonged care later.

Hypoglycemia

Hypoglycemia is less of a problem in the first hours of life, and more of a problem later secondary to inadequate nutritional intake, diarrhea, septicemia, etc. If severe, hypoglycemia can mimic meningitis with signs such as miotic pupils, ophisthotonus, seizures, etc. Glucose levels can be low with both conditions. If other causes of weakness and neurologic signs have been eliminated (hypothermia, acidosis, severe dehydration), a slow infusion of 0.5 mls/10 lbs body weight of 50% dextrose IV can be administered without the need of a catheter. If calves have simple hypoglycemia, they will usually respond to the dextrose by improving. If they do not respond, a CSF tap can be easily performed. If the fluid is grossly abnormal, the prognosis is poor, and the owner can factor this into treatment decisions. If the fluid looks grossly normal, the calf may still have meningitis, but the prognosis is good with treatment. If treatment is continued, these calves must have IV 2.5- 5% dextrose, since the 50% dextrose will cause a rebound hypoglycemia if dextrose isn't continued. Calves should be weaned form 5% dextrose slowly.

Failure of Passive Transfer

In an individual calf with failure of passive transfer (FPT), the most important problems are decreased growth rates and septicemia. Diagnosis of failure of passive transfer can be made at 24 hours up to about one week of age. One of the cheapest tests is serum protein, which should be > 6.0 g/dl in beef calves.

If the value of the calf warrants it, the only specific treatment for FPT is a plasma transfusion, or more practically, a whole blood transfusion. Plasma is of questionable benefit in healthy calves for prophylaxis because even with high volumes, immunoglobulin levels don't reach those of calves that received colostrums. This is further magnified if whole blood is given because of volume limits. However, there are other benefits of plasma or whole blood administration, especially in sick calves. The increase in protein levels helps prevent hypoproteinemia if IV fluids have to be given, and if fresh whole blood is given, benefits of cellular immunity, interferon, and other circulating non-specific immune factors may benefit the calf. Treatment with antibiotics prophylactically in healthy calves is controversial, and should be considered on a case by case basis.

Neonatal Septicemia

A potential sequella to failure of passive transfer is septicemia. Calves under 7 days of age are at greatest risk. The source of the bacteria can be the umbilicus, the GI tract, or the respiratory tract. Any organ system can be secondarily infected, but the

neurologic, musculoskeletal and ophthalmic systems are most likely. General clinical signs of septicemia are depression and reluctance or inability to stand. Anorexia, poor suckle reflex, +/- fever (more often hypothermia) are other general signs. Hypoglycemia may be present. Neurologic signs due to secondary meningitis are ophisthotonus, seizures, stiff extremities, nystagmus, and/or miotic pupils. Hypopyon, uveitis, synechia, and conjunctivitis may occur but are not life threatening, only a sign of serious problems. Single or multiple swollen joints, edema around the joints and osteomyelitis may occur. Musculoskeletal infections carry a poor prognosis unless caught early. Lameness in neonatal calves should be treated as an emergency. Infectious arthritis from septicemia is a more likely cause of lameness than injury/trauma. Diarrhea and pneumonia are not a common sequella to septicemia in beef calves, although they are common problems in calves with FPT.

Umbilical Infections

If an umbilicus becomes infected, and the calf is systemically ill or has poor growth, the umbilicus should usually be immediately removed surgically. Systemic antibiotics rarely work, and the longer the infected umbilicus stays, the higher the risk of the infection spreading to the joints, nervous system, etc. Unlike simple hernia repair, which can be done under sedation and local anesthesia, removal of an infected umbilicus can be more complicated, and general anesthesia is recommended. The surgeon should be prepared to resect infected or abscessed umbilical veins and arteries, and the urachus.

Field Diagnostics And Therapeutics For Cattle And Small Ruminants

DIAGNOSTICS

There is no substitute for a thorough physical examination when trying to determine what body systems are involved in making an animal sick. However, ancillary diagnostic procedures con often times help to more clearly characterize diseases.

Rumen Fluid Analalysis

Analysis of rumen fluid can help differentiate diseases of the forestomachs. An appropriate size orogastric tube can be can be passed via the oral cavity for fluid collection. Care must be taken to properly restrain the animal. Use of a mouth speculum is needed to prevent chewing of the tube, which if not prevented can lead to roughening of the tube surface and damage to the esophagus. Excessive chewing can also lead to a broken tube that can be swallowed. Rumen fluid can also be collected via percutaneous rumenocentesis. A 16 guage needle can be inserted in the rumen through the abdominal wall caudal to the xyphoid and to the left of midline. Fluid is then aspirated with a syringe. Local anesthesia and sedation of the animal may be needed. This technique avoids saliva contamination that can occur from collection with an orogastric tube and it appears to be less stressful. There is a slight risk of causing peritonitis with rumenocentesis, which can be minimized with proper restraint. Percutaneous rumenocentesisis should not be performed in pregnant females.

Once fluid is collected, it can be analyzed for color; odor; pH; protozoal species and motility; methylene blue reduction time (MBR); gram staining characteristics; and chloride levels. Anorexia may cause the fluid to look darker, the pH to increase, and the number and motility of protozoa to decrease. A grey color, low pH, and dead or no protozoa are seen with rumen acidosis from grain overload. The MBR will be prolonged with any type of indigestion. Large numbers of gram positive rods (*Lactobacilli spp.*) may also be seen with rumen acidosis. Elevated rumen chloride indicates an abomasal or proximal small intestinal obstruction (either functional or mechanical).

Abdominocentesis

Abdominocentisis is useful in discerning the causes of fluid distension in the abdomen. Two techniques can be utilized. The first technique is useful in ruling out a ruptured bladder as the cause of general ascites, and involves tapping the abdomen at the lowest point and slightly to the right of midline. Care should be taken to avoid the prepuce in males. The second technique is useful if peritonitis is suspected. Since localized peritonitis is more common than generalized peritonitis, four sites are tapped. The two cranial sites are slightly caudal to the xyphoid and medial to the milk veins on the left and right sides. The two caudal sites are slightly cranial to the mammary gland and to the left and right of midline. For either technique, manual

restraint with sedation is recommended, and the use of real time ultrasonography may help locate fluid pockets. A twenty-gauge needle or teat cannula can be used for fluid collection. Sterile preparation of the site is needed, and local anesthesia is necessary when a teat cannula is employed. Fluid should be collected into a small EDTA tube for analysis, and a sterile tube for culture. Fluid can be difficult to obtain, and is usually in small amounts. Care should be taken to minimize the ratio of EDTA to fluid, since EDTA can falsely elevate protein levels. Use of EDTA tubes made for small animals, or shaking excess EDTA out of large tubes will resolve this problem. Fluid can also be collected for culture. Normal values are similar to those for cattle (clear, colorless to slightly yellow, 1-5 gm/dl protein, < 10,000 cells). Cytologic examination is needed to characterize the cell population, and look for the presence of phagocytized bacteria.

Liver Biopsy

Liver biopsy in small ruminants is performed using the same technique and instruments as in cattle. However, sedation and ultrasound guidance are recommended. The biopsy can be performed in the 9th to 10th intercostal space slightly above an imaginary line from the tuber coxae to the point of the elbow. Local anesthesia with lidocaine hydrochloride at the site should be performed following sterile preparation. A small scalpel blade is used to make a stab incision through the skin. A 14 gauge liver biopsy instrument is inserted through the stab incision and intercostal muscles and into the liver. The biopsy instrument should be directed towards the opposite elbow in most cases, but utrasonography will help determine the direction and depth needed. Vessels along the caudal border of the ribs should be avoided. Samples can be submitted for culture (in a sterile plastic or glass vial or tube); histopathology (in formalin at a 10:1 ratio of formalin:tissue); and/or mineral analysis (in a plastic tube). When performing a liver biopsy for mineral analysis, the biopsy site should be rinsed with distilled and deionized water following sterile preparation to minimize contamination of the sample. Samples for mineral analysis should not be placed in formalin.

CSF Tap

A 20 gauge-1½ inch needle is used for neonates, an 18 gauge -1½ inch needle for adults. Ambulatory patients can be tapped standing. Non-ambulatory patients should be placed in lateral recumbency or in sternal recumbency in a "dog-sitting" position with the rear legs forward on either side of the animal. The pelvis needs to be straight and level. The lumbosacral area should be clipped and surgically prepared. Wearing sterile gloves, the indention of the lumbosacral junction should be palpated. A needle is inserted into the deepest part of the indention, directly on midline. Keep the needle perpendicular to the spine from the side view, and straight up and down from the rear view. If bone is encountered, redirect the needle slightly cranial or caudal until the needle drops into the lumbosacral space. Advance the needle slowly until a slight "pop" is felt. The animal usually jumps slightly when the needle punctures the dura mater. CSF should flow from the needle or can be gently aspirated with a syringe. If the needle is in the lumbosacral space and advanced until bone is encountered again, back the needle out 1-2mm and try to aspirate. Place the fluid in an EDTA tube for fluid analysis and a plain tube if cultures are desired. Normal, non-traumatically obtained fluid should be perfectly clear with no discoloration, sediment or turbidity. It is best to have the sample analyzed locally as soon as possible

(within one hour). If this is not possible, place half of the sample in an equal volume of 40% ethanol to preserve the cells (inform the laboratory that this has been done), or centrifuge half the sample to concentrate the cells and prepare slides to be sent with the rest of the fluid.

Four-Point Nerve Block:

Indications: Localization of lameness to the foot, anesthesia for surgery/therapy of the foot.

Materials: 20 gauge-1½ inch needles, lidocaine.

Procedure: 1. Insert the needle into the dorsal aspect of the pastern, in the groove between the proximal phalanges, just distal to the fetlock. Administer 5cc of lidocaine deep, and 5cc while pulling the needle out. 2. Repeat the previous block from the palmar/plantar aspect of the pastern, just distal to the dewclaws. 3. Palpate the nerve over the lateral aspect of the fetlock, approximately 2cm dorsal and proximal to the dewclaw. Administer 5cc of lidocaine over the nerve. 4. Repeat the previous block on the medial side.

Rectal Mucosal Biopsy:

Indications: Cytology or histopathology for diagnosis of Johne's Disease.

Materials: Rectal biopsy instrument, sharpened needle cap, or bottle cap.

Procedure: Place hand wrist deep into the rectum. Secure a fold of mucosa between two fingers and pinch off a piece of the mucosa with the biopsy instrument inserted with the opposite hand. Alternatively, pinch off a piece of mucosa between thumb and needle cap. Make impression smears for acid-fast staining or place the sample in formalin for histopathology.

Caution: Only go in wrist deep, staying in the retroperitoneal area to prevent a full thickness biopsy and peritonitis.

Fecal Smear:

Indications: Diagnosis of Johne's Disease or Cryptosporidiosis.

Materials: Acid-fast stain, slides

Procedure: Smear a small amount of feces on a slide. Heat fix the slide for Johne's and let it air dry for Cryptosporidiosis. Under 40x or 100x, look for small, acid-fast bacilli in clumps (Mycobacterium paratuberculosis), or round, refractile, acid-fast protozoa (Cryptosporidium).

Lung Aspirate:

Indications: Obtaining fluid for culture in a patient with respiratory disease.

Materials: 18 gauge-1½(calves) or 3½(adults) inch needle, syringe.

Procedure: Insert the needle through an intercostal space over an area where abnormal lung sounds are auscultated. Aspirate fluid and place on culturette of into blood culture bottle.

Caution: Stay close to the cranial border of the ribs and beware of the heart. Fatal hemorrhage from hitting a major vessel can occur.

THERAPEUTICS

Hoof Block

A wooden, plastic or rubber hoof block, placed on the normal claw removes the source of pain by preventing weight bearing on the affected claw and allows the animal to ambulate comfortably while healing takes place. This is an affective for treatment of P3 fractures, and is essential for treatment of sole ulcers and serious hoof cracks. Blocks 1-2 inches thick, cut to fit the shape of the claw, with grooves on both sides, are glued to the hoof with some type of bonding material. The wooden hoof blocks and Technovit® acrylic can be obtained from Jorgensen Laboratories. Bovi-Bond® is a newer product by Vettec that is very easy to use. The same company makes 1 inch plastic or rubber blocks that can be glued together if a taller block is needed. The black plastic blocks are the widest and fit beef bulls the best. Although Cow Slips® are used routinely in dairy cattle, they don't always fit beef cattle, especially bulls. All products can be found at Animart (www.animart.com or 800-255-1181).

Preparation of the claw is crucial for preventing the blocks from falling off too soon. The claw should be clean, dry and level. Shallow grooves can be made in the sole with a hoof knife but are not necessary in my opinion. The Bovi-Bond® sets up quicker than the Technovit®, so everything should be ready. In cold, wet weather, a hair dryer helps to quicken setup. When applying the acrylic, pay particular attention to the axial surface, making sure it's smooth and will not irritate the interdigital space or the axial surface of the opposite claw. Do not spread the acrylic onto the coronary band or up on the soft part of the heel. Make sure the block is positioned so that the animal does not rock back on its heel, and the toe doesn't tip up. Caution: If the lameness worsens with the hoof block on or if the animal develops a lameness after wearing the block for several days, , remove the block immediately and reevaluate the problem. The hoof block may be causing the lameness. Also, the heat produced during the hardening process can cause thermal necrosis of soft tissues under the hoof wall. Use sparingly on the hooves of young calves and small ruminants.

Other Uses of Acrylic

Acrylic can be used alone or in combination with wire to repair hoof wall defects. However, the hoof defect must be clean, dry, and free of infection before the acrylic can be placed over it. Hoof cracks are very common in beef cattle. Many times they are found during routine foot trimming. The author does not recommend any treatment of hoof cracks unless they are causing lameness, especially if the patient is a bull receiving a hoof trim immediately prior to breeding season and is not lame. Many times in paring out hoof cracks with a knife or rotary tool, sensitive lamina is encountered and the animal becomes temporarily lame. Many animals have hoof cracks for years with no lameness associated with them. If the hoof crack is causing lameness, then it must be treated.

Local Intravenous Anesthesia

Local intravenous anesthesia is the preferred technique for surgical procedures of the foot and pastern. Although clipping is not necessary, a surgical scrub should be performed prior to injection. A tourniquet is placed proximal to the fetlock immediately prior to injection (vein will be distended best immediately after the tourniquet is placed). Two sites of injection are available. One vein runs down the center of the dorsal aspect of the pastern and the other runs approximately 2cm dorsal to the dewclaw, on both the lateral and medial sides of the foot. A 20 gauge

needle or butterfly catheter is inserted into the vein and 15-20cc of lidocaine or carbocaine is administered. It is only necessary to administer an anesthetic into one of these veins to provide anesthesia to the entire area distal to the tourniquet. The tourniquet can be safely left on for up to 1 hour to provide hemostasis during surgical procedures.

In feet with severe cellulitis, local intravenous anesthesia can be difficult. In these cases, a four point nerve block or a simple ring block will also work. The two interdigital injections performed in the four point block can be used for removal of an interdigital fibroma.

Joint Injection and Lavage

Septic arthritis is a serious condition that requires immediate treatment. Although it is tempting to try systemic antibiotics alone first, the chances of this working are low, and if systemic antibiotics fail, the disease may have progressed to the point that the joint can't be saved. For joint lavage to be successful, it needs to be performed very early (first few days) in the course of the disease. Since veterinarians rarely see these cases early, this is not a common technique used for treatment of the coffin, pastern or fetlock joint in adult cattle. The coffin joint is especially difficult to tap in cattle. If a needle can be placed in the joint space, isotonic fluids can be administered under pressure to distend the joint. A second needle is then placed in the joint space and a through and through lavage is performed, preferably with about 500-1000 mls. The larger the needle (14 to 16 gauge) the better the lavage.

Joint Arthrodesis/Facilitated Ankylosis

When septic arthritis of the coffin or pastern joint has advanced to the point that there are radiographic changes in the bone surrounding the joint, two options exist. One is claw amputation and one is joint arthrodesis. Joint arthrodesis is preferable in valuable breeding animals, especially bulls, because it saves the claw. Although the procedure is easy to perform and relatively inexpensive, the aftercare (flushing wound, bandage changes, keeping a hoof block on and toes wired together, and/or cast application) can be prolonged (1-2 months), which increases the cost over amputation. Also, if significant cellulitis is present, or there is radiographic evidence of osteomyelitis proximal to the joint being athrodesed, the success rate of arthrodesis is poor, and amputation should be considered. The procedure is performed under local anesthesia, and only requires a shop drill and 1/4 and ½ inch drill bits (sterilized). There are several techniques described. Contact the author for references describing the details of the procedure. The owners should be aware that time to breeding soundness in bulls can be several months, even once the actual infection is cleared. The fetlock joint can also be successfully arthrodesed, although a splint or cast is needed for 6-8 weeks for support during healing.

Pregnancy Toxemia

Pregnancy toxemia (twin lamb disease, lambing or kidding sickness) is most common does with triplets, and/or are either thin or obese. Much of the abdominal space is occupied with multiple feti in the uterus during late gestation. Fat accumulation in the abdomen in obese animals also occupies space. Because of lack of space for the rumen, these females have difficulty consuming enough feedstuffs to satisfy their requirements. In late gestation, nutritional requirements increase to 150% of maintenance with a single fetus and 200% with twins. Late

gestation is usually during the winter months, when less pasture is available, and as a general rule, poorer quality feeds are available. Pregnancy toxemia is also seen following anorexia secondary to other diseases (ex. foot rot, ovine progressive pneumonia, caprine arthritis encephalitis virus), or with stresses such as bad weather, transporting, etc. There is also a genetic predisposition in some individual animals.

Very early signs of pregnancy toxemia are mild depression, anorexia and possibly limb edema. If left untreated, goats become anorexic and depressed, and soon become recumbent. Neurologic signs including blindness, circling, incoordination, star-gazing, and tremors. Constipation and teeth grinding can also occur Icreased respiratory rate develops if acidosis occurs. If left untreated, does become recumbent.

Azotemia, both from dehydration and secondary renal disease, is a common finding. A urinalysis will be positive for both ketones and protein. Ketoacidosis is common in small ruminants. Hypocalcemia and hypokalemia may be present due to anorexia. They are not always hypoglycemic. Liver enzymes are usually found to be within normal limits, but occasionally may be increased.

Diagnosis is based on clinical signs, the presence of multiple fetuses, and typical clinicopathologic findings. Differential diagnoses include listeriosis, hypocalcemia, polioencephalomalacia, hypomagnesemia, and meningeal worm infestation.

In small ruminants, very early cases (prior to recumbency) may be treated with oral glucose or glucose precursors. 60-100 mls of propylene glycol orally twice a day, or oral corn syrup or glycerol can be tried. Oral high energy calf electrolytes with bicarb can also be given orally. Rumen transfaunation, vitamin B complex (including B_{12}, biotin, and niacin) are also recommended treatments.

Once females show neurologic signs or become recumbent, treatment must be very aggressive. IV glucose, calcium borogluconate, and bicarbonate may be needed.

Glucocorticoids (15–20 mg dexamethasone) can help by causing gluconeogenesis, increasing appetite and inducing abortion. Prostaglandin ($PGF_{2\alpha}$) should also be used (5-10 mg) for induction of parturition in does. Flunixin meglumine (0.5-1 mg/lb) are indicated if endotoxemia is suspected from dead fetuses.

Removal of the fetuses is critical in these severe cases. Assessment of fetal viability with ultrasound helps with the decision to induce parturition or perform a C-section. Since a breeding date is rarely known, age of the fetuses is hard to determine. If the fetuses are alive, induction of parturition can be an option. However, if the fetuses are already dead, or the condition of the doe is severe, an immediate C-section is warranted. Fluid support during and after surgery is critical. Low birth weights of lambs, kids and calves at the beginning of the birthing season can indicate potential risk of pregnancy toxemia.

Prevention of this disease is through proper nutrition. Maintaining animals in proper body condition throughout the year, and making sure energy and protein levels are adequate in late gestation are important. For does in late gestation, hay should have protein content of at least 10%, and 1-2 lbs of concentrate should be fed per head per day. During periods of stress, particularly cold wet weather, concentrate may need to

be increased to 2-3 lbs/head/day (divided in two feedings). Parasite control, disease prevention and decreasing stress are important.

Ultrasonography can help determine which females have multiple fetuses, and these animals separated into groups and fed accordingly. Addition of an ionophore in a feed or mineral mixture will enhance the formation of the glucose precursor propionic acid, and improve effeciency of feed utilization.

Urolithiasis

Obstructive urolithiasis should be treated as an emergency. Immediate slaughter should be considered in feedlot or grade animals if rupture of the bladder or urethra has not occurred. If surgery is indicated, it should not be delayed. Dehydration and electrolyte abnormalities should be corrected with isotonic sodium chloride during surgery. If hyperkalemia is severe, adding dextrose or sodium bicarbonate to fluids may help decrease potassium. Calcium may also be needed. Nonsteroidal anti-inflammatory drugs are an important part of therapy. Not only do they help with pain, shock, and urethral swelling in the acute stages of the disease, they may also help decrease the amount of urethral stricture formation chronically. Broad-spectrum antibiotics should be administered prophylactically.

In cases of urethral obstruction, if the penis can be exteriorized, the urethral process can be removed with sharp scissors or a scalpel blade if it has not already necrosed off. Antibiotic and/or steroid cream can be applied to the penis. Removal of the urethral process may relieve the obstruction initially, but obstruction at the sigmoid flexure commonly occurs secondarily, so careful monitoring for recurrence is necessary. Sedation and/or caudal epidural may facilitate exteriorization of the penis. Acepromazine is the sedative of choice as it may also have antispasmodic effects on the urethra. Xylazine should be avoided due to its diuretic effects. Urethral catheterization is difficult to perform and should be performed with extreme caution to prevent rupture of the urethra. A mixture of 1 part 2% lidocaine and 3 parts saline may relieve some urethral spasms and facilitate flushing.

A more acceptable alternative to urethral catheterization if removing the urethral process fails to relieve the obstruction or the obstruction recurs is chemical dissolution of the calculi. Under general anesthesia, the bladder is located with ultrasound, and an 18 gauge 4 inch needle is inserted into the trigone of the bladder. Urine is aspirated until the bladder is small, and 30-60 mls of Walpole's solution is placed in the bladder and removed again until the turbidity of the urine is decreased. Then another 30 to 50 mls of Walpole's solution is infused into the bladder and the needle removed. Urine flow is usually seen in 24-36 hours, and is normal in 3-5 days. A second infusion of Walpole's may be needed in some cases.

Several surgical procedures are available for urethral obstruction. If the animal is destined for slaughter, temporary measures such as perineal or ischial urethrostomy or penile amputation can be performed. Urethrostomy can be difficult to perform in small ruminants, and stricture formation with obstruction at the surgical sight usually occurs in a few weeks to a few months. If the stones can be palpated in the urethra, a urethrotomy can be performed at this sight. However, stricture formation and reobstruction can still occur. For cases in which breeding is important, or in pets in which long term survival is important, a

tube cystostomy or bladder marsupialization is recommended.

If the tube cystostomy fails (estimated to fail in about 20% of cases), or the animal is a pet and breeding is not desired, the bladder can be marsupialized. A stoma is made lateral to the prepuce and as cranial as the bladder will allow. Note that this surgery can be difficult to perform following a failed tube cystostomy due to intra-abdominal adhesions. Although breeding success rates following marsupialization have not been reported, the author is aware of one animal that successfully bred females following bladder marsupialization.

The prognosis for urethral rupture is poor compared to simple obstruction or bladder rupture if these are treated appropriately. With urethral rupture, urine still needs an outlet, so a urethrostomy or penile amputation should be performed. Tube cystostomy or bladder marsupialization may not be able to be performed due to the severe ventral accumulation of urine, and tissue necrosis. Multiple incisions in the skin to allow drainage, and debridement of necrotic tissue is necessary. Severe adhesions of the prepuce and penis may interfere with subsequent breeding.

Prevention

Because preventive measures are dependent on the type of stone present, which is difficult to predict in small ruminants, it is important to have calculi analyzed. A thorough dietary history is necessary to determine any predisposing dietary factors. Castration of pet animals should be delayed as long as possible, preferably until the preputial attachment to the penis has broken down.

Increased water intake is indicated with any type of stone. This can be accomplished by adding salt to the diet at 4% to 5%. If free choice trace mineralized salt is the source of minerals, intake of these may drop when salt is added to the ration. Therefore, adequate minerals should be added to the ration. Clean water should be provided at all times. In winter, warm water can be provided to pet animals to keep intake up.

Pet small ruminants not used for breeding tend toward obesity, and can usually be maintained on *good quality* grass hay. Concentrate supplements should be reserved for breeding or feedlot animals. The concentrate in the diet should be limited to 25% of the ration (feedlot rations may be the exception). A 1.5 to 2:1 Ca:P ratio should be maintained in the entire diet. The total recommended amounts of calcium and phosphorus should not be exceeded, and magnesium should not be in excess. Adequate vitamin A should be included in the ration, especially if dry forages are the majority of the ration. Legume hay should be avoided, or limited to only what is needed if certain production systems require them.

Acidification of the urine can be accomplished several ways. For large numbers of animals, an anionic ration similar to that fed to dairy cattle to prevent hypocalcemia can be fed. Also, ammonium chloride can be added at 0.5% to 1% of the total ration, or 2% of the concentrate. For individual animals 5-10 grams/head/day should maintain the urine pH at approximately 6.5. This can be checked weekly by the owner with pH paper. Ammonium chloride is highly unpalatable, and should be mixed with syrup or put in gelcaps for administration (avoid molasses since it is high in cations). Vitamin C has to be administered several times per day in ruminants to be effective so is impractical.

Practical Fluid Therapy For The Field: Cattle And Small Ruminants

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suspected. Since localized peritonitis is more common than generalized peritonitis, four sites are tapped. The two cranial sites are slightly caudal to the xyphoid and medial to the milk veins on the left and right sides. The two caudal sites are slightly cranial to the mammary gland and to the left and right of midline. For either technique, manual restraint with sedation is recommended, and the use of real time ultrasonography may help locate fluid pockets. A twenty-gauge needle or teat cannula can be used for fluid collection. Sterile preparation of the site is needed, and local anesthesia is necessary when a teat cannula is employed. Fluid should be collected into a small EDTA tube for analysis, and a sterile tube for culture. Fluid can be difficult to obtain, and is usually in small amounts. Care should be taken to minimize the ratio of EDTA to fluid, since EDTA can falsely elevate protein levels. Use of EDTA tubes made for small animals, or shaking excess EDTA out of large tubes will resolve this problem. Fluid can also be collected for culture. Normal values are similar to those for cattle (clear, colorless to slightly yellow, 1-5 gm/dl protein, < 10,000 cells). Cytologic examination is needed to characterize the cell population, and look for the presence of phagocytized bacteria.

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needle or can be gently aspirated with a syringe. If the needle is in the lumbosacral space and advanced until bone is encountered again, back the needle out 1-2mm and try to aspirate. Place the fluid in an EDTA tube for fluid analysis and a plain tube if cultures are desired. Normal, non-traumatically obtained fluid should be perfectly clear with no discoloration, sediment or turbidity. It is best to have the sample analyzed locally as soon as possible (within one hour). If this is not possible, place half of the sample in an equal volume of 40% ethanol to preserve the cells (inform the laboratory that this has been done), or centrifuge half the sample to concentrate the cells and prepare slides to be sent with the rest of the fluid.

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Procedure: 1. Insert the needle into the dorsal aspect of the pastern, in the groove between the proximal phalanges, just distal to the fetlock. Administer 5cc of lidocaine deep, and 5cc while pulling the needle out. 2. Repeat the previous block from the palmar/plantar aspect of the pastern, just distal to the dewclaws. 3. Palpate the nerve over the lateral aspect of the fetlock, approximately 2cm dorsal and proximal to the dewclaw. Administer 5cc of lidocaine over the nerve. 4. Repeat the previous block on the medial side.

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Indications: Cytology or histopathology for diagnosis of Johne's Disease.

Materials: Rectal biopsy instrument, sharpened needle cap, or bottle cap.

Procedure: Place hand wrist deep into the rectum. Secure a fold of mucosa between two fingers and pinch off a piece of the mucosa with the biopsy instrument inserted with the opposite hand. Alternatively, pinch off a piece of mucosa between thumb and needle cap. Make impression smears for acid-fast staining or place the sample in formalin for histopathology.

Caution: Only go in wrist deep, staying in the retroperitoneal area to prevent a full thickness biopsy and peritonitis.

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Indications: Diagnosis of Johne's Disease or Cryptosporidiosis.

Materials: Acid-fast stain, slides

Procedure: Smear a small amount of feces on a slide. Heat fix the slide for Johne's and let it air dry for Cryptosporidiosis. Under 40x or 100x, look for small, acid-fast bacilli in clumps (Mycobacterium paratuberculosis), or round, refractile, acd-fast protozoa (Cryptosporidium).

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Hoof Block

A wooden, plastic or rubber hoof block, placed on the normal claw removes the source of pain by preventing weight bearing on the affected claw and allows the animal to ambulate comfortably while healing takes place. This is an affective for treatment of P3 fractures, and is essential for treatment of sole ulcers and serious hoof cracks. Blocks 1-2 inches thick, cut to fit the shape of the claw, with grooves on both sides, are glued to the hoof with some type of bonding material. The wooden hoof blocks and Technovit® acrylic can be obtained from Jorgensen Laboratories. Bovi-Bond® is a newer product by Vettec that is very easy to use. The same company makes 1 inch plastic or rubber blocks that can be glued together if a taller block is needed. The black plastic blocks are the widest and fit beef bulls the best. Although Cow Slips® are used routinely in dairy cattle, they don't always fit beef cattle, especially bulls. All products can be found at Animart (www.animart.com or 800-255-1181).

Preparation of the claw is crucial for preventing the blocks from falling off too soon. The claw should be clean, dry and level. Shallow grooves can be made in the sole with a hoof knife but are not necessary in my opinion. The Bovi-Bond® sets up quicker than the Technovit®, so everything should be ready. In cold, wet weather, a hair dryer helps to quicken setup. When applying the acrylic, pay particular attention to the axial surface, making sure it's smooth and will not irritate the interdigital space or the axial surface of the opposite claw. Do not spread the acrylic onto the coronary band or up on the soft part of the heel. Make sure the block is positioned so that the animal does not rock back on its heel, and the toe doesn't tip up. Caution: If the lameness worsens with the hoof block on or if the animal develops a lameness after wearing the block for several days, , remove the block immediately and reevaluate the problem. The hoof block may be causing the lameness. Also, the heat produced during the hardening process can cause thermal necrosis of soft tissues under the hoof wall. Use sparingly on the hooves of young calves and small ruminants.

Other Uses of Acrylic

Acrylic can be used alone or in combination with wire to repair hoof wall defects. However, the hoof defect must be clean, dry, and free of infection before the acrylic can be placed over it. Hoof cracks are very common in beef cattle. Many times they are found during routine foot trimming. The author does not recommend any treatment of hoof cracks unless they are causing lameness, especially if the patient is a bull receiving a hoof trim immediately prior to breeding season and is not lame. Many times in paring out hoof cracks with a knife or rotary tool, sensitive lamina is encountered and the animal becomes temporarily lame. Many animals have hoof cracks for years with no lameness associated with them. If the hoof crack is causing lameness, then it must be treated.

Local Intravenous Anesthesia

Local intravenous anesthesia is the preferred technique for surgical procedures of the foot and pastern. Although clipping is not necessary, a

surgical scrub should be performed prior to injection. A tourniquet is placed proximal to the fetlock immediately prior to injection (vein will be distended best immediately after the tourniquet is placed). Two sites of injection are available. One vein runs down the center of the dorsal aspect of the pastern and the other runs approximately 2cm dorsal to the dewclaw, on both the lateral and medial sides of the foot. A 20 gauge needle or butterfly catheter is inserted into the vein and 15-20cc of lidocaine or carbocaine is administered. It is only necessary to administer an anesthetic into one of these veins to provide anesthesia to the entire area distal to the tourniquet. The tourniquet can be safely left on for up to 1 hour to provide hemostasis during surgical procedures.

In feet with severe cellulitis, local intravenous anesthesia can be difficult. In these cases, a four point nerve block or a simple ring block will also work. The two interdigital injections performed in the four point block can be used for removal of an interdigital fibroma.

Joint Injection and Lavage

Septic arthritis is a serious condition that requires immediate treatment. Although it is tempting to try systemic antibiotics alone first, the chances of this working are low, and if systemic antibiotics fail, the disease may have progressed to the point that the joint can't be saved. For joint lavage to be successful, it needs to be performed very early (first few days) in the course of the disease. Since veterinarians rarely see these cases early, this is not a common technique used for treatment of the coffin, pastern or fetlock joint in adult cattle. The coffin joint is especially difficult to tap in cattle. If a needle can be placed in the joint space, isotonic fluids can be administered under pressure to distend the joint. A second needle is then placed in the joint space and a through and through lavage is performed, preferably with about 500-1000 mls. The larger the needle (14 to 16 gauge) the better the lavage.

Joint Arthrodesis/Facilitated Ankylosis

When septic arthritis of the coffin or pastern joint has advanced to the point that there are radiographic changes in the bone surrounding the joint, two options exist. One is claw amputation and one is joint arthrodesis. Joint arthrodesis is preferable in valuable breeding animals, especially bulls, because it saves the claw. Although the procedure is easy to perform and relatively inexpensive, the aftercare (flushing wound, bandage changes, keeping a hoof block on and toes wired together, and/or cast application) can be prolonged (1-2 months), which increases the cost over amputation. Also, if significant cellulitis is present, or there is radiographic evidence of osteomyelitis proximal to the joint being athrodesed, the success rate of arthrodesis is poor, and amputation should be considered. The procedure is performed under local anesthesia, and only requires a shop drill and 1/4 and ½ inch drill bits (sterilized). There are several techniques described. Contact the author for references describing the details of the procedure. The owners should be aware that time to breeding soundness in bulls can be several months, even once the actual infection is cleared. The fetlock joint can also be successfully arthrodesed, although a splint or cast is needed for 6-8 weeks for support during healing.

Pregnancy Toxemia

Pregnancy toxemia (twin lamb disease, lambing or kidding sickness) is most common does with triplets, and/or are either thin or obese. Much of the abdominal space is occupied with multiple feti in the uterus during late gestation. Fat accumulation in the abdomen in obese animals also occupies space. Because of lack of space for the rumen, these females have difficulty consuming enough feedstuffs to satisfy their requirements. In late gestation, nutritional requirements increase to 150% of maintenance with a single fetus and 200% with twins. Late gestation is usually during the winter months, when less pasture is available, and as a general rule, poorer quality feeds are available. Pregnancy toxemia is also seen following anorexia secondary to other diseases (ex. foot rot, ovine progressive pneumonia, caprine arthritis encephalitis virus), or with stresses such as bad weather, transporting, etc. There is also a genetic predisposition in some individual animals.

Very early signs of pregnancy toxemia are mild depression, anorexia and possibly limb edema. If left untreated, goats become anorexic and depressed, and soon become recumbent. Neurologic signs including blindness, circling, incoordination, star-gazing, and tremors. Constipation and teeth grinding can also occur Icreased respiratory rate develops if acidosis occurs. If left untreated, does become recumbent.

Azotemia, both from dehydration and secondary renal disease, is a common finding. A urinalysis will be positive for both ketones and protein. Ketoacidosis is common in small ruminants. Hypocalcemia and hypokalemia may be present due to anorexia. They are not always hypoglycemic. Liver enzymes are usually found to be within normal limits, but occasionally may be increased.

Diagnosis is based on clinical signs, the presence of multiple fetuses, and typical clinicopathologic findings. Differential diagnoses include listeriosis, hypocalcemia, polioencephalomalacia, hypomagnesemia, and meningeal worm infestation.

In small ruminants, very early cases (prior to recumbency) may be treated with oral glucose or glucose precursors. 60-100 mls of propylene glycol orally twice a day, or oral corn syrup or glycerol can be tried. Oral high energy calf electrolytes with bicarb can also be given orally. Rumen transfaunation, vitamin B complex (including B_{12}, biotin, and niacin) are also recommended treatments.

Once females show neurologic signs or become recumbent, treatment must be very aggressive. IV glucose, calcium borogluconate, and bicarbonate may be needed.

Glucocorticoids (15–20 mg dexamethasone) can help by causing gluconeogenesis, increasing appetite and inducing abortion. Prostaglandin ($PGF_{2\alpha}$) should also be used (5-10 mg) for induction of parturition in does. Flunixin meglumine (0.5-1 mg/lb) are indicated if endotoxemia is suspected from dead fetuses.

Removal of the fetuses is critical in these severe cases. Assessment of fetal viability with ultrasound helps with the decision to induce parturition or perform a C-section. Since a breeding date is rarely known, age of the fetuses is hard to determine. If the fetuses are alive, induction of parturition can be an option. However, if the fetuses are already dead, or the condition of the doe is severe, an immediate C-section is warranted. Fluid support during and after surgery is critical. Low birth weights of lambs, kids and calves at the beginning of the birthing season can indicate potential risk of pregnancy toxemia.

Prevention of this disease is through proper nutrition. Maintaining animals in proper body condition throughout the year, and making sure energy and protein levels are adequate in late gestation are important. For does in late gestation, hay should have protein content of at least 10%, and 1-2 lbs of concentrate should be fed per head per day. During periods of stress, particularly cold wet weather, concentrate may need to be increased to 2-3 lbs/head/day (divided in two feedings). Parasite control, disease prevention and decreasing stress are important.

Ultrasonography can help determine which females have multiple fetuses, and these animals separated into groups and fed accordingly. Addition of an ionophore in a feed or mineral mixture will enhance the formation of the glucose precursor propionic acid, and improve effeciency of feed utilization.

Urolithiasis

Obstructive urolithiasis should be treated as an emergency. Immediate slaughter should be considered in feedlot or grade animals if rupture of the bladder or urethra has not occurred. If surgery is indicated, it should not be delayed. Dehydration and electrolyte abnormalities should be corrected with isotonic sodium chloride during surgery. If hyperkalemia is severe, adding dextrose or sodium bicarbonate to fluids may help decrease potassium. Calcium may also be needed. Nonsteroidal anti-inflammatory drugs are an important part of therapy. Not only do they help with pain, shock, and urethral swelling in the acute stages of the disease, they may also help decrease the amount of urethral stricture formation chronically. Broad-spectrum antibiotics should be administered prophylactically.

In cases of urethral obstruction, if the penis can be exteriorized, the urethral process can be removed with sharp scissors or a scalpel blade if it has not already necrosed off. Antibiotic and/or steroid cream can be applied to the penis. Removal of the urethral process may relieve the obstruction initially, but obstruction at the sigmoid flexure commonly occurs secondarily, so careful monitoring for recurrence is necessary. Sedation and/or caudal epidural may facilitate exteriorization of the penis. Acepromazine is the sedative of choice as it may also have antispasmodic effects on the urethra. Xylazine should be avoided due to its diuretic effects. Urethral catheterization is difficult to perform and should be performed with extreme caution to prevent rupture of the urethra. A mixture of 1 part 2% lidocaine and 3 parts saline may relieve some urethral spasms and facilitate flushing.

A more acceptable alternative to urethral catheterization if removing the urethral process fails to relieve the obstruction or the obstruction recurs is chemical dissolution of the calculi. Under general anesthesia, the bladder is located with ultrasound, and an 18 gauge 4 inch needle is inserted into the trigone of the bladder. Urine is aspirated until the bladder is small, and 30-60 mls of Walpole's solution is placed in the bladder and removed again until the turbidity of the urine is decreased. Then another 30 to 50 mls of Walpole's solution is infused into the bladder and the needle removed. Urine flow is usually seen in 24-36 hours, and is normal in 3-5 days. A second infusion of Walpole's may be needed in some cases.

Several surgical procedures are available for urethral obstruction. If the animal is destined for slaughter, temporary measures such as perineal or ischial urethrostomy or penile amputation can be performed. Urethrostomy can be difficult to perform in small ruminants, and stricture formation with obstruction at the surgical sight usually occurs in a few weeks to a few months. If the stones can be palpated in the urethra, a urethrotomy can be performed at this sight. However, stricture formation and reobstruction can still occur. For cases in which breeding is important, or in pets in which long term survival is important, a tube cystostomy or bladder marsupialization is recommended.

If the tube cystostomy fails (estimated to fail in about 20% of cases), or the animal is a pet and breeding is not desired, the bladder can be marsupialized. A stoma is made lateral to the prepuce and as cranial as the bladder will allow. Note that this surgery can be difficult to perform following a failed tube cystostomy due to intra-abdominal adhesions. Although breeding success rates following marsupialization have not been reported, the author is aware of one animal that successfully bred females following bladder marsupialization.

The prognosis for urethral rupture is poor compared to simple obstruction or bladder rupture if these are treated appropriately. With urethral rupture, urine still needs an outlet, so a urethrostomy or penile amputation should be performed. Tube cystostomy or bladder marsupialization may not be able to be performed due to the severe ventral accumulation of urine, and tissue necrosis. Multiple incisions in the skin to allow drainage, and debridement of necrotic tissue is necessary. Severe adhesions of the prepuce and penis may interfere with subsequent breeding.

Prevention

Because preventive measures are dependent on the type of stone present, which is difficult to predict in small ruminants, it is important to have calculi analyzed. A thorough dietary history is necessary to determine any predisposing dietary factors. Castration of pet animals should be delayed as long as possible, preferably until the preputial attachment to the penis has broken down.

Increased water intake is indicated with any type of stone. This can be accomplished by adding salt to the diet at 4% to 5%. If free choice trace mineralized salt is the source of minerals, intake of these may drop when salt is added to the ration. Therefore, adequate minerals

should be added to the ration. Clean water should be provided at all times. In winter, warm water can be provided to pet animals to keep intake up.

Pet small ruminants not used for breeding tend toward obesity, and can usually be maintained on *good quality* grass hay. Concentrate supplements should be reserved for breeding or feedlot animals. The concentrate in the diet should be limited to 25% of the ration (feedlot rations may be the exception). A 1.5 to 2:1 Ca:P ratio should be maintained in the entire diet. The total recommended amounts of calcium and phosphorus should not be exceeded, and magnesium should not be in excess. Adequate vitamin A should be included in the ration, especially if dry forages are the majority of the ration. Legume hay should be avoided, or limited to only what is needed if certain production systems require them.

Acidification of the urine can be accomplished several ways. For large numbers of animals, an anionic ration similar to that fed to dairy cattle to prevent hypocalcemia can be fed. Also, ammonium chloride can be added at 0.5% to 1% of the total ration, or 2% of the concentrate. For individual animals 5-10 grams/head/day should maintain the urine pH at approximately 6.5. This can be checked weekly by the owner with pH paper. Ammonium chloride is highly unpalatable, and should be mixed with syrup or put in gelcaps for administration (avoid molasses since it is high in cations). Vitamin C has to be administered several times per day in ruminants to be effective so is impractical.

Basic Steps To Building Herd/Flock Health: Part I And II

INTRODUCTION: *The term "Herd Health" means different things to different people: both producers and veterinarians. Many times producers equate a herd health program with a vaccination program. However, much more that goes into preventive herd health programs than just a good vaccination program.*

It is most cost effective to personalize each program. Many ranches don't need every vaccine available, and in some cases recommendations don't fit the business model of the ranch. I stress to producers that they should not use "cookbook" programs found on the internet. They should consult their veterinarian to tailor the program to their ranch.

Good record keeping is extremely important. Herd records along with management changes and new information will allow the herd health program to be fine-tuned each year.

A key point for producers to understand is that health problems are not usually from a primary disease issue. Management issues (nutrition, biosecurity, genetic selection, etc.) usually allow a disease to take hold. It is futile to chase a disease problem if the underlying management issues are not also addressed.

Herd health programs can be divided into six parts: nutrition; parasite control; biosecurity; vaccinations; stress and genetics. .

NUTRITION

If nutrition is optimized, health and production will be also. If it is not optimized, diseases and production losses will be a problem. Poor nutrition (protein, energy, vitamins, minerals) depresses immunity to diseases and interferes with response to vaccination. Disease problems my be subclinical, but they will be there.

For cow-calf ranches, nutrition in the brood cow has a major impact on calf health and performance and ultimately the profitability of the ranch. One year of poor nutrition can have impacts for multiple years. When cows cannot maintain adequate body condition, dystocia problems increase. Thin cows have trouble pushing with enough force to have calves in a timely manner. This leads to more stillborn and weak calves. Weak calves are more likely to die of cold stress and have poor colostrum intake. Cows that cannot maintain their body condition also produce poor quality colostrum, further compounding failure of passive transfer problems. Failure of passive transfer leads to more disease and death in calves. Any calf that gets sick, even if it recovers, will not ever perform to its genetic potential. Calves that have failure of passive transfer but remain healthy still have decreased performance. Cows that calve thin will either not rebreed or will breed late. And heifers born to thin cows, even when managed with appropriate nutrition, will have decreased reproductive performance when compared to heifers born to cows in good condition. This all adds up to fewer pounds of calf weaned per cow for multiple years and decreased productivity of feeder calves and heifers beyond weaning.

PARASITE CONTROL

Good parasite control is essential for good health and productivity, especially in young animals. Controlling parasites increases weaning weights, milk production, and conception rates. Parasites are also immunosuppressive, so overall disease resistance and response to vaccines is decreased in parasitized animals.

BIOSECURITY

I do not know who to credit with this quote, but it is one of my favorites: "Most disease is bought and paid for". A good biosecurity program will protect a herd from diseases in which there is not a good vaccine available or what's available is very expensive. It is futile to try to eliminate a disease problem if you are not going to prevent it from coming back into the herd. Biosecurity plans can be challenging and time consuming to develop initially, but they are the cheapest and most effective means of disease control. No disease prevention program will work without biosecurity. There are different levels of risk and therefore biosecurity needs with different management/business models. It is up to the veterinarian to discuss the risks of certain management practices and business models and help producers develop practical biosecurity plans that fit each ranch.

Biosecurity plans do not have to be complicated. Since beef breeding animals are usually housed outdoors, the elements help with disease control. Some simple biosecurity recommendations are a good start: test purchased animals for diseases of concern (BVD, Trichomoniasis, etc.); quarantine new arrivals and any animals that are returning from shows or sales; avoid fence line contact with neighboring herds; and purchase breeding stock and embryo recipients from as few sources as possible.

VACCINATIONS

As mentioned before, there is no generic/cookbook vaccination program. Many programs are similar, but each should be tailored to the ranch. Management issues such as disease risk, breeding season, disease history, locale, etc. must all be taken into consideration. A "generic" vaccination program would have to cover all known diseases and be safe to recommend for all herds. The result would be a more costly but less effective vaccination program.

GENETICS

Genetically selecting animals that are more resistant to diseases would be attractive. This is an area of much interest and research is currently ongoing to investigate the genetics of disease resistance for problems such as respiratory disease and parasites.

Fetal programming and epigenetics are also areas of ongoing research. Fetal (or developmental) programming is the concept that a maternal stimulus or insult at a critical period in fetal development has long term impacts on offspring. For example, nutritional stress in the 1st and 2nd trimesters of pregnancy can lead to problems with fetal organ development

and vascularization/placental development. Epigenetics is the study of heritable changes in gene expression or cellular phenotype caused by mechanisms other than changes in the underlying DNA sequence. The resulting adverse long-term effects reflect a mismatch between fetal environmental conditions and the conditions that the individual will confront later in life. For example, when calves are born to thin cows, they may later have health and performance issues when placed on full feed in the feedyard. Knowing the implications of our management practices could lead to recommendations on matching cows to their ideal environment and managing feeder and breeding cattle for better performance.

Much recent research emphasis is being placed on genetic selection of "high immune responders"-animals that genetically are less likely to be diseased. Some progress is being made that has the potential to really improve animal health and welfare.

DECREASE STRESS

Other stressors such as castration, dehorning, weaning, commingling, handling should be minimized. Stress depresses the immune system making animals more susceptible to disease. It also interferes with vaccine response.

Índice

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Update On Reproductive Physiology And Pharmacology

INTRODUCTION: *Knowledge of basic reproductive physiology is essential to understand reproductive phenomena in health and disease. This knowledge is useful in designing or modifying estrus synchronization protocols or therapeutic plans of common reproductive disease that can be treated with hormones.*

ESTROUS CYCLES

The entire estrous cycle averages 21 days long, but it can be as short as 18 days and as long as 24 days. Estrus lasts 18 to 20 hours but may be shorter in hot, humid weather because of heat stress. Estrus can be particularly shorter in Bos indicus cows. Ovulation (Day 0) occurs 12 to 18 hours after the end of estrus. Estrus is followed by metestrus (days 1 to 3), which is the time of luteal development noted by rising concentrations of blood progesterone. A bloody discharge from the vulva may be noticed in nearly half of all cows and heifers 1 or 2 days after they are in estrus. The blood originates from diapedesis of erythrocytes leaking from caruncular capillaries in response to estrogen withdrawal. During metestrus, a corpus hemorrhagicum (CH) is formed at the site of ovulation. This structure will develop into a corpus luteum over the next few days. During metestrus the CL is not susceptible to the luteolytic action of a single administration of prostaglandin. The next phase is called diestrus (Days 4 to 18) is the time that the mature corpus luteum is functional and secretes progesterone. During diestrus there are waves of follicular growth that are important in understanding a cow's response to estrous cycle synchronization. At the end of diestrus, if an embryo is not present in the uterus to provide a pregnancy signal, the uterus releases prostaglandin that lyses the corpus luteum, resulting in a decline of progesterone and the cow returns to proestrus, which is a stage of the cycle that lasts 2 or 3 days until the cow becomes sexually receptive again (estrus).

FOLLICLE DYNAMICS ('WAVES')

Ovarian follicles develop and grown in a continuum characterized by periodic and constant appearance of tertiary follicles. This gradual and repeated growth of follicles occurs in cycles known as follicle waves. Most cattle have 2 or 3 follicle waves during an estrous cycle. A follicle wave is comprised of three distinct stages: recruitment, selection and dominance. A key factor for the success of estrus synchronization resides on the ability of veterinarians to pharmacologically induce a new follicle wave, so that an "young" and "fresh" oocyte is available for fertilization. This same concept is extremely important when one is attempting to superovulate cows in embryo transfer programs: treatment with superovulatory doses of FSH

must be initiated at the time of emergence of a new follicle wave.

HOW TO INITIATE OR INDUCE A NEW FOLLICLE WAVE?

- One practical way to induce the emergence of a follicle wave is to use treatments of GnRH in estrus synchronization programs. A new follicle wave starts in ~ 2 days
  - Refer to the estrus synchronization discussion
- Injections of estradiol (17-beta, benzoate or valerate) can successfully induce a follicle wave in ~ 4 days. This apparently protracted time from the treatment results from an initial down regulation of FSH secretion for ~ 48 hours following estradiol administration, followed by a resurgence of FSH concentrations by Day 2 to 3 following the estradiol treatment that results in the emergence of a new follicle wave by day 4. It is important to check with the regulatory agencies in your country to verify whether or not the use of estrogen in food-producing animals is approved.
- DRF refers to dominant follicle removal. The use of transvaginal ultrasonography to aspirate the preovulatory follicle or any follicle larger than 5 mm (follicle ablation) is very efficient in inducing a new follicle wave in ~ 1.5 days.

Table 1. Common drugs utilized in bovine reproductive management

Bovine	Drugs & dosage form	Route	Dose	Regimen
Induction of ovulation	hCG 1,000 IU / ml	I.V.; I.M.	1,000-2,500 IU	Single dose
	GnRH 50mcg/ml	I.V.; I.M.	100 mcg	
Estrus synchronization	Bioidentical $PGF_{2\alpha}$ (Dinoprost tromethamine; Lutalyse®, 5mg/ ml; or HighCon 12.5 mg/mL	I.M.	25 mg	Double injection 11 or 14 days
	Synthetic PGF2α (cloprostenol; Estrumate®, 250 mcg/ml) d-cloprostenol; 75 mcg/mL		500 mcg or 150 mcg	
	Controlled Internal Drug Release (CIDR; 1.9 g progesterone)	vaginal	One implant	Leave for 5 or 7 days; PGF2α upon removal
	Melengestrol acetate	Oral	0.5 mg/cow/day treat for 14 days	Risk reduced fertility
	*Estradiol benzoate *if approved in your country*	I.M.	2.5 mg	Induce follicle wave
Elective termination of pregnancy	Natural $PGF_{2\alpha}$ (Dinoprost tromethamine; Lutalyse®, 5mg/ ml	I.M.	25 mg	Single dose until 100 days of gestation and between 7 months until term
	Synthetic $PGF_{2\alpha}$ (cloprostenol; Estrumate®, 250 mcg/ml)	I.M.	500 mcg	
	Dexamethasone (Azium®, 2mg/ml)	I.M.	25-35 mg	Single dose 4 months until term; may add PGF2α

Clinical Management Of Ovarian And Uterine Disorders

FOLLICULAR CYSTS (CYSTIC OVARIAN DISEASE)

BACKGROUND

Bovine ovarian cysts are anovulatory structures persisting for varying periods of time that continue to be a significant source of financial loss in dairy cattle, and to a lesser extent in beef cattle. Cystic ovaries may affect cows for longer periods of time, causing a greater loss to individual herds and producers. The occurrence of COD continues to have significant economic impact in the bovine industry.

PATHOGENESIS AND CLINICAL SIGNS

Current considerations: Normal preovulatory follicles measure on average 15 to 20 mm in diameter. Follicular cysts have been traditionally defined as follicular structures that remain anovulatory for at least 7-10 days, measure > 20-25 mm diameter

•May have corpus luteum present

•Non-steroidogenic cysts may allow follicular waves to resume

•Pathogenesis

– Abnormal LH release
– Decreased circulating levels of estrogen, progesterone in high-producing dairy cows owing to high metabolic clearance
•Overall, largely unknown

CLINICAL SIGNS OF STEROIDOGENIC ACTIVE CYSTS

– Predominant estrogen production

•nymphomania

•decreased milk production

– Predominant progesterone production

•prolonged interestrus interval

•anestrus

TREATMENT

– GnRH (gonadotropin-releasing hormone); 100 mcg; IM

– hCG (human chorionic gonadotropin)

•2,000 to 3,000 IU IV or IM

– May associate with CIDR (progesterone)

•7-10 days

– Finish treatment with PGF2alpha at 7-10 days following GnRH/hCG treatment

CONSIDERATIONS

•Most commonly seen in dairy cows during the first 2 months post partum

– Good response to treatment
– Actually some resolve spontaneously
•What about beef cows?

– Some become chronically cystic

•Unresponsive to treatment

•Our approach for chronic conditions:

– hCG then 24 hours transvaginal follicle aspiration + CIDR for 14 days followed by recheck and PGF2alpha

Luteal Cyst

•Typically an old follicular cyst that becomes partially luteinized

•Diagnosis by palpation/ultrasonography

•Treatment

– Often responsive to PGF2alpha
– I recommend to treat luteal cyst as describe above for follicular cysts followed by PGF2alpha in 7-10 days
•Use of CIDRs may be optional

Cystic Corpus Luteum

•Not luteal cyst (pathologic)

•Suggestion for new terminology

– "corpus luteum with a cavity"
– Also known as "hollow CL"
– Physiologically normal; no treatment required
– Diagnosis by ultrasonography

GRANULOSA-THECA CELL TUMORS

•Most common ovarian tumors; somewhat rare in the bovine

•Benign

•Treatment: surgical removal; contralateral ovary enables reproductive potential

UTERINE DISEASES

Pyometra

•Associated with the presence of a functional corpus luteum

– Closed cervix

– Accumulation of pus in uterine horns

•Clinical signs

– Pseudopregnancy
– Anestrus
•Treatment

– PGF2alpha

Mucometra

•May or not be associate with CLs

•Often secondary to tubal, uterine or cervical pathology

– Salpingits
– Endometritis
– Cervical obstruction
•Treatment

– Difficult because underlying primary reason may be chronic

•Poor prognosis due to endometrial atrophy

Endometritis

•May be subclinical

•"repeat breeder"

•Diagnosis

– Ultrasonography
– Low volume lavage
•Culture

•Endometrial cytology

•Often performed to rule out uterine problems as cause of apparent infertility

Uterine abscess

•Relatively uncommon

•Secondary to severe endometritis or iatrogenic (AI, ET flushes, etc)

•Treatment difficult

•Poor prognosis

Fetal mummification

•It occurs after formation of the placenta and fetal ossification (~ 70 d gestation)

– Between the 3rd and 8th months of gestation
– Functional corpus luteum
– Closed cervix
•Causes are difficult to confirm

•Infectious: bovine viral diarrhea (BVD), leptospirosis, fungus,

•Mechanical: compression or torsion of the umbilical cord
– Uterine torsion
– Defective placentation
•Genetic anomalies, chromosomal abnormalities

•Diagnosis

– Transrectal palpation and ultrasonography
•appearance of a compact, firm, and immobile mass without placental fluid or placentomes.
– Often normal physical exam; Except rare decrease in milk production and a loss of weight
•Treatment

– PGF2alpha: I recommend 2 or 3 doses given once daily

– Efficacious; safe expulsion of fetus

•If treatment with PGF2alpha fails… (uncommon)

– Surgical removal

– Laparotomy or colpotomy (not for large fetuses)
•Our recommendation

– Treat with PGF2alpha for a couple of days

– Recheck in 3 to 5 days

•Dilate cervix with PGE2 (1000-1500 mcg misoprostol) if needed or dealing with "large" mummies

– Avoid injections of estradiol to dilate cervix

Fetal maceration

•Fetal death and retention

– Complete or partial following abortion with incomplete cervical dilation (less common)
– Bacterial contamination of uterine contents
•Fetid vaginal discharge; crepitation, fluid, fetal bones

•Treatment

– Not rewarding
– Hysterotomy
•Extensive endometrial damage invariably leads to infertility; culling often recommended

– Oocyte aspiration for IVF is an option for extremely valuable animals

Clinical Management Of Accidents Of Gestation In Cattle

FETAL MUMMIFICATION

Background

The overall incidence of fetal mummification varies from 0.43 to 1.8% of pregnancies but it can be as high as 3-4% in feedlot heifers due to incomplete regression of corpus luteum following induced abortion. Mummification of the fetus can only occur after calcification of the fetal skeleton, therefore, fetal death followed by mummification may occur anywhere from 3rd to 8th month of gestation; most cases occur during 4-6 months. Causes may include torsion of the umbilical cord, Campylobacter fetus, fungal infection, leptospirosis, Infectious Bovine Rhinotracheitis and Bovine Viral Diarrhea, etc. These infectious agents have in common the fact they do not induce placentitis and endometrial release of prostaglandin F2alpha; and are not pyogenic. Fetal mummification may be seen in ewes that become infected with toxoplasmosis during early gestation.

Pathogenesis and Clinical Signs

In mummification, fetal death occurs without luteolysis and adequate cervical dilation. It results in autolysis and fluid resorption in a non-pyogenic environment; fetus & membranes become dehydrated, resulting in a dark brown, leathery fetus with shrunken dried skin and bones. There is no odor or exudate present. Cows affected with mummified fetuses do not show signs of estrus because there is a persistent corpus luteum that not only keeps the cervix closed but also prevents signs of estrus, thus, mummified fetuses may be retained indefinitely. Spontaneous expulsion of mummified fetuses seldom occurs; affected cows typically have a history of failing to calve on time.

Treatment

The treatment of choice is PGF2alpha (25 mg dinoprost i.m); expulsion of mummified fetus is expected in 2 to 4 days. The breeding prognosis good; cows typically conceive in 1 to 3 months following expulsion of fetus. Surgical removal is indicated if fetus is not expelled after therapy with PGF2alpha.

FETAL MACERATION

Background and Pathogenesis

The incidence of fetal maceration is 0.09% of pregnancies. Fetal death followed by maceration may occur at any stage of gestation. If it occurs prior to calcification, the fetus will decompose in fetal tissue debris contained in a purulent liquid material. If fetal demise occurs after fetal calcification, and bacteria from the caudal reproductive tract gain access to the uterus, it will result in bacterial decomposition of autolyzing fetus and membranes. Clinically, there will be a compact mass of bones in a collection of purulent material. For these reasons, fetal maceration with a closed cervix and a functional corpus luteum is rare.

Clinical Signs

Usually a chronic, fetid reddish-gray watery or mucopurulent discharge from the vulva is seen over a period of several weeks to months. In some cases, there may be toxic metritis early but systemic illness is typically absent in later stages. Cows with macerated fetus may experience gradual weight loss and decline in milk production.

Treatment

No satisfactory treatment is available. Poor breeding prognosis due to severe endometrial damage. Surgical removal can be attempted if warranted by the animal's value but it is often a frustratingly unrewarding effort.

FETAL EMPHYSEMA

Similar to fetal maceration in that putrefactive bacteria invade the uterus through an open cervix.It is often detected in later term pregnant animals. Fetal death may be associated with dystocia or incomplete abortion in late gestation. Gross fetal changes include putrefaction, distension with fetid gases, crepitation, dry hair and coat secondary to extensive fluid loss and fetal dehydration. Dystocia involving a fetal emphysema is a complicated and grave condition that is commonly fatal to the dam. In ewes, Clostridium chauvoei may be involved; usually poor prognosis.

HYDRAMNIOS (HYDROPS OF THE AMNION)

Background and Pathogenesis

Normal amnion: The amniotic fluid is clear, colorless, and mucoid in nature. Under normal conditions, the volume of the amniotic compartment is regulated by the fetal swallowing. In early to mid-gestation the amniotic fluid is watery; in late gestation, the fetal bladder sphincter prevents urine outflow and the amniotic fluid becomes more viscid. The accentuated mucoid nature is owing to saliva and secretions of the nasopharynx from fetus.

Hydramnios is a relatively uncommon condition caused by autosomal recessive genes characterized by an abnormal accumulation of amniotic fluid. Pathologically the amount of amniotic fluid greatly increases up to 8-10 times (25-150 liters in cows).Its incidence is only 5-10% of uterine dropsy cases. The condition is associated with a genetic or congenitally defective fetus that has impaired swallowing. The increase of amniotic fluid is gradual. Hydramnios is seen most commonly in cattle, and occasionally in sheep.

Clinical Signs and Prognosis

Hydramnios is characterized by a gradual enlargement or filling of the amniotic cavity over several months during latter half of gestation. The gradual abdominal enlargement lasts leads to a pear-shaped abdomen when the cow is seen from her rear. It is often not noticed until parturition, when a large volume syrupy, viscous, meconium stained fluid is released during calving. In examining cows suspected to having hydrops, it would be important to differentiate hydrops of the amnion or the allantois compartment. In cows affected with hydramnions, the placentomes, and often fetus can be palpated because the chorioallantois, amnion and placentomes are normal. Dystocia is common due to uterine inertia and defective/abnormal fetus. Retention of placenta is a common sequela; milk production in subsequent lactation is generally poor.

- The prognosis for future breeding life of the dam is good, but the fetus is invariably defective and nonviable.

HYDRALLANTOIS (HYDROPS OF THE ALLANTOIS)

Background and Pathogenesis

Normal allantois: The allantois fluid is clear, watery, and amber colored. There is only a small amount of allantois fluid produced from the allantois epithelium prior to functional fetal kidneys. The allantois cavity stores fetal urine delivered through the umbilical cord via urachus. In late gestation and under normal conditions, the volume of fluid may reach 8 to 15 liters.

Hydrallantois is a much more common hydrops condition than hydramnios (85-90% of hydrops conditions); both beef and dairy cattle are affected. In hydrallantois, fluid accumulation may reach 50 to 200 liters. Excessive fluid has specific gravity and characteristics of a transudate due to vascular disturbance occurring in allantois. It is generally characterized by a diseased uterus with many non-functional caruncles and some placentomes greatly enlarged. Adventitial placentation is common, with portions that are necrotic and edematous. Cystic kidneys, hydronephrosis and dysfunction of the fetal tubules with resultant polyuria are seldom involved in pathogenesis of hydrallantois. It generally affects cows > 3 years, unless heifers are affected with congenital caruncle deficit. In older cows caruncle deficit may reflect prior uterine infection or injudicious removal of fetus and/or retained membranes leading to a defective endometrium and caruncles.

Clinical Signs and Prognosis

Occasionally develops as early as 5th month of gestation in severe cases. It usually develops rapidly over 1 to 3 weeks during late gestation; the distended uterus fills a tense, barrel-shaped abdomen (bilateral abdominal distension). The resulting marked abdominal enlargement leads farmers to question breeding dates or suspicion of triplets! Eventually, the abdominal distension leads to digestive symptoms such as anorexia, decreased ruminations and ultimately constipation. Hydrallantois can be misdiagnosed as indigestion, bloat, or traumatic gastritis. The pulse is elevated (90-140/min), accompanied by expiratory grunt.

Reproductive examination: the excessive fluid cause the uterus to palpate greatly distended and tense; the distension in the allantois compartment precludes the palpation of placentomes or fetus. Complications include uterine rupture, rupture of pre-pubic tendon, and ventral hernia. In mild cases, hydrallantois may not be diagnosed until term, when an excessive clear, watery, amber fluid with characteristics of transudate is passed during calving. A greatly enlarged and atonic uterus may cause dystocia. Fetuses are usually slightly smaller than normal and present with edema and ascites. Fetal membranes may be tough and difficult to rupture. The fetus is generally dead at birth or dies soon after. Fetal membranes may be heavy and edematous, and retained fetal membranes and septic metritis are common sequelae. These complications account for the relative high morbidity and mortality of hydrallantois. The prognosis is therefore guarded for life and fertility. Salvage by slaughter is the best option for most producers. Milk production in subsequent lactation is generally poor.

TREATMENT OF HYDRAMNIOS AND HYDRALLANTOIS

Varies with duration and severity of condition. In severe cases, an early decision to salvage is best while affected cows are still in good physical condition. Alternately, prompt termination of pregnancy is desirable and the best approach. Induction of parturition/abortion in affected cows can be achieved by administering 20 mg dexamethasone and 30 mg of PGF2alpha

that result in cervical dilation and abortion within 24 to 48hrs. Inducing abortion is more successful with hydramnios. Dystocia can occur in association with defective fetus (hydramnios) and uterine inertia secondary to uterine distension (hydrallantois). Weak abdominal muscles and absence of strong abdominal pressure are common; cervical dilation are often incomplete. A trochar or plastic tube can be used to draw fluid off slowly over 24 hours prior to Cesarean section. Rapid removal of large volume of fluid may induce shock. Appropriate fluid therapy in large volumes indicated before, during, and after surgery. When terminating hydrallantois by Cesarean section, the uterus may continue to fill with transudate for about 48 hours and it may require further draining. Retention of fetal membranes and secondary metritis is common; treat early with local and parenteral antibiotics and oxytocin to aid in continuing evacuation of the uterus.

Table 1. Hydrallantois and hydramnios

Characteristic	Hydrallantois	Hydramnios
Prevalence	85–95%	5–15%
Rate of development	Rapid (within 1 month)	Slow over several months
Shape of abdomen	(Bilaterally) round and tense	(unilaterally) pear-shaped, not tense
Palpation (per rectum) of placentomes and fetus	Nonpalpable (tense uterus)	Palpable
Gross characteristics of fluid	Watery, clear, amber-colored transudate	Viscid, may contain meconium
Fetus	Small, normal	Grossly abnormal
Placenta	Adventitious	Normal
Refilling after trocharization	Rapid	Does not occur
Occurrence of complications	Common	Uncommon
Outcome	Abortion or maternal death common	Parturition at approximately full term

Adapted from: M. Drost. Complications during gestation in the cow. Theriogenology 2007;68:487-491;

S.J. Roberts. Veterinary obstetrics and genital diseases (theriogenology) (3rd ed.) 1986:225;

V. Sloss, J.H. Dufty (Eds.), Handbook of bovine obstetrics, The Williams & Wilkins Co., Baltimore, MD 1980:89 .

UTERINE TORSION

Background and Pathogenesis

Uterine torsion is most common in dairy cows, but occasionally seen in beef cows, sheep and goats. The etiology involves anatomy, manner of lying down, and maybe sudden falls or rolling. The lesser curvature of uterus in late gestation is supported dorso-laterally by the broad ligament. The greater curvature lies free in abdominal cavity resting on abdominal floor, supported by rumen, the viscera, and abdominal walls. In ruminants the gravid horn is in the shape of an arc or a U-shaped loop with the vagina and ovary at the respective ends of the arc. The ovarian end of the gravid horn forms a narrow base upon which the uterus rests. Torsion involves the rotation of this arc on its transverse axis, and involves both gravid and non-gravid uterine horns. Each time cow lies down or rises, the gravid uterus is suspended in the abdominal cavity, and a sudden slip or fall could cause torsion (down front first; up back first). An increased incidence is seen in cows subjected to stall confinement in the winter. Torsion of the gravid uterus occurs more frequent in pluriparous than primiparous animals; it generally occurs in advanced pregnancy. Most torsions occur in late first stage or early second stage labor and may be associated with strong movements of the fetus. Uterine torsions <180º may be present for days or weeks without clinical symptoms until labor begins and dystocia results.

Clinical Signs and Prognosis

Usually history of prolonged 1st stage of labor (i.e. restless, colic behavior); abdominal straining is absent, or mild and intermittent as fetus cannot enter into birth canal and initiate the cervical dilation and contractions (Ferguson Reflex). Torsions or rotations of the uterus at 45 to 90º are often found during pregnancy and generally correct themselves. Unusual cases involve 180 to 360º torsions that leads to obstruction of blood supply to uterus → congestion, edema, shock, and may be gangrene of uterus. Under these conditions, fetal death is unavoidable.

Reproductive Examination: Diagnosis of uterine torsion and its direction is done via palpation per rectum. The amount of tension on the broad ligaments and arteries indicates severity of torsion. Vaginal walls spiral and a stenosis of the vagina is present but in pre-cervical torsions there may be no vaginal involvement. In ~ 75% of the cases, the cranial portion of the vagina will show a characteristic twisting of the vaginal folds. Torsions may be clockwise or counter-clockwise. Left torsions are more common than right. A left torsion means that the right uterine (gravid) horn moved to the left side. In most cases, the position of the fetus will be dorso-pubic in 180º torsions. Prognosis in torsions prior to term depends upon duration and degree of torsion, and severity of symptoms. If torsions > 180º are diagnosed and treated early, prognosis for dam and fetus are good. Prior to term, best methods of correction are rolling or via laparotomy. Complications include uterine rupture and hemorrhage from ruptured vessels. Only rarely does torsion recur in the subsequent pregnancy.

TREATMENT OF UTERINE TORSION

A. Rolling the Dam

- Disadvantage is manpower (3-6 needed).
- Caste cow in lateral recumbency onto side of torsion.
- Tie both hind limbs and both forelimbs together, leaving 8-10 foot of rope free for pulling.
- Hold head extended with halter.
- Rotate cow rapidly onto opposite side → body overtakes the more slowly rotating gravid uterus.
- If successful, the spiral folds and stenosis of birth canal will have disappeared. If cervix dilated, fetus may be palpated with ease and there may be a rush of fetal fluids.
- May require 2 or 3 rapid rotations to succeed.
- Schafer's method requires less assistance as the cow is rolled slowly with a plank holding uterus stationary.
- Cow caste on side of torsion.
- One end of a 3-4 meters plank (20-30 cm wide) placed over cow's para-lumbar fossa and assistant stands on plank.

B. Rotation of fetus and uterus per vagina

- Only possible if cow at term & has a partially dilated cervix.
- Must be able to introduce hand into uterus to grasp fetus – access depends on severity of twist.
- Rupture membranes first to release fluids → reduce size and weight of the uterus.
- Rock back and forth → momentum → vigorous twist in opposite direction to torsion.
- Detorsion rod – 1 cm steel rod, 80-100 cm long with eye at either end.
- Loop passed over one fetal limb and loop on other side of rod passed over 2nd limb.
- Aim to have loops just above fetlocks.
- Use short broom handle or large screw driver through eye in rod and wrap chain tightly around it.

C. Laparotomy

- Useful earlier in gestation, or when cervix closed.
- Try rolling first if assistance available.
- Open on side of torsion and pass hand down between uterus and abdominal wall and grasp fetal limb. Rock up and down → momentum → lift in direction opposite to torsion.

D. Cesarean Section

- Indicated when other methods fail or when the cervix not adequately dilated.
- In dystocia, cervix may have undergone constriction and emphysematous fetus now present.
- Generally cervix only partially dilated due to atony of cervix and uterus(circulatory disturbance).

E. Complications/Sequelae

- Uterine rupture with peritonitis - Internal hemorrhage - Retained fetal membranes - Septic metritis.

Principles And Applications Of Reproductive Ultrasonography

INTRODUCTION: *Although we are all familiar with reproductive ultrasonography today, it is valid to remember that was not until a couple of decades ago that this extraordinary technology revolutionized the knowledge of female bovine reproduction*

- *Increased knowledge of reproductive physiology*
- *Increased knowledge of reproductive pathology*

- *Potential applications*
  - *Routine clinical applications*
  - *Advanced clinical applications*
  - *Research applications*
- *This presentation will concentrate on the value of ultrasonography for routine clinical applications and examples of its use for advanced assisted reproduction in cows*

ROUTINE CLINICAL APPLICATIONS

- Palpation per rectum allied with transrectal ultrasonography
  - Powerful tool
  - Its routine use should be implemented
- Equipment
  - Portable units with linear-array transducer (typically 5 to 7.5 MHz) designed for transrectal use
  - Choose 5 MHz transducers If only one transducer is being purchased more versatile for general use
- Ovarian structures
  - Follicles
    - Average size of preovulatory follicles: 15-18 mm
  - Corpora lutea
    - It can be solid, uniformly echoic or with a cavity (7-10 mm)
  - Pathology
    - Follicular cysts
    - Luteal cysts
    - Hydrosalpinx
  - Ovarian dynamic events
    - Require serial examinations
    - Detection of follicle growth follicle selection, confirmation of ovulation, etc
  - Use in embryo transfer
    - Evaluation of the response to superovulation protocols
- Follicle development
- Number of corpora lutea before embryo collection
- Assessment of recipient's corpus luteum
- Uterus
  - *Invariably used for pregnancy diagnosis but bovine practitioners should develop an appreciation for interpreting normal and abnormal uterine changes in the non-pregnant cow*
  - Pregnancy
    - Adequate restraint

- Easily adapted by experienced practitioners
- High sensitivity and specificity especially during early gestation (between days 25 to 30)
- Diagnosis of twin pregnancies
- Management?
- Does not increase accuracy of pregnancy diagnosis > 45 days
- Except for inexperienced “palpators”
- Detection of early embryonic loss
- Repeat breeders
- Fetal sexing

ADVANCED CLINICAL USE

- Transvaginal ultrasound guided procedures
  - Aspiration of chronic ovarian follicular cysts
  - Aspiration of follicles for oocyte collection
- In vitro production of embryos

REASONS SUPPORTING THE ADOPTION OF ULTRASONOGRAPHY

- Professional
  - Ultrasound technology is not yet widely used by bovine practitioners
  - Increased diagnostic power
- Assessing breeding efficiency
- Trouble-shooting infertility
  - Use in combination with estrus synchronization protocols
  - Veterinarians are the only group that can perform ultrasound examinations in most states in the US
- Some farmers elect to purchase their own ultrasound equipment
- But they generally lack knowledge and adequate training to efficiently use this technology
  - Veterinarian and Client satisfaction
- Economics
  - Viable investment
- 1-3 years to pay for cost of equipment
- Not including fetal sexing
- Example: 7-14 days earlier diagnosis of non-pregnant animals

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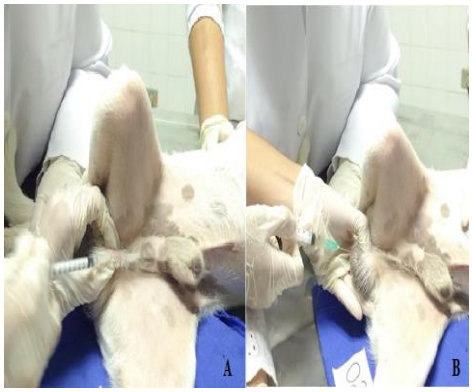
CASTRAÇÃO QUÍMICA EM CÃES COM ÓLEO DE CRAVO DA ÍNDIA

Raquel Freire Feix1 , Bruna Gabriela Kaiser Côrrea, Mayara da Silva Trentim2 , Rita de Cássia Lima Ribeiro, Ana Maria Quessada3 .

INTRODUÇÃO: *Atualmente, o método mais efetivo para controlar a população de cães errantes é a castração cirúrgica. No entanto, tal método apresenta limitações como necessidade de anestesiar o paciente, equipamentos cirúrgicos, instalações adequadas e a presença de um médico veterinário (CATHEY & MEMON 2010). Diante deste cenário, a castração química tem sido sugerida como uma alternativa rápida e de custo baixo (OLIVEIRA et al 2011). Neste procedimento são introduzidos agentes esclerosantes nos testículos (KUTZLER & WOOD, 2006). No entanto, a injeção intra-testicular de substâncias esclerosantes pode provocar dor intensa, mas a literatura é precária quanto à avaliação do processo inflamatório e da dor em animais castrados quimicamente (ROSSETTO et al., 2012). Recentemente foram divulgados estudos utilizando-se óleo essencial de cravo da índia em cães para realização de castração química (ABSHENAS et al 2013, ABU-AHMED, 2015).*

OBJETIVO*: O presente artigo tem o objetivo de analisar a morfologia testicular de cães submetidos à castração química com óleo essencial de cravo da índia, bem como mensurar a dor nestes animais por meio de escalas.*

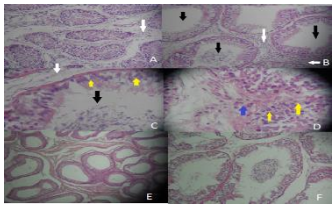
figura 1. Cão, sem raça definida, 10 kg, submetido à castração química com óleo essencial de cravo (Eugenia caryophyllus). A: administração de lidocaína (4mg/kg) no cordão espermático. B: Injeção intra-testicular de óleo essencial de cravo da índia (0,5ml em cada testículo).



RESULTADOS

Os pacientes apresentaram apenas dor leve nas duas escalas empregadas. Não se obteve diferença estatística significativa entre os dois grupos. A consistência dos testículos foi considerada firme para os cães dos dois grupos na palpação realizada antes dos procedimentos. Sete dias após, os testículos dos cães do grupo tratado (óleo de cravo) apresentaram consistência rígida. Quarenta e cinco dias após a castração química, a consistência testicular destes cães estava normal. Nos cães do grupo onde se injetou solução fisiológica a consistência dos testículos foi considerada normal em todas as avaliações. Os testículos dos animais do grupo tratado (óleo de cravo) apresentaram diferença estatística entre a mensuração basal (antes do procedimento) e sete dias após a aplicação do fármaco. No entanto, não houve diferença estatisticamente significativa entre os grupos em relação ao volume testicular ao longo do tempo. Os testículos dos cães em que foi injetada solução fisiológica

apresentaram aspecto macroscópico normal no corte longitudinal (Figura 2A). Os testículos em que foi administrado óleo de



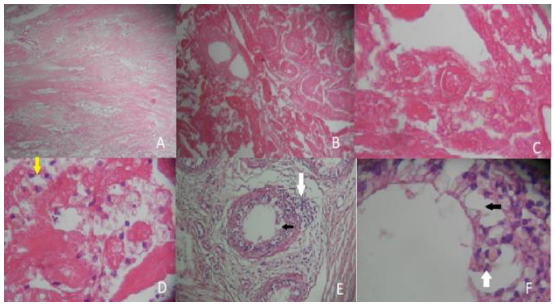
cravo (grupo tratado) apresentaram coloração amarelada e evidente degeneração testicular (Figura 2B). A histopatologia dos testículos dos animais em que foi injetada solução fisiológica (grupo controle) demonstrou aspecto normal (Figura 3). Nos animais tratados com óleo de cravo foram observadas severas alterações degenerativas (Figura 4).

METODOLOGIA

Foram utilizados doze cães, divididos em dois grupos iguais (grupo controle e grupo tratado). Realizou-se anestesia local infiltrativa (cordão espermático e intratesticular) (Figura 1A). Quinze minutos após a realização da anestesia foi introduzido óleo essencial de cravo da índia na dose de 0,5 ml em cada testículo para o grupo tratado (Figura 1B) e 0,5ml de solução fisiológica em cada testículo para o grupo controle. Os testículos de todos os cães foram avaliados por palpação e foi realizada biometria do saco escrotal. A avaliação da consistência e as mensurações da bolsa escrotal foram repetidas sete dias após os procedimentos e aos quarenta e cinco dias. Antes e após a introdução dos fármacos nos testículos, os animais foram submetidos a avaliações de intensidade da dor por meio do emprego de duas diferentes escalas: escala analógica visual e escala de Glasgow modificada. Aos 45 dias de realização dos procedimentos, os pacientes foram submetidos à orquiectomia bilateral. Os testículos foram colhidos e processados histologicamente sendo corados por hematoxilina-eosina. Os dados obtidos foram analisados estatisticamente.

Figura 3: Histopatologia de testículo de cão, submetido à injeção intratesticular de solução fisiológica (grupo controle) em estudo sobre castração química. A: Túbulos seminíferos sem alterações morfológicas em objetiva de 4 x. Estão separados por um delicado estroma de tecido conjuntivo frouxo e células intersticiais (setas brancas). B: Objetiva de 10 x, observando-se luz tubular (setas pretas). C e D:: Lâmina própria tubular, apoiada na membrana basal observando-se células de Sertoli (setas amarelas) e no interstício células de Leydig (seta azul). E: Ducto epididimário, células que revestem o ducto são ciliadas na luz do túbulo. Não há alterações morfológicas evidentes. Objetiva de 10x. F: Ducto epididimário sem alterações morfológicas evidentes. Objetiva de 40x. Células que revestem o ducto são ciliadas na luz do túbulo (seta azul). É possível encontrar espermatozoides (seta preta).

Figura 4: Histopatologia de testículo de cão, submetido à injeção intratesticular de óleo de cravo (Eugenia cariophyllata) (grupo tratado) em estudo sobre castração química. A: Objetiva de 4x. Nota-se alteração no tecido conjuntivo dos septos testiculares que separam os lóbulos dos túbulos seminíferos. Há uma proliferação de fibroblastos cicatriciais, aumentando o espaço do septo. B e C: Objetiva de 10x. Observa-se, necrose difusa nos túbulos e interstício, não evidenciando as células que compõem as estruturas do túbulo seminífero. D: Objetiva de 40x. Encontra-se moderado infiltrado inflamatório com predomínio de células mononucleares (seta amarela), apresentando distribuição multifocal. E: Objetiva de 10x e F: Objetiva de 40x. Ducto epididimário com células de revestimento tubular, apresentando núcleo preservado, porém o citoplasma vacuolizado (setas pretas). Degeneração hidrópica das células ciliadas. Observa-se infiltrado inflamatório misto (setas brancas).



CONCLUSÃO

A administração de óleo essencial de cravo (*Eugenia caryophyllus*) em testículo de cão produz alterações morfológicas macroscópicas e microscópicas, mas não causa dor com o emprego do protocolo analgésico proposto. Sendo assim, o óleo de cravo torna-se uma alternativa preferencial para ser usada em programas que utilizam a castração química como ferramenta para esterilização de cães.

VARIACIÓN EN LA CONCENTRACIÓN DE CORTISOL SÉRICO DE BOVINOS PRE Y POST TRANSPORTE EN CUNDINAMARCA

González-Rozo LV[1], Quiroga-Villarraga S[1], Rodríguez-Rodríguez MA[1], Córdoba-Parra JD[2]

INTRODUCCIÓN. *Actualmente existe una creciente preocupación pública sobre el bienestar de los animales en la mayoría de los países del mundo. Los estudios etológicos aplicados de la motivación, la cognición y la complejidad del comportamiento social en animales han dado como resultado el rápido desarrollo de la ciencia del bienestar animal (Broom, 2011). El embarque, transporte y desembarque son momentos que generan altos niveles de estrés en el bovino, provocando pérdidas económicas relacionadas con contusiones, mortalidad, bajo rendimiento de la canal y alteración de las variables organolépticas de la carne, entre otros (Gallo y Tadich, 2005). Los parámetros utilizados para evaluar el estrés o el bienestar se basan comúnmente en medidas simpato-adrenales, como la frecuencia cardíaca, hormonas plasmáticas, metabolitos e indicadores inmunes (Jacobson y Cook, 1998).*

OBJETIVO.

Evaluar la variación en la concentración sérica de cortisol en bovinos pre y post transporte desde Tabio a Facatativá, Cundinamarca.

MATERIALES Y MÉTODO.

Camión característico para la movilización de bovinos en Colombia. Elementos de restricción física (brete, lazos); Fundas o camisas de agujas de punción intravenosa-vacutainer; Tubos de recolección de sangre sin anticoagulante; un camión, 12 bovinos de la raza Gyrolando de 12-24 meses (nueve hembras, tres machos), equipo de transporte de muestras. Se procede a recolectar la muestra en los animales en un brete antes de embarcados, posteriormente son embarcados y transportados en un recorrido de 45 minutos desde Tabio hasta otra hacienda en Facatativá, Cundinamarca. Luego se desembarcaron y se tomaron nuevamente muestras de sangre en un brete. Las muestras se llevaron a un laboratorio Veterinario privado para procesarlas mediante la técnica de ultrafiltración.

RESULTADOS.

La concentración de cortisol sérico en los animales antes del transporte van desde 10,4 ng/ml hasta 18,8 ng/ml y la concentración de cortisol al momento de desembarque del transporte va desde 20,4 hasta 42,5 ng/ml.

DISCUSIÓN.

La concentración de cortisol total sérico en vacas en lactancia es de 4,5 ±0,7 ng/mL y de cortisol libre fue de 0,3 ng/mL (Shutt y Fell, 1985). En un estudio en Chile, en un transporte de 63 horas en terneros de carne, las concentraciones de cortiol en sangre fueron significativamente más bajas después de desembarcar los animales comparado con los resultados antes del embarque (Werner et al., 2013). En este caso, las concentraciones de cortisol sérico pre transporte fueron de 15,25 ± 2,4 ng/mL y post transporte de 32,65 ± 5,8, resultando casi en el doble el incremento de sus

concentraciones comparando los dos momentos.

Conclusión. El embarque y transporte generan incremento en las concentraciones séricas de cortisol en bovinos, incluso en trayectos cortos (45 minutos).

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PREVALENCIA DE MASTITIS EN UNA LECHERÍA EN CHIQUINQUIRÁ, BOYACÁ, COLOMBIA.

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INTRODUCCIÓN. *La mastitis es una enfermedad con una amplia distribución a nivel mundial, que se define como la inflamación de la glándula mamaria que generalmente se presenta como una respuesta a la invasión por microorganismos y se caracteriza por daños en el epitelio glandular, seguido por una inflamación clínica o subclínica (Pinzón A. , 2007). Esta enfermedad genera graves problemas económicos para la industria lechera, entre los cuales se encuentran la disminución en la producción de leche y costos de tratamientos, entre otros. Puede clasificarse como clínica o subclínica (Abebe, 2016). Una de las técnicas empleadas para evaluar la afección de las glándula mamaria es el California Mastitis Test (CMT)®, esta prueba estima un conteo total de células somáticas, por medio de un reactivo; es una prueba sencilla, de bajo costo, practica y se pueden obtener resultados diagnósticos de forma inmediata (Ballón 2013; Anderson 2014).*

OBJETIVO.

Determinar la prevalencia de mastitis en una lechería en Chiquinquirá, Boyacá, Colombia.

MATERIALES Y MÉTODOS.

Se realizó un chequeo de mastitis en noviembre de 2017 con un producto a base de sulfato de sodio y raqueta para evaluación de mastitis por cuartos, en 24 vacas en producción de leche raza Holstein. El ordeño se lleva a cabo en la lechería con un equipo mecánico. La lechería está ubicada en el municipio de Chiquinquirá, Departamento Boyacá, Colombia.

RESULTADOS.

Se encontró una prevalencia por animal de 66% (16 / 24 animales), de los cuales el 4% (una vaca) presentó mastitis clínica y las demás presentaron mastitis sub clínica. La prevalencia por cuartos fue de 47,9% (46 / 96 cuartos). El cuarto posterior izquierdo fue el más afectado (50%), seguido por el anterior izquierdo (45%), luego el anterior derecho (41%) y por último el posterior derecho (37%).

DISCUSIÓN.

La prevalencia de mastitis por vaca en este estudio fue de 66%. Pinzón (2007), encontró una prevalencia de mastitis clínica y sub clínica mayor al 50%. Dentro de los factores asociados se reporta el inadecuado manejo en la rutina de ordeño, ya que favorece la transmisión de microorganismos patógenos de cuartos infectados a cuartos sanos (Abebe, 2016). De acuerdo a los hallazgos de la afección por cuartos en este estudio, no concuerda con lo reportado por Bonifaz (2016), quien encontró que el cuarto más afectado es el posterior derecho, seguido del cuarto posterior izquierdo.

CONCLUSIONES.

Se encontró en el estudio una prevalencia del 66% de mastitis en la lechería, lo cual evidencia un problema a nivel sanitario relacionado con la salud de la glándula mamaria y posiblemente repercutiendo la calidad de la leche. La mastitis clínica fue de 4% y la sub clínica de 62%, reperestando en valores absolutos que de 16 vacas afectadas con mastitis, una corresponde con el mayor grado de severidad (clínica). Se recomienda revisar los posibles factores que estén influenciando la alta prevalencia de mastitis

e impementar nuevas estrategias para reducir este porcentaje.

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ESTUDIO PRELIMINAR DE TIPIFICACIÓN SANGUÍNEA EN CANINOS EN CASOS DE EMERGENCIA

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INTRODUCCIÓN: *La transfusión sanguínea en medicina veterinaria es una práctica médica que se ha desarrollado en los últimos años [(1)]. En la actualidad, fuera de la transfusión de sangre entera, se cuenta con la transfusión de hemoderivados como el concentrado de glóbulos rojos, plasma fresco, concentrado de plaquetas, entre otros [(1,2)]. También se han desarrollado productos sintéticos que sirven para casos de emergencia como la oxyglobina [(3)]. El sistema DEA es la clasificación de tipos de sangre aceptada internacionalmente para caninos, la cual reconoce 12 grupos de sangre, incluyendo DEA 1, 3, 4, 5 y 7 y el nuevo grupo llamado DA l [(4)] El grupo DEA 1 contiene tres antígenos DEA1.1, DEA1.2 y DEA 1.3; siendo el DEA 1.1 el más común y más antigénico en perros de diferentes países (45-64%)[(4)]; sin embargo, el DEA 1.2, y según algunos autores, el DEA 7 también se pueden considerar antigénicos[(2)]. No se han identificado aloanticuerpos naturales contra este antígeno por lo que reacciones inmunológicas en la primera transfusión son raras. Sin embargo, si se trasfunde sangre DEA 1.1 a un paciente que es negativo para este antígeno, se estimula una respuesta de anticuerpos. Frente a una segunda exposición todos los glóbulos rojos trasfundidos serán destruidos en menos de 12 horas [(1,2).] Durante la preñez y lactancia se pueden inducir aloanticuerpos para DEA 1.1 hasta en un 25% de los perros [(1,4).] Existen varios métodos para tipificación sanguínea [(5,6).] La tipificación en laboratorios de referencia hace que sea poco práctico y oneroso. La presencia de kits comerciales simplifica este proceso y se obtienen resultados rápidos y en la misma clínica [(5,6)]. Los caninos con presencia de antígeno DEA 1.1 son considerados receptores universales, mientras que los negativos para el antígeno (DEA 1.1-) son considerados donadores universales; y el pasaje de sangre de un paciente DEA 1.1 +, así sea en la primera transfusión, no está recomendado [(5,6)].*

OBJETIVOS:

Identificar el tipo de sangre de los pacientes caninos sometidos a una primera transfusión sanguínea en caso de emergencia.

METODOLOGÍA:

Se trabajó con 9 pacientes que llegaron a la clínica con un hematocrito menor a 20% y signos de descompensación asociados a la anemia o hipovolemia (membranas mucosas pálidas, tiempo de llenado capilar mayor a 2", taquicardia, afección de sensorio, alteraciones del pulso, signos eminentes de shock), con 6 unidades de sangre entera provistas por un banco de sangre comercial, con códigos de identificación diferentes, y 3 donadores (requisito no haber tenido transfusión previa). Previo a la transfusión se utilizó el kit comercial de tipificación sanguínea en caninos (*Alvedia® Blood typing DEA 1)* tanto para las unidades de sangre como para las muestras de sangre con EDTA de los pacientes y donadores. La técnica de tipificación es en base a inmunocromatografía utilizando

anticuerpos monoclonales específicos. Se coloca una gota de sangre del paciente y una del reactivo y se espera 2 minutos por los resultados. La tinción de la banda de control refiere que la prueba fue bien realizada. La presencia de dos bandas en la tira indica positivo para DEA 1.1+. Si la tinción es débil se considera DEA 1.1+ débil (DEA 1.2), si solo se pinta el control se considera DEA1.1-. Los resultados describen si el paciente presenta antígeno DEA 1.1 ó 1.2 (DEA 1.1+) o no los presenta (DEA 1.1-). Los resultados fueron tabulados y resumidos en frecuencia de tipo de sangre. También se realizó prueba crossmatch en los casos en los que se contó con donador (3 casos).

RESULTADOS Y DISCUSIÓN:

Se obtuvo un total de 8/18 (44.4%) animales positivos para el DEA 1.1+, de los cuales 1/8 fue débil considerándose DEA 1.1+débil (DEA1.2). Por otro lado, 10/18 (55.6%) fueron DEA 1.1-. Todos los receptores (100%) resultaron DEA1.1-; de los donadores el 88.9% (8/9) fueron positivos al antígeno DEA 1.1+ y 1/9 fue negativo para el mismo. Todas las pruebas crossmatch resultaron sin evidencia de aglutinación o hemólisis macro o microscópica. La tendencia de los grupos sanguíneos corresponde a la bibliografía revisada. En los casos donde los donadores fueron DEA 1.1+ y los receptores no, se consideró la búsqueda de nuevas unidades de sangre. Si bien, los caninos no presentan aloanticuerpos naturales que puedan generar una reacción transfusional aguda importante; bajo ninguna recomendación actual se sugiere, aún en la primera transfusión, el pasaje de sangre DEA 1.1+ a un individuo DEA 1.1-, en base a que el tiempo de sobrevida de glóbulos rojos es menor y se sensibiliza la sangre para un futuro. Consecuentemente este paciente estará sensibilizado de por vida y ante el marco de una segunda transfusión con sangre DEA 1.1+ el riesgo de reacciones transfusionales agudas, y de la vida del paciente es muy alto. Asimismo, estos anticuerpos van a ser trasladados por la leche materna (calostro) afectando la vida a futuro de los cachorros DEA 1.1-. Estudios de casos han señalado que la transfusión de DEA 1.1+ débil en individuos DEA 1.1- de igual manera genera una producción alta de anticuerpos contra el antígeno DEA1.1+.

CONCLUSIONES:

1) La tipificación sanguínea debe realizarse como parte de un control sanitario en etapa de cachorro.

2) La tipificación sanguínea debería ser parte de un protocolo de primera transfusión.

3) No se debe transfundir sangre DEA 1.1+ a un paciente DEA 1.1- bajo ninguna razón.

4) Se requiere realizar estudios con más pacientes para establecer porcentajes estadísticos de los tipos de sangre en caninos en nuestra población.

CARACTERIZACIÓN DEL MANEJO DE RESUCITACIÓN CARDIOPULMONAR (RCP) EN CANINOS Y FELINOS EN CLÍNICAS VETERINARIAS DE LIMA METROPOLITANA Y CALLAO

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La resucitación cardiopulmonar (RCP) es un protocolo organizado para proporcionar soporte artificial a la ventilación y circulación de un individuo ante un parto cardiorrespiratorio (PCR), hasta que este restaure la respiración y circulación espontánea. La ejecución de esta técnica e un reto tanto para médicos humanos y médico veterinarios alrededor del mundo debido a una baja de supervivencia. Menos del 6% de los perros y entre 2% a 10% de los gatos que generan un PCR salieron de alta. La Sociedad Veterinaria de Emergencias y Cuidados Críticos (VECCS) mediante la Campaña de Evaluación en Reanimación Veterinaria (RECOVER) presentó una serie de recomendaciones consensuadas para la aplicación y estandarización de la técnica de RCP en caninos y felinos. Debido a la falta de información de las técnicas utilizadas en Lima metropolitana y la Provincia constitucional del Callao, se realizaron encuestas con la finalidad de caracterizar el manejo de RCP en caninos y felinos en los centros veterinarios de estas áreas. De 100 médicos veterinarios encuestados, el 52% fueron mujeres, predominando profesionales menores de 35 años y con menos de 10 años de tiempo de ejercicio profesional. El 66% de profesionales no laboraban en centros con atención las 24 horas, y más del 50% no contaban con las medidas de preparación ni herramientas adecuadas para atender un paro cardiorrespiratorio. La intubación endotraqueal, el acceso venoso y el uso de compresiones torácicas laterales son técnicas frecuentemente utilizadas en una RCP. Sin embargo, no siempre se siguen las recomendaciones para la atención adecuada de una RCP. Se observó la tendencia por optar por una frecuencia de compresiones menor a 80cpm y una frecuencia respiratoria mayor a 16rpm, contrastando con lo recomendado de 100cpm y 10rpm. Más del 50% prefiere utilizar dosis alta de fluido terapia y fármacos como epinefrina a dosis alta, glucocorticoides y doxapram; terapéutica no recomendada para el manejo de una RCP. No existe diferencia entre médicos veterinarios que recibieron entrenamiento sobre RCP sobre aquellos que no. Se concluyó que el manejo de RCP se realiza de manera heterogénea y sin protocolos. Esto evidencia la falta de entrenamiento y capacitación profesional, reflejado un deficiente manejo del soporte vital tanto básico como avanzado. Con el fin de mejorar el desempeño de los médicos veterinarios en el área de emergencias y cuidados críticos, es necesaria la implementación de estrategias de capacitación continua.

INDICADORES DEMOGRÁFICOS Y ESTIMACIÓN DE LA POBLACIÓN DE CANES Y FELINOS DOMÉSTICOS CON DUEÑO EN EL DISTRITO DE SAN BORJA, LIMA-PERÚ, 2017

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RESUMEN: *El comportamiento de la tenencia de perros y gatos varía de acuerdo con el lugar geográfico en el que se desarrolla la estimación. Su conocimiento es importante para el desarrollo, promoción y planificación adecuada de los programas de salud preventiva, educación sanitaria y tenencia responsable de animales de compañía. El objetivo del estudio fue cuantificar los indicadores demográficos y estimar la población de canes y felinos domésticos en el distrito de San Borja. Para esto, se diseñó un estudio observacional con base en encuestas que consideraron como variables: tipo de viviendas, número de personas por vivienda, tenencia y número de canes y felinos domésticos, características demográficas (sexo, edad y raza), datos reproductivos y edad de fallecimiento del último can o felino. La recolección de la información se hizo en viviendas, que se seleccionaron con base en un muestreo estratificado aleatorio de manzanas o conjuntos habitacionales, encuestándose 10 viviendas en cada caso. Se recolectaron 871 encuestas válidas, las que provinieron principalmente de casas (57.7%) y departamentos (40.8%). Del total de las viviendas el 54.2% tenía perros y 11.6% gatos. El promedio de animales por vivienda fue de 1.4 para los canes y 1.7 para los felinos. Se obtuvo una relación persona: can de 5:1 y persona: felino de 19.5:1. El porcentaje de supervivencia de las crías fue estimado en 88.6% en perros y 88.4% en gatos y la esperanza de vida estimada fue de 9.44 y 7.38 años, para canes y felinos domésticos respectivamente, en el mismo orden. El principal uso de los canes y felinos fue como mascota (97.9% y 100% respectivamente). En canes predominó la tenencia de machos (57.2%), de raza pura (67.5%), no esterilizados (70.4%), tamaño pequeño (45.6%) y con una edad promedio de 4.26 años. En felinos, predominaron las hembras (55.3%), esterilizados (63.5%), raza cruzada (79.6%) y con una edad promedio de 2.52 años. Estos valores aportan información demográfica relevante de canes y felinos domésticos que habitan en San Borja, la cual servirá para mejorar la planificación de programas de vacunación y tenencia responsable de estas poblaciones.*

PALABRAS CLAVES: *San Borja, perros, gatos, población, rabia.*

ESTUDIO COMPARATIVO DE LA DETERMINACIÓN DE EDAD BOVINA POR CRONOLOGÍA DENTARIA Y LONGITUD DEL CARTÍLAGO DE LAS APÓFISIS ESPINOSAS DE LAS VÉRTEBRAS TORÁCICAS

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Susana Lem Guerra[1], *Raquel Watanabe Watanabe*[1], *Jorge Vilela Velarde*[1]

RESUMEN: *La evaluación en pie de los animales de abasto es una estimación importante de las características que presentan las carcasas. En el Perú, el sistema de clasificación y tipificación cárnica se basa en la descripción de las características físicas de la res, principalmente, por el patrón de erupción y desgaste dentarios. Aunque la estimación de la madurez por la dentición (evaluación de los dientes incisivos permanentes) es el método más utilizado, la edad fisiológica se puede estimar mejor por el grado de osificación de los cartílagos de las vértebras sacras, lumbares o torácicas. La osificación cartilaginosa es progresiva en sentido caudocraneal conforme el animal madura cronológicamente., iniciándose en la región sacra, continuando en la región lumbar para, posteriormente, finalizar en la región torácica (botones) de la canal a los 6 años de edad en promedio. El objetivo del presente estudio fue comparar la determinación de la edad por cronología dentaria con la longitud del cartílago de las apófisis espinosas de las vértebras torácicas en bovinos de las razas Brown Swiss, Cebú y criollo mejorado. Se evaluaron 375 carcasas de bovinos machos de las razas Cebú, Brown Swiss y Criolla, divididos en 5 grupos etarios acorde a la edad cronológica dentaria (dientes deciduos, dos, cuatro, seis y ocho dientes), y se determinó las longitudes de los cartílagos de las apófisis espinosas de las vértebras torácicas 1-4. Se encontró diferencia significativa entre el peso de carcasa y la longitud de la apófisis espinosa de la primera vértebra torácica de las razas Criolla y Brown Swiss ($p<0.0001$), y Criolla y Cebú ($p<0.0001$), mas no entre Brown Swiss y Cebú ($p=0.0855$). La correlación entre el peso y la longitud de las apófisis espinosas fue negativa y significativa ($p<0.001$). Asimismo, se observó diferencia estadística significativa entre los cinco grupos evaluados en relación a la longitud de los cartílagos de las apófisis espinosas de las primeras cuatro vértebras torácicas y la edad dentaria, lo que sugiere que el método de determinación de la edad mediante la evaluación de la madurez fisiológica del animal beneficiado es confiable.*

PALABRAS CLAVE: *bovinos, edad, dientes, apófisis espinosas, vértebras torácicas*

EVALUACIÓN TRANSCRIPTÓMICA DEL PERFIL INMUNE CELULAR EN LA MUCOSA INTESTINAL DE CRÍAS DE ALPACAS (VICUGNA PACOS)

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RESUMEN: *El objetivo del estudio fue estimar cuantitativamente la expresión génica de los factores transcripcionales (T-bet, STAT-1, STAT-4) y las citocinas (IL-2, IL-2, IFN-γ, TNF-α) involucradas en la activación y desarrollo de linfocitos Th1 de la mucosa intestinal de crías de alpacas de tres grupos etarios de 1-8, 10-21 y 22-47 días de edad, criadas en un sistema extensivo en los Andes peruanos. Se obtuvo muestras intestinales de cuatro centímetros de la porción media del yeyuno de las crías de alpaca de la Estación Experimental IVITA Maranganí (Cusco), las que fueron almacenadas a -196°C y procesadas en la Facultad de Medicina Veterinaria de la Universidad de San Marcos (Lima, Perú). Se extrajo los ARN totales y se realizó las RT-qPCR en tiempo real. La expresión cuantitativa del ARNm fue estimada estimó comparando los perfiles de expresión en el calibrador (feto) mediante el método $2^{-\Delta\Delta Ct}$ usando GAPDH como control endógeno. La expresión de STAT-1, T-bet, IL-2 y TNF-α se incrementó con la edad en el grupo de mayor edad, mientras que la expresión de IFN-γ excedió en cien veces la del calibrador ($p<0.05$). La expresión de STAT-4 e IL-12 no fue significativa. Las expresiones de ambos factores transcripcionales activados por IFN-γ, T-bet ($p<0.05$) y STAT-1 ($p> 0.05$), aumentaron con la edad, del mismo modo que las citocinas involucradas en la respuesta Th1. La expresión de IFN-γ superó en más de 100 veces la del calibrador, lo que evidencia la activación de los linfocitos Th1. No obstante, no puede descartarse la participación de células natural killer, LT $CD8^+$ y LTγδ en la producción de esta citoquina, así como el efecto de parásitos intracelulares y bacterias comensales que promueven la estimulación de las células dendríticas a través de la activación de TLRs y, en consecuencia, la producción de IFN-γ.*

PALABRAS CLAVE: *transcriptómica, respuesta inmune celular, mucosa intestinal, alpaca*

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ESTENOSIS INTESTINAL EN YEYUNO POR CUERPO EXTRAÑO EN CANINO SCHNAUZER DE 8 AÑOS

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INTRODUCCION: L*a estenosis refiere a la constricción/estrechamiento de un orificio/conducto corporal, congénito o adquirido. Puede ocurrir por cuerpos extraños, intususcepción y menos comúnmente por adhesión. La ingestión de cuerpos extraños en caninos se atribuye a hábitos alimenticios indiscriminados, dando como resultado desequilibrio de electrolitos, hipovolemia y toxemia que también se suman a mala digestión y mala absorción de nutrientes (Stagnant loop Syndrome o Síndrome de Asa Estancada).*

OBJETIVOS

Obtener casuística no muy común en nuestro medio, para mejorar el diagnóstico de pacientes con procesos crónicos y así seguir los casos posteriores de la mejor manera posible.

METODOLOGIA

Presentación del caso: Paciente canino de raza Schnauzer de 8 años de edad de nombre George, con historial de anorexia, emesis y diarrea intermitente además de pérdida progresiva de peso de noviembre de 2016 a Junio de 2017. Tiempo en el cual se realizaron exámenes complementarios como hemograma, coproparasitológico, bioquímica sanguínea y examen completo de orina con resultados no concluyentes.

En junio de 2017 se realizó ecografía abdominal donde lo más resaltante fue la evidencia de líquido libre en abdomen y presencia de líquido en asas intestinales, además de congestión de las mismas y a la par se realizó estudio radiográfico de abdomen donde se evidencia gran cantidad de gases en asas intestinales.

RESULTADOS

Se programó al paciente para cirugía, previa transfusión de sangre completa. En la laparotomía se observó estenosis intestinal a nivel de yeyuno, se realizó enterectomía de la zona afectada, el resto de órganos no presentaban alteraciones macroscópicos. En la muestra retirada se encontró material de textura dura y color negro insertado en las paredes del intestino.

CONCLUSIONES

Se concluye que muchas veces en el diagnóstico de cuerpos extraños ninguna prueba es concluyente al 100%, debiendo realizarse diferentes pruebas antes de llegar a un diagnóstico definitivo.

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REPORTE DE 25 CASOS EMPLEANDO TOMOGRAFIA COMPUTARIZADA COMO PRUEBA DIAGNÓSTICA

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Rosalinda Rubio Lezama3

INTRODUCCION : *La tomografía computarizada es una técnica de diagnóstico por imagen no invasiva, tecnología de exploración de rayos X que produce imágenes detalladas de cortes axiales del cuerpo, cada vez más usada en medicina veterinaria. Es utilizada en patologías de cavidad nasal y senos paranasales, medula espinal, fracturas y pacientes oncológicos.*

OBJETIVO

Mejorar el diagnóstico veterinario mediante el uso de pruebas complementarias como es el diagnostico por tomografía en diferentes áreas del organismo.

METODOLOGIA

VENTAJAS

1. Mayor resolución de contraste siendo superior a la radiografía convencional, mediante la toma de imágenes transversales, evita la superposición de estructuras. 2. Permite el uso de medios de contraste que servirán para aumentar la cantidad de información, mejorando la calidad y aumentando la disponibilidad de información morfológica. 3. Método de diagnóstico de elección en medicina veterinaria, debido a su rapidez, fiabilidad y versatilidad, para poder definir con precisión las situaciones y optimizar el tratamiento.

DESVENTAJAS

1. Requiere que los pacientes permanezcan quietos, lo cual quiere decir que deben permanecer sedados para la realización del examen. 2. El riesgo que conlleva este examen ya que, se ven relacionados con el uso de radiaciones ionizantes. 3. Los medios económicos con lo que se cuenta no son favorables.

El presente estudio se basa en el resultado de 25 pacientes caninos, de diferentes edades, raza y sexo, logrando obtener resultados en el 99% de casos y solo un fracaso del 1%, siendo 3 de columna lumbosacra, 13 de columna lumbar, 4 de columna dorso lumbar, 2 de abdomen completo, 2 de cráneo, 1 de tórax.

RESULTADOS

CONCLUSIONES

1. Cada vez más se toma en consideración la tomografía como prueba diagnóstica no solo en afecciones de columna.

2. El elevado costo de una sola prueba, en este caso la tomografía, no permite que esté al alcance de todos.

3. Hacen falta especialistas veterinarios en interpretación de imágenes tomográficas en nuestro medio, siendo muy útil como prueba diagnóstica.

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TUMOR VENÉREO TRANSMISIBLE (TVT) EN CAVIDAD NASAL DE CANINO SCHNAUZER DE 3 AÑOS

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INTRODUCCIÓN: *El tumor venéreo transmisible (TVT) es un tumor de células redondas de origen histiocítico que afecta a perros y otros cánidos como el lobo y coyote. Con mayor frecuencia en perros de 2 a 5 años sin raza o predisposición sexual. El tumor se transmite principalmente mediante la inoculación de células neoplásicas intactas en la membrana mucosa lesionada o en la piel durante el apareamiento, pero también se han reportado otras formas, morder, oler y lamerse. La enfermedad generalmente se considera benigna con predilección por los genitales externos (prepucio, pene y vagina), pero también hay informes de metástasis en otros órganos del cuerpo (piel, cavidad oral y nasal, conjuntiva, recto e hígado).*

OBJETIVOS

Identificar la signología poco común en nuestro medio, para poder compartir los alances y así seguir los casos posteriores mejorando la tánica diagnóstica en la clínica particular.

METODOLOGÍA

Presentación del caso: se presenta al Hospital de Mascotas para consulta y segunda opinión un macho canina de raza Schnauzer de 3 años de edad, llamado Tato. Reporte de vacunas y desparasitación al día. Al examen clínico presentó condición corporal 3/5, peso de 15 kg. Temperatura 38.6ºC. Reporta secreción nasal unilateral derecha hace 5 meses, durante los cuales estuvo en tratamiento con distintos antibióticos sin mayor éxito, además se observa inflamación en área afectada.

RESULTADOS

Se procedió a realizar una PAAF (punción aspiración con aguja fina) del área afectada y tomografía. El resultado final de la PAAF fue TVT canino y la tomografía evidencia masa en cavidad nasal unilateral derecha. Se procedió a tratamiento endovenoso con Vincristina, una dosis semanal con recuperación favorable en un mes de tratamiento (4 dosis).

CONCLUSIONES

La poca presencia de TVT canino en areas poco comunes como cavidad nasal pueden llevar al error diagnóstico, se debe obtener la mayor y mejor información de una buena anamnesis y realizar los exámenes auxiliares necesarios para determinar la patología final. Bibliografía 1. Ogilvie, G. K., & Moore, A. S. (2008). Manejo del Paciente Oncológico. Buenos Aires: Intermedica.

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INTRAOCULAR PRESSURE IN CAPTIVE MAGELLANIC PENGUINS (Spheniscus magellanicus) MEASURED BY REBOUND TONOMETRY

RCR Carazzatto1*, JOT Souza1,2,3, L Reisfeld4, CL Brunato1,5

INTRODUÇÃO: *Intraocular pressure (IOP) values are key diagnostic tools for ocular pathologies, and may differ between species of the same order, family or gender, these parameters may be above or below by normal considered. Intrinsic and extrinsic variables were described as influential for altering the ocular bulb and, consequently, the IOP of penguins, including: inflammation, cataracts, ocular trauma, corneal pathology, corneal thickness, circadian rhythm, body position and head, anatomical constriction, sedative and anesthetic drugs, and age. The establishment of basic health data is important to maintain the health of animals on professional care and to monitor the health of the wild population. However, there are no data described in the literature refering to Magellanic Penguins (Spheniscus-Magellanicus) species.*

PURPOSE:

The objective of this study is to describe the IOP (intraocular pressure) range found in a population of Magellanic Penguins (Spheniscus-magellanicus), to help to establish the normal range for the species.

METHODS:

A total of 9 animals (n=18 eyes) from the São Paulo aquarium (SP-Brazil) were used. They underwent an ophthalmic evaluation in order to exclude pathologies that interfered with the results. During the ophthalmic examination, the animals were held upright and 3 consecutive IOP measurements were performed using a rebound tonometer (Tono-Vet®- ICARE) without the use of topic anesthesia (figure 1 and figure 2). The measurements were performed in the afternoon at a room temperature of 25ºC and air humidity of 53%. The other data took into account body biometrics, weight, sex, and age. The analyzes were performed using the statistical program GraphPad Prism 5.

RESULTS:

It was possible to determine:

Average Values (9 animals)	
• Age	5.7±2.6 years
• Weight	3.79±0.53 kg
• Biometry - wing length	19.33±0.98 cm
• Biometry - chest circumference	44.33±1.96 cm
• Biometry - body length	59.08±1.96 cm
• IOP of the right eye (OD)	32.51±5.50 mmHg
• IOP of the left eye (OS)	32.51±7.12 mmHg

No statistical differences were found between the male and female range. No correlation was found between the IOP/Weight, nor the IOP/body length.

Figure 1: animal held upright and the IOP measurements

Figure 2: animal held upright and the IOP measurements

CONCLUSIONS:

The data obtained in this work can be used as a range reference and help future ophthalmologic evaluations in the species in question.

OCULAR ECOBIOMETRY IN CAPTIVE MAGELLANIC PENGUINS (Spheniscus magellanicus)

RCR Carazzatto1*, JOT Souza1,2,3, L Reisfeld4, CL Brunato1,5

INTRODUÇÃO: *Ocular ultrasonography or "ocular ultrasound" is indicated for opaque eyes, especially when it is impossible for complete ophthalmic examination, including corneal opacities, hyphema, cataract, hemorrhage and/or vitreous opacity, and in eyes with ocular trauma or suspicion of orbital disease; for diagnosis of primary or secondary diseases, anatomical location of intraocular or orbital wall anomalies, therefore, other indications involve biometric measurements, intraocular and orbital structures to establish values of normality and make the control and prevention of ocular diseases, possible. However, there are no data described in the literature refering to Magellanic Penguins (Spheniscus-Magellanicus) species.*

PURPOSE:

The present work aimed to describe the normal ultrasound ocular biometrics in captive Magellanic Penguin (Spheniscus magellanicus), through the following measures (distances) eye ecobiometrics of: M1 (corneal endothelium until anterior lens capsule); M2 (thickness of the lens); M3 (posterior lens capsule until vitreoretinal interface); M4 (corneal epithelium until vitreoretinal interface) (figure1).

METHODS:

A total of 9 animals (=18 eyes) from the São Paulo aquarium (SP-Brazil) were used. They underwent ophthalmic evaluation to exclude pathologies that interfered in the results. The animals were restrained in an prone position and ocular biometry was conducted Sonomed EscalonTM-VuPADTM,,mode A-B 10 mHz without topical anesthesia. The measurements were performed in the afternoon at a room temperature of 25ºC and air humidity of 53%.

The other data is based on weight, gender and age. The data was analyzed using the statistical program GraphPad Prism 5

RESULTS:

Average Values (9 animals/18 eyes)	
• **Age**	5.7±2.6 years
• **Weight**	3.79±0.53 kg
• **M1: right eye (OD)**	2.40±0.32 mm
• **M2: right eye (OD)**	4.17±0.45 mm
• **M3: right eye (OD)**	13.02±0.71 mm
• **M4: right eye (OD)**	19.60±0.59mm
• **M1: left eye (OS)**	2.15±0.25 mm
• **M2: left eye (OS)**	4.55±0.22mm
• **M3: left eye (OS)**	12.76±0.48 mm
• **M4: left eye (OS)**	19.46±0.42mm

No statistical differences were found between contralateral eyes neither between males and females. There was no correlation between body weight and ultrasound parameters.

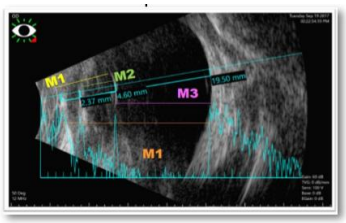

Figure 1: measurements of OD M1 (corneal endothelium until anterior lens capsule); M2 (thickness of the lens); M3 (posterior lens capsule until vitreoretinal interface); M4 (corneal epithelium until vitreoretinal interface).

CONCLUSIONS:

In our knowledge, this data are the first reported in that species and therefore serves as a reference for the ocular ecobiometric evaluation of the species studied.