2014 CVMA ANNUAL CONVENTION PROCEEDINGS

Welcome to the 2014 CVMA Convention in St. John’s, Newfoundland. The following papers are compiled to accompany the presentations scheduled in the continuing education sessions at the convention. The proceedings are organized by day and by stream as follows:

THURSDAY, JULY 10, 2014
Companion Animal - Emergency and Critical Care Medicine
Companion Animal - Canine and Feline Dentistry
Companion Animal - Rehabilitation and Pain Management for Osteoarthritis
Companion Animal - Principles of Fracture Repair
Equine - Field Ophthalmology for the Equine Practice
Bovine - Bovine Neurology
Bovine - Lameness and Infectious Disease
Animal Welfare - Decision Making in a Practice Setting
Animal Welfare - Dairy and Swine Codes of Practice
INTRODUCTION
There are many aspects of critical care that are unique to the cat. The physiologic response to shock, monitoring techniques used, and methods of resuscitation are different in cats in comparison to dogs. Although the underlying etiologies and pathophysiology of shock are similar in the two species, care must be taken to treat cats appropriately in order to avoid life-threatening consequences.

DEFINITION
The medical dictionary defines shock as “a medical emergency in which the organs and tissues of the body are not receiving adequate blood flow, thus depriving them of oxygen and allowing the buildup of waste products.” Essentially, shock occurs when the delivery of oxygen to the tissues does not meet their oxygen requirement. The body compensates by diverting blood flow to the heart and brain. Other organs, including the gastrointestinal (GI) tract, pancreas, kidneys, and lungs, sustain ischemic injury as a result. Ultimately, there is death of renal tubular cells resulting in kidney insufficiency, GI ischemia leading to bacterial translocation, and acute lung injury and impaired pulmonary gas exchange. In cats, the “shock organ” or organ that is most affected during shock is the lung. Thus, cats will often present with signs localizing to the respiratory system including tachypnea, respiratory distress, and pale or cyanotic mucous membranes. Cytokines (i.e., tumour necrosis factor-α, interleukin-1) are also released and cause a systemic inflammatory response. Anaerobic metabolism occurs leading to excess lactate production and subsequent metabolic acidosis. If left untreated, eventually shock will lead to cellular dysfunction/death, organ failure, and ultimately death.

CLASSIFICATION
There are several classification schemes used to categorize shock including those based on underlying etiology (e.g., cardiogenic, hypovolemic, hemorrhagic, septic, traumatic, anaphylactic), anatomic location affected (e.g., heart, blood [loss], vascular), and identification of the hemodynamic defect (e.g., hypovolemic, cardiogenic, distributive, obstructive). Classification by the hemodynamic defect (Table 1) is helpful to understand the pathophysiologic mechanisms of shock and to consider therapies that will reverse the dysfunctions. However, shock is complex and it is important to realize that most clinical forms of shock have many underlying mechanisms affecting the animal simultaneously. Therefore, there is no universal treatment for each category of shock that will suit every patient.

Table 1. Common causes of shock in cats

<table>
<thead>
<tr>
<th>Hypovolemic</th>
<th>Cardiogenic</th>
<th>Distributive</th>
<th>Obstructive</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Hemorrhage (trauma, coagulopathy)</td>
<td>* Cardiomyopathy (dilated, hypertrophic, unclassified)</td>
<td>* Sepsis</td>
<td>* Pericardial tamponade</td>
</tr>
<tr>
<td>* Profuse vomiting and/or diarrhea</td>
<td>* Severe arrhythmias</td>
<td>* Pancreatitis</td>
<td>* Restrictive pericarditis</td>
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<tr>
<td>* Severe polyuria</td>
<td>* Severe tissue trauma</td>
<td>* Severe tissue trauma</td>
<td>* Pulmonary thromboembolism</td>
</tr>
<tr>
<td>* Marked internal fluid loss (pleural, peritoneal, interstitial)</td>
<td>* Anaphylaxis</td>
<td>* Anaphylaxis</td>
<td></td>
</tr>
<tr>
<td>* Severe dehydration</td>
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</table>

Hypovolemic Shock
Hypovolemic shock is a common cause of shock in cats. It occurs due to an actual (absolute) or perceived (relative) loss of intravascular volume. Hemorrhage is considered an absolute cause of hypovolemic...
shock because it involves an actual loss of intravascular (blood) volume. Examples of clinical presentations for cats with absolute hypovolemic shock due to hemorrhage include trauma, coagulopathies, and gastrointestinal hemorrhage. Absolute hypovolemic shock can also be caused by non-hemorrhagic events that involve a large loss in plasma volume. Examples include severe dehydration, polyuria, vomiting/diarrhea, and third-space losses (e.g., abdominal or pleural effusion). Anaphylaxis also has characteristics of absolute hypovolemic shock as endothelial damage can enable the leakage of large volumes of plasma from the intravascular to the interstitial spaces, resulting in peripheral edema. Relative hypovolemic shock occurs when there is no loss of intravascular (blood or plasma) volume; however, because of venous or arterial dilation, there is a relative inadequacy of intravascular volume. This term is not used very commonly anymore as most people categorize this type of shock as distributive.

**Cardiogenic Shock**
Cardiogenic shock occurs due to a decrease in cardiac output and is most commonly seen in cats with cardiomyopathy, arrhythmias, or other conditions that adversely affect myocardial contractility. Many of these cats are concurrently in congestive heart failure. Because of this, the presenting clinical signs are not just characteristic of shock, but can also include additional signs such as pulmonary crackles, decreased lung/cardiac sounds due to pleural effusion, jugular distension, or a heart murmur.

**Distributive Shock**
Distributive shock occurs when there is dysfunction of the microcirculation due to inappropriate arterial or venous dilation resulting in abnormal blood flow. Most often vasodilation is a consequence of inflammatory mediators that are released and lead to the production of nitric oxide, a potent vasodilator. This can occur in cats with systemic inflammatory response syndrome (SIRS), sepsis, or severe pancreatitis. Conversely, anaphylaxis is another cause of distributive shock resulting in the IgE-mediated release of vasoactive substances (e.g., histamine, amine, tumour necrosis factor-α, prostaglandin, leukotriene) from mast cells, which cause massive vasodilation.

**Obstructive Shock**
Obstructive shock happens when there is an obstruction of blood flow from the heart or returning to the heart. Clinical examples of obstructive shock include pericardial tamponade due to pericardial effusion or pulmonary or aortic thromboembolism. The clinical signs associated with obstructive shock vary depending on the location of obstruction. For instance, pericardial tamponade can cause pulses paradoxus (i.e., a weaker pulse on inspiration and a stronger pulse on expiration), whereas aortic thromboembolism can result in an absence of femoral pulses.

**Clinical Stages and Signs**
Shock has been traditionally separated into three clinical stages that illustrate the progression: compensatory, early decompensatory, and decompensatory (terminal). Cats are different from dogs in that they rarely present with signs of compensatory shock, but are much more likely to show signs of early decompensatory or decompensatory shock. During the compensatory stage of shock, the body is able to maintain adequate cardiac output despite a mild to moderate loss of intravascular volume. However, the compensatory stage cannot last indefinitely without treatment and ultimately systemic vascular resistance will decrease, cardiac dysfunction will occur, and decompensation begins. Eventually, complete circulatory collapse occurs and death can occur.

**Compensatory Stage**
The signs of compensatory shock are often overlooked or mistaken as “normal.” These signs include a normal to mildly increased heart and respiratory rate, injected (bright pink to reddish) mucous membranes, shortened capillary refill time (< 1 sec), normal mentation, normal blood pressure, and normal to increased (bounding) pulses. Injected mucous membranes and increased pulse pressure are rarely detected in cats as opposed to dogs.
Early Decompensatory Stage
Cats and dogs most commonly present during the early decompensated stages of shock. Signs noted during this stage include tachycardia, normal to decreased pulse pressure, hypotension, pale mucous membranes, prolonged capillary refill time, decreased mentation, and decreased body temperature. Tachycardia does not occur in cats as often as dogs during this stage and should not be an expected finding. Cats that have all of the other signs should be considered in shock and treated accordingly.

Decompensatory (Terminal) Stage
Clinical signs of decompensatory shock include a low heart rate, severe hypotension, pale or grey mucous membranes, absent capillary refill, weak or absent pulses, decreased heart sounds, low body temperature, stuporous or comatose mentation, and decreased or absent urine production. If aggressive resuscitation is not provided, cardiopulmonary arrest and death will occur.

Cats typically present with a trilogy of signs during shock regardless of the stage that include hypothermia, hypotension, and bradycardia. Note: A heart rate < 160 bpm in a visibly sick cat is a concerning finding and the possibility of shock should be considered, especially if other signs of shock are seen.

Monitoring and Quick Diagnostic Tests
Thorough and frequent physical examinations focusing on perfusion parameters are important when monitoring patients during resuscitation from shock. Monitoring cats can be particularly difficult given that changes in their perfusion parameters (i.e., pulse quality, capillary refill time, mucous membrane color, extremity temperature) are often subtle compared to those in dogs. For example, while femoral and dorsal pedal pulses are easily detected in most dogs, dorsal pedal pulses can be difficult to palpate even in normal healthy cats. Likewise, detecting subtle changes in femoral pulse quality is a skill that only those with excellent tactile sensation possess. Nevertheless, it is important that attempts be made to palpate both dorsal pedal and femoral pulses in cats during shock resuscitation. A change in pulse strength or an absence or gain of a pulse can help gauge response to resuscitation efforts. Similarly, vital signs and other perfusion parameters should be monitored frequently to ensure an appropriate response to resuscitation (Table 2).

| Table 2. Perfusion parameters and the desired change during resuscitation |
|---------------------------------|------------------|------------------|
| Parameter                      | During shock     | Desired change   |
| Presence of pulse/quality      | Absent, poor/weak| Present, normal  |
| Heart rate                     | < 160 bpm        | > 160 bpm        |
| Respiratory rate               | Tachypneic       | Normal           |
| Extremity temperature          | Cool             | Normal           |
| Blood pressure                 | Systolic < 90 mm Hg | Systolic > 110 mm Hg |
| Mucous membrane colour         | Pale/grey/white  | Pale pink or pink |
| Capillary refill time          | Prolonged or absent | 1-2 seconds       |
| Rectal temperature             | < 37°C           | > 37°C           |
| Urine production               | Minimal or absent | Normal (1-2 mL/kg/hour) |
| Mentation                      | Depressed, stupor, coma | Alert and responsive |
| Lactate                        | > 2 mmol/L       | < 2 mmol/L       |

Blood Pressure
If blood pressure monitoring is available, it should be used to confirm the physical examination findings. It is important when measuring blood pressure using either Doppler or oscillometric devices to ensure that the cuff size is appropriate (width 30–40% of limb circumference), as small cuffs might overestimate blood pressure and large cuffs might underestimate blood pressure. Normal pressure is 120/80 mm Hg (mean arterial pressure [MAP] 90 mm Hg) and hypotension is typically defined as a systolic blood pressure [SBP] ≤ 90 mm Hg or a MAP ≤ 70 mm Hg.
Doppler ultrasonographic blood pressure devices measure the systolic blood pressure when the inflated cuff is released and blood flow and arterial wall motion occur creating the characteristic “whooshing” sound. Research studies have shown that in anaesthetized cats Doppler measurements more closely reflect the mean arterial blood pressure; however, this is debatable in awake or ill cats. A study investigating Doppler blood pressure in critically ill cats revealed that cats with an increase in Doppler blood pressure ≥ 20 mm Hg were more likely to survive.

Oscillometric blood pressure devices detect pulsatile arterial wall vibrations by an automatically inflating/deflating cuff. The cuff is inflated until no vascular vibrations (oscillations) are detected and then deflated. Throughout the deflation cycle, the average oscillations and corresponding cuff pressures are computer analyzed to determine the systolic, diastolic, and mean arterial pressures. Oscillometric devices are most appropriate for monitoring trends (they can be set to measure every 1–30 minutes). Oscillometric devices generally underestimate systolic blood pressure in cats, but are fairly accurate for mean and diastolic blood pressures. Unfortunately, their accuracy in measuring blood pressure in small or hypotensive animals is decreased. Therefore, they are most accurate in large cats (> 5 kg), but may not be as accurate with tachycardia, rapidly changing heart rates, or shock/vasoconstricted states.

Lactate
When perfusion and oxygen delivery are inadequate, anaerobic metabolism increases resulting in increased lactate production and a metabolic acidosis. Normal blood lactate levels are < 2 mmol/L. Mild hyperlactatemia is 2–4 mmol/L, moderate hyperlactatemia is 5–8 mmol/L, and severe hyperlactatemia is > 8 mmol/L. Lactate measurements can also be trended over time to assess the adequacy of fluid resuscitation or improvement in oxygen delivery during treatment for shock. Blood lactate can be easily measured in practice by the use of an iSTAT® or other handheld devices. When handheld devices were compared to standard laboratory analyzers using blood samples from cats, the iSTAT® and LactatePro® (www.vetlab.com/Lactate%20Pro.htm) were found to be the most accurate.

ECG
ECG monitors are becoming more frequently used in small animal practice. Many blood pressure and pulse oximetry machines have an ECG as well. An ECG is helpful for assessing cats with bradycardia, tachycardia, pulse deficits, or a history of heart disease, and for enabling moment-to-moment monitoring of heart rate during resuscitation from shock. If hyperkalemia is measured or suspected (e.g., urethral obstruction, uroabdomen, kidney failure), an ECG should be performed to assess for associated conduction disturbances such as small to absent P-waves, tall T-waves, or a widened QRS complex. Severe bradycardia (HR < 140 bpm) in critically ill cats is an emergency that requires immediate intervention.

Pulse Oximetry
Pulse oximetry is a noninvasive method for assessing oxygenation. It measures the peripheral oxygen saturation (SpO₂), which is an indirect measure of SaO₂ and thus PaO₂ using the oxygen-hemoglobin dissociation curve. A SpO₂ of 90% corresponds to a PaO₂ of 60 mm Hg (severe hypoxemia) and a SpO₂ of 95% corresponds to a PaO₂ of 80 mm Hg (normal).

Electrolyte and Glucose Measurements
Electrolytes can be measured on a chemistry analyzer or blood gas machine to rule out severe abnormalities (potassium < 3.0 or > 6.0 mEq/L, sodium < 140 or > 170 mEq/L, ionized calcium < 1.0 mmol/L). A blood glucose measurement should always be measured for cats in shock using a chemistry machine or glucometer. Hyperglycemia is common in cats due to the stress of hospitalization or illness; however, hypoglycemia can occur in cats with shock. A blood glucose measurement < 4.5 mmol/L in a cat in shock necessitates treatment with dextrose supplementation.

Focused Assessment Using Sonography for Trauma (FAST)
Abdominal FAST is a simple and rapid ultrasound examination that can be performed in cats in an emergency setting to detect intraabdominal free fluid suggestive of hemorrhage or organ rupture (e.g., uroabdomen). Veterinarians with limited previous ultrasound experience can perform a FAST examination in less than five minutes. It can be used with a scoring system (score 0–4) to evaluate for the
presence of fluid (anechoic) in four areas. With the cat in lateral recumbency (preferably right lateral), use the ultrasound probe to assess for fluid: 1) just caudal to the xiphoid process, 2) on the midline over the urinary bladder, and at the 3) left and 4) right flank regions. During the FAST examination, the urinary bladder and gallbladder can also be visualized. Note that even though an organ may be seen on radiographs, or ultrasound, and appear intact, organ rupture (e.g., urinary bladder, urethra, gallbladder) can still be present. FAST can be performed on initial presentation and serially thereafter to monitor for the presence of abdominal fluid.

If abdominal fluid is found, an abdominocentesis can be performed to obtain a sample of fluid for analysis. If the sample appears bloody, place it in a red-top blood tube to ensure that it does not clot. If the fluid sample clots, it is possible that a blood vessel or organ (e.g., spleen) was inadvertently aspirated. Otherwise, perform a PCV and TS on the fluid; a PCV > 10% is suggestive of hemorrhage. If uroabdomen is suspected, measure the potassium or creatinine concentration of the fluid and compare it to the periphery (serum). A fluid:serum ratio of potassium > 1.4:1 or creatinine > 2:1 is consistent with a uroabdomen.

Thoracic FAST can also be performed in cats presenting in shock and with increased respiratory effort or respiratory distress, to quickly determine if fluid (anechoic) is present in the thoracic cavity. Differentials for the fluid will depend on the history and presenting complaint, but could range from hemorrhage (i.e., traumatic shock) to pyothorax (i.e., septic shock) or chylothorax (i.e., cardiogenic shock). PCV and TS (if bloody) or fluid cytology will be helpful if a sample of fluid can be obtained for analysis.

**TREATMENT**

Early recognition and resuscitation are very important for the successful treatment of shock in cats. There are many facets of the treatment of shock in cats that are unlike that for dogs. Cats can be difficult to resuscitate and often require more conservative management to avoid complications such as fluid overload or pulmonary edema.

**Fluid Resuscitation**

Vascular access is a must when providing appropriate fluid resuscitation to cats in shock. Subcutaneous fluids are not appropriate in cats with evidence of shock and poor perfusion. Vascular access can be obtained via the cephalic, or medial saphenous, veins. The volume of fluid required for treating shock is based on the cat’s weight and estimated blood volume. If crystalloid fluids are used, one full blood volume could need to be administered over one hour. The blood volume of cats is estimated to be 45–60 mL/kg and is less than that of dogs. The response to fluid resuscitation should determine the volume of fluid that is administered. The recommended regimen for fluid resuscitation during shock is to divide the total volume to be given over one hour into smaller volumes and use frequent monitoring to assess the cat’s status. Divide the total fluid volume into four aliquots (10–15 mL/kg) and recheck the cat’s perfusion parameters every 15 minutes to determine the response. Not all cats in shock will require replacement of a full blood volume to treat their shock. If the clinical signs of shock resolve after the first 15 minutes, there is no need to proceed with another bolus of fluid. Consider giving a fluid bolus by drawing the amount of fluid up into a syringe and manually “pushing” it into the cat over 10–15 minutes. Conversely, a fluid pump can be set to deliver the volume over 15 minutes. Balanced electrolyte solutions (i.e., 0.9% NaCl, LRS, Plasma-Lyte 148, Plasma-Lyte A) are preferred over normal saline (0.9% NaCl); however, any isotonic fluid that is available can be used. Because cats in shock are typically hypothermic, it is beneficial to warm fluids prior to administration if possible.

Hypertonic saline (3–7%) is another fluid that can be considered during fluid resuscitation of shock. It is not commonly used in cats because they are small enough that isotonic fluids can be given rapidly and in large enough volumes to achieve the desired effect of intravascular volume expansion. However, hypertonic saline has other benefits including the reduction of intracranial pressure in cats with head trauma. In that case, a dose of 2–4 mL/kg is recommended over 15–20 minutes. Care should be taken not to administer hypertonic saline too quickly as it can cause bronchoconstriction. Hydroxyethyl starch (HES) solutions (e.g., pentastarch, tetrastarch) are colloid solutions containing hydrolyzed amylopectin dissolved in a crystalloid solution (typically NaCl 0.9%). They are administered for volume resuscitation
in patients with systemic inflammatory response syndrome (SIRS), sepsis, hypoalbuminemia, shock, trauma, or peripheral edema. HES solutions can be administered as part of a fluid resuscitation plan in patients that are hypoalbuminemic or not responsive to crystalloid therapy alone. Potential complications of HES solution administration include volume overload and coagulopathies. Typically, boluses are given in 2.5–5 mL/kg volumes over 10–15 minutes, not exceeding a total daily dose of 20 mL/kg/day. Cats are especially sensitive to boluses of HES solutions and will vomit if they are given too quickly.

**Blood Transfusions**
Cats in shock due to traumatic episodes might be anemic and hypoproteinemic. Additionally, if frequent blood sampling is performed for diagnostic tests, cats can become anemic. If the cat is indeed in hypovolemic shock due to hemorrhage, a whole blood transfusion is likely necessary. Remember that cats must be blood typed (or crossmatched) prior to receiving a blood transfusion of any kind, as type A-B incompatibilities in cats can result in lethal transfusion reaction. If the cat must be transferred to another hospital for the transfusion, it must be stabilized to the best of your ability (i.e., body temperature, heart rate, and blood pressure normalized) using the other recommended treatments.

**Analgesia**
Providing adequate pain relief to cats that are in shock is also extremely important. Cats that are in shock will not be able to elicit the same signs of pain that are recognized in otherwise normal cats (e.g., tachycardia, restlessness, irritability, withdrawn demeanor). Therefore, if the cat has experienced a traumatic event (e.g., dog attack, HBC) it must be assumed that the cat is painful and its pain should be treated accordingly. Due to the critical nature of cats that are in shock, as well as cats’ sensitivity to opioids, it is best to titrate pain-relieving medications to effect. Ideally, medications that are short acting and reversible should be used.

**Table 3. Suggested analgesics**

<table>
<thead>
<tr>
<th>Pain-relieving medication</th>
<th>Dose</th>
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<tbody>
<tr>
<td>Fentanyl</td>
<td>1–2 mcg/kg IV bolus followed by 2–5 mcg/kg/h IV continuous rate infusion</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>0.02–0.05 mg/kg IV or IM every 4 hours</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>0.005–0.02 mg/kg IV or IM every 6–8 hours</td>
</tr>
<tr>
<td>Ketamine</td>
<td>0.5–1.0 mg/kg IV bolus followed by 0.1–1.0 mg/kg/h IV continuous rate infusion</td>
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**Heat Support**
It is very common for cats in shock to be hypothermic. The low body temperature is often exacerbated by the low heart rate and subsequent low cardiac output. Unfortunately, the hypothermia will also heighten the bradycardia by depressing the sinus node. Therefore, it is very important that attempts be made to warm cats during shock resuscitation. This can be effectively achieved by applying external warming sources such as a circulating water blanket, BAIR Hugger®, or heated towel to the cat. Additionally, administering warmed (rather than room temperature) intravenous fluids can be helpful.

**Oxygen Therapy**
Oxygen supplementation should also be administered to cats that are in shock and whose SpO₂ is < 95%. This will help to improve oxygen delivery to tissues. It should be provided in such a way that will not cause stress to the cat. The easiest methods during the resuscitation period are flow-by (blow-by) or mask oxygen. Mask oxygen will enable a higher concentration of oxygen to be provided to the cat; however, not all cats will tolerate the mask. In those cases, directing the oxygen flow toward or near the cat’s nose and mouth is sufficient. If the cat is being housed in a cage during its resuscitation, an oxygen cage or oxygen hood may be used if available.
Electrolyte and Blood-Glucose Disturbances
Hypoglycemia should be immediately treated with an IV bolus of dextrose. Dilute 0.5–1 mL/kg of
dextrose 50% to a concentration of 12.5–25% and administer it over 2–3 minutes. Recheck the blood
glucose 10–15 minutes after the bolus to ensure that it is normal. If it is low again, administer another IV
bolus and add 2.5–5% dextrose to the IV fluids. Calcium gluconate 10%, 0.5–1 mL/kg IV, should be
administered over 10–15 minutes in cats with ionized hypocalcemia or hyperkalemia with associated
ECG abnormalities. Hyperkalemia should also be treated with IV dextrose (as above), as well as sodium
bicarbonate (1 mL/kg IV over 10–15 minutes) or regular insulin (0.5 U/kg) and IV dextrose 50% (2
mL/kg) concurrently.
**INTRODUCTION**

Large studies show that traumatic injuries account for approximately 10% of all veterinary teaching hospital admissions. Injuries vary depending on the nature of the trauma. Motor vehicle accidents (e.g., hit by car, train, tractor, boat, bike) or falls from height result in blunt trauma, with over 90% of blunt trauma patients presenting to veterinary hospitals after being hit by a motor vehicle. Conversely, animal altercations (e.g., bite wounds, BDLD), gunshot wounds, or accidental impalements are examples of causes of penetrating trauma.

Although trauma accounts for a large number of dogs treated at veterinary hospitals, a significant number of myths or misconceptions still exist surrounding the appropriate management and care of dogs with traumatic injuries. Some important facts regarding trauma in dogs will be outlined below and common myths will be dispelled with current recommendations for treatment of these patients.

**TRAUMATIC INJURIES**

A recent large retrospective study in dogs with blunt trauma suggests that dogs tend to be 2–3 years old and medium-large breed. The most common injuries following blunt trauma are thoracic (70%), abdominal (50%), extremity (40%), and head (30%) injuries. The most common thoracic injuries include pulmonary contusions and pneumothorax, each occurring in approximately 50% of dogs. Other thoracic injuries include hemothorax, rib fractures, pneumomediastinum, diaphragmatic hernia, pulmonary bullae, and flail chest. Extremity injuries include mostly superficial abrasions and lacerations, as well as fractures and luxations. The most common abdominal injury is hemoabdomen, followed by abdominal hernias, and rupture of the urinary tract.

**TRIAGE**

The term “triage” is derived from the French verb *trier*, which means “to pick” or “to sort.” In veterinary medicine, triage is defined as a systematic approach used to treat the most severely injured animals first, and to define the most life-threatening injury, with the hope of saving lives and decreasing morbidity. With regards to trauma patients, many dogs will present with multiple injuries that need to be prioritized. In general, injuries in order from most to least critical include arterial bleeding (rarely will dogs live long enough following the traumatic event for you to see this in practice), followed by injuries to the respiratory, cardiovascular, neurologic, abdominal, musculoskeletal, dermatologic, and ophthalmologic systems.

**PREPAREDNESS**

There is good evidence to suggest that injured patients benefit from preparedness of the hospital staff and a team approach to emergency cases. Ideally, one veterinarian and at least two technical support staff should be available in the event of a trauma emergency. Rapid assessment and resuscitation carried out during the first minutes following a traumatic event constitutes the “golden hour” of emergency medicine and is more easily achieved with appropriate preparedness and preplanning. A “ready area” that is set up for handling emergencies and providing immediate resuscitation should be designated in the hospital. This area should have good lighting and be organized in a way that most items needed are open and readily accessible. An oxygen source, crash cart, and monitoring equipment should be readily available. Supplies needed for catheter placement, fluid resuscitation, warming, and wound management should also be within reach. A backboard made of Plexiglas or plastic, or a commercially available stretcher, cart, or gurney can be helpful to transport nonambulatory patients.

A well-trained technician or veterinarian can make the following assessments immediately upon the dog’s arrival to the hospital:

- Level of consciousness
- Airway patency (listen for increased, decreased, or absence of airway sounds)
- Breathing rate and effort (watch chest movements)
- Strength and rate of pulses (palpation) or heart sounds (auscultation)
- Color of mucous membranes and capillary refill time
- Examination for external abnormalities such as hemorrhage, sucking wounds in the neck or chest, and abdominal distension

If the dog is deemed unstable, someone should move it to the “ready area” immediately. Dogs should be carried or moved on a stretcher or a trolley; avoid letting them walk in case they collapse. Do not place a leash around the neck of dogs with head, ocular, neck, or respiratory injuries or problems. Someone else can then stay with the owner to obtain an adequate history and ensure that the owner is calm. Conversely, the owner can accompany the pet to the “ready area” so that additional information can be obtained while early interventions are being accomplished.

Almost immediately, the following questions should be asked:
- Can we begin oxygen therapy and place an intravenous catheter?
- Can CPR be started if your pet arrests?

**Note:** Exam gloves should always be worn for the safety of the patient and technician. This is to avoid contamination of open wounds with bacteria from our hands. Additionally, trauma patients are often covered in blood, and it may be blood from the injured animal or that from the injured person who handled the dog.

**Obtain a Brief History**
When time is of the essence, the “AMPLE” is a handy acronym to help remember to ask some important questions:
- **A** = Allergy (to medications? blood products previously given? if so, reactions noted?)
- **M** = Medications (when was the last dose? when is the next dose due? nutraceuticals?)
- **P** = Past medical (are there any past medical or surgical conditions?)
- **L** = Last… (meal? urine? stool? observed healthy?)
- **E** = Events (that led up to the pet presenting to your clinic now including any visits to other veterinary clinics and treatments that were received there.)

**Trauma Patient Monitoring**
Several monitoring tools readily available in practice should be used to obtain objective assessments of the stability of every dog arriving to the hospital after trauma. In addition to vital signs (i.e., temperature, pulse, respiratory rate), a **blood pressure** (oscillometric or Doppler) should be measured to determine if the dog is in shock. A **pulse oximetry** (SpO2) measurement should also be obtained on the lip, tongue, or ear (or rectally if a reflectance probe is available), in order to assess the dog’s oxygenation. An **ECG** is helpful for assessing dogs with tachycardia, pulse deficits, or a history of heart disease and also for enabling moment-to-moment monitoring of heart rate during emergent fluid resuscitation. If available, ECG leads should be applied and the heart rate, rhythm, and any abnormal heartbeats recorded. Ventricular arrhythmias are the most common arrhythmias seen in injured dogs and can occur up to 48 hours after the traumatic event. They can be due to myocardial injury (i.e., traumatic myocarditis), metabolic acidosis, electrolyte disturbances (i.e., hypokalemia, hypomagnesemia), pain, hypovolemia, splenic disease (e.g., torsion, fracture), or heart disease.

While CBC and biochemistry profiles are likely unnecessary in most young and otherwise healthy dogs sustaining trauma, a minimum database should be performed using blood obtained from the catheter hub after the stylet is removed. An expanded laboratory database is not always necessary and can be performed later based on history, physical examination findings, and results of the minimum database. Perform a **PCV** and **total solids (TS)** to identify blood loss. **BUN** (Azostix) can be measured to establish if azotemia is present (especially if NSAIDs are going to be administered). **Glucose** (glucometer)
should also be measured, especially in small or toy breed dogs. Lactate can be measured (if possible) and used to gauge the response to fluid therapy and shock resuscitation.

The key to successful management of trauma patients is constant monitoring and reassessment of patient status. Dogs must be monitored frequently while recovering from trauma, even though they might appear stable, since they can decompensate at any time during the first 24 hours of care. Even a small change in patient status might indicate development of an arrhythmia, intraabdominal bleeding, or pain that requires immediate attention.

**Analgesia**

Traumatic injuries are painful and analgesia must be given promptly. Unless the dog is unconscious (unresponsive to verbal or painful stimuli), analgesia is recommended. Although opioids have mild respiratory suppressive tendencies, these effects are not clinically significant. Veterinary studies have also shown that the respiratory rate and pattern of dogs with traumatic injuries actually improves after analgesia administration. Therefore, opioids are the drug of choice for traumatic injuries and can be titrated to effect (given in small bolus amounts) to avoid side effects such as vomiting, nausea, panting, or profound sedation (Table 1).

**Table 1. Suggested analgesic medications**

<table>
<thead>
<tr>
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<td>Buprenorphine</td>
<td>0.01–0.02 mg/kg IV or IM every 6–8 hours</td>
</tr>
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</table>

**Diagnostic Imaging**

Radiographs should only be performed after a complete physical examination to identify the area in question and not until the dog is adequately stabilized! Remember that thoracic injuries occur in the majority of dogs experiencing trauma. Therefore, thoracic radiographs should be obtained on all dogs with traumatic injuries, but not until the dog is stable. Similarly, radiographs of fractures are not essential during the first 6–12 hours of care and should be delayed until the dog is stable.

Focused assessment using sonography for trauma (FAST) is a simple and rapid ultrasound exam that can be performed in dogs in an emergency setting to detect intraabdominal fluid suggestive of hemorrhage or urinary tract rupture. It can be used with a scoring system (score 0–4) to evaluate for the presence of fluid (anechoic) in four areas. With the patient in lateral recumbency (preferably right lateral), use the ultrasound probe to assess for fluid 1) just caudal to the xiphoid process, 2) on the midline over the urinary bladder, and at the 3) left and 4) right flank regions. FAST can be performed on initial presentation and serially thereafter to monitor for the presence of abdominal fluid, which can then be sampled using abdominocentesis to confirm whether it is blood or urine.

**Common Myths Regarding Trauma-Patient Management**

**Myth:** All trauma patients require their entire “shock volume” (blood volume) delivered during the first hour after arrival to the hospital.

**Fact:** Not all patients in shock will require replacement of a full blood volume to treat their shock. Clinical signs of poor perfusion (shock) include tachycardia, poor pulse quality, hypotension, prolonged capillary refill time, pale mucous membrane colour, hypothermia, and cool extremities. The volume of fluid required for treating shock is based on the patient’s weight and estimated blood volume. In the dog, this is 80–90 mL/kg. The dog’s response to fluid resuscitation determines the approximate volume that is administered. The recommended regimen for fluid resuscitation during shock is to divide the total volume to be given over one hour into smaller volumes and use frequent monitoring to assess the response. By dividing the total fluid volume into 1/4 volumes and monitoring every 15 minutes, it is easier to determine when emergent fluid resuscitation is complete. If the clinical signs of shock resolve after the first 15 minutes, there is no need to proceed with another bolus of fluid.
**Myth:** Fluids should be withheld from trauma patients with head trauma, pulmonary contusions, or hemorrhage.

**Fact:** While it is important to be cautious about overhydration and excessive increases in hydrostatic pressure in patients with pulmonary contusions, cerebral edema (e.g., traumatic brain injury), or hemorrhage, fluids are still needed to ensure adequate cerebral perfusion, pulmonary perfusion, and replace blood volume, respectively. Therefore, it is best to perform fluid resuscitation as outlined above, in terms of administered fluid in boluses over 15 minute periods and reassessing after each bolus to determine if further emergent fluid resuscitation is needed. Small volume resuscitation with hypertonic saline (NaCl 3–7%) might also be considered (4–6 mL/kg IV bolus over 15–20 minutes), in addition to the isotonic crystalloids fluid resuscitation, especially in dogs with traumatic brain injury (to decrease intracranial pressure).

**Myth:** Diuretics are often helpful for patients with pulmonary contusions.

**Fact:** Because contusions are the result of vessel damage and not increased hydrostatic pressure, diuretics are not effective and might even be harmful. Pulmonary contusions occur in approximately 40–50% of all dogs sustaining blunt trauma and are characterized by damage to the pulmonary vasculature and subsequent leakage of blood and plasma into the interstitium and alveoli followed by massive infiltration with inflammatory cells 24 hours later. Dogs with pulmonary contusions require supportive care, which includes oxygen therapy, pain management, and management of shock and concurrent injuries. Clinical signs usually resolve in approximately one week; however, the need for oxygen supplementation is usually only required for 2–3 days.

**Myth:** Trauma patients with hemoabdomen will usually require emergency surgery.

**Fact:** The vast majority of dogs with hemoabdomen after blunt trauma will not require surgery and can be managed supportively with cage rest and fluid resuscitation. With the use of FAST exams, up to 40% of dogs sustaining severe blunt trauma will be diagnosed with a hemoabdomen. The vast majority of those dogs will be successfully managed with cage rest and fluid resuscitation, while some of those dogs will require blood-product transfusion(s). Very few dogs will require an abdominal exploratory, unless they have a liver, or splenic, fracture with severe hemorrhage and cannot be stabilized with supportive care.

**Myth:** Dogs with penetrating trauma to the abdomen should be managed supportively without surgery.

**Fact:** Dogs experiencing penetrating trauma to the abdomen should undergo an exploratory laparotomy once they are stable, to investigate the extent of their internal injuries and perform surgical repairs. Injuries such as perforation of the liver, spleen, gastrointestinal tract (GIT), or bladder, as well as abdominal wall disruption or herniation, are common after penetrating trauma such as from gunshot wounds or animal altercations. These require an abdominal exploratory for proper diagnosis and repair.

**Myth:** Nonsteroidal antiinflammatory drugs (NSAIDs) are appropriate analgesics for all trauma patients.

**Fact:** While NSAIDs are excellent analgesics for many trauma patients, they should not be given during the first 12–24 hours of trauma or if any contraindications are noted. Absolute contraindications for NSAID administration include hypotension, shock, hypovolemia, dehydration, hemorrhage, azotemia, and liver failure. It is recommended to wait 12–24 hours after the traumatic event to confirm that the dog is stable before giving an NSAID. In the meantime, opioids can be given and if adjunctive analgesia is required, ketamine (IV), lidocaine (IV), gabapentin (PO), or local anesthesia can be considered.

**Myth:** Steroids are indicated for management of shock in trauma patients.

**Fact:** Steroids (glucocorticoids) are not recommended for trauma patients and might be detrimental in patients with head trauma. Although steroids have been previously recommended for resolution of shock, they are no longer recommended. Large clinical trials in people with head trauma have demonstrated a detrimental effect when people are treated with glucocorticoids. And because NSAIDs
are such effective analgesics, and administering NSAIDs and steroids concurrently is contraindicated, steroids are not recommended.

**Myth:** Trauma patients have a guarded prognosis for recovery from their injuries.
**Fact:** In general, the prognosis for survival after trauma in dogs is very good with over 85% of dogs surviving. The severity of the injuries and organ systems affected often determine the likelihood of survival following trauma. Dogs with polytrauma, traumatic brain injury, cardiac arrhythmias, body wall hernias, severe soft tissue injuries, vertebral fractures, or recumbency at admission have a higher mortality.
Taking the “Stress” Out of Respiratory Distress
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INTRODUCTION
Respiratory distress represents one of the most stressful and time-dependent emergencies that a veterinarian will face in practice. Regardless of the underlying condition, a respiratory emergency requires rapid identification of the primary cause, immediate alleviation of the sensation of difficulty breathing, and provision of information to the owners of the affected pet. Successful management of patients in respiratory distress is aided by “pattern recognition” of common conditions causing respiratory difficulty. Prompt recognition of the underlying condition will enable the provision of appropriate therapeutics to stabilize the patient and will ultimately improve outcome. A comforting fact is that all respiratory emergencies are initially handled identically until further information related to the underlying cause can be determined. Thus, if a veterinarian can recognize when a dog or cat is in respiratory distress and provide relief as soon as possible, these nerve-racking emergencies will become less stressful for everyone involved.

SIGNS OF RESPIRATORY DISTRESS
It is important first to ensure that all veterinarians and technicians in your practice easily recognize the signs of respiratory difficulty. Signs of respiratory distress include: extension of the head/neck, abduction of the elbows, open-mouth breathing (especially cats), retraction of the lips, flared nostrils, increased abdominal participation during breathing, paradoxical breathing (asynchrony of the thorax and abdomen), an increased respiratory rate, or an inability to sit/lie down.

INITIAL STABILIZATION
The first steps in handling a patient in respiratory distress include providing supplemental oxygen and administering appropriate sedation. The key is to provide the form of oxygen supplementation (outlined below) that gives the highest fraction of inspired oxygen (FIO₂) while causing the least amount of stress to the patient. Giving sedation to the patient will also help to relieve its anxiety and enable you to complete a physical examination without causing further distress. The sedation drug of choice is butorphanol (0.2–0.4 mg/kg IV or IM). If additional sedation is needed, a benzodiazepine (diazepam 0.25–0.5 mg/kg IV; midazolam 0.2–0.4 mg/kg IV or IM) can also be given. These drugs cause very little cardiopulmonary suppression and are extremely safe in all situations causing respiratory distress. Conversely, if the dog, or cat, is in respiratory distress due to traumatic injuries, a stronger opioid should be given (hydromorphone 0.025–0.05 mg/kg IV or IM; buprenorphine 0.01–0.02 mg/kg IV or IM).

OXYGEN SUPPLEMENTATION
Oxygen supplementation can be provided by several means, but must be regulated by a flow meter. Flow meters can be adapted to an oxygen tank to enable delivery of oxygen throughout a clinic. Ideally, oxygen should also be humidified when long-term administration is anticipated. Humidification can be achieved by attaching a commercial humidifier (plastic bottle filled with distilled water) directly to the flow meter.

FLOW-BY OXYGEN
Flow-by oxygen is the administration of oxygen by holding the oxygen tubing near (1–2 cm) the nose and mouth of the dog or cat. The flow rate should be set to approximately 100 mL/kg/min. This form of oxygen administration is usually temporary as it necessitates someone physically holding the oxygen near the patient and redirecting the oxygen as the patient moves. It is also associated with large losses of oxygen to the environment and a limited FIO₂ delivered to the patient. This method is well tolerated in the short term by most patients and easily performed upon arrival to the clinic, but might be difficult in an anxious or uncooperative dog or cat.
Facemask
Oxygen can also be provided via facemask by placing a cone (with or without the black rubber dam) over the patient’s mouth and nose. The mask should allow escape of heat, humidity, and CO$_2$, and the oxygen flow should be at least 100 mL/kg/min to avoid rebreathing. Poorly fitted facemasks might require flow rates as high as 300 mL/kg/min. Most patients do not tolerate oxygen administration by this method unless they are recumbent; however, some cats or small dogs will allow their entire face to be placed into the mask for oxygen administration. The FIO$_2$ is typically 50%, but may be up to 100% with a well-fitting mask.

Nasal Oxygen
To provide nasal oxygen, place a nasal cannula or nasal prongs and administer oxygen at a rate of 50–150 mL/kg/min. Nasal cannulas are placed to deliver oxygen directly into the nasal passage via the ventral meatus at the level of the distal nasal cavity or nasopharynx. As such, cannulas should be placed to the lateral canthus of the eye. An FIO$_2$ of 40–60% can be attained depending on the oxygen flow rate achieved. Typically, clear feeding tubes or red rubber catheters are used (cats: 3.5–5 F, dogs: 5–8 F).

Instructions for placement of a nasal oxygen cannula:
1. Administer proparacaine (ophthalmic anesthetic) drops (3–5 drops) or lidocaine (2%) injection (0.25 mL) into the nasal passage.
2. Premasure the cannula to the desired distance and mark the cannula with indelible marker or a piece of white tape.
3. Lubricate the tip of the nasal cannula with lidocaine (2%) jelly.
4. Direct the cannula ventrally and medially into the nasal passage (aiming for the base of the opposite ear in a cat or the opposite canine tooth in a dog).
5. The cannula should pass easily. An inability to pass the cannula past the medial canthus of the eye indicates placement within the dorsal meatus.
6. The cannula can be secured using a suture placed through tape immediately adjacent to the nares and again between the eyes on the forehead. Conversely, a very small amount of instant Krazy® glue may be applied.

Oxygen Hoods
Oxygen hoods are another form of oxygen supplementation that involve the placement of a Plexiglas, plastic, or cardboard container, or an Elizabethan collar (3/4 of the front covered by cellophane), over the head or entire anterior portion of the animal’s body. Oxygen is then supplied into the hood at flow rates of 1–10 L/min. These hoods can achieve an FIO$_2$ of approximately 50–100%, but vary dramatically. These hoods allow continued contact with the patient for auscultation, administration of medications, and performing procedures; however, they are not well tolerated by all patients. Additionally, these hoods can become warm and humid making them uncomfortable for long-term oxygen supplementation.

Oxygen Cages
Oxygen cages are a means of providing oxygen using commercial cages that are designed to provide an FIO$_2$ of 40–60%. Many reputable cages are heat and humidity controlled. Patients tolerate this form of oxygen supplementation very well as they can be placed inside the cage and monitored from the outside until their anxiety subsides. Disadvantages of this form of oxygen supplementation are that they are often only available at large emergency clinics or referral hospitals and that the FIO$_2$ will temporarily decrease each time the cage door is opened to assess the patient.

Intubation
Intubation allows immediate control of the airway to be obtained and an FIO$_2$ of 100% to be provided. This form of oxygen supplementation is necessary for patients with an upper airway obstruction, respiratory fatigue due to prolonged respiratory distress, respiratory failure, or respiratory arrest. Some patients will continue to breathe spontaneously once endotracheally intubated; however, others will require positive pressure ventilation using an AMBU bag or anesthetic machine. If long-term positive-pressure ventilation is required, the patient must be referred to a hospital that offers critical-care
(mechanical) ventilation. Induction drugs are often required for patients needing endotracheal intubation, although sometimes butorphanol sedation is enough in very debilitated patients. Ketamine:diazepam (1:2) 0.1–0.2 mL/kg IV titrated to effect is recommended for safe and rapid induction of patients in respiratory distress. Propofol induction is not recommended due to its respiratory suppressant effects.

**GENERAL PHYSICAL EXAMINATION**

Handling of all patients in respiratory distress should be performed in an area of the hospital where oxygen supplementation is readily available and emergency procedures could be performed rapidly if necessary. The initial assessment of the dog or cat in respiratory distress must be quick and not worsen the animal’s condition. Sometimes even a cursory exam is not possible without the animal becoming more stressed. If this occurs, administer sedation (if not already given), or consider giving additional sedation, and continue oxygen supplementation with minimal restraint or handling of the patient.

Once the patient tolerates handling, an exam should be performed focusing on the respiratory and cardiovascular systems including:

- Respiratory rate and character
- Thoracic auscultation (listen for increased/decreased lung sounds, crackles, wheezes)
- Cardiac auscultation (listen for murmurs, gallop rhythms, arrhythmias)
- Mucous membrane color and capillary refill time (CRT)
- Pulse quality and blood pressure (if tolerated)
- Extremity temperature
- Jugular vein assessment (for distension or pulses)
- Rectal temperature (if tolerated)

Although a complete physical examination is ideal, patients in respiratory distress are not candidates for a full physical examination including abdominal palpation, rectal examination, or neurologic or orthopedic assessments. Only when the patient is stabilized should an exhaustive physical examination be performed. Once the dog or cat tolerates a physical examination, placement of an intravenous (IV) catheter should be attempted. An IV catheter will enable the administration of additional sedation, induction medications if emergency intubation is required, or emergency medications in the face of cardiopulmonary arrest.

**HISTORY**

Obtaining an accurate history from the owner is very important for determining the underlying etiology for the respiratory distress. Although the precipitating cause of the respiratory distress might be obvious (e.g., trauma), there could be a more insidious underlying cause.

Owners should be asked about the following:

- Signalment including age
- Duration of clinical signs
- Past medical history (e.g., cardiac disease, megaesophagus, allergies, neoplasia)
- Routine veterinary care (i.e., heartworm prevention)
- Travel history
- Events leading up to the respiratory distress (e.g., exercise, hot/humid weather)
- Presence of concurrent abnormal clinical signs (e.g., inappetence, coughing, vomiting, lethargy)

**PATTERNS OF RESPIRATORY DISTRESS**

Diagnostic tests such as radiographs, lab work, and ultrasound can pose a serious threat to dogs, or cats, with respiratory distress. The physical examination and history are almost always enough to localize the cause of the respiratory distress. The most important observations are the respiratory pattern (i.e., type of distress) and auscultation. These preliminary findings will enable rapid determination of the origin of the respiratory distress.
<table>
<thead>
<tr>
<th>Type of respiratory distress</th>
<th>Most probable cause</th>
<th>Observations</th>
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<tbody>
<tr>
<td>Inspiratory distress</td>
<td>Upper-airway disease</td>
<td>Prolonged inspiratory phase Abnormal respiratory noises (e.g., stridor, stertor)</td>
</tr>
<tr>
<td>Expiratory distress</td>
<td>Lower-airway disease</td>
<td>Prolonged expiratory phase Abnormal lung sounds (e.g., wheezes)</td>
</tr>
<tr>
<td>Rapid, superficial breathing</td>
<td>Pleural-space disease</td>
<td>Fast expiratory phase Abnormal (i.e., dull, absent) lung sounds</td>
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<tr>
<td>Rapid, superficial breathing or concurrent</td>
<td>Pulmonary parenchymal disease</td>
<td>Abnormal lung sounds (e.g., increased, crackles)</td>
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<tr>
<td>inspiratory and expiratory distress</td>
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**Diagnostic Tests**
Only when the patient is considered stable should any diagnostic tests be performed. These tests might enable differentiating underlying causes of respiratory distress or monitoring response to treatment.

**Thoracic Radiographs**
Thoracic radiographs should not be performed until the patient is undoubtedly stable. If the patient resists handling or transport of any kind, radiographs should not be attempted. Sedation is often essential for enabling radiographs. Thoracic radiographs are helpful for confirming the presence of lower-airway disease, pulmonary parenchymal disease, or pleural-space disease. Thoracic radiographs are also recommended following any traumatic episode to document injuries (e.g., pulmonary contusions, rib fractures, pneumothorax, hemothorax, diaphragmatic hernia).

**Thoracic Ultrasound**
Thoracic ultrasound is often tolerated prior to thoracic radiographs because it can be performed with minimal patient manipulation. Thoracic ultrasound can be used to quickly determine the presence of pleural-space disease such as pleural effusion. Thoracic ultrasound can also help to identify pneumothorax, intrathoracic masses, pericardial effusion, or alterations in cardiac chamber size or contractility.

**Pulse Oximetry**
Pulse oximetry is a noninvasive method for assessing oxygenation of dogs and cats. It measures the peripheral oxygen saturation (SpO2), which is an indirect measure of SaO2 and thus PaO2 using the oxygen-hemoglobin dissociation curve. A SpO2 of 90% corresponds to a PaO2 of 60 mm Hg (severe hypoxemia) and a SpO2 of 95% corresponds to a PaO2 of 80 mm Hg (normal oxygenation). Pulse oximetry can be used for documenting hypoxemia and for monitoring the patient’s response to oxygen supplementation. In a normal dog or cat, the PaO2 should be 4–5 times the FIO2. For example, a patient breathing room air (FIO2 = 21%) should have a PaO2 of 80–100 mm Hg (SpO2 > 95%) and a patient on nasal oxygen (FIO2 ~ 40%) should have a PaO2 of 160–200 mm Hg (SpO2 ≥ 100%).

**Differential Diagnoses and Treatment of Common Respiratory Emergencies**
The initial treatment of every dog or cat with respiratory distress is the same regardless of the underlying cause. However, once the location of the respiratory disease is identified, differential diagnoses can be narrowed down and more specific treatment can be provided.

**Obstruction of the Upper Airways**
**Characteristics**
If the obstruction is complete, there is a total absence of airway sounds and intense effort is needed to breathe. If the obstruction is partial, breathing still requires intense effort, but loud respiratory noises (i.e., stridor or stertor) are common. Patients almost always breathe with their mouths open and inspiration is
generally prolonged. Affected dogs commonly present in respiratory distress after exercise or during hot/humid weather. A common concurrent finding is an elevated rectal temperature, because panting becomes a less efficient form of thermoregulation.

**Differential Diagnoses**
Brachycephalic syndrome; mass/foreign body in the pharynx, larynx, or trachea; laryngeal edema; laryngeal paralysis; laryngeal collapse; tracheal collapse; nasopharyngeal polyps (cats).

**Specific Treatments**
Sedation is highly effective for dogs and cats with upper-airway obstructions as sedation relieves anxiety and improves breathing. In addition to butorphanol, acepromazine (0.01–0.02 mg/kg IV or IM) can be helpful. If relief of the distress is not provided by sedation, induction and rapid endotracheal intubation or emergent tracheotomy might be required. Examine the oropharynx and larynx for the presence of foreign bodies or masses and, if intubation is performed, be sure to evaluate the laryngeal function. It is very important to monitor and control hyperthermia and provide active cooling (i.e., cool water/towels, fans, ice packs, IV fluids) if needed. The administration of corticosteroids (dexamethasone 0.1–0.15 mg/kg IV) can also effectively decrease upper-airway inflammation.

**Obstruction of the Lower Airways**

**Characteristics**
These patients typically present with a history of coughing or allergies and exhibit a prolonged expiratory phase of breathing. During auscultation, wheezes might be heard due to bronchoconstriction. Once performed, thoracic radiographs will reveal a bronchial pattern characterized by “doughnuts,” “railway tracks,” or “tram lines.”

**Differential Diagnoses**
Asthma (most common); bronchitis; foreign bodies; neoplasia

**Specific Treatments**
The stress of handling a patient (especially cats) with asthma will often cause decompensation. Sedation and oxygen are highly effective in providing initial relief from respiratory distress. The main objective is then to enable bronchodilation using inhalant or injectable medications. Salbutamol (inhalant) can be provided using a meter-dose inhaler (MDI) and pediatric mask and given as often as 1–2 puffs every 30–60 minutes during the initial crisis. Injectable bronchodilators include aminophylline (5 mg/kg IV slowly) and terbutaline (0.01–0.05 mg/kg IV, IM, SQ every 4–12 hours). Corticosteroids are often very effective in reducing inflammation due to airway inflammation (dexamethasone 0.1–0.15 mg/kg IV, IM or prednisone 1 mg/kg PO every 24 hours).

**Diseases of the Pulmonary Parenchyma**

**Characteristics**
Pulmonary parenchymal disease is caused by an accumulation of fluid, exudate, blood, or fibrous tissue within the pulmonary parenchyma. Almost all causes of pulmonary parenchymal disease lead to rapid, superficial breathing characterized by both inspiratory and expiratory distress. Auscultation often reveals abnormal lung sounds such as crackles or increased respiratory sounds. Once thoracic radiographs are obtained, they are useful in differentiating the underlying cause. Perihilar infiltrate (classic radiographic distribution in dogs) with distended pulmonary veins is indicative of cardiogenic edema. Cranioventral alveolar infiltrate is often associated with aspiration pneumonia. A nodular pattern is often associated with neoplasia or granulomatous/fungal disease.

**Differential Diagnoses**
Pulmonary edema (e.g., cardiogenic, noncardiogenic); pneumonia; pulmonary hemorrhage/contusions; neoplasia
Specific Treatments
Treatments administered for pulmonary parenchymal disease differ depending on the underlying cause. If cardiogenic pulmonary edema is suspected, diuretics (furosemide 2–4 mg/kg IV, IM, SQ every 2–4 hours) are indicated. Nitroglycerine (transdermal ointment - wear gloves!) can also be applied to the pinna for vasodilation and relief from pulmonary edema. For pneumonia, antibiotics, physiotherapy (i.e., chest coupage), fluid therapy, and humidification of the airways (i.e., nebulization) might be required. Pulmonary contusions require supportive care (i.e., oxygen therapy, analgesics) and typically resolve within 3–5 days.

Pleural-Space Disease
Characteristics
Any accumulation of air or fluid in the pleural cavity will reduce the lung’s ability to expand and cause rapid, shallow breathing sometimes characterized by prolonged inspiration and fast expiration. Auscultation reveals an absence of heart or lung sounds (ventrally if pleural effusion, dorsally if pneumothorax). Intestinal borborygmi might be heard in the thoracic cavity if a diaphragmatic hernia is present, but more commonly an absence of lung sounds is noted.

Differential Diagnoses
Pneumothorax (e.g., spontaneous, traumatic); pleural effusion (e.g., transudate, blood, exudates, chyle); diaphragmatic hernia

Specific Treatments
Thoracocentesis for pleural effusion or pneumothorax should be performed as soon as possible and will provide immediate relief from respiratory distress. Thoracocentesis can be diagnostic and performed simply with a 3-cc syringe and 1-inch 22-ga needle inserted into the chest. Otherwise, thoracocentesis can be therapeutic by removing large volumes of air or fluid from the pleural space. Thoracic ultrasound can be used to identify pockets of fluid, otherwise blind thoracocentesis is acceptable. If fluid or air builds up continuously requiring drainage or aspiration three times within a 24-hour period, placement of a thoracostomy tube or catheter should be considered. Fluid should always be saved for cytology ± culture. Thoracocentesis is generally performed with the dog or cat in sternal recumbency. Sedation beforehand is ideal to relieve respiratory distress and prevent movement during the procedure. Time permitting, analgesia can be provided by instilling a small bleb (0.25 mL) of lidocaine 2% at the thoracocentesis site 1–2 minutes prior to the procedure. Necessary equipment includes: clippers, sterile scrub solution, extension set, three-way stopcock, syringe (20–60 mL), and the desired needle/catheter. A butterfly catheter (cats and small dogs) or an over-the-needle 16- to 18-ga catheter (cats or dogs) is recommended. The catheter is inserted at the 7–9th intercostal space (ICS) in the dorsal portion of the chest if air is suspected or the ventral portion of the chest if fluid is suspected. Try to advance the catheter on the cranial aspect of the rib to avoid the nerves and vessels that run caudal to the rib. The catheter should never enter the chest ventral to the costochondral junction. Once fluid, or air, enters the syringe, the catheter should be advanced with care not to penetrate the lung (i.e., keep the needle or catheter parallel to the body wall). After the stylet is removed (over-the-needle catheters), aspiration should be continued until fluid or air can no longer be retrieved. Avoid performing needle redirection if using a butterfly catheter, to prevent lung puncture.

Nonrespiratory Causes of Respiratory Distress
Diseases that alter respiratory function without affecting the respiratory structures can cause respiratory distress. For example, diseases that impair neurologic or neuromuscular function (e.g., myasthenia gravis) can impair the ability of the chest wall to expand. Additionally, abdominal distension (e.g., abdominal effusion, GDV) or excessive compression from abdominal bandages, can compress the thoracic structures preventing normal chest expansion. Lastly, other conditions can cause hyperventilation, which can be mistaken for respiratory distress, such as cardiac tamponade, hypotension or shock, hyperthermia or fever, pain or anxiety, acidosis, and anemia.
Abdominal Fluid Collection and Analysis Made Easy
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INTRODUCTION
Dogs and cats often present to veterinary clinics because of the appearance of a pendulous or enlarged abdomen or for clinical signs secondary to abdominal fluid accumulation. Abdominal fluid can accumulate from a variety of disease processes including many serious conditions that require immediate intervention. Sometimes the condition that led to the accumulation of abdominal fluid is obvious, such as the collapsed elderly dog with the history of a large splenic mass, presumably ruptured and causing hemorrhage. Conversely, the underlying condition or cause of fluid accumulation is not always apparent. For example, a dog that was hit by a car could develop abdominal fluid because of a splenic or liver tear (hemoabdomen) or a bladder rupture (uroabdomen). In that situation, the treatment required differs depending on the underlying cause. In order to accurately determine the etiology of the effusion and advise the owner about the treatment options and prognosis for their pet, a sample of the fluid should be obtained by abdominocentesis and analysis of the fluid performed. Sometimes the analyses are easy and can be performed in-hospital, while other times cytologic assessment by a clinical pathologist is warranted. In any scenario, the collection and analysis of abdominal fluid will give a significant amount of information to help guide the management of these patients.

NORMAL ABDOMINAL FLUID PHYSIOLOGY
A small amount of abdominal fluid is normally present to allow for lubrication between abdominal organs during movement. Normal abdominal fluid is a low-protein, serous ultrafiltrate of blood that flows out of arteriolar capillaries into the abdominal cavity, and is mostly reabsorbed by the venous capillaries. The portion that is not reabsorbed, approximately 10%, is resorbed by the lymphatic system. The rate of fluid that exists within the abdominal cavity is largely determined by Starling’s forces, which are the hydrostatic and onotic pressures within the blood vessels and body cavity. The degree of lymphatic drainage, as well as mesothelial and endothelial permeability, also dictate the amount of abdominal fluid accumulation. Mesothelial and endothelial cells line the abdominal wall and abdominal organs, and the permeability across the surfaces is affected by hormones and cytokines released during different disease processes.

ABDOMINAL FLUID PATHOPHYSIOLOGY
Abdominal fluid accumulates when the rate of fluid filtration into the abdominal cavity exceeds the rate of fluid resorption from the abdominal cavity. The rate of abdominal fluid accumulation is increased with increased capillary hydrostatic pressure (e.g., heart failure), decreased capillary onotic pressure (i.e., hypoalbuminemia), increased endothelial permeability (e.g., peritonitis), and decreased lymphatic drainage (e.g., lymphatic obstruction). If abdominal fluid accumulation is severe, abdominal compartment syndrome can occur resulting in abdominal organ dysfunction (e.g., kidney failure). Abdominal fluid is resolved by restoring the pressure balance, lymphatic drainage, and permeability of the endothelium and mesothelium.

PHYSICAL EXAMINATION
It is important to perform a complete physical examination on all patients with confirmation or suspicion of abdominal fluid. Signs of abdominal fluid can be obvious including a distended or taught abdomen or palpation of a fluid wave. However, some patients will present with severe systemic illness (e.g., collapse, shock) secondary to blood or fluid loss, which leads to a suspicion of abdominal fluid accumulation. Findings from the complete physical exam might help to determine the underlying etiology of the abdominal fluid. For example, lymph node palpation might detect lymphadenomegaly and increase the suspicion of lymphosarcoma. Alternatively, if a heart murmur is detected and the jugular veins appear distended, the abdominal fluid might be secondary to congestive heart failure. Conversely, if the
abdomen is painful during palpation, this suggests that the abdominal fluid is likely secondary to a primary abdominal condition such as a ruptured gastrointestinal tract or severe pancreatitis.

**Diagnostic Imaging**

**Abdominal Radiographs**
Abdominal radiographs are often the first diagnostic imaging modality chosen in patients suspected to have abdominal effusion, probably because radiography is an available modality for most practitioners. If performed, abdominal radiographs must be adequate to assess all intraabdominal structures so that the abdomen can be thoroughly investigated for the presence of masses, intestinal obstructions, organ enlargement, absent abdominal organs (e.g., nonvisible urinary bladder), or free peritoneal gas (e.g., ruptured abdominal viscous). These signs, when seen with concurrent evidence of abdominal fluid such as loss of abdominal serosal (organ) margin detail, can assist in determining the cause of the abdominal fluid.

**Focused Assessment Using Sonography for Trauma (FAST)**
When abdominal fluid is suspected, ultrasound can be a secondary diagnostic modality used to confirm the presence of the fluid and assist with obtaining a sample for analysis. Focused assessment using sonography for trauma (FAST) is a simple and rapid ultrasound examination that can be performed to detect free fluid. Veterinarians with limited previous ultrasound experience can perform a FAST examination in less than five minutes. It can be used with a scoring system (score 0 to 4 out of 4) to evaluate for the presence of fluid in four areas.

With the patient in right lateral recumbency, use the ultrasound probe to assess for fluid (anechoic area around organs) in the following locations:
1. Diaphragmatico-hepatic (DH) view - just caudal to the xiphoid process
2. Spleno-renal (SR) view - left flank region
3. Cysto-colic (CC) view - on the midline over the urinary bladder
4. Hepato-renal (HR) view - right flank (most dependent) region

During the FAST examination, the urinary bladder and gall bladder should also be visualized. Note that even though an organ can be seen on radiographs or ultrasound and appear intact, organ rupture can still be present. FAST can be performed on initial presentation and serially thereafter to monitor for increases in abdominal fluid (i.e., increasing abdominal fluid score). In patients with lower abdominal fluid scores (1 or 2 out of 4), the DH and CC views are the most likely to reveal fluid.

**Diagnostic Abdominocentesis**
An abdominal fluid sample is generally accomplished by blind abdominocentesis, four-quadrant abdominocentesis, or ideally, ultrasound-guided abdominocentesis. With the patient in right lateral recumbency, the area around the umbilicus should be clipped and steriley prepped. When performing blind abdominocentesis, the ideal site is 2–3 cm caudal to the umbilicus and 2–3 cm from midline in the dependent region. With the patient in right lateral recumbency, accidental puncture of the spleen is less likely in this region. Standing abdominocentesis is not recommended, as it increases the chance of splenic puncture. Abdominocentesis should be performed using a needle (20–22 ga) and syringe (3–6 cc) advanced perpendicular to the skin. This closed technique is preferable to open techniques (using needles without attached syringes), which introduce air into the abdominal cavity thus making additional diagnostic imaging tests more difficult to interpret.

Once the needle is through the skin, apply suction on the syringe while advancing through the subcutaneous tissue and abdominal wall. Fluid should enter the syringe as soon as the abdominal wall is penetrated, if abdominal effusion is present. Alternatively, suction can be performed intermittently during advancement. If single blind abdominocentesis is unsuccessful, four-quadrant abdominocentesis can be performed by repeating the blind abdominocentesis in areas 2–3 cm cranial and caudal to the
umbilicus and lateral to midline (in a total of 4 quadrants). Finally, if ultrasound is available, the ideal method of abdominocentesis is ultrasound-guided into a pocket of fluid (i.e., anechoic region).

**Abdominal Fluid Diagnostic Tests**

Once an abdominal fluid sample is obtained, it can be placed into an EDTA (lavender top) tube, serum (red top) tube, and additional sterile (red top or other) tubes. Samples might need to be prioritized depending on the volume of fluid obtained and the suspected underlying disease. If the sample appears to be blood, it should be placed in a serum (red top) tube to evaluate for clotting. If the fluid was obtained from the abdominal cavity, it should not clot unless the hemorrhage is active/ongoing or the fluid has a high fibrinogen concentration. If the fluid was obtained from an accidental splenic or other vascular puncture, it will clot, similar to a venous sample.

**In-Hospital**

Fluid collected in EDTA tubes can have the PCV and total solids (TS) measured using hematocrit tubes and a refractometer. This is indicated immediately if the fluid appears hemorrhagic or serosanguinous. Unfortunately, some EDTA tubes contain additives that falsely increase the TS concentration. Similarly, if only a small amount of fluid was added to the tube, the PCV might be diluted. Therefore, these measurements can also be performed on the fluid in the syringe (prior to putting it into a tube) to avoid erroneous interpretation. Fluid turbidity from lipids, hemolysis, or cellular debris and refrigeration of fluid samples can also falsely increase the TS measurements. Therefore, turbid samples can be centrifuged and the TS measured on the supernatant. Analyses should always be performed on fluid samples at or near room temperature. Smears prepared for cytology (microscopic assessment) can be made directly from the fluid in the EDTA tube, especially if the fluid appears flocculent or turbid. However, if the fluid is clear or hazy, the tube should be centrifuged and smears made from the sediment of the sample. The smears can be stained with Diff-Quik or other Romanovsky-type stains for analysis.

Fluid collected into serum (red top) tubes can be used for measurement of total protein, albumin, bilirubin, creatinine, potassium, triglyceride, glucose, lactate, and lipase concentrations using an in-house laboratory or handheld device, if available. These measurements can be helpful in determining the underlying disease process, especially in comparison to the concurrent peripheral blood (serum, plasma) measurements. Note that a delay in sample processing can affect the results; fluid glucose levels will decrease and lactate levels increase as samples sit in the tube.

**Send-Out**

Fluid collected in EDTA tubes can be submitted for RBC count, total nucleated cell counts (TNCC), cytology (microscopic review by a clinical pathologist), or other advanced analyses if indicated (e.g., flow cytometry [neoplasia], PCR [feline infectious peritonitis]). Smears can be prepared (as outlined above) and unstained smears can also be submitted with the EDTA tube fluid to the laboratory. Preparation of slides at the time of sample collection is ideal to reduce artifactual changes in cell morphology.

Fluid samples collected into other sterile tubes (preferably red top or culture tubes) can be stored for aerobic and anaerobic bacterial, mycoplasma, and fungal cultures. Anaerobic cultures should not be refrigerated and are ideally processed within 24 hours of collection. Culture results are typically not available for 48–72 hours due to the length of time required to grow the organisms. Purple-top tubes should not be submitted for culture as the EDTA is bacteriostatic.

**Classification and Etiology of Abdominal Effusions**

Abdominal fluid is traditionally categorized by its protein concentration and cellularity (TNCC). Unfortunately, there is some overlap between the categories used (Table 1), which usually necessitates additional analysis of the fluid. However, when combined with the patient’s clinical picture, this information might be helpful in determining the etiology of the abdominal fluid, especially in patients with pure or modified transudates. For patients with exudates or more highly cellular fluids, cytology will help determine the predominant cell type(s) and underlying etiology. For example, exudates are often primarily composed of neutrophils; if the neutrophils appear degenerate, the smear should be thoroughly assessed for evidence of bacteria (intracellular), which would indicate a septic effusion. If
bacteria are not readily seen, other laboratory tests can be performed in-hospital (e.g., lactate, glucose measurements) to determine the likelihood of the effusion being septic. Additionally, there are many useful diagnostic techniques to differentiate nonseptic effusions, depending on the suspected underlying etiology.

### Table 1. Traditional classification of abdominal effusions and associated disorders

<table>
<thead>
<tr>
<th></th>
<th>Pure transudate</th>
<th>Modified transudate</th>
<th>Exudate</th>
<th>Chyle</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Colour</strong></td>
<td>Transparent - straw yellow</td>
<td>Transparent - yellow - reddish</td>
<td>Yellow - red Viscous</td>
<td>White - pink Cloudy</td>
</tr>
<tr>
<td><strong>Protein (g/L)</strong></td>
<td>≤ 25</td>
<td>≥ 25</td>
<td>≥ 30</td>
<td>≥ 25</td>
</tr>
<tr>
<td><strong>Total nucleated cell count (TNCC) x 10⁹/L</strong></td>
<td>≤ 1.5</td>
<td>1.5-7.0</td>
<td>&gt; 7.0</td>
<td>0.5-20.0</td>
</tr>
<tr>
<td><strong>Predominant cells</strong></td>
<td>Rare monocytes and mesothelial cells</td>
<td>Variable (monocytes, lymphocytes)</td>
<td>Polymorphonuclear neutrophils (PMNs) (possibly degenerative)</td>
<td>Mature lymphocytes, PMNs, macrophages</td>
</tr>
<tr>
<td><strong>Common causes</strong></td>
<td>Hypoalbuminemia, cirrhosis, portal hypertension</td>
<td>Heart failure, vasculitis, diaphragmatic hernia, portal hypertension</td>
<td>Bacterial or fungal infection, neoplasia, FIP, pancreatitis</td>
<td>Trauma, lymphatic obstruction, heart failure, idiopathic</td>
</tr>
</tbody>
</table>

### Septic Effusion

Septic effusion is typically the result of a ruptured gastrointestinal tract or penetrating wound to the abdomen. Patients with a septic effusion require immediate intervention including antibiotic administration and an exploratory laparotomy. Therefore, diagnosis of a septic effusion must happen as soon as possible. The TNCC for septic abdominal fluid is usually > 13.0 x 10⁹/L. If intracellular bacteria are not seen, additional tests can be performed to increase the index of suspicion of infection including measurement and comparison of abdominal fluid and peripheral blood lactate and glucose concentrations. Specifically, if a dog’s abdominal fluid lactate is 1.5 mmol/L higher than the concurrent peripheral blood lactate, or if the dog’s abdominal fluid glucose is more than 1.1 mmol/L less than the concurrent peripheral blood glucose concentration (without IV dextrose supplementation), this suggests that the effusion is septic. Unfortunately, these measurements are unreliable in cats, making cytology still the best test if a septic effusion is suspected.

### Nonseptic Effusions

Nonseptic effusions include abdominal fluid resulting from pancreatitis, feline infectious peritonitis (FIP), hemorrhage, chyle, neoplasia, or a ruptured abdominal viscous.

### Pancreatitis

Pancreatitis causes a nonseptic, suppurative inflammation that can have degenerate or non-degenerate neutrophils. It is often diagnosed in combination with clinical signs and ultrasound examination findings. A greater than 4-fold increase in abdominal fluid lipase compared to the upper end of the reference interval, or more than twice the peripheral blood lipase activity, suggest pancreatitis in a patient that has not experienced trauma.

### Feline Infectious Peritonitis (FIP)

Feline infectious peritonitis (wet form) causes a nonseptic effusion. The total protein of the fluid is typically 35–45 g/L and an effusion total protein ≥ 80 g/L is 90% specific and 55% sensitive for FIP. The
TNCC is usually low (2.0–6.0 × 10⁹/L). Unfortunately, FIP is very difficult to definitively diagnose and is typically a diagnosis of exclusion (i.e., ruling out bacterial infection and neoplasia). Abdominal fluid feline coronavirus antibodies (1:1600), gammaglobulin concentrations > 10 g/L, and albumin-globulin ratios ≤ 0.9 can be diagnostic, but with only 75–85% sensitivity and specificity.

**Hemorrhagic Effusions**

Hemorrhagic effusions typically have a PCV > 10% and can appear hemolyzed. If the cytology is consistent with peripheral blood including platelets and no erythrophagocytosis, then inadvertent splenic aspiration, venipuncture, or acute severe hemorrhage should be suspected. Hemorrhagic abdominal fluid is typically secondary to blunt trauma (e.g., hit by car, fall from height), coagulopathies (e.g., anticoagulant rodenticides), or neoplasia (e.g., ruptured splenic hemangiosarcoma). If there is no history of trauma, coagulation testing and diagnostic imaging should be performed to differentiate coagulopathies and neoplasia. Unfortunately, cytologic evaluation of hemorrhagic effusions is usually of low-diagnostic yield due to hemodilution.

**Chylous Effusions**

Chylous effusions are typically milky or opaque and result from reduced lymphatic drainage from the gastrointestinal tract into the cranial vena cava. Chylous effusions are characterized by an abdominal fluid triglyceride concentration > 1.1 mmol/L, abdominal fluid triglyceride concentration greater than the concurrent serum triglyceride concentration, or abdominal fluid cholesterol concentration less than the abdominal fluid triglyceride concentration. Chylous abdominal fluid can be the result of trauma, obstructed lymphatic drainage (secondary to masses), heart failure, or idiopathic.

**Neoplastic Effusions**

Neoplastic effusions are caused by exfoliation of neoplastic cells into the abdomen, most typically due to carcinomas, mesotheliomas, and round-cell neoplasms (e.g., lymphoma, mast-cell tumours, malignant histiocytosis). Lymphosarcoma is one of the more common forms of neoplasia in small animals that can cause neoplastic abdominal effusions and is typically characterized by a monomorphic population of immature or atypical lymphoid cells. These are usually larger than neutrophils and have a moderate amount of clear-blue cytoplasm, variably shaped nuclei with prominent and sometimes bizarre/angular nucleoli, and finely stippled nuclear chromatin. Clinical pathologists are often adept at diagnosing lymphosarcoma based on cytology, but additional diagnostic tests such as flow cytometry or PCR for analysis of antigen receptor rearrangements (PARR) can also be helpful and require an abdominal fluid sample stored in an EDTA (lavender top) tube.

**Ruptured Abdominal Viscous**

A ruptured abdominal viscous can result in a septic effusion (described above), uroabdomen, or bile peritonitis. When urine leaks from the urinary tract and into the abdomen, the resulting abdominal fluid is initially classified as a pure transudate. However, as inflammation progresses, especially if the urine is infected, neutrophils will accumulate. Creatinine, urea, and potassium are all concentrated in the urine compared to the peripheral blood, but because urea is a small molecule and easily passes across the mesothelium of the abdomen, abdominal fluid to peripheral blood creatinine or potassium ratios are recommended for diagnosing a uroabdomen. An abdominal fluid to peripheral blood ratio of creatinine > 2.0 or potassium > 1.4 is consistent with a uroabdomen.

Bile peritonitis is an inflammatory response to bile in the abdominal cavity, which usually occurs secondary to trauma, cholangitis, obstruction of the bile duct, or surgery (iatrogenic). Septic bile peritonitis results from the leakage of contaminated bile, which increases the severity of inflammation and has a worse prognosis. Bile peritonitis should be suspected when the abdominal fluid obtained from a patient is green, orange, or yellow-tinged, or the patient has a history of gall bladder disease, pancreatitis, trauma, or cholelithiasis. Cytology of bile peritonitis effusion often reveals gold, green, or black-brown pigment within macrophages or free in the background; however, measurement of fluid bilirubin concentrations is necessary to confirm the diagnosis. An abdominal fluid concentration that is more than 2-fold the peripheral blood bilirubin concentration is diagnostic. Cytologic evaluation for bacterial organisms should also be performed since this affects the prognosis for recovery.
Breaking the Silence: Disclosing Medical Errors
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Medical errors are an unfortunate occurrence in veterinary hospitals everywhere. Sometimes, despite the best veterinary care and intentions of the veterinary team, unintended adverse outcomes still occur. Medical errors are becoming increasingly recognized by veterinary care providers, who are under additional scrutiny from clients, given recent increases in public awareness of medical errors. While human-medicine regulating bodies have implemented guidelines for the disclosure of medical errors in order to learn from the mistakes and improve patient safety, no such guidelines currently exist in veterinary medicine. However, it is clear that documenting and disclosing medical errors has significant benefits for all people involved. Patients receive additional treatment required consequent to the medical error, concerns regarding otherwise unexplained problems are decreased, and the veterinary-client-patient relationship is strengthened. Ultimately, the veterinarians and technicians involved also have improved recovery from the emotional stress of making the error and clients will be less likely to file complaints or engage in litigation. Most importantly, the overall safety of the hospital will be improved when medical errors are recognized and steps are taken to avoid the same errors in the future.

Terminology
Medical errors are often described as any action or decision that retrospectively appears incorrect or results in a disappointing outcome. The Institute of Medicine defines a medical error as “the failure of a planned action to be completed as intended or the use of a wrong plan to achieve an aim.” An adverse event is defined as unanticipated harm (i.e., injury, illness, death) caused by medical care. A preventable adverse event can be attributed to a medical error. A negligent adverse event occurs when the medical care provided failed to meet the standard of care. In other words, an average or similarly trained veterinarian or technician would have been expected to recognize and prevent the error that caused the harm. It should be clarified that not all medical errors cause harm, not all adverse events causing harm result from medical errors, and not all adverse events resulting from errors are due to negligence. In some circumstances, an adverse event can be due to unreasonable client expectations, biological variability (e.g., animals treated the same way for the same disease will have different outcomes), or an unpredictable or low-likelihood complication (e.g., vaccine reaction).

Occurrence of Medical Errors
It is estimated that approximately 1.5 million preventable adverse events occur in the United States human medical field each year, with 44,000 to 98,000 human deaths each year being attributed to medical errors. It is likely that this number is actually even higher, given how many medical errors are unreported or undetected. It is estimated that one adverse event occurs in one-third of all admissions to human hospitals. Studies reveal that the incidence of adverse events in human hospitals is 3–36% and that the incidence of death due to these adverse events is 2–21%. The disparity among study findings is likely related to the different study methods in terms of data collection and patient selection, as well as the differing definitions of what is considered “preventable.” The highest medical error rates are said to occur in the intensive care unit (ICU), operating room, and emergency room.

Types of Medical Errors
Errors are often classified as diagnostic, treatment, preventive, or other. Specifically, diagnostic errors include an error or delay in making a correct diagnosis, failure to perform indicated tests, use of outdated tests, and failure to act on results of monitoring or testing. Treatment errors include errors in the performance of an operation, procedure, or test, errors in the administration of the treatment, errors in the dose or method of administering a drug, an avoidable delay in a treatment or responding to an abnormal test result, and inappropriate care or provision of care that is not indicated. Preventative errors include the failure to provide prophylactic treatment or inadequate follow-up after treatment and other
errors include failures in communication, equipment, or other system failures. The most commonly reported examples of adverse events in human medicine include drug reactions, improper transfusions, surgical injuries, wrong-site surgeries, restraint-related injuries, falls, burns, pressure ulcers, and mistaken patient identities.

**Documenting Medical Errors**
Detection of medical errors can occur by any of the following methods: chart review, malpractice claim analysis, observation of patient care, morbidity and mortality rounds including autopsy findings, clinical surveillance, administrative data analysis, electronic medical records, and error reporting systems. There are pros and cons to each of these systems, none of which have the ability to detect all adverse events that occur. Error or incident reporting systems (IRS) are voluntary and account for an estimated 10% of the total adverse events that actually occur. Studies reveal that the implementation of IRS in human hospitals allows for a global overview of adverse events in the hospital and has a positive effect on the safety of hospitalized patients because it enables hospital staff to learn from their mistakes and for adjustments to be made to improve patient safety in the future. Chart or medical-record review occurs retrospectively and is considered the gold standard of documenting an adverse event. Unfortunately, this process relies on accurate documentation by healthcare providers and because not everything is written in charts or records, adverse events or medical errors are still missed. Most human hospitals have a system employed to review the medical record of any patient that dies while hospitalized. Typically, a nurse will review the record to detect any possible medical errors and physicians will then confirm whether the medical error was avoidable or preventable.

Interestingly, studies comparing the different methods used to detect or document medical errors have found differences amongst the errors reported. IRS are more likely to document patient accidents, medical device errors, or medication errors, most likely because these reports rely on voluntary reporting from members of the healthcare team. Conversely, when reviewing patient complaints, the medical errors most likely found are due to inconsiderate behavior of healthcare providers, clinical administration (e.g., long waiting lists), insufficient resources (e.g., bed/staff availability), and clinical process (e.g., delayed or incorrect diagnosis). Likewise, retrospective chart reviews more commonly reveal medical errors due to clinical process (e.g., delayed or incorrect diagnosis, incidents during medical procedures).

**Etiology of Medical Errors**
Because of the complexity of the healthcare environment, the reasons underlying medical errors or adverse events are complex and due to multifaceted disease processes, number of staff, equipment, infrastructure, policies, and procedures. It should never be forgotten that some degree of error is inevitable with any human task. Nevertheless, there are various factors that contribute to an increased number of medical errors. These include increased workload, sleep deprivation, loud background noise, disorganized communication, too much or too little information, incomplete information during patient transfers, hierarchical barriers, different communication needs and expectations, failure to listen, equipment failure, negligence, insufficient training, and incompetence.

Most healthcare providers overestimate their ability to function while stressed. In fact, as stress increases due to anxiety or fatigue, thought processes and attention spans decrease and familiar exercises are performed poorly. Therefore, increased workload and sleep deprivation account for a large number of medical errors. During both physician and nursing shifts, studies show that more serious medical errors occur during longer shifts. Errors can also occur due to bias, which is actually more common with more experienced healthcare providers. Bias is difficult to remedy, but can be avoided by using an evidence-based approach to patient care.

**Reducing Medical Errors**
Several studies have investigated the impact of medical errors and adverse events on human patients. After adjustment for risk factor exposure time, even when medical errors occurred several times per patient, there was no impact on mortality. Conversely, having more than two adverse events was associated with a 3-fold increase in the risk of death. Therefore, while not every medical error will
increase the risk of death, medical errors resulting in adverse events will. And while the human ability to err cannot be changed, other circumstances in hospitals can be altered to help reduce medical errors. These include enhanced education related to medical errors, systems changes, improved teamwork and communication, and increased reporting of medical errors.

Studies reveal that only 1/3 of human hospitals have an environment that supports reporting of medical errors. As such, it is important to change the culture from one of “blame,” in which people are unwilling to accept responsibility for errors due to fear of criticism or punishment, to one of “just.” A “just” culture supports open dialogue and uninhibited reporting of medical errors as a commitment to quality patient care and to facilitate safer hospital practices. It involves a quick and thorough investigation of adverse events, extensive informal and formal communication, and hospital-wide learning including staff training and education.

MEDICAL ERRORS IN VETERINARY MEDICINE

There is considerably less information available with regards to the incidence, type, and handling of medical errors occurring in veterinary hospitals. Results from a survey sent to recent veterinary graduates in the United Kingdom were published in 2004 and revealed that 78% of veterinarians responding to the survey answered “yes” to the question: “Have you ever made a mistake (defined as an erroneous act or omission, resulting in a less than optimal or potentially adverse outcome for a patient) since starting work as a vet?” These errors were most commonly related to mistakes made regarding the diagnosis (e.g., appropriate diagnostic test not performed), during surgery, or during the management/treatment (e.g., administration of inappropriate drugs/medical therapy). Of the respondents who provided additional details of their error, 74% attributed the error to lack of experience, 36% to lack of time, 26% to lack of supervision, 21% to communication problems with colleagues, 16% to communication problems with owners, 15% to lack of information, and 12% to inadequate equipment. Interestingly, 37% of respondents did not discuss the mistake with the owner, whereas 93% of respondents discussed the error with friends, family, or colleagues.

Appropriate Handling of Medical Errors

Disclosure of medical errors requires courage and composure, effective communication skills, and the steadfast belief that an owner is obligated to know the truth. First and foremost, when a medical error is identified, ensure that the patient’s immediate needs are tended to. Second, recognize your own emotional needs and attempt to develop clarity regarding what happened. Third, prepare for the discussion with the owner, which will typically involve a team of people and a series of communications. Prior to the first conversation, it is important to try to understand the causes and effects of the medical error, which will determine the content of what will be discussed and who will be present and involved in the owner discussion.

The communication tools used to disclose a medical error are similar to those used during the delivery of bad news. It is important to maintain an empathic and non-defensive demeanor. Likewise, an accurate and honest explanation of what happened, as well as a discussion of the steps taken to prevent a reoccurrence must occur. Focus on what is known at the time (even if it is not entirely clear what happened) and assure the owner that future discussions related to new information will take place. The situation, problem, and all possible scenarios/outcomes for the pet and owner should be discussed. It is also imperative that a genuine apology is made that expresses acceptance of responsibility for what occurred. Thereafter, invite questions, listen, and answer truthfully. Generally, an agreement to waive fees related to the hospitalization and/or adverse event demonstrates commitment to helping the patient recover and decreases the perception that only an attorney can ensure that the system behaves fairly.

Practical Approach to Fluid Therapy

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**INTRODUCTION**
Animals presenting with either acute or chronic disease can exhibit a wide variety of fluid imbalances. The cat that has been chronically vomiting may be dehydrated but have normal perfusion. The dog that has been hit by a car may require intravascular volume replacement to treat hemorrhagic shock, but will have normal hydration. The puppy with parvoviral enteritis and severe diarrhea may require resuscitation of the intravascular space to improve perfusion, as well as replacement of interstitial volume deficits. It is often difficult to know where to start when determining which fluid to administer, when to reach for a colloid, and what fluid additives might be required. Information regarding the crystalloids and colloids currently available in Canada are provided and case examples will be used to illustrate their practical use.

**INTRAVENTOUS FLUID PHYSIOLOGY**

**Isotonic Crystalloid Solutions**
Isotonic crystalloid solutions (e.g., NaCl 0.9%, Lactated Ringer’s solution, Plasma-Lyte A) have essentially the same osmolality as body fluids. Therefore, there is no osmotic drive for water to either enter or leave the cells. So, the water and electrolytes remain within the extracellular fluid compartment (ECF). This means that after IV administration of this fluid, 25% of it stays within the intravascular space and 75% redistributes to the interstitial space. Therefore, effective fluid resuscitation of the vascular space with an isotonic fluid solution requires the administration of 4 times the vascular deficit (e.g., blood loss) to account for redistribution from the intravascular to the interstitial space, which occurs in approximately 30–60 minutes following the administration of IV fluids.

**Hypotonic Crystalloid Solutions**
Hypotonic crystalloid solutions (e.g., NaCl 0.45%, dextrose 5% in water [D5W]) have osmolalities that are less than that of body fluids. Therefore, hypotonic solutions will leave the intravascular space more readily than isotonic or hypertonic solutions, thus redistributing to both the interstitial and cellular compartments. Consequently, these fluids have no role in volume resuscitation or intravascular volume maintenance. Their administration is limited solely to the therapy of hypoglycemia, electrolyte abnormalities, chronic dehydration, or as maintenance fluid support. The dextrose contained in many hypotonic fluids is rapidly metabolized to free water and CO₂ when administered IV.

**Hypertonic Crystalloid Solutions**
Hypertonic crystalloid solutions (e.g., NaCl 3%–7.5%) have significantly more osmotically active particles per unit of volume compared to body fluids. Therefore, when given IV, hypertonic crystalloid fluids cause water movement from the interstitial and cellular compartments into the intravascular compartment. As a result, volume expansion of the intravascular compartment is increased greater than the amount of hypertonic crystalloid given (up to 8 times as much), as water is pulled from other body compartments. The movement of water from the intracellular compartment into the interstitium causes intracellular dehydration; therefore, hypertonic saline is contraindicated in patients that are dehydrated, hypernatremic, or hyperchloremic.

**Colloids**
Colloids contain large molecular weight substances (e.g., proteins, starches) that do not readily pass across capillary membranes and exert an oncotic pressure similar to that of proteins in the blood. Because the membrane between the intravascular and interstitial space allows for only limited passage of colloid molecules, colloid solutions (e.g., hydroxyethyl starches) administered IV tend to remain in circulation for longer periods of time (hours to days) when compared to crystalloids. Because of their oncotic properties, colloid solutions also have the ability to draw extravascular water into the intravascular space, thereby causing intravascular volume expansion above and beyond the administered amount (similar to hypertonic saline).
INTRAVENOUS FLUID SOLUTIONS

Dextrose Solutions
Dextrose 5% in water (D5W) is converted in the body to CO₂ (expelled by the lungs) and water. Therefore, the administration of D5W is equivalent to administering water (sterile water cannot be given IV as it will cause red cell hemolysis) and the water will redistribute to all fluid compartments including cells. Thus, the main indications for the use of D5W are to provide water to correct cellular dehydration, or to correct hypernatremia from a water deficit. In other cases, dextrose is added to IV fluids to help maintain normal blood glucose concentrations. Some veterinarians have the false perception that providing dextrose in IV fluids provides adequate nutrition to the patient. This is incorrect, as only 170 kcal is present in 1 L of D5W and at a maintenance fluid rate this is insufficient to meet daily calorie requirements. D5W is contraindicated as an emergent resuscitation fluid as most of it moves into cells, providing minimal expansion of the intravascular space.

Crystalloid Solutions
The term crystalloid refers to a fluid solution that contains sodium as its major osmotically active particle. Crystalloid solutions are typically a combination of sodium chloride and other electrolytes (i.e., potassium, chloride, magnesium, calcium). These fluids might also contain glucose and buffering agents such as lactate, gluconate, and acetate. Please refer to Table 1 for the components of the most commonly used crystalloids. Crystalloids can be classified as replacement or maintenance solutions. A replacement solution is a crystalloid solution that is required to treat the patient’s volume deficit (hypovolemia), whereas a maintenance solution is a crystalloid solution that is required to maintain the patient in a state of normal hydration and euvolement.

Table 1. Characteristics and contents of commonly used crystalloid solutions

<table>
<thead>
<tr>
<th>Solution</th>
<th>Type</th>
<th>Dextrose</th>
<th>Na</th>
<th>Cl</th>
<th>K</th>
<th>Ca</th>
<th>Mg</th>
<th>Buffer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertonic saline 7%</td>
<td>Replacement</td>
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<td>0</td>
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<tr>
<td>NaCl 0.9%</td>
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<td>154</td>
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<td>0</td>
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<tr>
<td>LRS</td>
<td>Replacement</td>
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<td>109</td>
<td>4</td>
<td>3</td>
<td>0</td>
<td>Lactate</td>
</tr>
<tr>
<td>Plasma-Lyte A</td>
<td>Replacement</td>
<td>0</td>
<td>140</td>
<td>98</td>
<td>5</td>
<td>0</td>
<td>3</td>
<td>Acetate, Gluconate</td>
</tr>
<tr>
<td>Normosol-R</td>
<td>Replacement</td>
<td>0</td>
<td>140</td>
<td>98</td>
<td>5</td>
<td>0</td>
<td>3</td>
<td>Acetate, Gluconate</td>
</tr>
<tr>
<td>5% dextrose</td>
<td>Maintenance</td>
<td>50 g/L</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<td>0</td>
</tr>
<tr>
<td>0.45% NaCl 2.5% dextrose</td>
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<tr>
<td>0.45% NaCl</td>
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<td>0</td>
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<td>77</td>
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<td>0</td>
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<td>0</td>
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<td>Normosol-M</td>
<td>Maintenance</td>
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<td>40</td>
<td>13</td>
<td>0</td>
<td>0</td>
<td>Acetate</td>
</tr>
</tbody>
</table>

Replacement solutions are isotonic and can be further classified into acidifying or alkalinizing solutions. An example of an acidifying solution is 0.9% NaCl because it does not contain a buffer. Alkalinizing solutions have a more similar electrolyte composition and tonicity to blood, as well as a buffer (e.g., Lactated Ringer’s, Plasma-Lyte A, Normosol-R). There is a slight variation in the electrolyte composition of the various alkalinizing solutions available, but all are “balanced” compared to the blood of animals and are subsequently referred to as “balanced electrolyte solutions.” These solutions contain a bicarbonate precursor (buffer) designed to reduce the dilution of the plasma bicarbonate and therefore prevent acidemia (dilutional acidosis). Lactated Ringer’s solution contains lactate as its bicarbonate precursor and Normosol-R and Plasma-Lyte A contain acetate and gluconate as their bicarbonate precursors. Lactate is metabolized in the liver to bicarbonate, whereas acetate is metabolized in the skeletal muscle and gluconate is metabolized in most cells within the body. Because their electrolyte composition is higher in sodium and chloride and lower in potassium, replacement fluids can be given...
rapidly and in large volumes to hypovolemic patients without altering the normal electrolyte concentration of the blood.

Maintenance solutions, such as Normosol-M and Plasma-Lyte 56, are hypotonic and are designed to meet the electrolyte requirements of patients with normal daily electrolyte losses. Animals normally lose 40–60 mEq/L of sodium and 15–20 mEq/L of potassium each day. Therefore, commercially available maintenance fluid solutions contain lower sodium and chloride and higher potassium concentrations than replacement fluids. Although the potassium concentration of maintenance solutions is higher than that of replacement solutions, it still may be inadequate to meet the average ill patient’s potassium requirements. Therefore, additional potassium supplementation to meet abnormal potassium losses associated with vomiting, diarrhea, or diuresis/polyuria might be required. Maintenance solutions are contraindicated in the treatment of shock due to their higher potassium content and hypertonicity, which prevents them from being given safely as fluid boluses.

**NaCl 0.9%**

NaCl 0.9% (physiologic saline) is often called “normal saline.” It contains sodium concentration similar to that of plasma (154 mEq/L), but abnormally high concentrations of chloride ions (154 mEq/L). NaCl 0.9% is used for shock resuscitation and is a useful replacement fluid in the treatment of hyperkalemia and hypokalemia, as seen in Addison’s disease. This fluid solution can also be used for the treatment of hypercalcemia and hypermagnesemia. Because NaCl 0.9% contains no bicarbonate precursor, it can be used in patients with metabolic alkalosis. Administration of large volumes of NaCl 0.9% can result in hyperchloremic acidosis. This fluid solution should not be used in animals with heart disease, hypertension, or liver disease as sodium restriction is typically part of their therapy.

**Lactated Ringer’s Solution**

Lactated Ringer’s solution (LRS) is a commonly used isotonic crystalloid. The lactate that is present in LRS does not contribute to the formation of lactic acidosis, nor does it alter the reliability of blood lactate measurements in normal animals. However, animals with severe liver disease might fail to convert lactate to bicarbonate. Therefore, administration of LRS to animals with liver failure or cats with hepatic lipidosis is not recommended. Animals with lymphosarcoma are also lactate intolerant and should not be given LRS. Because LRS contains calcium, it should be avoided in patients with hypercalcemia and should not be administered concurrently with blood products as the calcium can precipitate with citrate anticoagulant.

**Acetated Polyionic Solutions**

Acetated polyionic solutions (Plasma-Lyte 148, Plasma-Lyte A, Normosol-R) have a composition very similar to canine and feline blood. Since muscles and peripheral tissues metabolize the acetate and gluconate buffers it contains, this solution can be used in patients with severe liver disease. These solutions also contain magnesium instead of calcium. Rapid administration of these acetate-containing solutions can cause vasodilatation and hypotension in animals that already are hypovolemic. Although this is a rare event, monitoring blood pressure during bolus administration of acetated crystalloid solutions is recommended.

**Half-Strength Saline With or Without Half-Strength Dextrose**

Half-strength saline with or without half-strength dextrose (NaCl 0.45% + Dextrose 2.5%, NaCl 0.45%) are isotonic maintenance solutions. They are generally combined with potassium chloride to provide sufficient potassium to the patient. These fluids are an excellent choice for animals predisposed to sodium retention such as those with heart disease, hypertension, or liver failure. Since these fluids have little effect on maintenance of intravascular volume, they should not be used for emergency (shock) fluid resuscitation.

**Hypertonic Saline**

Hypertonic saline (NaCl 3%, 5%, 7%, 7.5%) is often used in the treatment of shock and trauma. Hypertonic saline also causes a decrease in intracranial pressure, which can be advantageous for patients with traumatic brain injury. Its effects are transient (lasting 30–60 minutes); however, more sustained
effects may be seen when combined with colloid solutions. Side effects of hypertonic saline include hypernatremia, hyperchloremia, hyperosmolality, and metabolic acidosis. Rapid infusions can cause bronchoconstriction and shallow breathing. The recommended dosage of hypertonic saline is 4–6 mL/kg (dogs) and 2–4 mL/kg (cats) given as a bolus over 5–10 minutes.

**Colloid Solutions**

Commonly used synthetic colloids include hydroxyethyl starch (HES) solutions or dextrans, whereas natural colloids include plasma or whole blood. Dextrans are rarely used due to a high incidence of allergic reactions and kidney injury. HES solutions are readily available and the most commonly used synthetic colloid.

**Hydroxyethyl Starch (HES) Solutions**

Hydroxyethyl starch (HES) solutions (pentastarch, tetrastarch, hetastarch) contain hydrolyzed amylopectin dissolved in a crystalloid solution (typically NaCl 0.9%). They are administered for volume resuscitation in patients with systemic inflammatory response syndrome (SIRS), sepsis, hypoalbuminemia, shock, trauma, hypotension, and peripheral edema. HES solutions can be administered as a small bolus in emergent situations, or as part of a fluid resuscitation plan in patients that are hypoalbuminemic or not responsive to crystalloid therapy alone. Potential complications of HES solution administration include volume overload (e.g., patients with heart disease or anuric renal failure) and coagulopathies. The larger molecules bind to platelets causing reduced aggregability and also inhibit the normal function of FVIII and vWF. Administering HES solutions at doses < 20 mL/kg/day is recommended to prevent the coagulopathic effects. Typically, boluses are given in 2.5–5 mL/kg volumes over 10–15 minutes. Cats are especially sensitive to boluses of HES solutions and may vomit if they are given too quickly.

In general, the oncotic effect and volume expansion of the colloid fluid increase with the number of small molecules in the solution (i.e., smaller molecules are more concentrated). Additionally, the half-life (duration of action) of the colloid solution will be longer as the size of the molecules in solution become larger (i.e., larger molecules are metabolized more slowly). The larger molecules also exert greater negative effects on the coagulation system. In order of largest to smallest molecules, hetastarch >> pentastarch > tetrastarch, which means that hetastarch lasts the longest and exerts the greatest negative effects on coagulation, whereas tetrastarch exerts the largest oncotic effect and lasts the least amount of time. HES solutions are becoming increasingly associated with acute kidney injury and need for hemodialysis in human patients; therefore, larger molecule solutions such as hetastarch are less readily available and HES solutions have been recently banned from the human market in Europe.

**Fluid Supplementation**

Supplemental electrolytes or dextrose can be added to crystalloids according to the patient’s requirements. The amount of potassium added should be based on the patient’s measured serum potassium. Normally, 20 mEq/L of potassium in maintenance IV crystalloids is sufficient to maintain normal potassium concentrations, but patients with preexisting deficits or ongoing losses of potassium can require much more. When rapidly replacing estimated fluid losses or giving shock boluses of fluids, it is important not to use fluids that have been supplemented with extra potassium because of the risk of hyperkalemia. **Potassium supplementation should not exceed 0.5 mEq/kg/h.** Remember that the amount of potassium administered to the patient will vary depending on the concentration in the bag and the rate at which the fluid is being administered. For maintenance rates of fluid, simple guidelines may be used to determine the amount of fluid added to the L bag (Table 2). When administering the fluids at rates above or below normal maintenance rates, the amount of potassium supplementation should be calculated on a mEq/kg/h basis (Table 3). However, avoid administering concentrations of KCl > 80 mEq/L via a peripheral vein as this can lead to thrombophlebitis.

**Table 2. General guidelines for potassium supplementation**
Serum potassium (mEq/L) | KCl added to the fluids (mEq/L)
--- | ---
Normal potassium | 20
Mild hypokalemia | 40
Moderate hypokalemia | 60
Severe hypokalemia | 80

Table 3. More accurate guidelines for potassium supplementation

<table>
<thead>
<tr>
<th>Serum potassium</th>
<th>KCl added to the fluids (mEq/kg/h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal potassium</td>
<td>0.05–0.1</td>
</tr>
<tr>
<td>Mild hypokalemia</td>
<td>0.15–0.2</td>
</tr>
<tr>
<td>Moderate hypokalemia</td>
<td>0.25–0.35</td>
</tr>
<tr>
<td>Severe hypokalemia</td>
<td>0.4–0.5</td>
</tr>
</tbody>
</table>

Dextrose supplementation (i.e., 2.5–5% in IV fluids) might be needed for patients exhibiting hypoglycemia including young animals, toy breed dogs, or patients with liver failure, Addison’s disease, insulin overdose, or xylitol toxicity. When adding 50% dextrose to crystalloid fluid bags to deliver dextrose supplementation, use the guidelines outlined in Table 4. Concentrations exceeding 5% administered long-term should be given via a central line (jugular vein) to avoid peripheral thrombophlebitis.

Table 4. General guidelines for dextrose supplementation

<table>
<thead>
<tr>
<th>Desired glucose concentration</th>
<th>50% dextrose added to the fluids (mL/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5%</td>
<td>50</td>
</tr>
<tr>
<td>5%</td>
<td>100</td>
</tr>
<tr>
<td>7.5%</td>
<td>150</td>
</tr>
<tr>
<td>10%</td>
<td>200</td>
</tr>
</tbody>
</table>
BLOOD SUPPLY TO THE BONE AND FRACTURE HEALING

I. NORMAL VASCULARIZATION
The normal blood supply is composed of three different systems:
- Afferent vascular system
- Intermediate vascular system
- Efferent vascular system

Afferent System
The afferent system carries the blood to the bone and consists of the nutrient artery, the metaphyseal arteries, and the periosteal arteries.

The periosteal arteries are normally a minor component of the afferent system and are mostly localized at muscle and fascial attachments to the bone.

In normal conditions, the periosteal arteries provide blood only to the outer cortex in the vicinity of their attachment.

Normally, the periosteal circulation is of lesser importance; its role should not be underestimated in the growing animal or after fracture.

Intermediate System
The blood vessels of the intermediate vascular system connect the afferent and efferent vascular system. It consists of canals of Havers and Volkmann and minute canaliculi, providing nutrients to the osteocytes.
**Efferent System**
Venous drainage occurs principally at the periosteum.
   The venous system present in the medullary cavity is mostly connected to the bone marrow.

In normal conditions, blood flow is essentially centrifugal.

**II. Vascular Response to Injury**
Response of vascularization following fracture depends on the complexity of the fracture.
- The afferent and efferent vascular systems hypertrophy. The arteries and veins left intact increase both in size and diameter.
- An *[extraosseous blood supply of the healing bone]* develops from the surrounding soft tissue. This blood supply is separate from the periosteal blood supply. It furnishes blood to devitalized fragment, devitalized cortex, and periosteal callus.
  After fracture stabilization and restoration of the normal afferent and efferent blood supplies, the extraosseous blood supply regresses.

Factors that interfere with blood supply will significantly impair bone healing. These factors can result from the initial trauma, improper surgical technique, inadequate reduction, and inadequate stabilization....

**III. Bone Healing**
Three types of bone healing are recognized:
- Primary bone healing (primary osteonal reconstruction)
- Gap healing (primary osteonal reconstruction)
- Secondary bone healing

**Primary Bone Healing**
Primary bone healing or direct bone union occurs in area of bone contact with high compression forces.
The gap between the fragments must be smaller than 0.1 mm and the fragments must be very stable (< 2% strain). When these conditions are met, osteons from one fragment will directly invade the other fragment and cortical remodeling will occur. No callus will be identifiable radiographically.

Although primary bone healing may appear ideal, in clinical situations, true primary bone healing is rarely achieved.

Bone healing by direct osteonal reconstruction regains strength slowly.

**Gap Healing**
Gap healing occurs when the gap between the fragments is less than 1 mm and the gap is very stable (< 2% strain). The gap is originally filled with blood clot, followed by loose connective tissue. After a few weeks, osteoblasts deposit lamellar bone in the gap between bone fragments. This lamellar bone is initially weak. After 3 to 4 weeks, the lamellar bone is progressively replaced by osteonal bone originating from both fragments.

**Secondary Bone Healing**
If the gap is too wide or if there is any movement between the fragments, secondary bone healing will occur. Secondary bone healing will progress through several steps, eventually leading to the restoration of a normal bone.
1. Hemorrhage and formation of a clot.
2. Inflammation and edema.
   **In areas of intermittent contact, there is initial resorption of bone to enlarge the gap.**
3. Proliferation of pluripotential mesenchymal cells (callus formation).
4. Fibrous tissue formation.
5. Metaplasia into fibrocartilage.
7. Osteonal bone formation and remodeling.
This process allows the fracture gap to be filled with successively stronger and stiffer tissue, until normal Haversian bone can be restored.

The entire healing process is under the control of mediators, chemoattractants, angiogenic factors, and growth factor, mechanical and electrical stimuli.

Callus can be subdivided on the basis of locations into medullary bridging callus, periosteal bridging callus, or intercortical bridging callus.

Although all three types of callus will usually form, the relative amount of each type will vary markedly in response to circumstances (i.e., bending instability is usually associated with a large periosteal callus).

IV. Average Time to Clinical Union

<table>
<thead>
<tr>
<th>Age</th>
<th>IM pins</th>
<th>Bone plate</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 3 mo</td>
<td>2-3 weeks</td>
<td>4 weeks</td>
</tr>
<tr>
<td>3-6 mo</td>
<td>4-6 weeks</td>
<td>6-12 weeks</td>
</tr>
<tr>
<td>6-12 mo</td>
<td>5-8 weeks</td>
<td>12-16 weeks</td>
</tr>
<tr>
<td>&gt; 1 yr</td>
<td>7-12 weeks</td>
<td>16-30 weeks</td>
</tr>
</tbody>
</table>

Although this table can be used as a rough guide, each patient must be assessed individually and the decision to remove the implants must not be made before complete assessment of the fracture and confirmation of healing.

Do not remove the implants before clinical union!

V. Complications of Bone Healing

Delayed Union
A delayed union is a fracture that has not healed in the expected time when compared with other similar fractures treated similarly in comparable patients.

Causes: Physical and Systemic Factors
Instability and vascular impairment are major causes of delayed union. Constant instability, beyond the acceptable strain will cause disruption of the delicate vasculature and will prevent callus metaplasia.

Vascularity impairment at the fracture site at the time of injury, or during reduction and internal fixation, will significantly delay bone healing.
Infection at the fracture site delays the healing process by promoting tissue ischemia and necrosis. Bones poorly covered by muscles tend to heal more slowly (radius, tibia) because the available source of vessels for recruitment into the healing process is limited. A large gap at the fracture site due to poor reduction, severe comminution, bone loss, and soft tissue interposition are other causes of delayed union.

**Treatment**
Surgery is indicated if the main fracture fragments are significantly malaligned and cannot be realigned by closed reduction or if the implants have loosened and instability is significant. Fibrous callus within the fracture gap need not be excised if adequate stability, compression, and reduction can be achieved. Delayed union may be treated conservatively if the alignment of the main fragments is adequate, the implants are still firmly attached to the bone, and instability at the fracture site is minimal. Radiographic progresses are reevaluated every 4–6 weeks and union is expected to develop within 8 to 12 weeks.

**Nonunion**
A nonunion is an ununited fracture characterized by a pseudoarthrosis at the fracture site.

**Causes**
Persistent instability, impaired vascularization, large gaps, interposition of soft tissue, and infection are major causes in the development of nonunions.

The radius is responsible for the majority of the nonunions and represents 60% of all nonunions (tibia 25%, femur 15%).

**Surgical Treatment**
Nonunions should be treated surgically as soon as they are diagnosed. Rigid stabilization of the fragments and cancellous bone graft are mandatory for success.

Treatment of nonunion is difficult and the prognosis is usually guarded.

**Malunion**
A malunion is a fracture that has healed in an abnormal position. It may result from:
- Spontaneous healing of a fracture
- Implant failure
- Improper fixation

Although dogs and cats can tolerate up to 20% shortening of their leg, angular and rotational deformation may cause significant dysfunction.

**Correction of a malunion is difficult and may require multiple surgeries.**
Surgical correction is performed only if a significant improvement in leg function can be expected.

**Characterization of the Fracture**
It is important to be able to accurately describe a fracture. Not only does this facilitate communication between clinicians, it is the first step in determining what forces will be acting on the fracture and choosing a successful repair technique. A minimum of two good-quality, orthogonal radiographs centered on the affected bone are needed to formulate this description.

**I. What Bone Is Involved**
This may seem obvious, but the surgical approach to a pelvis is significantly different from a tibia and the front limbs must bear more weight than the hind limbs. Similarly, stabilization techniques applicable to the axial skeleton may be quite different to those available for long bones.

**II. Extent of the Fracture**
A complete fracture disrupts the cortex 360 degrees and is usually accompanied by some displacement of the fractured ends. A greenstick fracture (seen mainly in young animals) indicates only one side of the bone is broken while the opposite cortex is merely bent. Greenstick fractures are innately stable and may be amendable to external coaptation. Fissures are longitudinal cracks that are not displaced, leaving the
periosteum intact. Stabilizing fissures should be the first step in internal fixation as failure to do so will often result in iatrogenic comminution of the fracture.

**III. Soft-Tissue Injury**

Closed fractures have an intact, viable soft-tissue covering. Open fractures have some degree of communication between the fracture and the external environment. Grade I open fractures may have an intact covering of nonviable skin or a small (< 1.0 cm) full-thickness wound. Grade II fractures are associated with larger lacerations, often created by the ends of the fractured bone. Grade III fractures have extensive soft-tissue injury with gross exposure of the underlying bone.

The greater the soft-tissue injury, the more damage will have been done to the local blood supply and the slower will be the fracture healing. Likewise, open fractures will have greater contamination and an increased risk of infection. Open fractures are generally not treated by external coaptation and extra effort must be made to ensure fragment stability.

**IV. Part of the Bone Affected**

Designations of epiphysis, metaphysis, and diaphysis are useful, but may have limited application in mature animals. Describing a fracture as located in the proximal, middle, or distal third of the bone is generally adequate. Of specific importance is involvement of the growth plate. Physeal injuries in immature patients may disturb future longitudinal bone growth potential in these animals.

The Salter-Harris classification is used to describe physeal fractures and is based on the extent of damage done to the growth plate and surrounding bone. Type I fractures form transversely along the layer of hypertrophying cells in the growth plate and do minimal damage to the dividing cells. Early, accurate reduction of these fractures results in rapid healing and continued longitudinal development of the bone. Type II fractures pass along the physis, but include a triangular portion of the metaphysis. The potential for continued growth in these bones remains good. In Type III fractures, the fracture line travels from the physis, through the epiphysis to the articular surface. Extension of fractures into the joint is important because, with the current technology, accurate and stable fixation of these fractures often requires the use of interfragmentary lag screws. Type IV fractures are a combination of type II and III with involvement of both the metaphyseal and epiphyseal portions of the bone. Type V fractures result in crushing of the physis and cessation of growth. The most common form of this injury is the ‘occult’ damage that occurs in the distal ulnar growth plate after radial fractures in immature animals. These patients will show premature closure of their distal ulnar physis weeks after the injury and are at risk of developing angular limb deformities.

**V. Number of Fragments**

Fractures are generally referred to as ‘two-piece,’ comminuted, multiple, or segmental. As the name implies two-piece fractures have only two fragments (it does not rule out fissures). Comminuted fractures may have single or multiple smaller fragments with a variety of fracture lengths. One of the most common types of comminution, the ‘butterfly’ fragment, is characterized by a large segment of cortical bone isolated by two converging, long oblique fracture lines. Bones with multiple or segmental fractures are broken into three or more pieces with fracture lines that do not intersect.
As a general rule, a larger number of bone fragments is associated with a more difficult repair, more extensive soft-tissue injury, and delayed healing.

VI. Obliquity

Transverse
By definition, a transverse fracture forms perpendicular to the long axis of the bone and the fracture length is roughly equal to the diameter of the bone. These fractures are innately resistant to compressive forces and may do well when treated by intramedullary pinning or external coaptation. Conversely, treatment of avulsion fractures at the insertion of ligaments or tendons needs to include some means of overcoming on-going traction.

Short Oblique
The length of the fracture line in a short-oblique fracture is less than twice the diameter of the bone. This obliquity affords some resistance to rotational forces, but compression will cause over-riding of the fragments. Hemicerclage wires are generally used with intramedullary pins to stop this slippage. The use of full cerclage wires, or lag screws, is contraindicated when treating short-oblique fractures because interfragmentary compression will cause slippage along the fracture line and displacement of the fragments.

Long Oblique
The length of the fracture line in a long-oblique fracture is at least two to three times the diameter of the bone. These bone fragments have no resistance to over-riding. Full cerclage wires or interfragmentary screws can achieve compression along the fracture line resulting in greater stability of the repair.

BIOMECHANICS OF FRACTURES
Under normal circumstances, bone is subjected to external forces (hit by car, etc.) and internal forces (muscle contraction). Bones can deform relative to the loads or stresses applied and remodeling occurs
with repeated or chronic stress. Variations in bone anatomy reflect the different physiologic forces that act on individual bones as they perform their normal functions.

**Stress** is defined as a local force and **strain** is the deformation created by that stress. **Normal stress**, in long bones, is defined as a force applied parallel to the weight bearing axis of the bone. **Shear stress** is force applied perpendicular to the weigh bearing axis.

When discussing forces on a bone, we divide the stresses into: compression, tension, bending, rotation, and shear. When these stresses exceed the bone’s structural limits, fractures will occur. The type of stress that created it may predict a fracture’s pattern. **Traction** is often associated with the insertion of tendons or ligaments and results in transverse fractures. **Compression** generally creates a ‘normal stress’ and is the force best tolerated by long bones. Excess compression will cause oblique fractures. **Bending** forces will generate both traction and compression on opposite sides of the same bone. This combination of forces can create butterfly fragments at the fracture line. **Rotation** around the central axis of a bone creates ‘shear stresses’ along a spiral line and is the force least tolerated by long bones.

As a general rule, the greater the energy of the force, the larger number of fracture fragments will be present and the more soft tissue injury will occur (slowing healing and increasing the risk of infection).

**REFERENCES**

ORTHODONTIC (BITE) PROBLEMS
Orthodontic problems are not unusual in dogs, but are fairly uncommon in cats. They may be purely cosmetic or can result in trauma to the lips, gums, palate, or teeth.

By far, the most common cause of malocclusions is hereditary. Additional genetic causes include tongue size as well as lip and cheek tension.

These patients often do not show any overt clinical signs other than the jaws or teeth being out of alignment. Depending on the type and severity of the problem, oral trauma may be present and can result in bleeding, oral pain, gum disease, tooth death, and even nasal infection.

Therapy for malocclusions is relative to type and severity of the disease process. Options include:

- No therapy (if purely cosmetic)
- Extraction of the offending tooth or teeth
- Orthodontic correction using appliances
- Lowering the tooth and then protecting the root canal (coronal amputation and vital pulp therapy)

Strictly cosmetic correction is certainly possible; however, it may not be in the patient’s best interest. The pain associated with orthodontic adjustment, and the numerous anesthetics required, often make orthodontic therapy a disservice to the otherwise healthy patient.

PERSISTENT DECIDUOUS TEETH
Persistent deciduous teeth are very common, especially in small and toy breed dogs. However, they can occur in any breed, as well as cats. They create both orthodontic and periodontal problems if not treated promptly. It used to be believed that the persistent deciduous caused the permanent tooth to become maloccluded. Studies have shown, however, that it is the permanent tooth erupting incorrectly that causes the deciduous to be persistent.

It has been reported that orthodontic problems begin within two weeks of the permanent canines starting to erupt. This is due to the deciduous tooth being in the place that the adult wishes to occupy.

The periodontal issues occur due to a disruption of the normal maturation of the periodontium. When there is a persistent deciduous tooth, one area of the periodontium is not attaching to the permanent, therefore the periodontal attachment in that location will not be normal. It has been reported that the damage begins within 48 hours of the permanent teeth starting to erupt!

Therefore, the adult tooth does not need to be completely erupted for these problems to occur, and they should be extracted as early as possible, do not wait until six months of age to perform the extractions along with neutering. In fact, we recommend that the owners of breeds prone to retain their teeth be instructed to watch for eruption of the permanent teeth and to present the patient for therapy as soon as this occurs.

ROTATED AND CROWDED TEETH
Rotated and crowded teeth can occur alone, in which case the malocclusion is classified as class 1, or in combination with other malocclusions. Rotated and/or crowded conditions can occur in a single tooth, in multiple teeth, or in any combination of teeth (incisors, canines, premolars, and molars). It is not uncommon to find crowded mandibular incisors in brachycephalic breeds. Another common finding in many breeds, but especially in brachycephalics, is maxillary third premolars crowded with maxillary fourth premolars or the mandibular fourth premolars crowded with first molars. The maxillary third and mandibular fourth premolars are usually also rotated in this condition. Other common conditions include
incisors crowded together, but also against the canine teeth. Finally, impaction of the distal shoulder of the maxillary third premolar into the furcation of the mesiobuccal and mesiopalatal roots of the fourth premolar is often seen in brachycephalic breeds.

Dogs with small jaws commonly have incisor crowding. This has been assumed to be an inherited condition. Often the second incisor on both sides on the mandible will be displaced out of the normal curve. Also, third incisors can be crowded with canines and maxillary premolars can be crowded together. Commonly recommended treatments for some of the most frequently found crowding situations include extraction of the lateral incisors to protect the canines, extraction of the maxillary third premolars to save the maxillary fourth premolars, or extraction of one or more of the more crowded mandibular incisors.

Rotation and crowding can cause pain from chronic tooth on tooth contact. This might be compared to the pain that humans experience from a caries that has been overfilled by their dentist, resulting in trauma to the opposing tooth during mastication. It is a condition that generally does not result in clinical signs of pain or anorexia; however, it can be quite painful. The chronic trauma resulting from tooth on tooth contact can lead to tooth non-vitality. Nonvital teeth must be either extracted or receive endodontic therapy.

Rotation and crowding can also result in tooth on soft-tissue contact, which can be not only painful, but can result in soft-tissue defects. This condition can progress and produce a defect that can collect food and stay inflamed, and if severe, can progress further to produce a fistula. Fistulas, when they continue into the maxillary sinus, can become oral-nasal fistulas.

Periodontal disease is commonly an eventual result of rotated and crowded teeth. Although human studies have shown that, with good home care, teeth can be maintained with some attached gingival lacking, a complete collar of attached gingiva around each tooth is ideal for ongoing periodontal health. This is often lacking for the rotated/crowded tooth. With these normal gingival attachments absent, the tooth is more prone to periodontal disease. On the maxilla, rotation situates the tooth in an abnormal position relative to the mucosa of the hard palate, often creating a pseudopocket that can trap food and debris. This can further complicate the lack of attached gingiva that is also a result of its rotation. Furthermore, crowding can result in a lack of interdental papilla, a part of the normal gingival collar between two teeth. Without this protective collar, both teeth are susceptible to periodontal disease. Teeth affected by rotation and crowding have lowered defenses to periodontal disease because of their ability to trap food, plaque, and calculus resulting in early onset of infection and inflammation.

Our canine and feline patients are anisognathic, meaning they have maxillary and mandibular dental arches or jaws that are of different sizes. Normal anisognaths have a pinking shear premolar orientation and a scissor incisor occlusion. This configuration promotes a self-cleaning mechanism. In the rotated and crowded situation, this normal cleaning mechanism is impaired or absent, further promoting plaque and calculus accumulation and adding fuel to the fire of periodontal disease. Left untreated, generalized periodontal disease is often the eventual result.

If intervention in the form of extraction occurs early in the course of the disease, the more functionally important tooth can be saved. For example, in the case of the rotated and/or crowded maxillary third premolar with fourth premolar, if the third premolar is extracted early it can save the maxillary fourth premolar. Delayed treatment will often result in the extraction of both teeth due to periodontal disease. Selected extractions are the treatment of choice for this condition.

**Fractured Teeth**

The two main types of crown fracture seen in veterinary medicine are complicated and uncomplicated. Both types require therapy; however, treatment for each is often different.

The tooth crown is made up of 3 layers. The innermost layer is the pulp chamber (an extension of the root canal). It is filled with blood vessels and nerves that originate from the maxillary or mandibular artery and nerve. The outermost layer is called enamel. It is 97% inorganic material. It has no sensory ability; however, it also has no ability to regenerate if lost. Between the pulp chamber and the enamel is dentin. Dentin makes up the majority of tooth structure in mature patients. Dentin is a living structure in that it has the ability to respond to stresses and has sensory ability. This sensory ability is due to the fact
that there are dentinal tubules which run at right angles to the root canal system ending at the dentinal-enamel junction (DEJ). There are 45,000 tubules per mm$^2$ in coronal dentin. This means that a defect 1 cm in diameter will result in the exposure of 1,000,000 odontoblasts.

The hydrodynamic mechanism of dentin hypersensitivity is the currently accepted explanation for pain associated with dentin exposure. Dentin exposure changes the fluid dynamics within the tubules. This change in fluid velocity is translated into electrical signals by the sensory fibers located within the tubules or subjacent odontoblast layer. These signals result in the sensation of pain (or sensitivity) within the tooth. It is rare for veterinary patients to show this discomfort, but occasionally anorexia will be the presenting complaint. Finally, the exposed dentinal tubules may act as a conduit for bacterial infection of the pulp, thus initiating endodontic disease.

Over time, the tooth will respond to this exposure by laying down a layer of reparative dentin. There is no study that documents the time for an effective layer to be placed in veterinary patients. One human study found that reparative dentin is seldom found prior to 30 days following exposure of dentinal tubules and completion of formation is generally around 130 days. It is not known, however, if this layer of reparative dentin is effective in decreasing tooth sensitivity.

All teeth with direct pulp exposure (complicated crown fractures) should be treated with endodontic or exodontic therapy; ignoring them is not an option. Prior to tooth necrosis, the viable nerve is excruciatingly painful. Following tooth death, the root canal system will act as a bacterial super-highway creating not only local infection, but also a bacteraemia which has been linked to more serious systemic diseases (see the article on Updates on Periodontal Disease for further information). The owners of these patients will be reluctant to pursue therapy as “It does not seem to bother the dog.” Fractured and/or infected teeth do bother the pet and they will act better following therapy. Veterinary patients are known for being stoic, and therefore lack of outward signs of oral pain should not be misinterpreted as a benign state. Therefore, you must be a patient advocate and recommend therapy.

Uncomplicated crown fractures are also a very common finding on oral exam, particularly in large breed dogs. These fractures will result in direct dentinal exposure. The exposed dentinal tubules will create significant pain for the patient. The currently accepted means by which this sensitivity is created is via the theory of fluid dynamics. In addition, some of these teeth will become nonvital due to the traumatic incident, pulpal inflammation, or direct pulpal invasion via the dentinal tubules. For these reasons, it is recommended that these teeth be radiographed to ensure vitality. If the teeth are nonvital (evidenced by periapical rarefaction or a widened root canal) endodontic, or exodontic, therapy is required. If the teeth appear vital, the application of a bonded composite is recommended to decrease sensitivity.

**INTRINSICALLY STAINED TEETH**

Endodontic disease is also manifested by intrinsic staining. This can appear as pink, purple, yellow, or grey. A study by Hale showed that only 40% of intrinsically stained teeth had radiographic signs of endodontic disease; however, 92.7% are nonvital. Nonvital teeth lose their natural defense ability and are often infected via the bloodstream, which is known as anachoresis. Therefore, do not rely on radiographic appearance to determine vitality; all teeth should be definitively treated via root-canal therapy or extraction.

**CARIES**

True bacterial caries are rare in dogs and almost unheard of in cats. They are most common on the occlusal surface of the upper first molars, but can be seen on any tooth. In addition, the most common breed is a German Shepherd Dog. Early lesions can mimic wear, and are best diagnosed by tactile feel of the defect with a sharp explorer. If it is sticky, like wax, it is likely a caries lesion. These lesions can progress into the endodontic system resulting in pain and infection (see fracture teeth above). Treatment options are restoration (composite or amalgam) or crown therapy (± endodontic therapy); or extraction.
**ENAMEL HYPOCALCIFICATION (HYPOPLASIA)**

Enamel is a very thin (< 1 mm) material on the surface of tooth crowns. It is formed and deposited on the dentin by the enamel forming organ which consists of cells called ameloblasts. Enamel is only formed prior to tooth eruption and cannot be naturally repaired after eruption into the mouth.

Hypoplasia/hypocalcification results from disruption of the normal enamel development. Ameloblasts are very sensitive and minor injuries can result in enamel malformation.

The most common acquired cause of enamel hypocalcification of one or several teeth is trauma to the unerupted tooth. This may be due to any external trauma, but is most often associated with the extraction of a deciduous tooth. In traumatic cases, one or several adjacent teeth may be affected. Additional causes of this pattern are infection or inflammation from a deciduous tooth.

A severe systemic infectious, or nutritional problem, may also result in improper enamel production. In these cases, most or all of the teeth are affected, but only a small part of the crown, usually a horizontal circumferential strip. Canine distemper was a common cause of this condition in the past.

Finally, enamel hypoplasia may result from a hereditary condition known as **amelogenesis imperfecta**. This condition is created by a decrease in the amount of enamel matrix applied to the teeth during. In these cases, nearly all teeth are involved on all surfaces.

Areas of enamel hypocalcification will generally appear stained a tan to dark brown (rarely black) color, and may appear pitted and rough. The tooth surface is hard, however, as opposed to the soft/sticky surface of a caries lesion. The areas of weakened enamel are easily exfoliated which will expose the underlying dentin, resulting in staining. Dentin exposure will result in significant discomfort for the patient (see uncomplicated crown fractures above).

The roughness of the teeth will also result in increased plaque and calculus retention, which in turn leads to early onset of periodontal disease.

For all of these reasons, prompt therapy of these teeth is critical to the health of the patient. Treatment is aimed at removing sensitivity, avoiding endodontic infection by occluding the dentinal tubules, and smoothing the tooth to decrease plaque accumulation. The most efficient and effective way to accomplish these goals is placement of a bonded composite restoration.

If the damage is severe and the client is interested in a permanent correction, crown therapy can be performed. Alternatively, extraction may be performed; however, this is not the recommended course of therapy if the root structure is normal with no evidence of endodontic infection.

**FELINE TOOTH RESORPTION**

Tooth resorptions (TRs) are a very common malady. Reports vary as to their incidence, but approximately 60% of cats over 6 years of age have at least one, and those that have one typically have more. These lesions are caused by odontoclasts which are cells that are responsible for the normal remodelling of tooth structure. These cells are activated and do not down regulate, resulting in tooth destruction.

There are currently two recognized forms of resorptive lesions, type 1 and type 2. Clinically, they appear very similar, as dental defects that are first noted at the gingival margin. However, advanced cases will show significant tooth destruction and may appear to be a fractured tooth. The best diagnostic tool for differentiating between types is dental radiology. With type 1 lesions, there is no replacement of the lost root structure by bone, whereas with type 2 there is generally marked replacement of the lost tooth structure.

Type 1 TRs are typically associated with inflammation such as L/P stomatitis or periodontal disease. In these cases, it is thought that the soft-tissue inflammation has activated the odontoclasts. The inciting cause of class 1 lesions is a cemental defect. Odontoclasts move in and destroy the dentin, leading to secondary enamel loss and a resorption lacuna. The weakened crown will eventually fracture, and in these cases the root canal system stays intact resulting in continued pain and infection for the patient.

Type 2 lesions are generally seen in otherwise healthy mouths; however, the lesions will create local gingivitis. The etiology of type 2 TRs remains unproven. The two major current theories are abfraction injuries from eating hard food and excess vitamin D in the diet. Type 2 TRs show histological evidence of
simultaneous repair of the defect by osteoblasts at the same time that tooth is being resorbed by odontoclasts.

Historically, restoration was a recommended therapy; however, due to the progressive nature of the disease, extraction is now the treatment of choice. Extractions can be very difficult in these cases due to tooth weakening and ankylosis. Additionally, in some cases, there is little to no tooth structure remaining. In cases with significant weakening and or ankylosis, performing the extractions via a surgical approach is recommended to speed the procedure and decrease the incidence of fractured and retained roots (see extraction article: Dental Extractions Made Easier).

Recently, crown amputation has been suggested as an acceptable treatment option for advanced type 2 lesions as it results in significantly less trauma and faster healing than complete extraction. This procedure, although widely accepted, is still controversial. Most veterinary dentists employ this technique, however, in widely varying frequency. Veterinary dentists typically employ this treatment option only when there is significant or complete root replacement by bone. Unfortunately, the majority of general practitioners use this technique far too often. Crown amputation should only be performed on teeth with radiographically confirmed advanced type 2 TRs which show no periapical or periodontal bone loss. Crown amputation should not be performed on teeth with: type 1 TRs, radiographic or clinical evidence of endodontic or periodontal pathology, inflammation, or infection; or in patients with L/P stomatitis. Those practitioners without dental radiology capability should not perform crown amputation. In these cases, the teeth should either be fully extracted or the patient referred to a facility with dental radiology.

**MISSING TEETH**

There are several reasons that teeth may be missing. These reasons include: congenitally missing, previously extracted, fractured (or extracted) with retained roots, or impacted. The first two scenarios do not require therapy, whereas the latter two may necessitate intervention. Therefore, dental radiographs are indicated in all cases of “missing teeth.”

If dental radiographs reveal retained roots and evidence of inflammation, or infection (clinical or radiographic), the teeth should be surgically extracted. If they are “quiet,” the owners should be informed and given the option of having the teeth surgically extracted.

Impacted teeth are defined as any tooth that has not erupted by its normal time. This is generally considered to be the time when the surrounding or contralateral teeth have already erupted. The most common cause of impaction is the presence of an overlying structure that interferes with normal eruption. These structures may be bone, soft tissue, or even tooth/teeth that interfere with the normal eruption path. The most common interference is an area of thick and firm gingiva called an operculum.

Impactions occur most commonly in the maxillary cuspid and premolar teeth (especially PM1). They also occur most often in toy and small breeds as well as brachycephalic dogs.

These patients generally have no overt clinical signs other than a missing tooth in a young animal. Alternatively, there may be a persistent deciduous tooth present.

On occasion, an unerupted tooth may lead to the development of a dentigerous cyst. The incidence of this is unknown in veterinary medicine; however, pathologic changes were noted in 32.9% of cases in one human study. Consequently, the presenting complaint, or oral examination finding, may be a swelling in the area of a “missing” tooth.

A dentigerous cyst is a fluid-filled structure which develops from the enamel forming organ of an unerupted tooth. Small dentigerous cysts are generally asymptomatic, and often go undiagnosed without dental radiology. If clinical, these cysts will generally be seen as swellings in the area of a missing tooth in a young patient. Dentigerous cysts can become quite large and disfiguring, requiring major surgical correction.

In addition, these cysts may become infected, resulting in acute swelling and pain. These cases are often misdiagnosed as abscesses. Finally, dentigerous cysts have reportedly to undergone neoplastic transformation. Dental radiographs are generally diagnostic, revealing a unilocular radiolucent area that is associated with the crown of an unerupted tooth. An aspirate obtained for fluid analysis and cytology...
will be supportive of a cyst. Definitive diagnosis can be achieved with histopathologic analysis of the
cystic lining.

Prognosis for these lesions is excellent if diagnosis and treatment are achieved relatively early in the
disease course.

Surgical removal of the offending tooth and careful debridement of the cystic lining will prove
curative. It is important to avoid leaving any of the cystic lining behind, as this could allow the cyst to
reform. Early surgical intervention will result in the least invasive surgery possible.

**ORAL NEOPLASIA**
The oral cavity is the fourth most common place to encounter neoplastic growths. The most common oral
growths are the epulides (fibromatous and ossifying). These are benign overgrowths of the periodontal
ligament (hamartomas). These can grow very large, but are not aggressive. Acanthomatous
ameloblastomas (epulides) are locally aggressive. They do not metastasize and are mildly aggressive
locally. They respond well to local excision with ½-cm margins and enjoy a 90% control rate with
radiation therapy.

Benign tumors are exceedingly rare in cats. By far the most common malignant oral tumor in cats is
a squamous cell sarcoma. Fibrosarcomas are a distant second. Both of these tumors are typically seen in
older cats, are locally aggressive, and are late to metastasize. The only therapeutic option at this point is
early, aggressive surgery (2-cm surgical margins).

The above tumors are also seen in dogs. Their behaviour and therapy is similar to cats; however,
these tumors respond better to radiation therapy in dogs. In dogs, the most common malignant tumor is a
melanoma which is typically seen in older dark pigmented dogs. Melanomas are not only locally
aggressive; they also metastasize very early in the course of the disease. A combination of aggressive
surgery, radiation therapy, and chemotherapy is the best way to treat this disease process. In addition, a
vaccine has been recently released that shows promise as an adjunct therapy for this disease process.

**EOSINOPHILIC GRANULOMA COMPLEX**
The true etiology of these conditions is unknown; however, a local accumulation of eosinophils is thought
to initiate the inflammation and necrosis. The accumulation may result from a local (food) or systemic
allergies; although these lesions have been seen in cases where allergic disease has been ruled out.
Additional causes include a response to irritation, such as chronic grooming or traumatic malocclusion.
There may also be a genetic predisposition.

Indolent ulcers are the most common oral manifestation, and they will present as brownish-red
lesions on the upper lip or around the maxillary canine teeth.

Linear granulomas can be single or multiple; the most common sites are the lips, gingiva, palate,
and tongue. They are generally nonpainful, but can become secondarily infected. The typical presentation
is a raised, lobulated, yellow-pink mass; however, they can also appear ulcerative causing severe damage
to the oral mucosa and underlying bone. This may lead to severe periodontal loss, pathologic fractures, or
oronasal fistulas.

**CAUDAL STOMATITIS**
This is another relatively recent disease process in cats that is frustrating us at present. The best
description is a severe, immune-mediated reaction to dental tissues, but we really don’t know. Some feel
that this may actually be a group of disease processes that look the same clinically which is why they can
be very frustrating to treat.

The history will generally include anorexia, drooling, gagging, and pain during mastication.
Physical exam will typically include a thin pet with unkempt fur. The oral exam will reveal severe
stomatitis usually over all teeth. The inflammation will most commonly be worse on cheek teeth than
canines and incisors. However, faucitis is the key clinical finding. Severe, hyperplastic inflammation to
the gingiva can result from periodontal disease; however, faucitis will not be present.
**Medical Therapy**

Most medical therapies will work for a while; however, in general, resistance will start within a year or less. In addition, most therapies have side effects worse than the disease process in and of itself. In general, medical therapy is very frustrating to the practitioner and client.

Corticosteroids are the mainstay of most medical therapy today. It is generally very effective at first and is relatively inexpensive for the client. In my experience, injectable (Depo-Medrol 5–10 mg IM) is much more effective than oral preparations in my experience. However, they will typically lose effectiveness after a year or so requiring higher and higher doses at shorter increments. This generally results in significant deleterious effects. About 10% of stomatitis cases we treat are already diabetic!

Antibiotics are safer than steroids, but much less effective, especially in long-term therapy. They are generally disappointing in their success. Metronidazole and clindamycin are the mainstays of therapy; however, Clavamox and amoxicillin can be used as well. Metronidazole may be the antibiotic of choice due to its antiinflammatory effect. Other immune suppressives such as Imuran, Cytoscan, gold salts, and cyclosporine have been used. However, they are all very expensive with numerous adverse side effects (myelosuppression).

**Surgical Therapy**

Extraction is currently the only effective long-term treatment for this disease process in cats. In our experience, the sooner this is done, the better that cats do both postoperatively as well as long term.

For extractions to be successful, the teeth must be completely removed. Therefore, postoperative radiographic confirmation of complete extraction of the tooth roots is recommended. Following the insurance of complete removal of the teeth, perform alveoloplasty to remove the periodontal ligament and smooth rough bony edges. This is typically performed with a rough diamond bur.

Studies report a 60% success rate when all teeth caudal to the canines are extracted; however, our experience has not been as good. However, whole-mouth extractions have a success rate of approximately 90-95% for clinical remission. Slight faucitis may remain, but pets are comfortable. In addition, the rare cases that don’t completely respond are generally much more responsive to medical therapy.

If there is no inflammation to the canines or incisors (which is rare), then the owner is given the option of leaving the canines. However, if these are inflamed, all teeth should be extracted. If the teeth are ankylosed, complete root pulverization may be necessary.

Sixty to seventy percent of the strength for the rostral mandible is contained in the canine tooth roots. Extracting both mandibular canines at the same time has resulted in jaw fracture on occasion. Therefore, consider extracting all but 2 ipsilateral canines on the first visit. Often this will be sufficient and cats will never have the last two teeth extracted.

Why leave a maxillary canine if the goal is to decrease the inflammation? This is because about 1/3 of cats who have maxillary canines extracted surgically will develop lip trauma from the mandibular canine. In many instances, this has necessitated further therapy in a cat where the stomatitis was resolved.
Updates on Periodontal Disease
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INTRODUCTION
Periodontal disease is the number one health problem in small-animal patients. By two years of age, 70% of cats and 80% of dogs have some form of periodontal disease. However, there are generally little to no outward clinical signs of the disease process, and therefore, therapy typically comes very late in the disease. Consequently, periodontal disease may also be the most undertreated disease in our patients. Additionally, unchecked periodontal disease has numerous local as well as systemic consequences. Local consequences include: oronasal fistulas, class II perio-endo lesions, pathologic fractures, ocular problems, osteomyelitis, and increased incidence of oral cancer. Systemic diseases which have been linked to periodontal disease include: renal, hepatic, pulmonary, and cardiac diseases, osteoporosis, adverse pregnancy effects, and diabetes.

PATHOGENESIS
Periodontal disease is generally described in two stages, gingivitis and periodontitis. Gingivitis is the initial, reversible stage in which the inflammation is confined to the gingiva. The gingival inflammation is created by plaque bacteria and may be reversed with a thorough dental prophylaxis and consistent homecare. Periodontitis is the later stage of the disease process and is defined as an inflammatory disease of the deeper supporting structures of the tooth (periodontal ligament and alveolar bone) caused by microorganisms. The inflammation results in the progressive destruction of the periodontal tissues, leading to attachment loss. This can be seen as gingival recession, periodontal pocket formation, or both. Mild to moderate periodontal pockets may be reduced, or eliminated, by proper plaque and calculus removal. However, periodontal bone loss is irreversible (without regenerative surgery). Although bone loss is irreversible, it is possible to arrest its progression. However, it is more difficult to maintain periodontally diseased teeth in comparison to healthy teeth. Additionally, periodontal attachment loss may be present with, or without, active inflammation.

Periodontal disease is initiated by oral bacteria which adhere to the teeth in a substance called plaque. Plaque is a biofilm, which is made up almost entirely of oral bacteria, contained in a matrix composed of salivary glycoproteins and extracellular polysaccharides. Calculus (or tartar) is basically plaque which has secondarily become calcified by the minerals in saliva. Plaque and calculus may contain up to 100,000,000,000 bacteria per gram. Bacteria within a biofilm do not act like free living or “planktonic” bacteria; and in fact are 1,000 to 1,500 times more resistant to antibiotics than are planktonic bacteria. Plaque on the tooth surface is known as supragingival plaque. Once it extends under the free gingival margin and into the area known as the gingival sulcus (between the gingiva and the teeth or alveolar bone), it is called subgingival plaque. Supragingival plaque likely affects the pathogenicity of the subgingival plaque in the early stages of periodontal disease. However, once the periodontal pocket forms, the effect of the supragingival plaque and calculus is minimal. Therefore, control of supragingival plaque alone is ineffective in controlling the progression of periodontal disease.

Initial plaque bacteria consist of predominately nonmotile, gram-positive, aerobic facultative rods and cocci. Gingivitis is initiated by an increase in the overall number of bacteria, which are primarily motile, gram-negative rods and anaerobes. In established periodontal disease, gram-negative rods account for approximately 74% of the microbiotic flora. Finally, elevated numbers of spirochetes are found in almost all periodontal pockets, and anaerobic organisms compose 90% of the bacterial species in chronic periodontal disease.

Classically, periodontal disease was thought to be caused by an increase in the overall numbers of bacteria. The nonspecific plaque hypothesis was based on the fact that periodontal disease is associated with an increased level of plaque and calculus. It was thought that low levels of plaque bacteria were controlled by the host response. It was further noted that the concentration of bacteria in periodontally diseased sites is twice as high as in healthy sites. However, recent studies point to a few, virulent strains of bacteria as being responsible for the attachment loss seen with periodontal disease. The specific
The plaque hypothesis is based on the fact that these few species are seen in virtually all cases of chronic periodontal disease. These findings have led to the development of the “one-stage, full-mouth disinfection” treatment as well as a vaccine against these organisms. However, the cornerstone of therapy is still meticulous plaque control.

The bacteria in the subgingival plaque secrete toxins as well as metabolic products. Also produced are cytotoxins and bacterial endotoxins which can invade tissues on their own, and in turn cause inflammation to the gingival and periodontal tissues. This inflammation causes damage to the gingival tissues and initially results in gingivitis. Eventually, the inflammation can lead to periodontitis (i.e., the destruction of the attachment between the periodontal tissues and the teeth). In addition to directly stimulating inflammation, the bacterial metabolic byproducts also elicit an inflammatory response from the animal. White blood cells and other inflammatory mediators migrate out of the periodontal soft tissues and into the periodontal space due to increased vascular permeability and increased space between the crevicular epithelial cells. White blood cells fight the infection by phagocytizing bacteria, but may also release enzymes to destroy the bacterial invaders either by design or after their death. When released into the sulcus, these enzymes will cause further inflammation of the delicate gingival and periodontal tissues. In fact, the progression of periodontal disease is determined by the virulence of the bacteria combined with the host response. It is the host response that often damages the periodontal tissues. However, patients with deficient immune systems typically have more severe periodontal disease than those individuals in good health. HIV, diabetes, and stress are significant risk factors for severe periodontitis in humans, and could likely be extrapolated to our animal patients.

The inflammation produced by the combination of the subgingival bacteria and the host response damages the soft tissue attachment of the tooth, and decreases the bony support via osteoclastic activity. This causes the periodontal attachment of the tooth to move apically (towards the root tip).

As periodontal disease progresses over time, the attachment loss continues in a nonlinear pattern as active stages of destruction are followed by quiescent phases (burst). The end stage of periodontal disease is tooth loss; however, the disease has created significant problems prior to tooth exfoliation.

**Clinical Features**

It is important to be familiar with normal features in order to identify abnormal findings. Normal gingival tissues are coral pink in color (allowing for normal pigmentation), and have a thin, knife-like edge with a smooth and regular texture. In addition, there should be no demonstrable plaque or calculus on the dentition. Normal sulcal depth in a dog is 0 to 3 mm and in a cat is 0 to 0.5 mm.

The first clinical sign of gingivitis is erythema of the gingiva. This is followed by edema, gingival bleeding during brushing or after chewing hard/rough toys, and halitosis. Gingivitis is typically associated with calculus on the involved dentition, but is primarily elicited by plaque and thus can be seen in the absence of calculus. Alternatively, widespread supragingival calculus may be present with little to no gingivitis. It is critical to remember that calculus itself is essentially nonpathogenic. Therefore, the degree of gingival inflammation should be used to judge the need for professional therapy. As gingivitis progresses to periodontitis, the oral inflammatory changes intensify.

The hallmark clinical feature of established periodontitis is attachment loss. In other words, the periodontal attachment to the tooth migrates apically. As periodontitis progresses, alveolar bone is also lost. On oral exam, there are two different presentations of attachment loss. In some cases, the apical migration results in gingival recession while the sulcal depth remains the same. Consequently, tooth roots become exposed and the disease process is easily identified on conscious exam. In other cases, the gingiva remains at the same height while the area of attachment moves apically, thus creating a periodontal pocket. This form is typically diagnosed only under general anesthesia with a periodontal probe. It is important to note that both presentations of attachment loss can occur in the same patient, as well as the same tooth. As attachment loss progresses, alveolar bone loss continues until tooth exfoliation in most cases. After tooth exfoliation occurs, the area generally returns to an uninfected state, but the bone loss is permanent.
Severe Local Consequences

The most common of these local consequences is an oral-nasal fistula (ONF). ONFs are typically seen in older, small-breed dogs (especially chondrodystrophic breeds); however, they can occur in any breed as well as felines. ONFs are created by the progression of periodontal disease up the palatal surface of the maxillary canines; however, any maxillary tooth is a candidate. This results in a communication between the oral and nasal cavities, creating an infection (sinusitis). Clinical signs include chronic nasal discharge, sneezing, and occasionally anorexia and halitosis. Definitive diagnosis of an oronasal fistula often requires general anesthesia. The diagnosis is made by introducing a periodontal probe into the periodontal space on the palatal surface of the tooth. Interestingly, this condition can occur even when the remainder of the patient’s periodontal tissues is relatively healthy (including other surfaces of the affected tooth). Appropriate treatment of an ONF requires extraction of the tooth and closure of the defect with a mucogingival flap. However, if a deep periodontal pocket is discovered prior to development of a fistula, periodontal surgery with guided tissue regeneration can be performed to save the tooth.

Another potential severe consequence of periodontal disease can be seen in multi-rooted teeth, and is called a class II perio-endo abscess. This occurs when the periodontal loss progresses apically and gains access to the endodontic system, thereby causing endodontic disease via bacterial contamination. The endodontic infection subsequently spreads through the tooth via the common pulp chamber and causes periapical ramifications on the other roots.

This condition is also most common in older small and toy breed dogs; however, this author has personally treated a case in a Labrador Retriever. The most common site for a class-II perio-endo lesion to occur in small-animal patients is the distal root of the mandibular first molars.

The third potential local consequence of severe periodontal disease is a pathologic fracture. These fractures typically occur in the mandible (especially the area of the canines and first molars), due to chronic periodontal loss, which weakens the bone in affected areas. This condition is again, most commonly seen in small breed dogs, mostly because their teeth (especially the mandibular first molar) are larger in proportion to their jaws as in comparison to large-breed dogs. Pathologic fractures occur most commonly as a result of mild trauma, or during dental extraction procedures. However, some dogs have suffered from fractures while simply eating.

Although this is typically considered a disease of older patients, this author has personally treated three cases in dogs less than three years of age.

Pathologic fractures carry a guarded prognosis for several reasons including: lack of remaining bone, low oxygen tension in the area, and difficulty in rigidly fixating the caudal mandible. There are numerous options for fixation, but the use of wires, pins or plates is generally required. Regardless of the method of fixation, the periodontally diseased root(s) must be extracted for healing to occur.

Awareness of the risk of pathologic fractures can help the practitioner to avoid problems in at risk patients during dental procedures. If one root of an affected multi-rooted tooth is periodontally healthy, there is an even greater chance of mandibular fracture due to the increased force needed to extract the healthy root. An alternate form of treatment for these cases is to section the tooth, extract the periodontally diseased root, and perform root canal therapy on the periodontally healthy root. In cases where periodontitis involving a mandibular canine, or first molar, is identified during a routine prophy, it is best to inform the owners of the possibility of a jaw fracture prior to attempting extraction of the offending tooth.

The fourth local consequence of severe periodontal disease results from inflammation close to the orbit which could potentially lead to blindness. The proximity of the tooth root apices of the maxillary molars and fourth premolars, places the delicate optic tissues in jeopardy. In cats (especially brachycephalic), the apices of the maxillary canines lie in this area and can create similar issues.

The fifth local consequence is described in recent studies which have linked chronic periodontal disease to oral cancer. The association in this case is likely due to the chronic inflammatory state that exists with periodontitis.

The final significant local consequence of periodontal disease is chronic osteomyelitis, which is an area of dead, infected bone. Dental disease is the number one cause of oral osteomyelitis. Furthermore,
once an area of bone is necrotic, it does not respond effectively to antibiotic therapy. Therefore, definitive therapy generally requires aggressive surgical debridement.

In some cases, the bacterial infection may also result in a septicemia. In one case treated by this author, the patient had white blood cell counts of > 50,000 on several occasions, which responded transiently to antibiotics. However, each time the antibiotics were discontinued the infection returned. On clinical presentation, the patient had halitosis, but no obvious dental disease other than a small fistulous tract. A dental radiograph revealed significantly mottled bone in the area, which prompted aggressive surgical debridement of the area of necrotic bone. Histopathology was consistent with osteomyelitis, and after a course of clindamycin, the patient did not relapse.

In another case treated by this author, the patient presented with an entire hemimandible which was necrotic secondary to osteomyelitis. In this case, the patient required a complete hemimandibulectomy.

**Severe Systemic Manifestations**

Systemic ramifications of periodontal disease are also well documented. The inflammation of the gingiva and periodontal tissues that allows the body’s defenses to attack the invaders also allows these bacteria to gain access to the body. Recent animal studies suggest the possibility that these bacteria negatively affect the kidneys and liver, leading to decrease in function of these vital organs over time. Furthermore, it has also been suggested that these bacteria can become attached to previously damaged heart valves (IE valvular dysplasias) and cause endocarditis, which in turn can result in intermittent infections, and potentially thromboembolic disease. Other studies have linked oral bacteremias to cerebral and myocardial infarctions and other histological changes. Additional human studies have linked periodontal disease to an increased incidence of chronic respiratory disease (COPD) as well as pneumonia. There are many studies that strongly link periodontal disease to an increase in insulin resistance, resulting in poor control of diabetes mellitus as well as increased severity of diabetic complications (wound healing, microvascular disease). Additionally, it has been shown that diabetes is also a risk factor for periodontal disease. Periodontal disease and diabetes are currently viewed as having a bidirectional interrelationship where one worsens the other.

Finally, it has been proven in animal studies that periodontal disease can elicit an increase in inflammatory lipids, as well as an overall lipidemic state. This is a state of overall body inflammation leading to chronic disease processes and an abnormal immune response.

While some of these studies are not definitive, we know that periodontal disease is an infectious process and that affected patients must deal with these bacteria on a daily basis, which in turn can lead to a state of chronic disease. Therefore, we must learn to view periodontal disease as not just a dental problem that causes bad breath and tooth loss, but as an initiator of more severe systemic consequences. As one author states, “Periodontal disease is clearly an important and potentially life-threatening condition, often underestimated by health professionals and the general public.” Only by thinking in these terms can we fully appreciate the scope of the disease process and discuss the problem with clients so that they can appreciate the depth of the problems their pets face. This information will significantly increase client compliance with homecare and dental prophylaxis, as well as advanced dental procedures.
The basis of periodontal therapy today is plaque control. This is accomplished via two to four components depending on the stage of the disease. These include a thorough dental prophylaxis, periodontal surgery, homecare, and extraction. There are numerous variations and treatment options for therapy. This lecture will cover the basics and touch on the available options.

The cornerstone of periodontal therapy is a thorough dental prophylaxis. This must be performed under general anaesthesia including a properly inflated endotracheal tube. The prophylaxis should include the following steps.

**Step 1: Presurgical Exam and Consultation**
This is often a much neglected step of a professional dental prophylaxis. The veterinarian should perform as complete as possible physical and oral exam. The physical exam (along with preoperative testing) will help ensure anesthetic safety. The oral examination will identify obvious pathology (fractured, intrinsically stained, or mobile teeth; oral masses; and resorptive lesions) as well as allow for a preliminary assessment of periodontal status. The veterinarian can then discuss the various disease processes found and the various available treatment options. Based on the physical findings, the practitioner can create a more accurate estimate (both financial and time). Both of which will decrease problems with over scheduling and client finances during the anesthetic event. This small time investment will improve the experience of everyone involved (veterinarian, technician, receptionist, client, and patient).

**Step 2: Chlorhexidine Lavage**
The oral cavity is a contaminated area and a dental cleaning is a mildly invasive procedure. In this way, it often results in a transient bacteremia. For this reason it is recommended to rinse the mouth with a 0.12% solution of chlorhexidine gluconate prior to commencing the prophylaxis to decrease the bacterial load.

**Step 3: Supragingival Cleaning**
This can be performed via mechanical or hand scaling. The mechanical scalars decrease anaesthetic time and include both sonic and ultrasonic types. The most common type of mechanical scaler in veterinary dentistry today is the ultrasonic scaler. There are two main types magnetostrictive and piezoelectric. Both of these scalers vibrate at approximately 45,000 hertz. They are very efficient and have an additional benefit of creating an antibacterial effect in the coolant spray (cavitation). They can, however, be more damaging to the tooth, and may leave some calculus behind. Thus, it has been recommended that hand scaling be performed after ultrasonic scaling to ensure the complete removal of calculus. Sonic scalers run on compressed air and vibrate at 8–18,000 hertz. They are safer, but slower than sonic scalers and do not offer cavitation. The area of maximum vibration is 1–3 mm from the tip. Do not use the tip or back of the instrument as these are not effective for calculus removal and can potentially damage the tooth. The instrument is placed on the tooth and left on the tooth for up to 15 seconds. Once the instrument loses contact with the tooth, the scaler can no longer be effective. Run the instrument slowly over the tooth surface in wide sweeping motions to cover every mm² of every tooth surface.

Hand scaling is performed with a scaler. This is a triangular instrument with a sharp cutting edge. In addition, the tip is very sharp. Scalers are designed for supragingival use only. The scalers (as well as curettes below) are held with a modified pencil grip. The instrument is gently held at the gnarled or rubberized end with the thumb and index finger tips. The middle finger is placed near the terminal end of the shaft and is used to feel for vibrations which signal residual calculus or diseased/rough tooth/root surface. Finally, the ring and pinkie fingers are rested on a stable surface.

Hand instruments are used with a gentle touch and are run over the tooth numerous times in overlapping strokes until the tooth feels smooth. This step may be performed with a curette and combined with subgingival scaling (see below).
**Step 4: Subgingival Plaque and Calculus Scaling**

This step is best performed by hand with a curette. A curette has 2 cutting edges and a blunted toe and bottom. In this way, it will not cut through the delicate periodontal attachment as long as excess force is not applied. The proper curette is selected based on its angulation. The lower the number (i.e., 1–2) the less the angle and the further rostral in the mouth the instrument is used. The face of the instrument is placed flat against the surface of the tooth and inserted gently to the base of the sulcus or pocket. Once there, the instrument is rotated so that the shaft is parallel to the long axis of the tooth. This will engage the calculus as well as place the instrument in the proper position for root surface and subgingival debridement. This is repeated with numerous overlapping strokes until the root feels smooth. Cleanliness can be further evaluated by gently directing compressed air into the sulcus. Any remaining calculus will appear chalky. This is a very technically demanding procedure and the practitioner is directed to continuing education programs to hone their skills.

Traditional ultrasonic scalers should not be used subgingivally due to thermal damage to the gingiva and pulp. This occurs because the water coolant cannot reach the tip of the instrument. However, sonic and ultrasonic scalers with specialized periodontal tips have been developed for subgingival use. These are much easier to use and therefore will likely result in superior cleaning in the hands of novices. Like supragingival scaling, it is recommended to perform mechanical scaling first to remove the majority of the plaque and calculus first, and then follow up with hand scaling.

**Step 5: Polishing**

Scaling (especially mechanical) leaves the tooth surface (and especially the root) rough, which increases plaque attachment. Polishing will smooth the surface of the teeth which will retard plaque attachment. Polishing is typically performed with a prophy cup on a slow-speed handpiece with a 90-degree angle. The handpiece should be run at a slow rate and no greater than 3,000 RPM. Ensure that adequate polish is used at all times. Running the prophy cup dry is not only inefficient, it may also overheat the tooth. Just like with scaling, every mm² of tooth surface should be polished. In addition, slight pressure should be placed down onto the tooth to flare the edges of the prophy cup so as to polish the subgingival areas. One tooth may be polished for a maximum of five seconds at a time to avoid overheating. The tooth can be further polished after a short break (while other teeth are polished).

**Step 6: Sulcal Lavage**

The cleaning and polishing steps will result in debris such as calculus and prophy paste (some of which is bacteria laden) to accumulate in the gingival sulcus. In some cases there are visible deposits, but in all cases there is microscopic debris. These substances will allow for continued infection and inflammation. Therefore, a gentle lavage of the sulcus is strongly recommended. The lavage is performed with a blunt ended cannula which is placed gently into the sulcus and the solution is injected while slowly moving along the arcades. The typical lavage solution is sterile saline, although some authors favor a 0.12% chlorhexidine solution.

**Step 6 (a): Fluoride Therapy (Optional)**

This is a controversial step with some dentists recommending that it be performed in all cases and some that it never be done. The positive aspects of fluoride include antiplaque and antibacterial activities, hardening tooth structure, and decreases tooth sensitivity. The latter activity is most important in patients with gingival recession and secondary root exposure. When root planing is performed, cementum is removed which may expose underlying dentin. In this case, sensitivity may result from the hydrodynamic theory of tooth sensitivity. Application of fluoride should help decrease this sensitivity.

**Step 7: Periodontal Probing, Oral Evaluation, and Dental Charting**

This is a critical, however, often poorly performed and underappreciated step. The entire oral cavity must be systematically evaluated using both visual and tactile senses. Careful visual examination should be performed during the periodontal evaluation. The periodontal probe should be inserted at six spots around every tooth to identify periodontal pockets. This is performed by gently inserting the probe into the pocket until it stops and then “walking” the instrument around the tooth. The normal sulcal depth in a dog is 0–3mm, and a cat is 0–0.5 mm. All abnormal findings must be recorded on the dental chart.
Dental charting should be performed 4-handed. This means that one person evaluates the mouth and calls out pathology to the assistant who records it on the chart. Using the Modified Triadan System will greatly increase efficiency of this step. Dental charts must be of sufficient size to allow for accurate placement of pathology. The minimum size for an acceptable dental chart is 1/3 of a page; however, veterinary dentists use full-page charts. Samples of these may be downloaded at www.vetdentalrad.com/downloads/index.html. (VIN editor: Original like was modified 3-31-14.)

**Step 8: Dental Radiographs**
Dental radiographs should be performed on any pathology noted on dental exam. This includes any periodontal pocket which is larger than normal, fractured or chipped teeth, masses, swellings, or missing teeth. Dental radiographs are a critical aid in the evaluation of dental pathology. Help is available for any questionable cases at www.vetdentalrad.com.

**Step 9: Treatment Planning**
The practitioner, utilizing all available information (visual, tactile, and radiographic) then decides on appropriate therapy. Additionally, the prudent veterinarian will keep in mind the patient as a whole, the owner’s wishes and willingness to perform homecare, and necessary follow-up. Following the creation of a dental plan for the patient, an estimate is created and the client contacted for consent.

**Step 9 (a): Additional Therapies**
Based on the oral examination and client wishes, any additional therapy is performed. If this is extensive and would result in a long anesthesia or the practitioner to be unduly rushed, rescheduling the remainder of the work is an acceptable alternative. There are numerous possibilities for this (including referral), and the reader is directed to texts or hands-on labs for more information on these procedures. In this issue we will only cover periodontal therapy (see below).

**Step 9 (b): Barrier Sealant (OraVet®)**
A barrier sealant has recently been introduced as a means to decrease plaque and calculus accumulation. This is a waxy sealant that has been proven clinically to decrease plaque and calculus. While it has not been proven to decrease gingivitis and therefore periodontal disease, due to its placement at and below the gingival margin, it should theoretically work. Following a prophylaxis, the teeth are dried and then the product is applied. Following this, the client applies the home version on a weekly basis.

**Step 10: Client Education**
The postsurgical release is an important step in periodontal therapy. Use this opportunity to go over radiographs (and pictures if available) with the client. This will not only reenforce your findings and treatment, it will also allow you to discuss periodontal disease. This discussion should not only include immediate postoperative instructions; but also cover periodontal disease and long-term periodontal care.

**Home Care**
This is a very important part of periodontal therapy. A recent study has shown that periodontal pockets are reinfected within 2 weeks of a prophylaxis if homecare is not performed. Therefore, homecare must be discussed with each client following a prophylaxis.

There are two divisions of homecare active and passive. They both can be effective if performed correctly; however, active homecare is still the gold standard in homecare.

Active homecare consists primarily of tooth brushing. There are various veterinary brushes; however, a soft child’s toothbrush is also effective. There are numerous veterinary toothpastes available. These increase the palatability of the toothbrush, and many add a cleaning aid. Human tooth pastes are generally not recommended. There are also antimicrobial preparations that can be used in certain cases. Technique: use a circular motion with the brush at a 45-degree angle to the gingival margin.

Frequency: once a day would be ideal, as this is required to stay ahead of plaque formation, but for most owners this is unrealistic. Three days a week is considered the minimum frequency for patients in good oral health. If the patient has periodontal disease, daily brushing is necessary. One other option for active homecare is to rinse with a chlorhexidine solution. This has been shown to decrease gingivitis if
done consistently over time. Even though brushing and rinsing greatly improves periodontal health, it does not completely eliminate the need for professional cleanings.

Passive homecare is the other option for minimizing periodontal disease. Since this requires no work by the owner, compliance is more likely. This is especially important since long-term consistency is the most important factor in the effectiveness of dental care. There are currently several diets that decrease tartar and plaque build-up. In addition, tartar-control chews and treats have been developed.

All of these products have been shown to decrease plaque and calculus; however, they are most effective on plaque and tartar on the cusp tips not at gingival margin. Supragingival plaque and calculus is in general nonpathogenic. Of the available products, only two have been clinically proven to decrease gingivitis.

The downfall of all passive homecare products is that the patient is not likely to chew with the entire mouth; therefore, areas will be missed. Passive homecare is most effective on the carnassial and surrounding teeth, where chewing is concentrated. Active homecare, in contrast, is most effective in controlling plaque and calculus on the incisor and canine teeth, likely due to the ease in accessing these teeth. Therefore, a combination of active and passive homecare is likely ideal.

**PERIODONTAL SURGERY**

Any pockets greater than normal for the species are pathologic and in need of therapy. It is important to note that this is a separate procedure from the prophylaxis and the practitioner should be charging for this. Periodontal therapy is aimed at removing the infection from the root surface (plaque, calculus, and granulation tissue) as well as smoothing the diseased root surface. This will allow for reattachment and decrease in pocket depth.

In the canine patient, pockets between 3 and 5 mm which do not have mobility, or other issues, are best treated with closed root planing and subgingival curettage. This step is performed in a similar manner to subgingival scaling above, with a combination of mechanical and hand scaling. This should be meticulously performed in order to achieve as clean a tooth as possible to promote healing.

An additional way to promote reattachment is the instillation of a sustained-release doxycycline product. This has been shown to temporarily locally control the microorganisms as well as decrease inflammation. It is performed by mixing the product according to package directions and then inserting the product into the pocket until it is just over flowing. The product is then wetted, which will harden it, and tapped gently into the sulcus. If some of the product extrudes from the pocket, it should be rewetted and then placed. This should continue until the pocket is full.

Pockets greater than 5 mm require direct visualization of the root surface for effective cleaning. If the tooth is not effectively cleaned, the infectious agents remain along with the plaque and calculus. Visualization is best accomplished via periodontal flap procedures. These procedures are very effective in animal patients. If the clients are interested in salvaging the teeth, periodontal surgery can be performed. These are advanced procedure, but can be learned by general practitioners. However, the reader is encouraged to attend a hands-on wet lab prior to undertaking these surgeries.

**EXTRACTION**

The final modality for the therapy of periodontal disease is extraction. While extreme, it is the only true cure. Without a commitment to homecare or routine professional cleanings, advanced periodontal surgery should likely not be attempted. Depending on the stage of periodontal disease, the involved teeth should be extracted.
Dental Radiology Simplified
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Dental Radiograph Units
Radiographic exposure is controlled by 3 components: kVp (kilovolt peak), mA (milliamperage), and exposure time. kVp controls the “quality” of the X-ray beam. This is the power of each particular X-ray particle which controls the penetration of the beam through tissues.

The quantity of the exposure is controlled by mA and time of exposure. The higher the mA, the more X-rays produced over the time period. Multiply this number by the exposure time and you will get the total number of X-ray units.

Since there is not a significant amount of variation of tissues in oral radiology, the kVp and mA are set constant on dental radiology units. The only variable factor is time. This is measured in seconds or pulses. One pulse is equal to 1/60 of a second. Most standard (human) dental radiology units have a digital control for the exposure, and it is set by the operator based on a technique chart. Recently, however, veterinary-specific machines have become available which have a computer that sets the exposure based on the size of the patient, the speed of dental film used, and the particular object tooth. This can take a lot of the guesswork out of the exposure setting. However, with a little experience and practice, it is easy to figure out a setting.

Dental Radiographic Film
Dental film is nonscreen film. This means that it is directly exposed by the X-ray and does not require an intensifying screen. This gives much more detail than standard radiographic film, but requires a higher amount of exposure. It is packaged in its own paper or plastic sleeve to protect it from light and the oral environment.

There are two types of dental film commonly used in dental radiology. These are Ultra-speed “D” and Ektaspeed “E” film. Recently “F” speed film has become popular. The difference is in the size of the silver halide crystals and secondary to this the amount of exposure required to expose the dental film. “E” speed film requires approximately ½ the amount of radiation for exposure than “D” speed film, and “F” speed even less. This decreases exposure to the patient and staff as well as decreases the wear and tear on the X-ray unit. There is a slight decrease in resolution with faster films due to the larger crystal size, but according to most experts, the difference is negligible. Therefore, it is recommended in human dentistry to use “E” or “F” speed to decrease exposure time. They are more technique-sensitive, however, in both the exposure and development of the image. This may be frustrating for the novice; therefore, it is generally recommended that practitioners start with “D” speed and advance to “E” or “F” speed when they are more comfortable with the settings and positioning.

There are several different sizes of dental film available (4, 3, 2, 1, and 0). The most common sizes used in veterinary medicine are 4, 2, and 1. Size 3 are bite wings and are generally not used in veterinary medicine. Size 4 (occlusal) film is the largest available; it is used mostly in large-breed dogs or when taking whole-mouth radiographs. For small dogs and cats and most any single-tooth radiograph, size 2 (standard) is commonly used. For the mandibular first and second premolars and very small cats and puppies, size 1 (or 0) (periapical) are used.

Another consideration in selecting film size is cost. Size 4 film is about 3 times the cost of size 2. Therefore, if you can use a size 2, it is recommended. However, it is much easier to position size 4 films, allowing for much more latitude in positioning. This will result in less retakes. Therefore, the less experienced may consider practicing with size 4 film and graduating to size 2 when a level of skill is obtained.

Digital Dental Radiology
There are numerous human veterinary digital systems. These are excellent means of obtaining dental radiographs. The only major problem currently is the lack of a number 4 sensor. The major advantages to these systems are the decrease in radiation exposure, rapidity of the development, and that you can
reposition the sensor if the view is not correct the first time. There is one company, however, which makes a size 4 phosphor plate (CR).

**TAKING A DENTAL RADIOGRAPH**

**Step 1: Patient Positioning**
Position the patient so that the area of interest is convenient to the radiographic beam. In general this is where the object is “up.” For maxillary teeth, the patient should be in ventral recumbency. For mandibular canines and incisors the pet should be in dorsal recumbency. Finally, for maxillary cheek teeth, the patient should be in lateral recumbency with the affected side up. This being said, in our practice virtually all radiographs are exposed in lateral recumbency. This takes some getting used to, but decreases the number of times a patient must be rolled when doing surgical or endodontic procedures.

**Step 2: Film Placement Within the Patient’s Mouth**
There is an embossed dot on the film. The convex side of this should be placed towards the X-ray beam. In most films, this side is pure white. The opposite or “back” side of the film will usually be colored (purple or green). Place the film in the mouth so that the entire tooth (crown and entire root surface) is covered by the radiograph. Remember, the roots of all teeth are very long. This is especially true of canine teeth, which are longer than you think. Always err on the side of having the film too far in the mouth to ensure you do not cut off the root apexes. The film should be placed as near as possible to the object (generally touching the tooth and gingiva) to minimize distortion.

**Step 3: Positioning the Beam Head**
There are two major techniques for positioning the beam head in veterinary patients. Both of these techniques are used daily in veterinary practice.

**Parallel Technique**
This is where the film is placed parallel to the object being radiographed and perpendicular to the beam. This is how standard (large) films are taken. This gives the most accurate image. Unfortunately, this is only useful in the lower cheek teeth in the dog and cat. This is due to the fact that these patients don’t have an arched palate. The film cannot be placed parallel to the tooth roots because of the palate’s interference. Therefore, this technique is not always possible.

**Bisecting Angle Technique**
This is the most common type of dental radiograph taken in veterinary patients. This uses the theory of equilateral triangles to create an image that accurately represents the tooth in question. To utilize this technique, the film is placed as parallel as possible to the tooth root. Then the angle between the tooth root and film is measured. This angle is cut in half (bisected) and the beam placed perpendicular to this angle. This gives the most accurate representation of the root.

If this angle is incorrect, the radiographic image will be distorted. This is because the X-ray beam will create an image that is longer or shorter than the object imaged. The best way to visualize this is to think of a building and the sun. The building will create a 90-degree (right) angle to the ground. The bisecting angle in this case is 45 degrees to the ground.

Early and late in the day, the sun is at an acute angle to the building and casts a long shadow. In radiology this occurs when the angle of the beam to the object is too small and is known as elongation. At some point in the late morning and early afternoon, the sun is at a 45-degree angle to the building, which is the bisecting angle. This gives an accurate representation of the building height. As the sun continues up in the sky, the shadow shortens. This occurs in veterinary radiology when the angle is too great and is known as foreshortening. Finally, at noon, the sun is straight up from the building, which gives no shadow.

**Simplified Technique**
The “simplified technique” as developed by Dr. Tony Woodward does not utilize direct measurement of any angle, instead relying on approximate angles to create diagnostic images. There are only 3 angles used for all radiographs in this system: 20, 45, and 90.
Mandibular premolars and molars are exposed at a 90-degree angle, maxillary premolars and molars at a 45-degree angle, and incisors and canines at a 20-degree angle.

To initiate any radiograph, place the film in the mouth and set the positioning indication device (PID) perpendicular to the film. For mandibular cheek teeth, this is the correct placement. For the maxillary premolars and molars, rotate the beam to a 45-degree angle. For the incisors and mandibular canines, rotate 20 degrees. For the maxillary canines an additional rotation 20 degrees lateral is necessary to avoid superimposition of the first and second premolars.

**Step 4: Setting the Exposure**

If you are using a machine where you set the exposure manually, you will need to set up a technique chart similar to one for a standard (large) unit. The good news is that there is only one variable that needs to be adjusted.

If you are utilizing the computer-controlled system, set the buttons for the species, size of the patient, and tooth to be imaged. If you have correctly set the machine and the image is incorrectly exposed, the easiest way to adjust is to change the \( f \) setting. By pressing this button, you will see the numbers go up on both sides. The one on the left is the \( f \) number and the one on the right is the exposure time. If you continue to press the button it will continue to increase the exposure until you reach 9 when it will markedly lower and the \( f \) number will go back to 1. If the radiograph is overexposed (too dark), lower the \( f \) number by 1. If it is underexposed (too light), increase the number by 1. Continue this process until you have the film that you want. Generally, the \( f \) number will be the same for all radiographs once you have discovered the correct setting for your machine; start at that number in future sessions.

**Step 5: Exposing the Radiograph**

Dental radiograph machines have a handheld switch to expose the radiograph. If it is possible, leave the room prior to exposing the radiograph. If it is not, stand at least 6 feet away at a 90- to 130-degree angle to the primary beam (meaning to the side or back of the tube head, not in front or behind). Once everything is set, press the button. It is important to remember that these switches are “dead man’s”. This means if you let up during the exposure, it will stop the production of X-ray beams. On a standard unit, this will make a light radiograph; on a computer-controlled one, it will give an error message and you will need to start over. Make sure you hold the button down until the machine stops beeping.

**Step 6: Developing the Radiograph**

The most economical way to develop the radiograph is coffee cups filled with dental developing solutions in your darkroom. *(Using chemicals other than products for dental radiology will result in inferior film quality.)*

Although developing films in a darkroom can produce quality films, the use of a chairside developer has several distinct advantages:

1. The chairside developer also allows you to easily judge when development time is correct and be able to evaluate your films in only 1–2 minutes.
2. The technician does not leave the room and can still monitor the patient.
3. The units take up very little space, minimize chemistry use, clean up easily, and store quickly.

To develop films, begin by peeling back the covering layers from the film, taking care to handle the film only by the edges. Use a film clip to grasp the corner of the film and place it in the developer. When developing a size 4 film, make sure to immerse the entire film in the liquid to ensure that the whole film gets developed. Develop the film until an image is just visible (sight developing). Then rinse the film briefly in a water bath, and place the film in the fixer for one minute until partially fixed. The film may be evaluated at this time, but should be placed back in the fixer for an additional 10 minutes to ensure complete fixation (archival quality). When completely fixed, the film becomes clear and will lose all traces of a greenish color. The film should then be thoroughly rinsed in running water or placed in a clean water bath for 10–15 minutes. This is followed by a final rinse to remove all traces of fixer. Be sure to remove the clip and rinse all film surfaces thoroughly. Traces of fixer remaining on a dental film give it a characteristic “slick” feel; therefore, rinse the film under running water while gently rubbing the film
between your fingers for a few seconds until the film does not feel slick. The film is then placed in drying clips overnight to dry. Make sure to dry the film completely to ensure that they do not stick together.

Be sure to change the solutions whenever the developing and fixation times seem to be slowing down. This will occur after you have developed and fixed around 20 smaller (# 0 or # 2) films, or 10–15 larger (# 4) films. Use of exhausted chemistry results in poor image quality and hazy images.
Dental Radiograph Interpretation
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Interpreting dental radiographs can be daunting, but it is very similar to interpreting a standard bony radiograph. The major difference is that dental radiographic changes are often more subtle. In addition, there are pathologic states that are unique to the oral cavity. Finally, there are several normal anatomic structures that may mimic pathologic changes.

This lecture concentrates on the most common pathologies, which are illustrated by classic examples. Note that in practice, these lesions are often less obvious. The reader is directed to additional continuing education meetings to further their expertise. In addition, www.vetdentalrad.com is an excellent resource for questionable cases.

Determining Which Teeth Were Imaged
The first step in radiographic interpretation is determining which teeth have been imaged. This requires a firm knowledge of oral anatomy as well as the architecture of dental films. Digital systems with veterinary templates do not require this step as long as the images are properly placed (do not assume this was done correctly). If your system does not support a veterinary template, there is a mark on the image which is in a consistent location. Review the owner's manual for instructions on its use.

The key to properly identifying the imaged teeth is the embossed dot, which is on one corner of the film. When exposing a radiograph, if the film is properly positioned, the convex surface will point towards the radiographic tube head. There is no way to expose a diagnostic radiograph with the film in backwards, due to the lead sheet on the back side of the film. Therefore, when interpreting the film, the embossed dot is facing out of the mouth.

First, place the dot towards you (this is done for you on most digital systems). This means you are looking at the teeth as if you are the beam.

Next, rotate the film so that the roots are in their natural position (up on maxillary and down on mandibular).

Canines and incisors: this orients the film so the right side of the mouth is on the left, and right side is on the left. This is like a VD abdomen radiograph.

Molars and premolars: ascertain mesial from distal. If the mesial side is on the left side of the film, it is a radiograph of the left side of the patient and vice versa for the right.

Normal Radiographic Anatomy
There are numerous structures within the oral cavity that mimic pathologic states depending on the projection. Knowledge of normal radiographic anatomy will help avoid over interpretation.

Normal alveolar bone will appear gray and relatively uniform throughout the arcade. It is slightly more radiopaque, “darker”, than tooth roots. In addition, it appears slightly, but regularly mottled. Alveolar bone should completely fill the area between the roots (furcation) and end at the cementoenamel junction (CEJ). The root canals should all be the same width; allowing for differences in the diameters of the root. There should be no radiolucent areas in teeth or bone. A regular thin dark line (periodontal ligament) should be visualized around the roots.

There are several normal anatomic findings that are commonly misinterpreted in dental images as pathologic. On radiographs of the mandibular cheek teeth, a thick, horizontal radiolucent line courses parallel to and just coronal to the ventral cortex of the mandible. This is the mandibular canal. In addition, there are three circular radiolucent areas seen in the area of the apices of the first three premolars, which are the mental foramina (rostral, middle, and caudal). On rostral mandibular views, a radiolucent line will be present between the central incisors. This is the fibrocartilaginous mandibular symphysis. In the rostral maxillary area: there are paired radiolucent areas distal to the intermediate incisors, which are the palatine fissures. Finally, a significant widening of the periodontal ligament at the apex of the cuspid teeth is normal. This may appear to be a periapical lesion, but is differentiated from pathology because it is very regular and V-shaped, as opposed to irregular and round.
Any questionable areas should be evaluated by exposing a comparative view.

A suspicious periapical lucency (especially in the area of the mandibular premolars) should be evaluated with an additional film exposed at a slightly different angle (in the horizontal or vertical plane). If the lucency is still centered on the apex, it is likely real. If the lesion moves off the apex or disappears, it is an artifact. Suspect changes in the diameter of the root canal of a tooth should be compared against surrounding as well as contralateral teeth. Surrounding teeth can be seen on the same film with the “lesion.” The contralateral view should be taken at the same angle as the original. It is important to note that root canals are not exact cylinders (especially cuspids). A lateral view may have a much different canal width than a V/D view.

PERIODONTAL DISEASE
Periodontal bone loss results from the combination of bacterial-induced inflammation and host response creating osteoclastic resorption of bone. This resorption will result in crestal bone loss to a level below the cementoenamel junction. This decrease in bone height may also create furcation exposure. Horizontal bone loss is the most common pattern in veterinary patients is horizontal. This appears as generalized bone loss of a similar level across all or part of an arcade. The other pattern is angular (vertical) bone loss. The radiographic appearance of angular bone loss is one area of recession below the surrounding bone. The surrounding bone may be normal or be undergoing horizontal bone loss. Therefore, it is common to have a combination of the two types in the same arcade.

Bone loss does not become radiographically evident until 30–50% of the mineralization is lost. Therefore, radiographic findings will always underestimate bone loss. In addition, bone loss only on the surface (i.e., lingual, palatal, or facial) may be hidden by superimposition of bone or tooth. This may result in a nondiagnosed bony pocket. Always interpret radiographs in light of the complete oral examination findings.

ENDODONTIC DISEASE
Endodontic disease may be demonstrated radiographically in several ways. An individual tooth may have one, some, or all of the different changes listed below. However, only one need be present to establish a presumptive diagnosis of endodontic disease. Radiographic changes can be broken into two major classifications: 1) changes in the surrounding bone or 2) changes within the tooth itself.

Bony Changes
The classic and most obvious finding is periradicular rarefaction. This appears as a radiolucent area surrounding the apex of a root. On rare occasions, this may also be seen mid-root, but these will virtually always be associated with periapical disease. Other, more subtle changes include a widened periodontal ligament, a thickened or discontinuous lamina dura, or even periradicular opacities. It is important to be aware of superimposed lucencies which are artifactual. These structures (i.e., mental foramina) can be imaged over an apex and falsely appear as osseous rarefaction. There are several clues that superimposed lucencies are artifactual. First, superimposed artifacts are typically seen on only one root, whereas it is very rare to find a true periapical lesion on only one root of a multirooted tooth. In addition, artifacts tend to be regular in appearance, whereas true periapical lesions are ragged.

If any area is in question, it is best to expose an additional film with a slightly different angle. If a periradicular lucency is still centered over the apex, it is likely real and not an artifact.

Tooth Changes
The most common change in endodontic disease within the tooth itself is a root canal with a different diameter. As a tooth matures, secondary dentin production will cause a decrease in canal width. When a tooth becomes nonvital, this development stops secondary to the death of the odontoblasts. Consequently, nonvital teeth have wider root canals than the surrounding vital teeth. Conversely, on rare occasions, pulpitis may result in increased dentin production, and create an endodontically diseased tooth with a smaller root canal. This is especially common in teeth that are also periodontally diseased. This could potentially lead to a misdiagnosis of the endodontically diseased tooth as healthy and vice
versa with the contralateral tooth. Hence, it is important to evaluate the adjacent teeth as well as the contralateral.

Width discrepancy can be compared to any tooth (taking the size of tooth into consideration), but it is most accurate is to compare to the contralateral tooth.

Endodontic disease may also be manifested radiographically as internal resorption. This results from osteoclastic activity within the root-canal system due to pulpitis. These changes create an irregular, enlarged region within an area of the root-canal system. Finally, external root resorption can be seen with endodontic disease. It will appear as a defect of the external surface of the root, generally accompanied by a loss of bone in the area. External resorption most commonly occurs at the apex in companion animals and is quite common in cats with chronic endodontic disease.

**Feline Tooth Resorption (TRs)**

TRs are the result of odontoclastic destruction of feline teeth, and are classified as either type 1 or type 2. In type 1 there is no replacement by bone, whereas in type 2 there is replacement of the lost root structure by bone.

TRs are very common in our feline patients. Studies have reported up to a 70% incidence in felines over 6 years of age! The etiology at this point is unknown. They are not bacterial in nature, although in some cases the inflammation which activated the odontoclasts may have been bacterial in nature. There are numerous theories; however, none have been proven at this time. Osteoclastic resorption will generally begin at the cervical line of the tooth and progress at varying rates until in some cases no identifiable tooth remains.

Type 1 TRs are typically associated with inflammation such as gingivostomatitis or periodontal disease. Thus, they are commonly associated with periodontal bone loss on dental radiographs. In these cases, it is believed that the soft-tissue inflammation activated the osteoclasts. The teeth will have normal root density in some areas and a well-defined periodontal space. In addition, there is often a definable root canal in the intact part of the tooth. This type will have significant resorption of the teeth and tooth roots that is not replaced by bone.

Type 2 TRs are usually associated with only localized gingivitis on oral exam, in contrast to the more severe inflammation due to periodontal disease or gingivostomatitis seen with type 1. In these cases, the gingival inflammation is secondary to the TR. The radiographic appearance is that of teeth which have a different radiographic density as compared to normal teeth, as they have undergone significant replacement resorption. Findings will include areas with no discernable periodontal ligament space (dentoalveolar ankylosis) or root canal. In the late stages, there will be little to no discernable root structure (ghost roots). In these cases, the lost root structure will be replaced by bone.

The importance of dental radiography in TR cases cannot be overstated. Type 1 lesions typically retain a viable, root-canal system, and will result in pain and endodontic infection if the roots are not completely extracted. However, the concurrent presence of a normal periodontal ligament makes these extractions routine. With type 2 lesions, there are areas lacking a normal periodontal ligament (ankylosis) which also demonstrate varying degrees of root resorption, which makes extraction by conventional elevation difficult to impossible. The continued resorption in type 2 teeth is the basis for crown amputation therapy. It is this author’s opinion that teeth with an identifiable root canal on dental radiographs must be extracted completely, while teeth with no discernable root canal may be treated with crown amputation. If there is any question, always err on the side of complete extraction.

**Neoplasia**

Neoplasia is defined as the abnormal growth of cells that is not responsive to normal growth control. Neoplasms can be further classified by their biologic behavior as benign or malignant.

**Benign Masses**

Most benign neoplastic growths will have no bony involvement on dental radiographs. If bone involvement does occur with a benign growth it will be expansive, resulting in the bone “pulling away”
from the advancing tumour leaving a decalcified soft tissue filled space in the tumour site. Bony margins are usually distinct. Finally, this expansive growth will typically result in tooth movement.

**Cysts**
Cystic structures will appear as a radiolucent area with smooth bony edges. Similar to other benign growths, they grow by expansion and thus displace the other structures (e.g., teeth). Dentigerous cysts are typically seen as a radiolucent structure centered on the crown of an unerupted tooth.

**Malignant Neoplasia**
Malignant oral neoplasms typically invade bone early in the course of disease, resulting in irregular, ragged bone destruction. Initially, the bone will have a mottled “moth-eaten” appearance, but radiographs late in the disease course will reveal a complete loss of bone (the teeth will appear to float in space). If the cortex is involved, an irregular periosteal reaction will be seen.

Histopathologic testing is always necessary for accurate diagnosis of oral masses since a variety of benign, or malignant, tumors appear radiographically similar. In addition, osteomyelitis can create the same radiographic findings as malignant tumours. Finally, aggressive tumours will show no bone involvement early in the course of disease. The prudent practitioner will note the type and extent of bony involvement (if any) on the histopathology request form (and may include copies of the radiographs and pictures) to aid the pathologist. It is key to interpret the histopathology result in light of the radiographic findings. A diagnosis of a malignancy without bony involvement should be questioned prior to initiating definitive therapy such as aggressive surgery, radiation therapy, or chemotherapy. Conversely, a benign tumor diagnosis with significant bony reaction should be further investigated prior to assuming that the patient is safe.

Additional diagnostic tests in questionable cases include complete blood panel, urinalysis, bacterial and/or fungal culture, as well as fungal serology.

**Retained Tooth Roots**
Persistent tooth roots following extraction attempts are a common occurrence in veterinary medicine. In the vast majority of cases, there are no outward clinical signs; however, the patient suffers regardless. In rare cases, the retained root may abscess, resulting in significant morbidity to the patient and possible legal action from the client.

Dental radiographs must be exposed following all extractions. Regardless of the appearance of complete extraction, there is still a possibility of retained roots or other pathology. Therefore, postoperative radiographs are critical in all cases. In addition, they will serve as a legal document in cases of complications.
**Dental Extractions Made Easier**
Brook A. Niemiec, DVM, DAVDC, Fellow Academy of Veterinary Dentistry
Southern California Veterinary Dental Specialties, CA, USA

**STEP 1: OBTAIN CONSENT**
Never extract teeth without owner consent (preferably written), no matter how bad the problem, or how obvious the decision is. Make sure that you have a valid daytime number (or numbers) for the client and inform them they must be available during surgery hours. Consider loaning pagers to clients for the day, as this author has found this to be a very effective means to contact clients. If the client cannot be reached and prior consent was not obtained, do not pull the tooth. Document the problem, recover the patient, and reschedule the work. Remember, the tooth can always be extracted later, but it cannot be put back in!

**STEP 2: DENTAL RADIOGRAPHS**
Dental radiographs should be exposed on all teeth prior to extraction. Dental radiographs are invaluable resources for the practitioner. Radiographs allow the practitioner to determine the amount of disease present, any root abnormalities, or ankylosis. Help with radiographic interpretation is available while the patient is under anesthesia at www.vetdentalrad.com. In addition, the radiographs will serve as evidence for the extraction in the medical record. Radiographs should also be exposed postextraction to document complete removal of the tooth.

**STEP 3: OBTAIN PROPER VISIBILITY AND ACCESSIBILITY**
The patient should be positioned in such a way as to allow maximum visibility of the area as well as make the surgeon most comfortable. Note that during the extraction procedure the ideal position may change and the patient should be adjusted appropriately. The lighting should be bright and focusable on the surgical field. Suction, air/water syringes, and gauze should be utilized continually to keep the surgical field clear, and mouth gags can be used to hold the mouth in proper position for surgery. Finally, magnification may help the surgeon locate furcations or retained root tips.

**STEP 4: PAIN MANAGEMENT**
Extractions are surgical procedures and are moderately to severely painful for the patient. Depending on patient health, a multimodal approach (combination of opioids, NSAIDs, local anesthetics, and dissociative) should be employed, as this provides superior analgesia. Preemptive analgesia is proven to be more effective than postoperative, and it is therefore important to administer the drugs before the painful procedure.

**SINGLE-ROOT EXTRCTIONS**

**STEP 5: INCISE THE GINGIVAL ATTACHMENT**
This is accomplished with a scalpel blade (number 11 or 15), elevator, or luxator. The selected instrument is placed into the gingival sulcus with the tip of the blade angled toward the tooth (this will help avoid going outside the bone and creating a defect or cutting through the gingiva). The blade is then advanced apically to the level of the alveolar bone, and the instrument is carefully worked around the entire tooth circumference.

This step is very helpful as the gingival attachment contributes approximately 15% of the retentive strength of the periodontal apparatus. More importantly, however, this procedure will keep the gingiva from tearing during the extraction procedure. This is most important with mobile teeth where little elevation is needed, but one edge is still attached. Gingival tearing can cause defects that require closure or can make a planned closure more difficult.

**STEP 6: ELEVATE THE TOOTH**
Elevation is the most dangerous step in the extraction procedure. Remember that you are holding a sharp surgical instrument and working in an area of numerous critical and delicate structures. There have been
many reports of eyes that have been gouged and lost by extraction instruments as well as at least one confirmed fatality due to an elevator puncturing a patient’s brain. The index finger is placed near the tip of the instrument to avoid causing iatrogenic trauma in the event of instrument slippage or encountering diseased bone. In addition, the jaw should be gently held with the opposite hand to provide stability and avoid mandibular fracture.

First, select an instrument which matches the curvature and size of the root. There are numerous instruments available including the classic elevator, the luxating elevator, and the winged elevators. Classic elevators and winged elevators are used in an “insert and twist” motion to tear the periodontal ligament, whereas luxators are used in a rocking motion during insertion to fatigue as well as cut the periodontal ligament. Luxators can be gently twisted for elevation, but they are not designed for this and can be easily damaged when used in this manner.

Elevation is initiated by inserting the elevator, or luxator, firmly yet gently into the periodontal space. The insertion should be performed while keeping the instrument at about a 10- to 20-degree angle toward the tooth, to avoid slippage. Once in the space between the bone and the tooth, the instrument is gently twisted with two-finger pressure. This is not to say that the instrument should be held with two fingers, rather the entire hand should be used to hold the instrument. Twist only with the force that you could generate when holding with two fingers. Hold the position for 10–30 seconds to fatigue and tear the periodontal ligament.

It is important to note that the periodontal ligament is very effective in resisting intense, short forces. It is only by the exertion of prolonged force (i.e., 10–30 seconds) that the ligament will become weakened. Heavy stresses only serve to put pressure on the alveolar bone and tooth which can result in the fracture of one of these structures, so it is important not to use too much force.

After holding for 10 to 30 seconds, reposition the instrument about 1/8 of the way around the tooth and repeat the above step. Continue this procedure 360 degrees around the tooth, each time moving the elevator apically as much as possible. Depending on the level of disease and the size of the tooth, a few to several rotations of the tooth may be necessary.

The key point to successful elevation is patience. Only by slow, consistent elevation will the root loosen without breaking. It is always easier to extract an intact root than to remove fractured root tips.

**STEP 7: Extract the Tooth**
Removing the tooth should only be attempted after the tooth is very mobile and loose. This is accomplished by grasping the tooth with the extraction forceps and gently pulling the tooth from the socket. Do not apply undue pressure as this may result in root fracture. In many cases, especially with premolars, the roots are round in shape and will respond favorably to gentle twisting and holding of the tooth while applying traction. This should not be performed if there are root abnormalities (significant curves, weakening) seen on the preoperative radiograph.

It is helpful to think of the extraction forceps as an extension of your fingers. Undue pressure should not be applied. If the tooth does not come out easily, more elevation is necessary. Start elevation again until the tooth is loose enough to be easily removed from the alveolus.

**STEP 8: Alveoloplasty**
This step is performed to remove diseased tissue, or bone, as well as rough boney edges that could irritate the gingiva and delay healing. Diseased tissue can be removed by hand with a curette. Bone removal and smoothing is best performed with a carbide, or preferably, a coarse-diamond bur on a water-cooled, high-speed, air-driven handpiece. Alternatively, rongeurs, or bone files, may be used if a high-speed dental unit is unavailable. Next, the alveolus should be gently flushed with a 0.12% chlorhexidine solution to decrease bacterial contamination. After the alveolus is cleaned, it may be packed with an osteopromotive substance.

**STEP 9: Closure of the Extraction Site**
This is a controversial subject among veterinary dentists, and thus some texts recommend suturing only in large extractions, other authors (including this one) recommend suturing almost all extraction sites.
Closure of the extraction site promotes hemostasis and improves postoperative discomfort and aesthetics. It is always indicated in cases of larger teeth (e.g., canines, carnassials), or any time that a gingival flap is created to allow for easier extraction. This is best accomplished with size 3/0 to 5/0 absorbable sutures on a reverse cutting needle. Closure is performed with a simple interrupted pattern with sutures placed 2 to 3 mm apart. It is further recommended to utilize one additional throw over manufacturer’s recommendations to counteract tongue action.

In regards to flap closure, there are several key points associated with successful healing. The first and most important is that there must be no tension on the incision line. If there is any tension on the suture line, it will not heal. Tension can be removed by extending the gingival incision along the arcade (called an envelope flap) or by creating vertical releasing incisions and fenestrating the periosteum. The periosteum is a very thin fibrous tissue which attaches the buccal mucosa to the underlying bone. Since it is fibrotic, it is inflexible and will interfere with the ability to close the defect without tension. The buccal mucosa is very flexible and therefore will stretch to cover large defects. If there is no tension, the flap should stay in position without sutures.

If at all possible, the suture line should not be made over a void. If sufficient tissue is present, consider removing some on the attached side to make the suture line over bone. Always suture from the unattached (flap side) to the attached tissue, because this avoids tearing the flap as the needle dulls. Finally, ensure that all tissue edges have been thoroughly debrided as intact epithelial tissues will not heal.

**Extraction of Multirooted Teeth**

Section all multirooted teeth into single-rooted pieces. The roots of almost all multirooted teeth are divergent and this will cause the root tips to break off if extractions are attempted in one piece. Root fracture can occur even if a tooth is relatively mobile to start with. With mobile teeth, the sectioning step alone often allows for simple extraction.

The best tool for sectioning teeth is a bur on a high-speed, air-driven handpiece. Besides being the quickest and most efficient tool for the job, it also has air and water coolant that will avoid overheating the tooth. Many different styles of burs are available; however, this author prefers a cross-cut, taper-fissure bur (699 for cats and small dogs, 701 for medium dogs, and 702 for large breeds).

The best way to section the teeth is to start at the furcation and work towards the crown of the tooth. This method is used for two major reasons. First, it avoids the possibility of missing the furcation and cutting down into a root, which subsequently weakens the root and increases the risk of root fracture. In addition, this method avoids the possibility of cutting through the tooth and inadvertently damaging the gingiva or alveolar bone.

After the tooth has been properly sectioned, follow the above steps for each single rooted piece. In some cases, the individual tooth pieces can be carefully elevated against each other to gain purchase.

**Surgical Extractions**
The more difficult extractions are best performed via a surgical approach. This includes canine and carnassial (maxillary fourth premolar and mandibular first molar) teeth, as well as teeth with root malformations or pathology, and finally retained roots. A surgical approach allows the practitioner to remove a small amount of buccal cortical bone, promoting an easier extraction process.

A surgical extraction is initiated by creating a gingival flap. This can be a horizontal flap along the arcade (an envelope flap) or a flap with vertical releasing incisions (a full flap). An envelope flap is created by releasing the gingival attachment with a periosteal elevator along the arcade including one to several teeth on either side of the tooth, or teeth, to be extracted. The gingiva along the arcade is released to or below the level of the mucogingival junction (MGJ) and the flap is connected by incising the gingiva in the interdental spaces. The advantage to this flap is that the blood supply is not interrupted and there is less suturing.

The more commonly used flap includes one or more vertical releasing incisions. This method allows for a much larger flap to be created, which (if handled properly) will increase the defects which can be covered. The vertical incisions are created at the line angle of the target tooth, or one tooth mesial and
distal to the target tooth. The incisions should be made slightly apically divergent (wider at the base than at the gingival margin). Furthermore, it is important that the incisions be created full thickness, in one motion (rather than slow and choppy). A full-thickness incision is created by incising all the way to the bone, and the periosteum is thus kept with the flap. Once created, the entire flap is gently reflected with a periosteal elevator. Care must be taken not to tear the flap, especially at the mucogingival junction.

Following the flap elevation, a small amount of buccal bone should be removed (approximately 1/3 to 1/2 of the root length depending on the situation) to the depth of the root. This should only be performed on the buccal side. Next, the teeth should be sectioned if multirooted and the teeth then extracted as described above. After the roots are removed, the alveolar bone should be smoothed and the defect closed.

Closure is initiated with a procedure called fenestrating the periosteum. The periosteum is a very thin fibrous tissue which attaches the buccal mucosa to the underlying bone. Since the periosteum is fibrotic, it is inflexible and will interfere with the ability to close the defect without tension. The buccal mucosa, however, is very flexible and will stretch to cover large defects. Consequently, incising the periosteum takes advantage of this attribute. The fenestration should be performed at the base of the flap, and must be very shallow as the periosteum is very thin. This step requires careful attention, as to not cut through or cut off the entire flap.

After fenestration, the flap should stay in desired position without sutures. If this is not the case, then tension is still present and further release is necessary prior to closure. Once the release is accomplished, the flap is sutured normally.
INTRODUCTION
The discipline of human physiotherapy is a relatively recent pursuit. Physiotherapy was first recognized as being important in the early 1900s focusing on treatment of individuals suffering from polio. It became even more important following World War 1 as those returning from war with injuries were required to return to the work force. Formal physiotherapy training began in the United States in 1918. Interest in canine rehabilitation came much later starting initially in the 1980s throughout the 1990s and resulting in the formation of the Animal Physical Therapy Association (APTA). The current practice now draws from the knowledge and skills of veterinarians, veterinary technicians, human physiotherapists, and physiotherapy assistants. Formal training and accreditation is now available for veterinarians and veterinary technicians, and advanced specialty training is now available with the newly formed American College of Veterinary Sports Medicine and Rehabilitation (ACVSMR). These developments will move the discipline forward to better treat our veterinary patients and make rehabilitation an important option just like prescribing a medication or performing surgery.

WHY IS REHABILITATION IMPORTANT?
Our veterinary patients are no different than the human population. They experience injury and degenerative processes that result in changes in structure and function in the body. Our patients undergo surgery for fractures, ligament ruptures, neurological conditions, and soft-tissue excisions. They also are affected by nonsurgical, soft-tissue injuries and conditions, such as obesity and osteoarthritis. Many of these conditions are devastating and complete recovery may not be possible, but rehabilitation can help to improve day-to-day function and improve quality of life.

Benefits of Rehabilitation
The following are perceived benefits of rehabilitation:
- Increased speed of recovery following injury or surgery
- Improvement in quality of movement and performance
- Increased strength and endurance
- Improvement in flexibility and biomechanics
- Reduction in the incidence of future injury both due to restoration of normal biomechanics and through client/owner education
- Reduced pain
- Positive psychological effect for both the pet and for the owner
- Minimally invasive approach
- Minimal complications if administered properly

Where Rehabilitation Is Beneficial
Orthopedic conditions:
- Young dogs - developmental orthopedic diseases
- Mature dogs - osteoarthritis
- Hip and elbow dysplasia - medical and postsurgical management
- Postoperative-fracture patients
- Stifle surgery - cruciate and patella conditions
- Osteoarthritis

Neurological conditions:
- Intervertebral disc disease
- Degenerative myelopathy
- Lumbosacral instability
- Peripheral nerve disorders - radial and sciatic nerve trauma

Trauma:
- Fractures - articular, physeal, long bones
- Tendon, ligament, joint capsule injuries
- Muscular injuries

Skin:
- Reconstructive surgery following tumour excision
- Burn victims

Wellness:
- Obesity and weight-loss management

**Rehabilitation Techniques and Modalities**
Current modalities used in rehabilitation include:
- Thermal modalities - cryotherapy and heat
- Therapeutic exercises - passive range of motion, strengthening, hydrotherapy
- Massage
- Therapeutic ultrasound
- Electrical stimulation
- Laser therapy
- Shockwave therapy

**How Can We Achieve Success Using Rehabilitation?**
The pursuit of rehabilitation in our veterinary practice has potential to improve the outcome of both surgical and nonsurgical conditions. By accepting and incorporating rehabilitation techniques in our treatment protocols we can reduce pain in our patients, improve patient quality of life, increase owner satisfaction, and the owner’s enjoyment of their pet. In order to do this we must have a thorough knowledge of medical conditions and appropriate treatments as well as what can be expected in terms of the response of tissues to injury and response of tissue to disuse and remobilization. Rehabilitation is not a panacea that cannot be expected to replace other important forms of treatment. A diagnosis is essential before designing a suitable rehabilitation protocol.

The following key points for success must be kept in mind:
- Get a diagnosis.
- Use rehabilitation as an adjunct therapy in your pain-management protocols.
- Stay positive and be creative
- Reevaluate - if something is not working for a particular patient modify your approach. “Cookbook” approaches often will result in disappointment. Recognize that all patients are individuals.
- Follow-up with all aspects of treatment.
- Do it - when used appropriately there are no serious side effects to rehabilitation.
INTRODUCTION
Osteoarthritis (OA) can be described simply as a deterioration of a joint associated with pain and dysfunction. In reality OA is a much more complex condition with biochemical, physical, and pathological alterations. All of these factors require consideration when approaching treatment. Traditionally the treatment approach has been to palliate painful symptoms. In fact, the pathologic changes including bone and soft-tissue alterations that result in lameness and clinical signs may not coincide with the degree of pathologic or radiographic change. This makes utilization of multimodal techniques essential for the treatment of OA.

MULTIMODAL THERAPY
Multimodal treatment is the practice of combining multiple analgesic drug classes or techniques to target different points along the pain pathway. It takes advantage of additive or synergistic, analgesic effects that optimize analgesia and improve patient comfort, while lowering the dose of individual analgesic agents reducing the potential for undesirable side effects. When choosing and combining therapies it is a good practice to know where each drug or modality is expected to have its effect avoiding duplication or omission of a potential target.

OSTEOARTHRITIS TREATMENT
Treatment decisions are affected by both owner factors and clinician factors. Owner factors include willingness and ability of the owner to implement the plan. Treatment plans must consider finances, schedule, physical environment, geographical location, and owner’s physical abilities to name a few. Clinician factors include expertise, personal successes and failures, and availability of treatment. As clinicians, we should strive to attain research-based practice where our treatment decisions and recommendations are based on data related to effectiveness and safety.

Current treatments for OA include:
The use of nonsteroidal antiinflammatory drugs (NSAIDs) for the treatment of OA in dogs and cats is widespread in veterinary medicine. The efficacy of NSAIDs in treating OA is unquestioned. This class of drugs is found to decrease pain and improve function with minimal side effects while providing high client satisfaction.

Other drugs such as tramadol, amantadine, gabapentin, amitriptyline, and acetaminophen are all used to varying degrees for the treatment of osteoarthritis in our veterinary patients, but the reality is that there is very little in the literature that supports the use of these drugs in relation to both efficacy and safety. Other therapeutic agents with potential for treatment of osteoarthritis include a polysulfated glycosaminoglycan (PSGAG) (Adequan®) and a pentosan polysulfate (PPS) (Cartrophen®). The evidence supporting effectiveness of these products in the veterinary literature is also minimal at this time. Finally, veterinary diets that make claims regarding effective OA treatment in dogs and cats have been available for decades. The reality is that the strength of evidence for many of these diets and other nutraceuticals is still weak. This is supported by a recent systematic review that looked at the evidence to support use of these products for treatment of osteoarthritis in veterinary patients. (Vandeweerd et al. Systematic review of efficacy of nutraceuticals to alleviate clinical signs of osteoarthritis. J Vet Intern Med. 2012;26:448–456.) The overwhelming message was that: “The evidence of efficacy of nutraceuticals is poor, with the exception of diets supplemented with omega-3 fatty acids in dogs” and that “…four randomized controlled trials in dogs concerning diets supplemented with omega-three fatty acids, which were of high quality and demonstrated a significant effect on clinical signs of OA.”

Another such review stated that:
- Weight management should be part of the first-line approach in patients with osteoarthritis.
- High levels of the omega-3 fatty acids EPA and DHA may help to modulate inflammation and improve clinical signs.
- Diets fortified with glucosamine and chondroitin may provide benefit, but supplementation may be needed to reach therapeutic dosages.
- Supplements such as antioxidants and green-lipped mussel may provide additional benefits, but more research is needed.

These statements should also not be over interpreted. These reviews both state that there is a shortage of quality research that supports the use of many of these diets or products. It is possible that these nutraceuticals could be effectively used to treat OA. The current state of affairs would be that more quality investigation of the effectiveness is required.

Of major importance is the role that weight management plays in the prevention and treatment of osteoarthritis in our veterinary patients. Proper nutritional counseling for our patients and maintaining healthy body condition and activity level may have the most profound impact on preventing and managing OA of all treatment options that are currently available.
The Role of Rehabilitation in the Management of Osteoarthritis
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INTRODUCTION
Rehabilitation is a relatively new pursuit in veterinary medicine with the first interest gaining momentum in the late 1980s through to the present. Many of us have experienced first-hand the benefits of physiotherapy following injury and/or surgery and thus it would seem logical that our veterinary patients would experience the same benefits. That being said, there is a paucity of quality publications in the literature that provide strong evidence that rehabilitation is beneficial. With increasing client interest in veterinary rehabilitation, the availability of training courses for veterinarians and veterinary technicians and the recent formation of the American College of Veterinary Sports Medicine (ACVMSR), hopefully we see strong evidence supporting this practice.

Currently established physiotherapy techniques that are used to manage canine patients that have OA have the following perceived benefits:
- Reduce pain
- Resolving inflammation
- Improve strength and balance
- Increase range of motion
- Prevent muscle spasms
- Help to restore more normal joint function
- Preventing or minimizing muscle atrophy
- Preventing periarticular contraction
- Increasing blood and lymph flow through the affected area
- Providing positive psychologic effects for the patient and owner

The ultimate goal for any rehabilitation protocol would be to restore function to as close to normal as possible. In many cases, physiotherapy also helps to reduce the dose of analgesics necessary to maintain patient comfort.

BENEFITS OF REHABILITATION
It is well established that joint dysfunction that arises from osteoarthritis can range from minor discomfort to complete debilitation. In veterinary practice we see patients all along this spectrum. In many cases OA is advanced and progressing before owners report clinical signs and many pathological changes have already occurred in both the joint and the periarticular structures such as tendons, ligaments, and the joint capsule. An obvious example of this is the ‘medial buttress’ seen on the medial aspect of the canine stifle with cranial cruciate ligament rupture and subsequent instability and advancement of osteoarthritis. With this fibrosis and scarring we see a reduction in range of motion both from physical restriction, but also from the discomfort associated with movement of the joint. This vicious spiral leads to disuse, muscle atrophy, further loss of joint support, and further deterioration in the range of motion and quality of movement. Therefore, the goal of any therapy should be to reduce discomfort, improve range of motion, and thus quality of movement, minimizing the negative effects of loss of use. It is important to realize that these periarticular structures are well innervated and must be considered a source of discomfort that are major contributors to the pain associated with OA. Anyone that has had a joint immobilized in a cast, or joint surgery, can attest to the discomfort associated with a joint with a reduced range of motion. This is especially apparent when the well-meaning physiotherapist uses manual stretching to help restore normal range of motion!
REHABILITATION TECHNIQUES AND MODALITIES

Cryotherapy
When tissues are inflamed, pain management and rehabilitation start with cryotherapy. Cryotherapy is an inexpensive and readily available modality that is effective for reducing swelling and inflammation for tissues that are chronically inflamed, recently injured, or postoperatively. It can consist of ice packs, ice wraps, and cold compression wraps and can be as simple as a bag of frozen peas, a Ziploc™ bag with two parts water and one part alcohol, or as complicated as a Game Ready™ cold compressive therapy unit. Using compression such as an elastic wrap can further decrease the temperature of the deeper tissues.

Cryotherapy is most effective when inflammation is present and some of the perceived benefits of cryotherapy include:
- Promotion of vasoconstriction and skeletal muscle relaxation and decreases nerve conduction.
- Vasoconstriction limits blood flow into the area, thereby reducing edema.
- Muscle relaxation can decrease edema formation by improving venous return and by preventing endothelial damage caused by local acidosis.
- Decreased nerve conduction produces mild analgesia.

Moist Heat
Moist heat can be applied using warmed oat bags in a moistened towel, using commercial, microwaveable moist heat packs, or using moist heat bags from a hydrocollator commonly found in physiotherapy facilities. It is best used after acute inflammation has resolved. It is very useful when applied before stretching, massage therapy, passive range-of-motion (PROM) exercises, or active exercise.

The benefits of moist heat include:
- A reduction in muscle spasms and increase in blood flow to the treatment region.
- Penetration to a tissue depth of 1 to 2 cm.
- Causes vasodilation, mild sedation, relief of muscular pain, resorption of extravasated fluids, and increased local circulation.
- Enhances local metabolism and improves the delivery of nutrients.
- Increases the compliance of joint capsules, tendons, and scar tissue and reduces joint stiffness, thereby countering much of the stimulus for pain.

Passive Range of Motion Exercises (PROM)
The purpose of passive range of motion activities is to advance the joint through a comfortable range of motion. This is not considered stretching and is not intended to exceed the limit of comfortable joint movement or the ‘end point’ as it is called. Common questions that arise related to PROM activities include use of sedation or muzzles during this activity. In the majority of cases neither is required. Animals with temperaments such that a muzzle is required may not be good candidates for this type of manipulation.

PROM is intended to:
- Maintain normal range of motion in joints
- Prevent contracture
- Improve blood and lymphatic circulation
- Stimulate sensory awareness
- Reduce the catabolic effects of immobility on articular cartilage

Stretching
When additional pressure is applied at the end points of the ROM then it becomes stretching. The goal of stretching is to increase tissue extensibility. These activities are ideally performed several times daily after the application of moist heat or therapeutic ultrasound therapy. Trained individuals can perform both passive ROM and stretching. Caution should be exercised when instructing owners to perform stretching
exercises, as there may be some discomfort associated with manipulating a joint past its end point if limitations in joint range of motion are present.

**Massage**

Massage techniques are adjunct therapy that may have direct and indirect effects on pain sensation. These techniques do not have any direct effect on muscle mass, strength, or rate of muscle atrophy, but when used in conjunction with pain management modalities and exercise will increase mobility and improve function.

Direct effects on pain include:
- Stimulation of sensory afferents
- Counterirritant theory
- Psychological effects

Indirect effects on pain include:
- Increasing extensibility of tight muscles, scar tissue, joint capsule, tendons, ligaments
- Assisting venous and lymphatic flow

**Therapeutic Ultrasound**

The underlying principle of therapeutic ultrasound is the conversion of sound waves to heat as they are absorbed in the tissues that are being treated. A depth of effect of 5 cm is reported, causing an elevation in temperature to 40°C to 45°C and allowing treatment of deeper structures such as the hip joint. Ultrasound is also thought to promote healing by stimulating fibroblastic activity, increasing cellular metabolism, improving circulation, and increasing the strength and pliability of tendons.

Ultrasound’s role in pain control is thought to be a result of:
- Altering its transmission or perception
- Modifying the underlying condition causing the pain
- Modulation of the inflammatory process

**Laser (LLLT)**

Low-level laser therapy (LLLT) is a modality gaining popularity in general veterinary practice with multiple and widespread claims. LLLT is the directing of red and near infrared light over tissue in hopes of improving healing and reducing pain, and reducing bacterial counts. The mechanisms of action for these effects are poorly understood at this time, but some believe that laser directs biostimulative light energy to the body’s cells which the cells then convert into chemical energy to affect biological processes.

**Electrical Stimulation (EStim)**

Electrical stimulation (EStim) is commonly used in human physiotherapy to increase muscle strength, improve joint range of motion, reeducate muscles, and decrease edema and pain. There are both pain management and muscle stimulation modes for this modality. Transcutaneous electrical nerve stimulation (TENS) is commonly used to treat a specific area of pain or to stimulate a particular muscle in order to combat muscle atrophy. These TENS units are readily available, battery powered, and inexpensive.

**Hydrotherapy**

Hydrotherapy in the form of the underwater treadmill is one of the most effective methods of providing controlled and targeted therapy for our patients. Its benefits in pain relief in postoperative, neurological, and chronic OA patients are a result of the effects of buoyancy, hydrostatic pressure, and temperature. It provides safe, controlled, supportive, and non-explosive activity that is ideal for weight loss, hip dysplasia, and FHO patients. The one unfortunate drawback of underwater treadmills is the expense, space requirement, and maintenance of the equipment.
**Therapeutic Exercises**
The true gains made in any rehabilitation protocol are made through exercise. Most of the modalities mentioned previously are intended to provide better quality and more comfortable movement allowing our patients to exercise to regain strength, range of motion, and muscle mass whose loss is associated with injury, disuse, or chronic conditions. Unlike the underwater treadmill, these activities can be done with little financial investment with a bit of ingenuity and creativity. Activities as simple as walking and trotting over different inclines and terrain, walking over cavaletti rails, walking with resistance provided by water, elastic bands, sand, snow, and sit to stand exercises are all exercises that can be incorporated into a dry-land rehab program. Many of these activities are essential to a well-directed home exercise program. Stairs, exercise balls, and peanuts, orange safety cones, and broom sticks can all be modified and used inexpensively to set up a dry rehabilitation area.
COMpanion animal – principles of fracture repair

Fracture fixation - some basic considerations of biology and classification
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Blood supply to the bone and fracture healing

I. Normal vascularization
The normal blood supply is composed of three different systems:
- Afferent vascular system
- Intermediate vascular system
- Efferent vascular system

Afferent system
The afferent system carries the blood to the bone and consists of the nutrient artery, the metaphyseal arteries, and the periosteal arteries.

The periosteal arteries are normally a minor component of the afferent system and are mostly localized at muscle and fascial attachments to the bone.

In normal conditions, the periosteal arteries provide blood only to the outer cortex in the vicinity of their attachment.

Normally, the periosteal circulation is of lesser importance; its role should not be underestimated in the growing animal or after fracture.

Intermediate system
The blood vessels of the intermediate vascular system connect the afferent and efferent vascular system. It consists of canals of Havers and Volkmann and minute canaliculi, providing nutrients to the osteocytes.
**Efferent System**
Venous drainage occurs principally at the periosteum.
   The venous system present in the medullary cavity is mostly connected to the bone marrow.

In normal conditions, blood flow is essentially centrifugal.

**II. Vascular Response to Injury**
Response of vascularization following fracture depends on the complexity of the fracture.
   - The afferent and efferent vascular systems hypertrophy. The arteries and veins left intact increase both in size and diameter.
   - An *extraosseous blood supply of the healing bone* develops from the surrounding soft tissue. This blood supply is separate from the periosteal blood supply. It furnishes blood to devitalized fragment, devitalized cortex, and periosteal callus.
     After fracture stabilization and restoration of the normal afferent and efferent blood supplies, the extraosseous blood supply regresses.

   Factors that interfere with blood supply will significantly impair bone healing. These factors can result from the initial trauma, improper surgical technique, inadequate reduction, and inadequate stabilization....

**III. Bone Healing**
Three types of bone healing are recognized:
   - Primary bone healing (primary osteonal reconstruction)
   - Gap healing (primary osteonal reconstruction)
   - Secondary bone healing

**Primary Bone Healing**
Primary bone healing or direct bone union occurs in area of bone contact with high compression forces. The gap between the fragments must be smaller than 0.1 mm and the fragments must be very stable (< 2% strain). When these conditions are met, osteons from one fragment will directly invade the other fragment and cortical remodeling will occur. No callus will be identifiable radiographically.

   Although primary bone healing may appear ideal, in clinical situations, true primary bone healing is rarely achieved.
   - Bone healing by direct osteonal reconstruction regains strength slowly.

**Gap Healing**
Gap healing occurs when the gap between the fragments is less than 1 mm and the gap is very stable (< 2% strain). The gap is originally filled with blood clot, followed by loose connective tissue. After a few weeks, osteoblasts deposit lamellar bone in the gap between bone fragments. This lamellar bone is initially weak. After 3 to 4 weeks, the lamellar bone is progressively replaced by osteonal bone originating from both fragments.

**Secondary Bone Healing**
If the gap is too wide or if there is any movement between the fragments, secondary bone healing will occur. Secondary bone healing will progress through several steps, eventually leading to the restoration of a normal bone.
   4. Hemorrhage and formation of a clot.
   5. Inflammation and edema.
   6. Proliferation of pluripotential mesenchymal cells (callus formation).
   7. Fibrous tissue formation.
   8. Metaplasia into fibrocartilage.
   9. Lamellar bone formation.
  10. Osteonal bone formation and remodeling.
This process allows the fracture gap to be filled with successively stronger and stiffer tissue, until normal Haversian bone can be restored.

The entire healing process is under the control of mediators, chemoattractants, angiogenic factors, and growth factor, mechanical and electrical stimuli.

Callus can be subdivided on the basis of locations into medullary bridging callus, periosteal bridging callus, or intercortical bridging callus.

Although all three types of callus will usually form, the relative amount of each type will vary markedly in response to circumstances (i.e., bending instability is usually associated with a large periosteal callus).

IV. AVERAGE TIME TO CLINICAL UNION

<table>
<thead>
<tr>
<th>Age</th>
<th>IM pins</th>
<th>Bone plate</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 3 mo</td>
<td>2-3 weeks</td>
<td>4 weeks</td>
</tr>
<tr>
<td>3-6 mo</td>
<td>4-6 weeks</td>
<td>6-12 weeks</td>
</tr>
<tr>
<td>6-12 mo</td>
<td>5-8 weeks</td>
<td>12-16 weeks</td>
</tr>
<tr>
<td>&gt; 1 yr</td>
<td>7-12 weeks</td>
<td>16-30 weeks</td>
</tr>
</tbody>
</table>

Although this table can be used as a rough guide, each patient must be assessed individually and the decision to remove the implants must not be made before complete assessment of the fracture and confirmation of healing.

Do not remove the implants before clinical union!

V. COMPLICATIONS OF BONE HEALING

Delayed Union
A delayed union is a fracture that has not healed in the expected time when compared with other similar fractures treated similarly in comparable patients.

Causes: Physical and Systemic Factors
Instability and vascular impairment are major causes of delayed union.

Constant instability, beyond the acceptable strain will cause disruption of the delicate vasculature and will prevent callus metaplasia.

Vasculature impairment at the fracture site at the time of injury, or during reduction and internal fixation, will significantly delay bone healing.
Infection at the fracture site delays the healing process by promoting tissue ischemia and necrosis. Bones poorly covered by muscles tend to heal more slowly (radius, tibia) because the available source of vessels for recruitment into the healing process is limited. A large gap at the fracture site due to poor reduction, severe comminution, bone loss, and soft tissue interposition are other causes of delayed union.

**Treatment**
Surgery is indicated if the main fracture fragments are significantly malaligned and cannot be realigned by closed reduction or if the implants have loosened and instability is significant. Fibrous callus within the fracture gap need not be excised if adequate stability, compression, and reduction can be achieved. Delayed union may be treated conservatively if the alignment of the main fragments is adequate, the implants are still firmly attached to the bone, and instability at the fracture site is minimal. Radiographic progresses are reevaluated every 4–6 weeks and union is expected to develop within 8 to 12 weeks.

**Nonunion**
A nonunion is an ununited fracture characterized by a pseudoarthrosis at the fracture site.

**Causes**
Persistent instability, impaired vascularization, large gaps, interposition of soft tissue, and infection are major causes in the development of nonunions.

The radius is responsible for the majority of the nonunions and represents 60% of all nonunions (tibia 25%, femur 15%).

**Surgical Treatment**
Nonunions should be treated surgically as soon as they are diagnosed. Rigid stabilization of the fragments and cancellous bone graft are mandatory for success. Treatment of nonunion is difficult and the prognosis is usually guarded.

**Malunion**
A malunion is a fracture that has healed in an abnormal position. It may result from:
- Spontaneous healing of a fracture
- Implant failure
- Improper fixation

Although dogs and cats can tolerate up to 20% shortening of their leg, angular and rotational deformation may cause significant dysfunction. Correction of a malunion is difficult and may require multiple surgeries. Surgical correction is performed only if a significant improvement in leg function can be expected.

**Characterization of the Fracture**
It is important to be able to accurately describe a fracture. Not only does this facilitate communication between clinicians, it is the first step in determining what forces will be acting on the fracture and choosing a successful repair technique. A minimum of two good-quality, orthogonal radiographs centered on the affected bone are needed to formulate this description.

**I. What Bone Is Involved**
This may seem obvious, but the surgical approach to a pelvis is significantly different from a tibia and the front limbs must bear more weight than the hind limbs. Similarly, stabilization techniques applicable to the axial skeleton may be quite different to those available for long bones.

**II. Extent of the Fracture**
A complete fracture disrupts the cortex 360 degrees and is usually accompanied by some displacement of the fractured ends. A greenstick fracture (seen mainly in young animals) indicates only one side of the bone is broken while the opposite cortex is merely bent. Greenstick fractures are innately stable and may be amendable to external coaptation. Fissures are longitudinal cracks that are not displaced, leaving the
periosteum intact. Stabilizing fissures should be the first step in internal fixation as failure to do so will often result in iatrogenic comminution of the fracture.

III. Soft-Tissue Injury
Closed fractures have an intact, viable soft-tissue covering. Open fractures have some degree of communication between the fracture and the external environment. Grade I open fractures may have an intact covering of nonviable skin or a small (<1.0 cm) full-thickness wound. Grade II fractures are associated with larger lacerations, often created by the ends of the fractured bone. Grade III fractures have extensive soft-tissue injury with gross exposure of the underlying bone.

The greater the soft-tissue injury, the more damage will have been done to the local blood supply and the slower will be the fracture healing. Likewise, open fractures will have greater contamination and an increased risk of infection. Open fractures are generally not treated by external coaptation and extra effort must be made to ensure fragment stability.

IV. Part of the Bone Affected
Designations of epiphysis, metaphysis, and diaphysis are useful, but may have limited application in mature animals. Describing a fracture as located in the proximal, middle, or distal third of the bone is generally adequate. Of specific importance is involvement of the growth plate. Physeal injuries in immature patients may disturb future longitudinal bone growth potential in these animals.

The Salter-Harris classification is used to describe physeal fractures and is based on the extent of damage done to the growth plate and surrounding bone. Type I fractures form transversely along the layer of hypertrophying cells in the growth plate and do minimal damage to the dividing cells. Early, accurate reduction of these fractures results in rapid healing and continued longitudinal development of the bone. Type II fractures pass along the physis, but include a triangular portion of the metaphysis. The potential for continued growth in these bones remains good. In Type III fractures, the fracture line travels from the physis, through the epiphysis to the articular surface. Extension of fractures into the joint is important because, with the current technology, accurate and stable fixation of these fractures often requires the use of interfragmentary lag screws. Type IV fractures are a combination of type II and III with involvement of both the metaphyseal and epiphyseal portions of the bone. Type V fractures result in crushing of the physis and cessation of growth. The most common form of this injury is the ‘occult’ damage that occurs in the distal ulnar growth plate after radial fractures in immature animals. These patients will show premature closure of their distal ulnar physeal weeks after the injury and are at risk of developing angular limb deformities.

V. Number of Fragments
Fractures are generally referred to as ‘two-piece,’ comminuted, multiple, or segmental. As the name implies two-piece fractures have only two fragments (it does not rule out fissures). Comminuted fractures may have single or multiple smaller fragments with a variety of fracture lengths. One of the most common types of comminution, the ‘butterfly’ fragment, is characterized by a large segment of cortical bone isolated by two converging, long oblique fracture lines. Bones with multiple or segmental fractures are broken into three or more pieces with fracture lines that do not intersect.
As a general rule, a larger number of bone fragments is associated with a more difficult repair, more extensive soft-tissue injury, and delayed healing.

**VI. Obliquity**

**Transverse**
By definition, a transverse fracture forms perpendicular to the long axis of the bone and the fracture length is roughly equal to the diameter of the bone. These fractures are innately resistant to compressive forces and may do well when treated by intramedullary pinning or external coaptation. Conversely, treatment of avulsion fractures at the insertion of ligaments or tendons needs to include some means of overcoming on-going traction.

**Short Oblique**
The length of the fracture line in a short-oblique fracture is less than twice the diameter of the bone. This obliquity affords some resistance to rotational forces, but compression will cause over-riding of the fragments. Hemicerclage wires are generally used with intramedullary pins to stop this slippage. The use of full cerclage wires, or lag screws, is contraindicated when treating short-oblique fractures because interfragmentary compression will cause slippage along the fracture line and displacement of the fragments.

**Long Oblique**
The length of the fracture line in a long-oblique fracture is at least two to three times the diameter of the bone. These bone fragments have no resistance to over-riding. Full cerclage wires or interfragmentary screws can achieve compression along the fracture line resulting in greater stability of the repair.

**Biomechanics of Fractures**
Under normal circumstances, bone is subjected to external forces (hit by car, etc.) and internal forces (muscle contraction). Bones can deform relative to the loads or stresses applied and remodeling occurs
with repeated or chronic stress. Variations in bone anatomy reflect the different physiologic forces that act on individual bones as they perform their normal functions.

**Stress** is defined as a local force and **strain** is the deformation created by that stress. **Normal stress**, in long bones, is defined as a force applied parallel to the weight bearing axis of the bone. **Shear stress** is force applied perpendicular to the weigh bearing axis.

When discussing forces on a bone, we divide the stresses into: compression, tension, bending, rotation, and shear. When these stresses exceed the bone’s structural limits, fractures will occur. The type of stress that created it may predict a fracture’s pattern. **Traction** is often associated with the insertion of tendons or ligaments and results in transverse fractures. **Compression** generally creates a ‘normal stress’ and is the force best tolerated by long bones. Excess compression will cause oblique fractures. **Bending** forces will generate both traction and compression on opposite sides of the same bone. This combination of forces can create butterfly fragments at the fracture line. **Rotation** around the central axis of a bone creates ‘shear stresses’ along a spiral line and is the force least tolerated by long bones.

As a general rule, the greater the energy of the force, the larger number of fracture fragments will be present and the more soft tissue injury will occur (slowing healing and increasing the risk of infection).

**REFERENCES**

**Fracture Fixation - Coaptation Devices for Small Animals**
Thomas Gibson, BSc, BEd, DVM, DACVS
Associate Professor of Small Animal Surgery, Ontario Veterinary College, University of Guelph, Guelph, ON, Canada

**Fracture Management Objectives**
The following are the major objectives of fracture management in any species:
- Anatomic reduction
- Prevention of displacement, angulation, and rotation of bone fragments
- Preservation of soft tissues
- Prevention of fracture disease
- Maintain the use of as many joints as possible during the healing period
- Early return to full limb function

The common methods available to use for management of fractures include internal fixation, external skeletal fixation, and **external coaptation**. In order to achieve successful fracture management with coaptation it is essential to understand the principles of both wound management and fracture management.

One must consider the following:
- The benefits and limitations of coaptation
- The potential complications
- The skill and materials required for application of the coaptation

**Coaptation Advantages and Disadvantages**
Ultimately, the use of coaptation should enhance patient comfort, healing, and function. One of the advantages of external coaptation is that the fracture site is not disrupted, as in the case of surgery, avoiding further compromise to the blood supply in the region. This should facilitate more rapid healing. Another advantage is that no implants are being placed removing the risk of implant loosening or implant associated infection. Finally, some report that the cost of external coaptation is less than that of internal fixation. This is certainly an item for debate depending on the necessary frequency of changing the coaptation device as well as the cost associated with any related complications.

Effective coaptation requires the immobilization of the joint proximal and distal to the fracture. Prolonged immobilization can result in severe disuse atrophy and fracture disease. It can also result in soft-tissue complications. Selection of an effective fracture management technique begins with both patient and fracture assessment.

**Patient Assessment**
As with any type of injury, one must consider the signalment of the animal, patient history, and perform a thorough physical and orthopedic examination.

Animals younger than 1 year of age generally heal quickly, limiting time in coaptation, and therefore may be ideal candidates for external coaptation as a primary method of fracture repair. Consideration of the breed involved is also important. For example, small and toy breed dogs have up to an 83% incidence of nonunion and malunion when external coaptation is used for primary repair for radius/ulna fractures. Constructing external coaptation devices for chondrodystrophic and obese animals can also be a challenge. Other things that must be considered are the presence of concurrent soft-tissue injuries, if multiple limbs are involved, the temperament of the patient, and if any other concurrent medical therapy could have an impact on successful treatment.

**Fracture Assessment**
A minimum of two radiographic views is required. If joints and ligaments are involved, ‘stressed’ radiographs under heavy sedation or general anesthesia may be necessary.
The fracture ‘environment’ must be established with consideration of the following:

- Location
- Type
- Displacement
- Comminution
- Fracture forces

Fracture forces include bending, rotation, shear, compression, and distraction or tension. Regardless of the method of repair, forces present at a particular fracture site must be adequately neutralized by the repair method for healing to occur. Consideration of these forces should be part of a ‘mental checklist’ when deciding if a particular type of coaptation will be adequate to allow healing to occur. External coaptation can effectively counteract bending and rotational forces, provided that the joints proximal and distal to the fracture are immobilized. Paired bone situations such as the radius/ulna and tibia/fibula may provide some advantages. If bones adjacent to a fracture (e.g., ulna, fibula) are intact, they can provide some stability by reducing bending, rotation, and shearing forces. Fractures subject to compression, shear, and/or distractive forces require internal fixation, as external coaptation alone cannot adequately counteract these forces.

**EXTERNAL COAPTATION FOR FRACTURE REPAIR - MUST HAVES**

- Fracture reduction
- Fracture alignment
- Neutral standing position
- Joints proximal and distal

**TEMPORARY SPLINT USE**

For fractures distal to the elbow and stifle joints temporary splintage can offer improved patient comfort, decrease or prevent edema, and minimize additional trauma if reduction and fixation is delayed. Temporary splintage may also be beneficial for open fractures to allow wound treatment, to reduce the risk of infection, and to decrease soft tissue swelling before definitive repair is performed. For other types of fractures all that may be required is cage rest and sedation and/or analgesia until definitive repair is performed. If an animal is required to travel a distance before a fracture can be repaired then use of a temporary splint is recommended for patient comfort as well as protection of the soft tissues.

**OTHER REASONS FOR EXTERNAL COAPTATION**

- Adjunctive therapy in managing swelling and edema
- After removal of external skeletal fixation devices
- For stabilization and support of orthopedic soft-tissue injuries or repairs
- Wound management

**COMPLICATIONS AND CARE**

Complications can arise when using external coaptation for fracture management or for temporary stabilization.

These are often a result of:

- Poor patient and fracture assessment
- Improper device application
- Case mismanagement
- Patient and/or owner compliance issues

Complications may be minor such as skin irritation and mild dermatitis, or major and life and limb threatening, such as fracture disease or necrosis secondary to swelling and vascular compromise. Owner education is essential and should be well documented. Coaptation must be applied and monitored
appropriately. Communications with owners must convey the gravity of home monitoring for potential complications, proper coaptation care, and diligent follow-up.
Fracture Fixation - Pin and Cerclage Techniques
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CERCLAGE Wires
Cerclage wires are ancillary fixation devices. They are made of pliable stainless steel. They come in
different diameters, from 27 ga (gauge) to 14 ga. The sizes most often used in small animal surgery are
from 22 ga (0.025 in, 0.64 mm), 20 ga (0.032 in, 0.81 mm) to 18 ga (0.04 in, 1.0 mm), 16 ga (0.049 in, 1.25
mm).

The term “cerclage” means to encircle. The wire is placed around the bone and is tightened to
provide static, interfragmentary compression of the fragments. Cerclage wires are called “full cerclage”
when they completely surround the bone fragments. They are called “hemicerclage” when they are
passed through holes drilled into one fragment of the bone and encircle the other fragment, and are
called interfragmentary wires when they are passed through a hole in each of the fragments.

Cerclage wires are never used as the sole method of fixation as the last wire would serve as a
stress riser and cause a pathologic fracture. In order to effectively compress the bone fragments, the full
cylinder of bone must be reconstructed anatomically. Any defect in the cortex or movement of any of
the fragments under the wire will automatically and irreversibly result in loosening of the wire and loss of
the entire reduction.

Wires are used principally on simple (1 or 2 pieces), long oblique, and spiral fractures. They can
also be used as a temporary method of reduction in more complex fractures during plate application.
Cerclage wires are never used alone, but always as an ancillary fixation device with IM pin, interlocking
nail, external fixator, or bone plate.

Studies on their effects on bone vascularization have shown that, when correctly applied they only
minimally disturb blood circulation to the bone. Anatomical studies of the blood supply to the bone
showed that venous drainage from the periosteum or the “extraosseous blood supply of the healing
bone” that develops soon after fracture enters and leave the bone at right angles. Therefore, a tight wire
does not cause significant vascular impairment. A loose wire, on the other hand can significantly disturb
vascular supply to the bone, causing delayed union or nonunion. Multiple wires placed too close together
will also cause blood flow impairment and can equally contribute to delay healing. (Blass CE, et al. Micro
vascular and histological effects on cortical bone of applied double loop cerclage. JAAHA. 1991;27:432–
434.)

Wire Application
The three most common methods of wire tightening are: twist, single loop, and double loop.

Twist
For the twist, the first 2 or three twists are started by hand then continued with an old needle driver or a
pair of pliers. The remaining twists are made by pulling the wire firmly away from the bone with the
pliers as the twisting motion is applied. Even tension on the strands will ensure that the wire twists evenly. When further twisting fails to improve tension and a slight matting of the wire occurs due to stretching, twisting is stopped and the wire is cut leaving approximately 3 to 4 twists. It is better not to push the twist flat against the bone as this will result in loss of initial tension. If it is absolutely necessary to flatten the wire, it is usually done while applying the last twist to maintain tension as the wire is flattened.

**Single Loop**
The single-loop technique uses a wire with an eyelet at the end. The wire is wrapped around the bone and the free end of the wire is fed through the eyelet. The wire is fed through a wire tightener. The crank is turned until the wire is tight. Once tight, the tightener is bent over so that the end of the wire is bent over itself. While maintaining tension, the crank is reversed to expose 1 cm of wire and the wire is cut off and pushed flat against the bone.

**Double Loop**
The double-loop technique uses a wire bend in its middle. The two free ends of the wire are passed through the loop formed by the bend and fed through the 2 cranks on the wire tightener. Both cranks are turned to evenly tighten the wire. Finishing the wire is similar to the single-loop technique. Because a double wire is used, this method is significantly stronger than the single loop or the twist and is therefore recommended when strong fixation is required.

**Hemicerclage, Interfragmentary Wires, and Skewer Pins**
To form a hemicerclage, the wire is passed through holes drilled into one of the fragment, then wrapped and secured around the bone. The portion inside the medullary cavity should wrap around the pin so that when the wire is tightened, it will pull the pin against the endosteal surface of the fragments. Although this may improve reduction of the fragments, it does not result in a significant-mechanical advantage.

When placing a hemicerclage, you must consider that the holes drilled into the bone weaken the ends of the bone. Nevertheless, hemicerclage may be useful in some situations because they do provide some protection against rotation and shearing and they do not have the tendency to slip when placed near the cone-shaped bone of the metaphysis.
Cerclage can also be placed in a way similar to sutures, holding bone fragments together. A hole is drilled in each fragment so that the wire will be perpendicular to the fracture line once tightened. These wires are tightened carefully as they can easily tear through the bone. Interfragmentary wires only provide marginal stability and they are therefore reserved for simple fractures with good interdigitation. These “interfragmentary wires” are often used to repair fractures of flat bones such as the skull or mandible. (Metelman et al. A mechanical evaluation of the resistance of various interfragmentary wire configurations to torsion. *Vet Surg.* 1996;25:213–220.)

Full cerclage should not be used on short-oblique fractures (less than 2 x the diameter of the bone) because the fragments being secured are likely to shear while the wire is tightened. The shearing of the fragments reduces the overall diameter of the bone, resulting in loosening of the wire. If cerclage wires are to be used in short-oblique fractures, their orientation should be changed so that they apply their compression perpendicular to the fracture line. This can be achieved with interfragmentary wires or with the use of a small K-wire, also called a “skewer pin.” The pin is inserted across the fracture, as perpendicular to the fracture line as possible. By passing the wire above and below the K-wire, the orientation of the wire can be maintained during tightening, reducing the risk of shearing.

**Rules for Cerclage Wire Use**
- A wire should never be used alone.
- Wire fixation should be restricted to fractures where the length of the fracture line is at least twice the diameter of the bone (*long oblique*).
- Full cerclage wires can be used only if the full diameter of the bone can be reconstructed and only if the wire maintains the reduction.
- The wire must be tight. A loose wire must be removed.
- Two adjacent wires should be spaced by a distance equal to approximately ½ to 1 x the diameter of the bone and at least 5 mm from the fracture edge.
- In most cases, at least 2 wires per fragments are used.
- The twist must be performed under tension so as to produce uniform twist of both wires.
- The wire is cut 3–4 twists from the bone and left upright. If there is insufficient soft tissue coverage or if the wire may interfere with a vital structure (nerve, blood vessel…) the wire may be left longer and bent flat while twisting.
- Bending the wire significantly decreases its initial tension.
**TENSION-BAND WIRING**

A boney projection or ‘apophysis’ marks the insertion site of many major tendons. In veterinary surgery the most common of these are the greater trochanter, greater tubercle, olecranon, and tibial crest. The growth plates associated with these landmarks are prone to traction fractures in young animals and osteotomy of these prominences can be useful for accessing otherwise well hidden joints and boney surfaces in mature patients.

The goal of tension-band wiring is to convert distraction forces caused by muscle pull, into compression at the fracture/osteotomy site. Sharp reduction forceps may aid in maintaining reduction, but be careful not to split the fragment by applying too much pressure. Two parallel Kirschner wires (0.035, 0.045, 0.062 or 0.8 to 2.0 mm) are placed relatively perpendicular to the fracture/osteotomy with the pins at the periphery of the cut. This orientation will best facilitate interfragmentary compression and limit interference with the tendon. If possible, the K-wires should engage the opposite cortex. Holes are drilled transversely through the bone’s main shaft, distal and parallel to the fracture/osteotomy. The distance between these holes and the fracture/osteotomy is roughly equal to the distance from the fracture/osteotomy to the tip of the apophysis.

![Diagram of tension-band wiring](image)

A wire (22, 20 or 18 gauge or 0.8, 1.0, or 1.2 mm) is passed through the hole in the shaft, crossed on the lateral cortex and under the tendon, as close to the K-wires as possible. Although some authors feel twisting the wire on only one side of the figure-8 pattern is adequate, forming a knot on both sides gives more even tightening. To simplify this, I use two separate wires to make the figure-8. One wire is passed through the distal holes and crossed on the lateral cortex, and a shorter wire passed under the tendon next to the pins. The ends of the separate wires are twisted together and tightened by alternating between the two knots until both are secure. The K-wires are then bent laterally 90 degrees, cut off leaving 3–5 mm and rotated medially to minimize skin irritation.

Although these implants may be left in place indefinitely, figure-8 wire breakage or loosening and migration of the K-wires are not uncommon. In this situation removal of both pins and figure-8 wire is recommended.
**Intramedullary Pins: Principles and Application**

The use of intramedullary pins for fracture fixation started in the early 40s. Because of its widespread availability, intramedullary pinning quickly became a popular method of fixation and, despite its limitations is still widely used today. The key for successful application of pins and cerclage wire is an acute awareness of their shortcomings in stabilizing fractures. Once recognized and dealt with, IM pin and wires can be a very successful method of fixation that can be used for many fractures with minimal complications.

There are several advantages of pin and wire fixation over bone plating in veterinary medicine. Pin and wire fixation is much less expensive than bone-plate fixation. Their application requires only minimal equipment. Most pin and wire placement requires a smaller surgical approach than plating, resulting in less vascular damage and soft tissue trauma, and ultimately, may result in an improved healing. Although, initial placement of the pin may interfere with the medullary vascularization, it certainly does not obliterate it and revascularization soon occurs. If the implants are stable, good intramedullary vascularization is restored within a week or two after implantation. It must be noted, however, that a loose, unstable pin or a pin filling the entire medullary cavity will seriously impair bone vascularization and may cause delayed and nonunions.

Often, the surgery is faster than plating, allowing for shorter anesthesia time. Although pins don’t always need to be removed, their removal is often relatively straightforward and can often be performed under heavy sedation as opposed to a general anesthesia for a bone plate.

Disadvantages of fracture fixation: most disadvantages are linked to the biomechanical properties of intramedullary pins. Overall, the fixation is less stable than with bone plates. One of the major disadvantages is the fact that IM pins cannot act as a buttress and therefore, the fracture must be axially stable or collapse will happen.

Advantages of intramedullary fixation vs. bone plating:
- Low cost
- Minimum of equipment necessary
- Less surgical exposure than bone plates
- Often faster
- Easier to remove
- Less trauma to the vasculature

But:
- Less stable than bone plates
- Cannot act as a buttress

**Pin Types**

A few different pins exist. Steinmann pins and Kirschner wires are similar, but differ in size. Kirschner wires (a.k.a. K-wires) are smaller and their diameter are expressed as 0.035, 0.045, and 0.062 inches (0.9 to 1.5 mm) while Steinmann pins are usually identified by a fraction of an inch (1/16 to 1/4 inch)

They can be smooth or partially threaded. When used intramedullarily, the thread offers no biomechanical advantages and certainly does not act like a screw. Furthermore, the thread creates a weak spot where the thread meets the shank and failure can be observed at the interface if this area of junction falls near the fracture site. (Howard PE, et al. An *in vitro* comparison of the holding strength of partially threaded vs. nonthreaded intramedullary pins. *Vet Surg*. 1983;12(3):119–122.)

The tip of the pin is designed to cut through the bone to allow insertion. The two most common ends are trocar and chisel. The 3-sided trocar tip is usually easier to start because of its sharp point and symmetrical design. The chisel point is harder to start, particularly at a shallow angle; however, its design allows better cutting of the bone and therefore generates less heat during introduction.

The most desirable pins are those with a different point at each end so that you can choose the point.
**Pin Diameter**
Although filling the entire medullary cavity appears to offer some advantages as far as stability is concerned, it presents some major drawbacks. The pin would completely obliterate the medullary cavity causing widespread devascularization of the bone, leading to delay union. The curvature found in many bones of the dog also does not allow the use of a large diameter pin. For this reason, it is recommended that the pin fill 60 to 75% of the medullary cavity at its smallest diameter. This diameter pin would provide the stability required for fracture healing and, if stable, would allow rapid and almost complete revascularization of the medullary cavity and bone cortex. When in doubt, a small pin diameter can be used. This is also the case for the tibia, in which the pin has to bend along the caudal cortex. A large pin would not allow this bending to occur and a smaller pin (50% of the medullary cavity) must be used for that purpose.

**Seating of the Pin**
To be properly seated, the pin should span the entire medullary cavity and the extremities should be seated into the cancellous bone of the epiphysis. Driving the pin too far results in penetration of the distal cortex and, in most cases, invasion of the joint surface. When this happens, the pin must be removed and redirected into the distal fragment. Simply retracting the pin without reorienting it will result in migration of the pin back into the joint.

**Cutting the Pin**
Often a sterilized bolt cutter is used to cut the pin. It is important to cut the pin as short as possible (5 mm from the bone end) to avoid unnecessary damage to the soft tissue covering the pin. Leaving the pin too long often results in soft tissue trauma, seroma formation or nerve entrapment.

- Soft tissue often interferes with the tip of the bolt cutters and it is often difficult to cut the pin short. One way to circumvent this difficulty is to properly seat the pin and determine the position of the cut. Retract the pin by a known distance allowing easy insertion of the bolt cutter (~ 2 cm). Cut the pin with the bolt cutter then gently impact the pin again to its final position using a mallet.
- Metal saws are generally awkward to use and result in a large amount of metal particles being produced. They often induces significant tissue trauma and their use is usually not recommended.

**Pin Insertion**
Pins can be inserted in long bones by either retrograding or normograding.
Retrograding
The pin is inserted from the fracture side in either end of the bone. The pin is driven through the fragment and retracted up to the level of the fracture. The fracture is reduced and cerclage wires are applied. The pin is then driven back through the fracture line, into the other bone segment while maintaining reduction of the fragments. Inserting the pin in the second fragment before reducing the fracture will invariably result in the impossibility to properly reduce the fracture once the pin is seated.

Normograting
The pin is started at one end of the bone and driven through the bone. Once the pin reaches the level of the fracture, the bone is reduced and stabilized, then, the pin is pushed past the fracture and into the opposite segment.

The femur and the humerus can be pinned using both methods, but are often retrograded because the medullary cavity provides a natural guide for pin placement. In these bones, the proximal articular surfaces are offset relative to the axis of the medullary cavity and therefore joint penetration by the pin is rarely a problem. On the other hand, the tibia is usually normograde because the medullary cavity is in direct alignment with the cranial cruciate ligament, the menisci, and the articular surfaces of the femur. Insertion of a pin from the medullary cavity into the proximal segment would likely result in the pin being directed straight into these structures. Instead, a smaller pin is chosen and inserted normograde, starting cranial to the articulation. If properly inserted, the pin will bend along the caudal cortex and follow the medullary cavity of the distal segment. The same technique is also used for IM fixation of metacarpal/tarsal fractures to avoid the distal metacarpophalangeal articular surfaces.

Pin Removal
Pins do not necessarily need to be removed unless they are causing a problem or are migrating out. Pins that are migrating out often create a small seroma and cause mild lameness. They have the potential, however, to cause severe and sometimes irreversible damage on their way out if they interfere with nervous structures (sciatic nerve), vascular structures or articular cartilage. Pins that are migrating are usually easy to retrieve under short general anaesthesia or sedation.

Pins that are stable may present a challenge to retrieve as they are often difficult to localize and difficult to extract.
**CROSSED AND DYNAMIC PINS**

A useful way of counteracting rotational forces when treating metaphyseal fractures with intramedullary fixation is by using crossed or Rush pins. For the sake of this discussion, distal femoral physeal fractures will be used as an example of this type of injury/repair.

The femur is the most commonly fractured long bone in the dog, and in immature dogs the growth plate is the weakest part of the bone. Salter-Harris type I & II fractures of the distal femoral physis account for 37% of all physeal fractures in dogs.

A study of pinning techniques done at the OVC compared a single intramedullary pin to crossed pins, convergent pins, and ‘dynamic’ (Rush pin-like) fixation pins. All of the pins had the same diameter except the single pin, which was twice the diameter of the double pins. Results of biomechanical testing in torsion showed that crossed pins gave the strongest fixation and were 117% of the normal intact bone. Convergent pins tested at 105%, dynamic pins were 91%, and a single pin was 72% of the intact control. Failure of the crossed and convergent pins resulted in fracture of the associated bone while the dynamic and single pins failed by simple separation at the fracture site. Interestingly, when the testing stress was released from the dynamic pin samples, the fragments tended to realign, albeit slightly distracted.

Attention to detail is necessary for the successful use of a crossed pinning technique.

11. Repair of the fracture should be done early, whenever possible within three days of the injury. Delaying surgery will allow muscle contraction making accurate, atraumatic reduction more difficult.

12. Pins should not be placed through the joint capsule as this can limit postoperative mobility of the stifle. An arthrotomy is needed in most cases.

13. Flexing the stifle and extending the hock will reduce the muscle tension on the distal fragment and make reduction easier. Although the growth plate will have sustained damaged and its future growth potential limited, take care not to crush the soft bone of the epiphysis, creating Salter-Harris III or IV fractures.

14. Avoid under-reduction of the fracture. Over-reduction (placing the distal fragment slightly cranial to its normal location) will ensure the patella contacts only a smooth articular surface.

15. Small diameter pins should be used: 0.045 and 0.062 wires can be used for small and medium-sized dogs. One eighth-inch pins are adequate for larger dogs.

16. Insert the lateral pin just proximal to the articular surface, midway between the long digital extensor tendon and the lateral collateral ligament. The pin(s) should form a 20- to 45-degree angle
with the long axis of the bone depending on whether you are trying to create a dynamic- or crossed-pin technique.

17. Drive the lateral pin across the fracture line, but do not seat it fully into the bone. There is no landmark for the second (medial) pin, but it is placed symmetrically to the lateral pin. The medial pin can be driven fully followed by complete seating of the lateral pin.

18. Ideally the pins should cross above the fracture line to maximize antirotation.

Pre and Postoperative Fracture Assessment

Preoperative Radiographs

To accurately describe a fracture, a minimum of two good-quality orthogonal radiographs centered on the affected bone are needed. This description is the first step in determining what forces will be acting on the fracture, picking the least traumatic surgical approach to the fracture site, choosing the appropriate implants, and planning a successful repair technique. Combined with physical examination, preoperative radiographs will also help estimate the amount of soft-tissue injury, the need for perioperative antibiotics, and the potential for postoperative complications and exercise restrictions.
Postoperative Radiographs

Two-view radiographs of the surgical site should be taken immediately after surgery to document the appropriate repair and provide a baseline for comparison with future radiographic evaluations. The early detection of surgical errors is important to identify areas for technical improvement and suggest modifications to postoperative care.

Assessment of a fracture repair and healing has been divided into four categories: alignment, apposition, apparatus and activity.

Alignment of the bone fragments and associated joints is evaluated for maintenance of bone length, valgus and varus angulations, cranial or caudal bowing, and torsion or rotation around the central axis of the bone.

Apposition relates to the quality of the fracture reduction. Normal healing generally requires a minimum of 50% overlap of the fracture ends with a displacement equal to less than 25% of the diameter of the bone. Joints are less accommodating and the surgeon must strive to achieve perfect reduction of articular surfaces.

Apparatus is the evaluation of the type, size, placement, and stability of the implants selected.

Activity refers to the biological activity that is present at the fracture site (is it healing or not) and will help determine how much, when, and what type of exercise should the patient be allowed.
Follow-up radiographs should be taken 4 weeks after surgery to confirm stability of the implants and fracture fragments. Except for very young animals, there will be little evidence of healing at this time. In the early stages of healing, fracture lines often appear to widen because of normal loss of calcium and it is difficult to radiographically distinguish healing from infection.

Fracture healing is affected not only by the type of repair and its presumed stability, but also the species, breed, and weight of the animal, its age, the amount of soft-tissue injury, the presence of infection, and cooperation of the owner. Most fractures will heal within 3 to 4 months of repair, but those with multiple fragments and damage to the surrounding muscles may take significantly longer. **Delayed unions** are characterized by having excessive callus with a persistent fracture gap. Delayed unions generally occur in fractures that have an adequate blood supply, but inadequate stabilization for the amount of patient activity. Treatment of these cases is centered on increasing the stability of the fixation although a bone graft should be added if treatment involves surgical exposure. **Nonunions** are fractures that have had inadequate stabilization and compromised blood supply. The result is partially resorbed ends of the bone with no osteogenic potential. Treatment of true nonunions must include debridement of the fractured bone ends, bone grafting, and stabilization.

Additional radiographs should be taken every 4–6 weeks to monitor healing. The sudden onset of lameness or pain at the surgical site is an indication for additional radiographs and may be associated with broken, loose, or migrating implants. Infection (primary or secondary) may be associated with failed implants and bacterial culture of loose metal is always recommended.

Removal of metal implants following boney union is not routine unless they are causing problems. Indications for implant removal include loosening, migration, infection, or lameness in cold weather. Loss of bone substance from under the implant because of stress protection becomes problematic because of the potential for refracture after the implant has been removed.
Radiographs should be taken immediately after implant removal to document healing and serve as a baseline for comparison with future radiographic evaluations. These films often guide recommendations for the patient’s return to normal activities.

Whenever possible, owners should be shown the patient’s radiographs. It has been my experience that seeing the preoperative fracture facilitates their choice to proceed with surgical correction and gives them a better understanding of the need for restricted activity during postoperative healing. Most owners need very little guidance to accurately interpret the healing process seen on most radiographs and involving clients in the decision making process will increase their satisfaction with the final outcome.
Understanding the Anatomy of the Equine Eye

1. The three layers and other items inside the globe can be thought of like a sandwich
   - Outer layer is connective tissue: sclera and cornea (think of as the “bread”).
   - Middle layer is vascular uvea: iris, ciliary body, and choroid (think of as “tomato”).
     - Uvea is derived from the Latin term for grape - pigmented, juicy!
   - Inner layer is sensory: retina and optic nerve - the business layer (think of as “meat”).
   - Lens splits the eye into anterior and posterior segments (think of as “lettuce” inside sandwich).
   - Ocular media are wet sources of nourishment (can compare to “mayonnaise or butter”).

2. There is normally a blood-ocular barrier. The interior of the eye is immune privileged.
   - Lens proteins are not recognized as “self.” If discovered by immune surveillance, intense inflammation results.
   - Similarly many proteins in the retina are regarded as “foreign” and will incite an immune reaction if accessed by immune-monitoring elements.
   - Some proteins found in nature (e.g., leptospiral bacterial proteins) mimic molecules found in the eye (e.g., corneal or retinal proteins). If the immune system mounts a reaction to a foreign protein encountered outside the eye, it will “remember” and react to any similar proteins inside the eye if the blood-ocular barrier is compromised.

3. Chambers inside the eye normally hold clear media that are in liquid or gel form
   - Every clear substance that light passes through has a refractile influence. Light passes through tear film, cornea, aqueous humor, lens, and vitreous to reach the retina.
   - The volume of the aqueous is about 3 ml and is normally a clear liquid that is an ultrafiltrate of plasma. The volume of the vitreous is about 26 ml and is normally a clear gel, but often liquefies in aging horses or in disease conditions.
   - Disruption of blood-ocular barrier lets components into these media that do not belong in the eye: ensuing altered clarity of ocular media, or obvious accumulation of cells in the chambers of the eye, is proof of a compromised barrier. Manifestations include hyphema (accumulation of RBC), flare, hypopyon (accumulation of WBC), fibrin clots in the anterior chamber and vitritis, accumulation of cells and debris in the posterior segment.

4. Cornea
   - Cross section of the cornea includes the precorneal tear film plus several tissue layers: epithelium, basement membrane (Bowman’s), stroma, basement membrane (Descemet’s), endothelium. All layers add up to only about 1 mm thickness in the non-inflamed eye.
     - Of these layers, epithelium is thin with only 6–8 layers of cells. The stroma represents over 90% of the corneal thickness. The combined thickness of Descemet’s membrane and the endothelium is only 21 microns (equivalent of 3 RBC).
     - Clinicians must judge both the depth of corneal ulcers and the layers affected.
   - Healing of noninfected, epithelial defects can be very fast. The whole corneal epithelium normally turns over in about one week.
   - Healing of stromal defects can be very slow. Deep, stromal defects may get covered with epithelium but persist as “facets” until fibrosis and remodeling is complete. Normally, the stroma is made of layers of connective tissue arranged in regular, geometric planes that are transparent. Fibrotic areas will have irregular geometry and be opaque. Areas where inflammatory cells or infectious elements have infiltrated within the layers will be opaque.
The endothelium is responsible for normal cornea transparency, as activity of the Na+-K+ ATPase pump across this boundary layer of cells keeps the corneal stroma relatively dehydrated. If the endothelium decompensates the cornea will “steam up” in the compromised area.

A normal cornea is avascular, but contains many nerve fibers that trigger pain if exposed. Nerve fibers are most abundant in the upper stromal layers and near the limbus.

5. Uvea: Iris
- The iris has no epithelium on the anterior face - heavily pigmented stroma and abundant blood vessels are readily visible. The posterior iris has two layers of epithelium.
- The walls of uveal blood vessels are normally sealed by tight junctions between component cells. Additionally, there are active mechanisms that “police” vessel boundary activity to maintain the blood-ocular barrier. With inflammation (uveitis) the uveal blood vessels become congested and the tight junctions are compromised. The vessels become leaky. Red blood cells, white blood cells, inflammatory cytokines, and other plasma components then enter the ocular tissues and media.
- An inflamed iris is a “sticky” tissue. Iridal tissue can adhere to other parts of the eye like a weld. Most commonly, the tissue sticks in a posterior direction to form posterior synechia between the posterior iris epithelium and the lens. Occasionally, iris tissue migrates forward and forms anterior synechia between the iris stroma and the corneal endothelium.
  - When used to treat inflammation, atropine not only pulls the iris out of harm’s way by dilating the pupil, but it also reduces the “leakiness” of the blood-ocular barrier, and lessens ocular pain by blocking ciliary spasm.
- If iris tissue prolapses out a hole in the cornea it looks like focal granulation tissue - more red than brown. In some cases this may effectively seal a leaking globe, but in other cases it is part of a complete endophthalmitis that will necessitate enucleation or globe compromise that will require advanced surgery.

6. Uvea: Ciliary Body
- The ciliary body (CB) is one of the biggest multitaskers in the body!
  - It actively produces aqueous humor and filters plasma from circulating plasma, acting as a continuous “soaker hose” to inflate the anterior chamber.
  - It is bordered anteriorly by the ciliary cleft and trabecular meshwork, and thus is adjacent to the region that is responsible for the outflow drainage of aqueous out of the eye.
  - If inflamed, it acts as a regional immune-surveillance site, harboring clusters of infiltrating lymphocytes.
  - It anchors the lens with zonular fibers arranged like the springs in a trampoline.
  - The CB musculature pulls on the zonular fibers in response to visual stimuli, altering the lens curvature and resultant refractive properties.

- When the CB is inflamed, all these functions are affected:
  - Aqueous production decreases.
  - Infiltrating lymphocytes form organized clusters that function as regional “nodes” for expansion of inflammatory activity.
  - The lens may subluxate or fall down completely.
  - Altered CB function can result in a globe that has too little fluid (hypotony from reduced production), or too much fluid (glaucoma from a drainage angle that is clogged with inflammatory debris, blocking aqueous outflow).

- In end-stage ocular disease the CB stops functioning. The eye then deflates and becomes atrophied and scarred, a condition called “phthisis bulbi.”

7. Uvea: Choroid
- A tremendous blood flow occurs through the network of choroidal vessels. A layer of wider vessels that opposes the sclera is adjacent to an inner complex of capillary-sized vessels, the choriocapillaris.
The choroid in the horse is responsible for supplying nutrition to the entire retina, except the immediate peripapillary area. Disruption of the blood-ocular barrier can result in exposure of retinal antigens to immune surveillance and leakage of blood between the choroid and the retina. When the blood-ocular barrier is compromised, infiltrating WBC gain access to intraocular antigens. Some of the most reactive antigens are found within the retinal pigmented epithelium (RPE), the retinal layer adjacent to the choroid.

8. Lens

Normally, the lens is a clear disc that is oriented in a vertical plane and held up by the zonular fibers, like a trampoline that is resting on its edge.
- The lens can also be compared to a disc-shaped candy (M & M or Junior Mint). The outer coating is similar to the lens capsule and the inner sweet is analogous to the cortex.
- The lens is nourished by the aqueous humor. The lens is transparent because it is maintained in a dehydrated state by the metabolic activity of the lens epithelial cells.
- If enzymatic activity or lens metabolism is altered, a cataract ensues.
- A lens that is diseased manifests as one or two problems: loss of position (luxation) or loss of transparency (cataract).
- Intraocular inflammation can cause both conditions as the supporting zonular fibers fail if the ciliary body is compromised, and the metabolic and enzymatic activity of the epithelial cells of the lens is affected by inflammatory activity in the aqueous.
- Lens cortical proteins are autoantigens that cause intense immune reaction and may further cause cataractogenesis if exposed to intraocular WBC.

9. Neurosensory Retina

The retina is adherent to the outer layers of the eye in only two places: a boundary ring around the optic disc and a ring “behind” the ciliary body.
- Disruption of the blood-ocular barrier can result in leakage of fluid between the loose plane that separates the choroid and the retina, causing retinal detachment and vision loss.
- The retinal layer called the retinal pigmented epithelium (RPE) contains abundant quantities of autoantigens, notably molecules called IRBP, S antigen, and CRALBP.
- Lymphocytes that are found inside the eye of horses with uveitis mount intense responses to these antigens. This activity contributes to retinal degeneration and vision loss in ERU.
- Visual signals from the retinal photoreceptor cells are relayed towards the brain by a large population of retinal ganglion cells that comprise the nerve fiber layer of the retina.
- Increased IOP in glaucoma causes interruption of axoplasmic flow in the optic nerve. This interruption eventually causes RGC death and vision loss. ERU is the primary risk factor for development of glaucoma.

Setting the Stage for Examination of the Equine Eye

Examination of the equine eye is not difficult! Unlike a reproductive or musculoskeletal exam, a thorough examination of the globe is noninvasive and requires only simple, inexpensive tools.

- Start creating an “exam room” in the field. Find a darkened area protected from the wind.
- Assemble all anticipated tools and equipment on a clean, elevated surface. This author uses folding dog-grooming tables for this purpose: www.champagnetales.com/ring.html.
- If diagnostic tests or a sedated examination are anticipated, have all materials at the ready and build an “equine ocular exam table” to support the horse’s head by stacking bales or rolling a barrel, or tote, into the examination area.

Instrumentation to Examine the Equine Eye

Most general equine practitioners use a direct ophthalmoscope for their examination of the equine eye. www.welchallyn.com/wafor/students/Optometry-
Students/Ophthalmoscope_2007_05_09.htm or www.heine.com/eng_US./PRODUCTS/PRODUCT-OVERVIEW/Instruments-d-Ophthalmologie/Direct-Ophthalmoscopes. The Welch Allyn line of products are in broad use in North America and a good coaxial instrument can be purchased for about $350.

- Another important tool for examination is a bright light source. A 3.5 V Halogen fiber-optic transilluminator costs less than $150 and can be ordered with a removable cobalt blue filter as an “attachment” for a direct ophthalmoscope. www.welchallyn.com/apps/products/product.jsp?id=11-ac-100-000000010102
  The halogen light source attaches to the handle of the direct ophthalmoscope and is used to illuminate the periorcular region and corneal surface, as well as transilluminate or retroilluminate the interior of the eye. Another handy item is a simple headband magnifying head loupe. The Optivisor® provides reasonable magnification of ocular detail; these devices are available through most veterinary distributors for < $50 www.doneganoptical.com/products/optivisor.

- A tonometer is a handheld instrument used to measure intraocular pressure. Two models are available. The Tono-Pen www.danscottandassociates.com/index.php?id=11971 measures IOP through applanation (the force required to flatten the ocular surface as the instrument is hand-tapped on the ocular surface) to measure IOP and the TonoVet www.jorvet.com/video-tutorials/tonovet-tonometer-for-easy-animal-eye-pressure-measuring uses a rebound system (an instrument calculation after a small magnetized probe is gently thrust by the tonometer on and off the ocular surface). This author has found the TonoVet to be most practical in field situations. Each of these instruments costs around $4000.

- Specialists use sophisticated, indirect ophthalmoscopy equipment to perform fundus examination. Simple, indirect fundus examination can be performed with a halogen transilluminator and a handheld 14 or 20 diopter lens. www.danscottandassociates.com/files/Product%20Photos/GPP/volk.pdf

- Clinicians with a special interest in ophthalmology will want to invest in a handheld slit lamp ($4000–5000, www.danscottandassociates.com/index.php?id=11336) for biomicroscopy and slit beam assessment of the ocular anatomy.

- One essential, inexpensive, but often overlooked tool for equine eye examination is a digital camera. Recording images of the eye is as important as taking an X-ray of a leg! Captured images can be magnified on the camera to show the client what the problem is. Sequential images can be shared with the owner to report progress and explain changes over time.

**THE EXAMINATION PROCESS**

An ocular examination starts with an unsedated general assessment of the whole animal, with particular care paid to body condition, general “bloom,” neurologic status, skin lesions, and any clues of systemic disease that might accompany or influence ocular issues. The examination then moves to the head and periorbit. Skull symmetry, sinus anatomy, ocular position, and gaze of both eyes are assessed. A basic assessment of cranial nerve function is performed. Pupils are inspected and compared, and a bright light source is used to test dazzle and papillary-light responses. Mildly threatening hand motions are used to assess menace responses of both eyes. The skin around the eyes is inspected closely for masses, alopecia, swelling, or inflammation. Blepharitis, tearing, and excessive nasal discharge are noted. The angle of the lashes on both upper eyelids is compared, as a drooping of one side may indicate ocular pain. The conjunctiva and third eyelids are inspected for color, masses, and inflammation.

Once a general assessment of the body and adnexa is complete, the examination moves to the globe itself. Many horses will need to be sedated for thorough globe inspection, and some will require regional anesthesia of the auriculopalpebral nerve to block blepharospasm. This author uses xylazine as a sedative for simple ocular examination of most horses. Detomidine is used if the horse is extremely painful, or requires deep corneal debridement, and/or insertion of an SPL system. Thorough globe examination is aided by pupil dilation. This is achieved by instilling 0.5 ml of tropicamide drops onto the ocular surface. A normal pupil takes about 20 minutes to dilate; this time delay should be factored into the examination plan.
The globe examination begins with a bright light source (transilluminator or slit lamp set on “spotlight” setting at one of the lowest illumination levels). All elements of the three-dimensional globe should be examined in a logical and systematic fashion with the operator consciously thinking about the anatomic region (i.e., “cornea,” “anterior chamber,” “iris,” “lens”) in sequence. When an abnormal finding is noted, the operator should localize it in terms of its geography on the anatomical region (e.g., “axial,” “paralimbal,” “equatorial,” etc.) and its depth on the parent structure (e.g., “anterior,” “subepithelial,” “subcapsular,” “posterior,” “mid-stromal,” etc.) and be prepared to record the pertinent findings in a similar fashion in the medical record, taking care to include a thorough description of the character and size of the lesion(s). If a slit lamp is available, a narrow beam of light is used to systematically scan “slices” of the anterior segment, looking for cellular infiltrates in the tissue or ocular media, and assessing tissue areas that are swollen, thin, or edematous, characterizing any abnormal infiltrates or masses from a three-dimensional perspective. The surface of the iris is inspected, as is the drainage angle behind the limbus. The lens is illuminated to look for cataracts or displacement.

An assessment of the posterior segment follows. Most practitioners will use a direct ophthalmoscope for this purpose. As the bulk of observable pathology is located in the peripapillary region, this is acceptable technique for field purposes. However, indirect ophthalmoscopy using a Finnoff transilluminator and handheld lens is a simple technique that will expand the observable geography of the interior of the eye. Specialists often augment these techniques with inspection of the fundus using a hand lens that is illuminated by an indirect ophthalmoscope mounted on a headband.

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**STALL-SIDE DIAGNOSTIC TESTS**

Stall-side tests may include tonometry, Schirmer tear testing (STT), fluorescein, rose Bengal ocular surface dye tests, corneal culture, and corneal cytology. In some cases, blood may be drawn for serologic or other analysis.

**Tonometry**

Tonometry is used to measure intraocular pressure. The author uses a rebound tonometer (TonoVet, cost @ $4000). Although the soft tapping pressure of the instrument tip on the cornea is tolerated without topical anesthesia, most horses are not very cooperative about having a piece of equipment the size of a small hammer very close to their globes. This author prefers to perform tonometry on patients who have been given light sedation (150–200 mg of xylazine, IV). Results are most consistent when the horse head is supported on a bale table in a normal resting position, and the upper eyelid motion is stopped by the use of an auriculopalpebral eyelid block.

**Schirmer Tear Testing**

Schirmer tear testing is used to quantify the volume of tear production. Fold the paper strip at the notch and then insert the notched end over the lower eyelid. Record how fast the strip is wet at 15, 30, and 60 seconds by the capillary wicking action of the paper strip, which has a metric ruler calibrated in millimeters. Normal tear production in horses is copious. Healthy horses have been reported to have a range of up to > 30 mm of wetting in one minute and 15–20 mm/minute in 30 seconds.

**Fluorescein Dye Testing**

Fluorescein dye testing is used to assess integrity of the corneal epithelium. The orange end of the paper fluorescein dye strip can be applied to the bulbar conjunctiva as this will allow dye to merge with the tear film where it will turn green. Alternatively, the orange end of the strip can be torn off, and put inside a 3-ml syringe and mixed with a small volume of sterile saline. The dyed saline is then sprayed on the ocular surface through the hub of a broken-off needle. Any area of the cornea that is devoid of epithelium will stain bright green. Subtle lesions are best viewed through a cobalt-filter blue light.

**Rose Bengal Dye Testing**

Rose Bengal dye testing is used to assess the mucin layer of the tear film. The red RB dye is irritating so the paper strip should be torn off, and put inside a 3-ml syringe and allowed to mix with a small volume of sterile saline. The dyed saline is then sprayed on the ocular surface through the hub of a broken-off needle attached to the syringe. Areas with abnormal tear film will stain a rosy pink pattern, which is often stippled or irregular. Corneas that have positive RB staining characteristics are suspect for keratoconjunctivitis sicca or for surface fungal colonization.

**Corneal Cultures**

Corneal cultures from corneal ulcers should be taken prior to cytology sampling. It is best to sample a cornea that has not been treated with topical anesthetic, but sometimes this is not possible due to patient resistance. Calcium alginate swabs are preferable to other sampling devices. Alternatively, a sample for in-house analysis can be taken by scraping a small portion of diseased/disrupted cornea with the sterile blunt end of a scalpel blade. The blade can then either (a) be used to directly apply the contents to bacterial culture plates by making a series of small “C”-shaped carvings into the growth agar, or (b) be dropped in sterile fashion into a tube of thioglycollate broth and placed in an incubator at 38 degrees C. If the broth becomes turbulent, the resultant growth can be plated out onto agar plates for identification and antimicrobial sensitivity analysis.
Corneal Cytology

Corneal cytology is a recommended diagnostic test for all significant corneal ulcers and also for some suspect neoplastic lesions. The sampling procedure is a simple skill that all equine practitioners should master. The horse should be sedated, and the mandible should be supported by a bale table. If the horse has severe blepharospasm, or is uncooperative, an auriculopalpebral nerve block should be performed. About 0.5 ml of topical anesthetic (Proparacaine® or Tetracaine®) is applied to the corneal surface through the hub of a broken-off needle attached to a small syringe. The blunt end of a scalpel blade is applied at a 45-degree angle to the target lesion, with the operator using the foil wrapper of the scalpel blade to hold the blade in a sterile fashion and cover the sharp end. A firm scraping motion is used to dislodge cellular material from the corneal surface. The material is transferred from the blade edge to the dry surface of two glass microscope slides. The scraping process is repeated several times and the slides are inspected to make sure that several areas of visible material that is at least 0.5–1 mm in diameter is observed. The slides are placed in a slotted plastic slide box www.heathrowscientific.com/catalog/product?deptId=MICROSCOPY+SUPPLIES&prodId=15982 and allowed to air dry. After the slides are dried, one is stained with Diff-Quik stain and the other is Gram stained. Clinicians are urged to gain comfort with interpretation of corneal cytology - the skills needed for most cases are simple and rapid access to this diagnostic information will provide key information for effective therapeutic decision making.

Cytology analysis should ask three questions:
19. Are inflammatory cells present? If so, what kind - PMNs, bands, eosinophils, mast cells?
20. Are infectious agents (bacteria or fungi) present or absent? If so, what are the staining and morphologic features?
21. Are there any foreign bodies or other noncellular elements present in the sample (plant material, crystals of calcium, parasites, etc.)?

Simple Ocular Imaging for Road Warriors

Digital Photography

The advent of high-quality, inexpensive, compact digital cameras has brought the capability of field imaging within the range of all practitioners.

A few tips for great ocular photography:
- The most important concept is an understanding of the autofocus system. If the camera is set on the program (P = automatic) setting with the macro option (flower icon) selected, the autofocus will be optimized for taking pictures of objects that are 12–20 cm away from the lens. This autofocus system is engaged when the shutter button of the camera is pushed halfway down. This action causes an infrared beam to be emitted from the camera. This beam bounces off of the object that is in the center of the camera viewfinder and is “read” by a computer inside the camera. The processor then adjusts the lenses and the light aperture for optimum imaging of the object in the center of the viewfinder. It signals the operator that it is focusing on the area of interest by projecting a bracketed outline on the viewfinder. If the camera shutter is depressed fully after the autofocus is engaged, and the distance from the camera lens to the eye has not changed at all, the image will be in sharp focus.
- Experimentation with a given camera model will demonstrate the optimum focal distance for imaging the eye. The autofocus will not engage if the camera is held too close to the eye. The operator will know this because the bracket will not appear on the viewfinder.
- Horses become restless if too much time is spent “setting up” an image, so the best practice is to take several images in rapid sequence, making sure the autofocus is operating for each one. The operator can then review the images on the viewfinder and decide if the quality is acceptable. If necessary, shots can be repeated, or slightly different angles can be taken to image the area of interest.
Photographs will be of the highest quality if they are taken indoors in a dark area with a flash. The operator must be aware of background and foreground detail that may impair quality - the corneal surface is glossy and reflective and it will pick up windows or other reflections that are present behind the operator.

The digital zoom feature should not be used when the picture is taken. However, the digital zoom feature is very useful to use after the image is obtained to demonstrate lesions to the owner. The clinician can use the camera “review” feature to scroll through the images on the LED screen on the back of the camera, selecting the best ones. Then the digital zoom and positional buttons can be used to center and enlarge the area of interest to fill up the LED screen. Owners can then look at the lesions in a magnified view. **Showing the problem to the owner on the camera LED screen is a very important part of the treatment plan.** It is hard for most owners to “see” lesions on the live horse, but it is easy for them to appreciate pathology on a camera screen. Treatment compliance and acceptance of the expense and effort involved in handling a tough problem will be enhanced by the stall-side review of images.

Images obtained in the field should be downloaded to a viewing computer at the end of the day. The images will have superfluous detail of the animal’s head that will need to be cropped with editing software. This task is easily performed with a variety of software programs (Apple iPhoto, Aperture, Microsoft Photo Editor, Adobe Photoshop). The detailed, magnified images can then be transferred to the medical record and/or emailed to the owner.

**Progressive photography** of lesions that are being treated or followed is important. Assessing progress (or worsening) of a lesion is difficult when the operator is relying on memory for judgment, but is straightforward when sequential images are compared side by side.

**Ultrasound of the Globe, Orbit, and Periorbit**
Ultrasound can be performed with standard machines that ambulatory clinicians use for reproductive or musculoskeletal evaluation. Ultrasound is appropriate for cases where the clinician is trying to check for orbital fractures, assess tissue density in a swollen eyelid to check for abscesses, or assess globe size in cases of exophthalmos or suspected orbital tumors. It is also useful for inspecting the anatomy inside the globe to look for evidence of cataract, lens luxation, intraocular masses, or retinal detachment. The author favors the use of a 7.5-MHz curvilinear probe for most ocular imaging, but also uses a 5.0-MHz linear probe for assessment of tissue behind the globe. A transpalpebral approach is suitable for most ambulatory cases. The author always performs ocular ultrasound with the horse’s head supported by a bale table, as this practice simplifies restraint. Images can be captured on a jump drive, or photographed directly from the machine screen. Clinicians should be aware that many referral institutions have access to **high-frequency ultrasound machines** that can obtain very high detail of ocular structures. Certain cases may benefit from referral for this procedure.

**Medical Record of Equine Eye Examinations**
An examination is never complete without a **medical record**: findings that detail various regions of the cornea and globe should be recorded. Many clinicians use premade outline drawings of the cornea, iris/pupil, lens, and fundus as templates on which to depict findings. Drawing can be supplemented with written detail. Reviewing ocular photographs will aid the description process for complicated findings.

A few tips:
- A STT strip makes a handy ruler to measure findings.
- For reference, the average width of the equine palpebral fissure is about 40 mm and the width of the cornea is about 35 mm. The height of the cornea is about 25–27 mm.
- Any ocular region that is circular or ovoid (iris, cornea, optic disc, observable fundus) can be related to a clock face where findings can be compared to their “clock-hour” position. Findings can also be related to a point of reference like the optic disc (e.g., “the fundic lesion is located ½-disc diameter away from the 4:00 border of the optic nerve and occupies a region that is ¼-disc diameter in width”).
A few vocabulary words reflect common anatomic descriptions. “Axial” describes a line bisecting the center of the cornea and the rear of the globe. “Limbal” describes the intersection between the cornea and sclera. “Temporal” describes the “outside” of the eye (what might be thought of as “lateral”). “Nasal” describes the “inside” (often also called “medial”). The lens has a “capsule” that acts an outer skin and a “cortex” (everything inside the capsule, but thought of in terms of anterior and posterior regions). It also has an “equator,” which is a term that describes the outer thin edge of the vertical disc-shaped structure which is usually covered by iris.

Terminology for areas of altered color or appearance on the cornea, lens, or fundus may be quite descriptive. Examples of words used to describe the shape of ocular findings include: floriform, stellate, focal, pinpoint, speck-like, vermiform, serpiginous, dendritiform, staghorn, geographic, elliptic, and coralliform. Examples of terms that may be used to describe the character of an ocular opacity found within a normally transparent structure include lacy, steamy, stippled, smoke-like, opalescent. Ophthalmic medical record detail will bring out your inner creative writer!

**CONDITIONS COMMON TO VARIOUS EQUINE LIFE-STAGES**

Practitioners should be alert to detecting the following conditions as they examine horses through the stages of their lives:

**Neonatal Congenital Conditions**

Present at birth
- Microphthalmos
- Lacrimal puncta agenesis or duct atresia
- Congenital strabismus
- Dermoids
- Aniridia
- Persistent papillary membranes (PPMs)
- Anterior segment dysgenesis (may not be noticed till maturity)
- Congenital cataracts
- Coloboma
- Persistent hyaloid artery
- Congenital glaucoma or retinal detachment (rare)

**Neonatal Acquired Conditions**

Follow birth process or develop in the first few days of life
- Entropion (be vigilant for this in sick foals)
- Subconjunctival hemorrhage
- Retinal hemorrhage
- Uveitis secondary to septicemia
- Jaundice secondary to neonatal isoerythrolysis (scleral icterus)
- Various manifestations of hypoxic ischemic encephalopathy (HIE)
- Ulcers: uncomplicated, melting, infected, or persistent erosions
- Secondary manifestations of adenovirus, botulism

**Pediatric Conditions**

Conditions that are found in sucklings or weanlings
- Blunt head trauma - concussive during pasture roughhousing or training accidents - can cause acute blindness
- Blunt globe trauma - as above
- Sharp facial or lid trauma
- Uveitis secondary to *R. equi* or strangles
- Corneal ulcers
- Vitiligo
**Mature Horse Conditions**

Conditions that commonly occur during adulthood

- Trauma: blunt and sharp
- Corneal ulcers
- Uveitis
- Squamous cell carcinoma
- Sarcoid

**Geriatric Horse Conditions**

These conditions are often found in aged patients

- Sinus disease with ocular manifestations
- Periocular neoplasia
- Indolent ulcers
- Cataract
- Glaucoma
- Insidious uveitis
- Vitreal syneresis
- Asteroid hyalosis/synchesis scintillans
- Senile retinopathy
- Proliferative optic neuropathy

**References**

ORBIT AND PÆRIORBIT

The most common problems seen in the periorbital region involve trauma.

Chronic facial deformity reflecting past trauma is often encountered in mature horses in the form of dents or abnormal contour of the facial bones, especially in the sinus region. Practitioners must be alert to any acute facial deformity that could indicate recent fractures of the frontal, temporal, or zygomatic bones, sequestra formation, or local abscessation in the soft tissue. Foreign body, abscess, or fracture detection may require imaging with ultrasound, radiology, computed tomography, or MRI. Occasionally sinus or orbital trauma will compress the extraocular muscles and other soft tissue structures around the globe, causing a strabismus (deviant direction of gaze) and altered globe position.

Horses with acute sinus fractures or sinus infections secondary to trauma or dental disease are at risk for orbital cellulitis. Sinus fracture cases require aggressive antibiotic therapy for several weeks and/or sinus trephination and lavage. Horses with infections secondary to dental disease may require tooth extraction and/or sinus surgery. Horses with impaired eyelid function secondary to periorbital oculomotor (CN VII) nerve trauma are at risk for corneal ulceration secondary to exposure keratitis. They require frequent applications of topical lubricants and may need to have the corneal surface protected with a temporary tarsorrhaphy while nerve function is impaired. Horses that have sustained head trauma with hyphema where more than half of the anterior chamber is filled with blood have a guarded prognosis. If bleeding recurs the eye has a poor prognosis and may become phthisical. Horses that show any restriction or deviation in eye position, or movement, following blunt trauma are candidates for referral. Horses that have sustained enough trauma to the head to cause optic neuropathy may lose vision in the affected eye.

Horses may occasionally present with exophthalmos caused by orbital tumors or masses. Exophthalmos can be easily differentiated from buphthalmos by measuring globe diameter with transpalpebral ultrasound with a 5.0- or 7.5-MHz probe. Ultrasound of the orbit can also give an indication of the consistency and dimensions of any abnormal tissue behind the globe. The most common tumors that have been reported in the orbit are neuroendocrine tumors and extraadrenal paraganglioma. Other neoplasias that have been reported in the orbit include squamous cell carcinoma, anaplastic sarcoma, and lymphosarcoma. Rare reports of malignant rhabdoid neoplasia, fibroma, angiosarcoma, adenocarcinoma, and juvenile neuroectodermal tumor exist. While some orbital neoplasias are solitary, others can spread to, or originate from, other regions within the skull, including the sinuses, periorbital tissues, and pharynx. Imaging of these areas with endoscopy and computed tomography is encouraged prior to surgery for prognostic reasons. Practitioners who attempt enucleation of horses with presumptive orbital tumors should be advised that hemorrhage during removal of orbital neoplasia may be excessive, particularly if the tumor is an extraadrenal paraganglioma.

EYELIDS AND ADNEXA

The most common problems seen in the eyelid region involve trauma, allergy, and neoplasia.

Eyelid margin tears commonly occur when stabled horses rub their heads on prong-like objects and avulse the eyelid margin. The incidence of eyelid trauma can be greatly reduced if owners tape up the J-shaped bases of the handles of stall buckets. Repair of eyelid lacerations in the field will be facilitated if a “surgery table” is constructed for head support using stacked bales of hay or shavings. A bright LED headlight, or tripod halogen light, positioned near the patient will aid visualization. Practice tips for effective closure include cleansing the wound with liberal application of 2% Betadine solution, minimal sharp debridement with a small pair of Metzenbaum scissors, and closure of the subcutaneous tissues with knots that are buried and placed sparingly. Precise apposition of the torn tarsal margin is critical and should be achieved with careful placement of a figure-of-eight suture that does not penetrate the conjunctiva or have tags that rub on the cornea. The author obtains excellent cosmetic results with the use of 4-0 to 5-0 absorbable suture for all layers, placed using a 5½-inch Olsen Hegar needle holder and a
small pair of forceps. Repair of chronic lacerations that are several days old is often successful if done with great care for the preservation of anatomy.

Owners often call for emergency examinations of horses with very swollen eyelids. Many of these horses are suffering dramatic acute eyelid edema related to insect allergy or other seasonal irritant. If edema is the only problem, the pupil of the affected eye will be midrange and reactive, and the surface of the cornea will be transparent and show no fluorescein dye uptake. This problem responds quickly to topical corticosteroid application, but must be differentiated from chemosis accompanying a corneal ulcer or a deeper problem in the globe.

Neoplasia of the eyelid or periocular region is often a difficult challenge for the practitioner. The most common tumors are squamous cell carcinomas and sarcoids, though melanoma, lymphoma, fibroma, mast cell tumors, and other neoplasias may occur. Patients at risk for squamous cell carcinoma include draft horses (particularly Belgian Drafts), Haflingers, and color dilute breeds like Paints, Pintos, Appaloosas, and Hackney ponies that lack pigmentation in the lid region. Exposure to a high level of solar radiation is an additional risk factor. Field therapy for smaller eyelid tumors should include careful excision followed by local immunotherapy, cryotherapy, or infiltration of the region with chemotherapeutic agents (cisplatin or 5-fluorocytourasil). However, many patients with periorbital neoplasia present when the tumors are advanced. Horses that present with lesions that occupy more than one third of an eyelid margin may be best served by prompt referral to a veterinary ophthalmologist who can perform excision with reconstruction and administer adjunctive therapies. Current adjunctive therapies include cryotherapy, hyperthermia, photodynamic therapy, brachytherapy, and intrallesional chemotherapy. Early referral gives the best chance of good long-term results.

Nictitans
The most common problems seen in the nictitans are trauma, neoplasia, and burdock pappus bristle keratopathy.

Minor tears of the nictitans may not cause a clinical problem. Lacerations that cause the nictitans to evert intermittently outside the eyelid margin may require excision or surgical revision of the leading edge of the third eyelid.

The most common neoplasia affecting the nictitans is squamous cell carcinoma. This tumor presents as a raised or ulcerated, reddened region of abnormal mucosa. Most SCC of the nictitans originates on the leading edge of one third eyelid. The extent of the tumor may not be appreciated until the nictitans is everted with forceps for examination. Affected horses may show profuse, mucopurulent discharge. Occasionally, horses will present with bilateral SCC of the nictitans, particularly Belgian Drafts and Haflingers. Lymphoma and melanoma have also been reported as originating on the nictitans.

Treatment of tumors of the nictitans is either local excision of the mass (for small masses on the leading edge) or complete excision of the nictitans (for larger masses). Surgery may be performed as a standing procedure in the sedated horse. Practice tips for successful removal include supporting the horse’s head on a table made of stacked bales, heavy sedation, doing the surgery within stocks, and judicious infiltration of the mucosa of the nictitans with local anesthetic using a tuberculin syringe attached to a 25-ga needle. The base of the nictitans is attached to a large pad of orbital fat which will be pulled out of the fornix during the surgery. Complications from orbital fat prolapse are rare if the fat pad that is attached to the gland is amputated along with the nictitans.

Burdock pappus bristle keratopathy is often seen in the fall in horses that live in temperate climates like the Northeast where burdocks are a common pasture weed. The tiny bristles of the mature plant can become embedded in the mucosa of the nictitans that apposes the cornea or in the cornea itself. A hallmark of this condition is a cobblestone appearance of the inner mucosa of the nictitans, and adjacent scarring and ulceration of the apposing naso-ventral corneal epithelium. Treatment involves topical anesthesia of all affected surfaces, eversion of the nictitans, and debridement of both the nictitans mucosa and the affected cornea. Cytology samples should be taken to check for associated infection. Practice tips for successful resolution include using the open jaw of a small hemostat as a scraping instrument for the mucosa, and following hemostat debridement with judicious rubbing of the mucosa with a bit of gauze stretched over a gloved finger.
**NASOLACRIMAL SYSTEM AND CONJUNCTIVA**

Dacryocystitis is a common problem in the field. Retrograde flushing of the system can be performed using a variety of thin tubes that attach to a syringe containing wash fluid including plastic teat cannulas, IV catheters, tomcat catheters, and intranasal vaccine applicators. Stubborn blockages can benefit from passing a small 5-Fr nasogastric feeding tube (Mila International, NG522S, 5 Fr x 55 cm) as far up the duct as it will pass. Practice tips for smooth flushing include the use of an LED headband light for illumination of the nasal puncta, application of a small dab of local anesthetic gel onto the nasal mucosa around the puncta and inclusion of a small amount of local anesthetic in the first volume of sterile wash that is flushed into the duct. Cases that present seasonally with a lot of mucopurulent discharge may benefit from the topical application of an ocular preparation containing 1% hydrocortisone or 1% dexamethasone that will medicate the system as tears drain down the duct.

Conjunctivitis is also a common field problem. Practitioners must understand that conjunctivitis is rarely a primary diagnosis - it is usually a secondary symptom of more widespread ocular inflammation. Practitioners should look for allergic, infectious, immune-mediated, neoplastic, or parasitic disease elsewhere in the eye as the inciting cause.

Prolapse of orbital fat is a condition that is occasionally seen following trauma or surgery. Weakened episcleral fascia allows fat to herniate under the bulbar conjunctiva or the conjunctiva of the nictitans, forming a fluctuant mass that may be mistaken for a tumor. The character of prolapsed fat is soft and pillow-like, much like a marshmallow. Cytology on material aspirated from the fluctuant region of conjunctiva will differentiate this benign condition from neoplasia. The prolapsed fat is not harmful, but will have an undesirable cosmetic appearance; an ophthalmologic surgeon might be able to resect the prolapsed fat and suture the underlying fascia to restore good cosmesis.

**ORBITAL IMAGING**

Orbital imaging has advanced considerably in the last decade. General practitioners are usually limited to using ultrasound and digital radiology to assess this region, but many universities and specialty hospitals are able to produce excellent three-dimensional images of this complex region with computed tomography. Ultrasonography can be used to assess periorbital masses, eyelid swelling or abscess, orbital fractures, and look for foreign bodies. Computed tomography produces the lifelike reconstructions of the orbit, and gives excellent detail when there is a need to image the periorbital sinuses, an orbital mass, or orbital fracture. Practitioners should offer patients with these problems the option of referral to a university for computed tomography, but must make the owners aware that the procedure is fairly expensive, and horses must undergo general anesthesia for this type of imaging examination.

**SURGERY OF THE ORBIT**

Many general equine practitioners offer enucleation surgery as an option for their patients. In recent years, many clinics have been performing enucleation as a standing surgery, restraining the horse in stocks and providing deep anesthesia through either a CRI infusion of sedation, or intermittent bolusing of sedatives. The author has performed over 50 enucleations in standing patients with good results. Several reports have been published that detail practical techniques for this procedure.

In addition to advanced imaging, referral hospitals offer options for certain types of orbital surgery, including orbital exploration, repair of orbital fractures, and enucleation, including exenteration (removal of all orbital contents). A few horses have undergone orbital implantation where a cosmetic conformer (artificial corneal scleral prosthctic) is fitted to an orbital implant that is made of hydroxyapatite.

**REFERENCES**


Tracks of My Tears: Equine Corneal Disease
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**GENERAL KEY POINTS**
Veterinarians are frequently called out to examine and treat horses with corneal disease. A normal cornea is transparent and covered with smooth epithelium. Abnormal findings may include disruption of the normally smooth ocular surface (ulceration), and/or opacification of the normally transparent corneal layers.

Trauma is the most common cause of disruption of the equine ocular surface, but neoplasia, foreign bodies, and some immune-mediated conditions may also cause ulceration. Infection, mechanical damage, and host proteinase activity are frequent complications that complicate healing. Clinicians presented with a horse that has ulcerative corneal disease should perform diagnostic tests to determine the etiology of the condition and prescribe treatment based on analysis of those tests. Therapy must be administered several times a day and multiple recheck appointments may be required for successful resolution. Certain cases may require intense medical or surgical therapy that is best provided by referral institutions. An understanding of the differential diagnosis of ulcerative corneal disease, coupled with results of clinical diagnostic tests and response to rational treatment will dictate case management and optimize the chance for successful outcomes.

Numerous factors may cause loss of corneal transparency. Opacity of the epithelium may result from neoplastic transformation of the surface or focal edema. Opacity of the stroma may reflect fibrosis, cellular infiltrate, foreign bodies, neoplasia, or focal edema. Opacity of the deepest aspects of the cornea may reflect changes in the contour of Descemet’s membrane or edema accompanying endothelial dysfunction.

**KEY CLINICAL DIAGNOSTIC POINTS**
- Digital photography of corneal problems is essential for documenting disease and assessing response to therapy, especially on deep, or melting, ulcers. Many models of inexpensive digital cameras take very good images. Lecture 2 (Ophthalmology for Equine Road Warriors, Part 2: Diagnostic Testing and Imaging) contains more information on digital photography.
- Head support will optimize the ocular exam and facilitate the collection of diagnostic samples. A simple “table” can be built with bales of hay or shavings. Horses with very painful eyes may require sedation and periocular nerve blocks for completion of the examination and diagnostic tests, as well as administration of stall-side treatments or device insertion.
- Fluorescein dye tests for integrity of the corneal epithelium. Rose Bengal dye tests for integrity of the mucin layer of the tear film. Both tests provide needed diagnostic information. Rose Bengal can be irritating and should be diluted by tearing off a piece of dye strip and mixing it with saline in the barrel of a small syringe. The dyed saline can be dripped onto the ocular surface.
- A Schirmer tear test (STT) should be performed on all eyes that appear to have dull or dry corneal epithelium. Normal equine corneas have an abundant tear film that will soak over 15 mm of the STT strip in less than 30 seconds.
- Cytologic analysis is beneficial for all but the most superficial corneal erosions. Collection of corneal cells for cytology should be done with a Kimura spatula or the blunt end of a sterile scalpel blade - samples should be spread on 2-3 slides which are then stained with Diff-Quick and Gram stains. Cytology should ask three questions:
  - Are inflammatory cells present? If so, what kind - PMNs, bands, eosinophils, mast cells?
  - Are infectious agents (bacteria or fungi) present or absent? If so, what are the staining and morphologic features?
  - Are there any foreign bodies or other noncellular elements present in the sample (plant material, crystals of calcium, parasites, etc.)?
Culture is indicated for deep or persistent ulcers in addition to standard cytologic analysis. Culture samples should be obtained before cytologic samples and debridement, and may be plated directly or transported in broth which is then plated out if it becomes turbid with microbe growth.

Vascularization of the cornea occurs in many disease processes. Superficial vessels are individual, long-branching vessels that lie within the epithelium or anterior stroma. Deeper, mid-stromal vessels are short and straight and present as a brush border along a portion of the limbus or along the entire circumference. Very deep vessels that rest on the Descemet’s membrane interface are individual, long-branching vessels.

Descemetoeceles (very deep ulcers) will show a crater-like contour and lack of fluorescein stain uptake centrally. Eyes with descemetoeceles are at risk for perforation: all manipulation should be done with caution.

Host proteinases play an important role in the pathogenesis of corneal ulcers. Elevated levels of matrix metalloproteinases (MMPs) and serine proteinases such as neutrophil elastase (NE) are present in the precorneal tear film of eyes with corneal ulcers. While some tissue lysis and remodeling is essential for corneal healing, excessive proteinase activity can lead to keratomalacia or melting. Autologous serum is an effective treatment that contains high levels of growth factors and anti-proteinases. Some clinicians also use other anti-proteinase therapies like EDTA or 5–10% acetylcysteine.

**Key Etiologic and Pathophysiologic Points**

**Cornea:** The cornea consists of several layers of tissue with an average total thickness of about 1 mm; thinnest in the center.

- The outer layer is a relatively impermeable epithelium which is richly innervated and very thin (0.14 mm and 6–8 cell layers thick) with an underlying basement membrane. The epithelium is covered by the precorneal tear film that some anatomists consider a “fifth layer” of the cornea.
  - Healing of noninfected superficial defects in the epithelium usually is rapid and occurs initially in a “sliding leapfrog” motion of adjacent cells to cover the adjacent wound bed and then later by basal cell mitosis.
  - Healing of deeper defects where the basement membrane is disrupted involves reepithelialization, followed by a longer period of remodeling of the deeper layers.

- The next layer of the cornea is the stroma which is the thickest layer, comprising over 90% of the corneal thickness. Stroma is composed of type I collagen fibrils that are arranged in a parallel lamellar lattice pattern. Disruption of the lattice causes opacity.
  - Healing of stromal defects involves a balance of resorptive remodeling (facilitated by the proteinases that are released from bacteria, corneal cells, and infiltrating PMNs) and restorative repair where fibroblasts lay down collagen to fill in the defect. Successful healing of defects is followed by several months of collagen remodeling that may return the tissue to a degree of its original transparency. In deep lesions, or lesions where healing is delayed, the collagen is laid down in a thick, random fashion, making an opaque scar. In severe cases, remodeling by proteinase activity is excessive, resulting in keratomalacia (melting) or perforation.

- The next deepest layer of tissue is a thin basement membrane called Descemet’s membrane.

- The final layer of the cornea is a very thin monolayer of cells, the endothelium. This layer of cells has a Na+–K+ activated ATPase-dependent electrolyte pump that constantly works to keep the corneal stroma relatively dehydrated. Disruption of the normal pump activity results in edema of the endothelium and overlying stroma that can be permanent.

**Common Problems of the Cornea Include**

**A. Superficial Corneal Erosions**

Erosions are defects that do not break into the stroma. If these do not get infected, they heal quickly, without visible scars. Topical mydriasis and antibiotic therapy are indicated.
B. Superficial Keratitis
Superficial keratitis may present as punctate areas of stain uptake, as focal vascularization, as pigment deposition, or as focal superficial opacities. **Punctate keratitis** may have a herpetic, fungal, or an idiopathic etiology. The lesions may be painful or comfortable. Epithelium shows fluorescein stain uptake in a dot-like pattern that is scattered over the corneal surface. A trial of topical idoxuridine may improve the condition. Topical NSAIDs, especially 0.1% diclofenac, may be very helpful. Some forms of keratitis will respond to topical NSAIDs or antivirals, while others respond to topical antibiotics.

C. Ulcerative Keratitis
**Ulcers** are defects that extend through the corneal epithelial layers and basement membrane into the stroma. Healing of these defects is a balancing act: ideally, tear film proteinases remodel the stromal defect and native fibroblasts restore stromal integrity. Bacterial or fungal infections, as well as various host factors, may tip the balance towards excessive resorption, resulting in melting of stromal collagen or even perforation of the globe. Ulcers are very painful and are accompanied by secondary uveitis, so the syndrome is complicated by patient objection to topical therapy. Adjunctive surgical therapy may involve debridement or keratectomy. Complex cases may need conjunctival grafts, amniotic membrane grafts, or tarsorrhaphy and thus may best be handled as referral cases. Very serious cases may require corneal transplantation by penetrating keratoplasty (PK), penetrating lamellar keratoplasty (PLK), or deep anterior lamellar keratoplasty (DALK).

**Ulcers with bacterial infection** can be diagnosed by cytology. Initial therapy choices are dictated by the type of bacteria seen on slides, and later may be adjusted according to clinical response and results of lesion culture/sensitivity. Therapy is intense, usually 4–6 x per day. Antibiotics are combined with mydriatics and topical anti-proteinases. Systemic NSAIDs help control pain. Subconjunctival injection may be used to supplement topical therapy. Treatment of cooperative patients without obvious keratomalacia may be via ointments administered at home, and resolution may be straightforward. Treatment of fractious patients, or patients with deep defects, may be via liquid medications administered through an SPL tube at home or at a referral hospital. Frequent monitoring will be necessary until it is clear that healing is occurring. The most common antibiotic drugs used on bacterial keratitis are fluoroquinolones (ciprofloxacin, ofloxacin, moxifloxacin), chloramphenicol, cephalizin, tobramycin, gentaminic, and amikacin (Link to Drug Table Dwyer). Atropine application should be to effect. Topical antiproteinase therapy using serum application is routine and may include a combination of other MMP inhibitors such as EDTA or acetylcysteine. Debridement should be judicious, but may need to be repeated weekly.

**Melting ulcers**: In some cases an ulcer may be sterile, but host proteinase activity is severe enough to cause corneal “melting” that threatens globe integrity. In other cases, corneal melting may be related to bacterial infections, particularly those caused by beta-hemolytic *Streptococcus* spp., or *Pseudomonas aeruginosa*, as both of these infections progress rapidly and incite extensive collagenolysis. Melting ulcers are expensive and time consuming as treatment must be immediate and aggressive. Outcome may be optimized if the horse is sent to a referral center where anti-infective and antiproteinase therapy (serum, EDTA, acetylcysteine, ilomastat) is administered every 1–2 hours around the clock.

**Fungal keratitis**: Ulcers with fungal invasion are common in humid southern climates. While less common in northern or desert regions, reports of fungal keratitis have increased in these areas in recent years. The presence of septate branching hyphae on a corneal cytology sample is diagnostic, but sequential cytology samples may be needed to discover fungal elements, and treatment is often begun if the index of suspicion for fungus is high. Antifungal sensitivities vary from one region to the next, so it is helpful to know what medications have been most effective in a specific geographic area. Fungal infections are very painful and the host inflammatory reaction is extreme. Fungi have the ability to tunnel down to Descemet’s membrane where topical drug activity may be limited. Prognosis is always guarded; early surgical intervention may be necessary for resolution. Surgical treatment involves keratectomy with conjunctival grafting or corneal transplantation. Topical antifungal therapy may involve the use of voriconazole, miconazole, natamycin, itraconazole, or silver sulfadiazine. Systemic antifungal therapy may be instituted using fluconazole or itraconazole.
Nonhealing indolent ulcers: Shallow ulcer defects may fail to heal in some older horses if the epithelium does not generate a normal basement membrane for secure adherence. These cases may be helped by debridement with a swab or blunt blade, or temporary tarsorrhaphy. If a noninfected ulcer does not heal in two weeks, diamond burr debridement with an AlgerBrush II® or a superficial linear keratotomy may stimulate healing. Both forms of debridement create small defects in the superficial stroma and thus provide a lattice for adherence of new epithelial cells. It is important to perform cytology on any nonhealing lesion to rule out infection before attempting one of these procedures.

D. Corneal Tumors
Squamous cell carcinoma is the most common tumor of the corneal surface, especially in Appaloosa, Paint, or draft breeds. The limbus is a common area of tumor. Other tumors include melanoma (most common in gray horses), hemangioma, hemangiosarcoma, mast-cell tumors, and lymphosarcoma. Prompt referral for surgery, with added adjunctive therapy like beta irradiation, cryotherapy, photodynamic therapy, or laser ablation is advised. Enucleation may be the best treatment for some cases.

E. Corneal Foreign Bodies
Horses occasionally present with foreign bodies embedded in the stroma or epithelium. Superficial foreign bodies can often be “wicked” out with a sterile Q-tip, flushed out with a pressure lavage of saline pushed through a broken-off hub of a 25-ga needle, or “scooped out” by careful undermining with the bevel of a 20-ga needle or small 2-mm biopsy punch. The remaining ulcer should be swabbed and lavaged with 2% povidone iodine/saline solution and treated medically. Very deep or penetrating foreign bodies must be referred for surgery and supportive care.

F. Eosinophilic Keratoconjunctivitis
Eosinophilic keratoconjunctivitis is a condition of probable immune-mediated etiology where a horse presents with limbal granulation tissue, chemosis, mucoid discharge, and/or limbal, axial, or paraxial corneal ulcers that may have a rubbery texture, or may have white or yellow raised plaques with gritty chalk-like material embedded along the margins. Cytology will reveal an abundance of intact eosinophils and loose eosinophilic granules and also may show scattered mast cells. Therapy includes debridement/debulking of the plaques (possibly using an AlgerBrush II® battery-powered tool), atropine, antibiotics, topical mast-cell inhibitors, and topical steroids. Note that this is the one condition where steroids are often used in the face of ulcerated epithelium - this therapy is appropriate, but must be monitored closely. Many clinicians have found that a tapering dose of systemic steroids in addition to systemic NSAIDs is beneficial. Recent reports on the addition of a systemic, oral human antihistamine (cetirizine or Zyrtec®, 0.4 mg/kg PO BID) have been encouraging.

G. Burdock Pappus Bristle Keratopathy
Horses that live in regions where burdock is a common pasture weed often present in the fall with tiny pappus bristles embedded in the cornea or nictitans. Large, burr-like burdock thistles are commonly found tangled in the tail and mane. Affected horses present with signs of corneal ulceration or erosion, particularly in the medial canthus under the nictitans. The tiny burdock bristles are not visible in field conditions, but in chronic cases there may be vessel patterns on the nictitans conjunctiva or on the cornea that “point” to their location. All suspect areas should be debrided. Nictitans debridement is facilitated by everting the whole membrane with a small towel clamp or hemostat, and gently scraping the conjunctiva with the serrated edge of a sterile hemostat until it bleeds. Resolution is prompt if the bristles have been completely removed. Treatment involves topical atropine and antibiotics, plus systemic NSAIDs.

H. Calcific Band Keratopathy
Some cases of chronic uveitis are complicated by mineralization of the cornea with deposits of calcium. This “calcific band keratopathy” may be related to repeated topical steroid application. Gritty plaques of Ca+ are deposited in the corneal epithelium and upper stroma in the central axial region where the lid aperture exposes a central band of corneal tissue. Often the plaques protrude through the epithelium and are associated with erosions and ulceration. Removal of the irritating deposits may require superficial keratectomy and/or chelation with topical 2% EDTA. Recurrence is common.
I. Nonulcerative Corneal Disease - Stromal Abscesses

One of the most serious corneal conditions that presents without ulceration of the epithelium is a **stromal abscess (SA)**. Stromal abscesses are seen as single or multiple, non-staining, focal, fluffy, yellow to tan-white densities which reflect microabscesses located deep within the corneal stroma. These focal lesions are very painful and are usually accompanied by an intense ingrowth of deep vessels from the closest region of the limbus. Many of these lesions have been found to contain fungal hyphae, so aggressive antifungal therapy is advised in addition to antibacterial and mydriatic treatment, and systemic NSAIDs. Superficial SAs may respond to intense topical therapy, but deeper SAs may be completely refractive to medical therapy or debridement, and many patients with SAs end up requiring enucleation if they are not treated surgically at a specialty hospital. Surgical options for SAs include several different types of corneal transplantation procedures including penetrating keratoplasty (PK), penetrating lamellar keratoplasty (PLK), or deep anterior lamellar keratoplasty (DALK). Outcome is improved if referral is made early in the course of disease, and if the referral institution has broad experience in treating equine patients.

J. Non-Ulcerative Corneal Disease - “Immune-Mediated Keratitis” (IMMK)

Less serious, but still problematic transparency changes are seen in the group of conditions known as the **immune-mediated keratopathies**. This group of diseases is poorly understood. The variants termed IMMK demonstrate focal or diffuse regions of opacity limited to the stroma or endothelium that retain an intact epithelium and thus do not take up ophthalmic stains. Many of these conditions are recurrent. Confocal microscopy of opaque regions usually reveals an infiltrate of lymphocytes and plasma cells within the affected stroma and an absence of infectious elements. Although some cases are bilateral, most are unilateral, and a careful history may reveal a previous episode of trauma in the affected eye. The observed opacity may reflect a cellular immune reaction to antigens expressed within the stroma. The intensity and regional pattern of observed opacification often varies over time. Areas that were previously opaque may clear only to have other geographical regions of the cornea lose transparency. Often there is vascularization of the opaque areas that will also change pattern over time. Pain is variable - some horses never exhibit pain while others may have episodes of severe pain that accompany increasing opacity.

**Key Therapeutic Points**

- **Debridement of the cornea** should be routinely performed in all but the most superficial ulcerative conditions. It can be as simple as swabbing the lesion with a sterile Q-tip, or as involved as actually cutting away some cornea with a # 15 or # 63 blade. Opinions vary on intervals between debridement sessions, but reassessment at weekly intervals is common.

- The **AlgerBrush II®** is an inexpensive, battery-powered tool that is being used with increasing frequency by equine ophthalmologists for light debridement of selected corneal lesions. It works like a miniature Dremel tool when it is fitted with a round diamond pterygium burr. Data are accumulating as to the best cases to select for burring, but good success has been reported in cases of indolent ulcers and eosinophilic keratitis. [www.algercompany.com/brush](http://www.algercompany.com/brush)

- **Subconjunctival injections** of antibiotic or antifungal drugs are occasionally used as adjunct topical ulcer therapy. Injection administration is safest if the horse’s head is supported by a table made of bales, and the handler, who is standing on the contralateral side of the horse, uses the ear of the horse to tilt the top of the head away from the clinician, thereby exposing target sclera.

- **Application of topical anesthetic** via sterile Q-tip and a small volume squirted onto the target surface with a small syringe fitted with the hub of a broken-off 25-ga needle will facilitate subconjunctival injections and debridement or inspection of the nictitans.

- **Lavage catheters (SPL tubes)** are useful ways to deliver medication to the surface of the eye, especially in fractious horses. Mila International ([http://milainternational.com](http://milainternational.com)) supplies catheters in two sizes that have swedged on tubing that is very easy to insert. Lecture 6 (Coping Strategies: Use of Subpalpebral Lavage Systems and Management of Blind Horses) covers the use of these devices.
Management of IMMK cases requires lifelong monitoring. Treatment may involve systemic NSAIDs and topical antiinflammatory therapy for horses that present with pain or increasing areas of opacity. Some horses will respond well to topical NSAIDs (diclofenac); others may be helped by topical immunomodulators (compounded cyclosporine [2%] or tacrolimus). Topical steroids (prednisolone acetate or dexamethasone) help some cases, but the use of these agents requires close monitoring, as the presence of an undetected infectious agent deep in the stroma can never be fully ruled out, and topical steroids can exacerbate a flare-up of infectious keratitis.

KEY PROGNOSTIC POINTS

- Corneal ulcers can be simple traumas that resolve uneventfully or progressive problems that are among the most complicated and expensive that horses experience. Often, prompt assessment and aggressive therapy ward off disaster. Practitioners should commit to seeing painful eyes on the date of occurrence and should perform appropriate diagnostic tests to make rational treatment choices.
- Very painful, large, deep, or melting ulcers may best be handled at referral institutions. Early referral/consultation may result in the best outcome.
- The prognosis for fungal keratitis is guarded: successful resolution often requires surgical intervention. Referral is encouraged.
- IMMK cases rarely resolve, but also rarely advance to the point where they cannot be managed. Observed corneal opacities will ebb and flow. Owners should be coached that these horses require lifelong monitoring, and may require intense treatment if the eye is very painful or opaque.

REFERENCES

The Many Faces of Uveitis
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INTRODUCTION
- Recurrent or persistent uveitis is not one disease, but a whole umbrella complex of diseases.
- Uveitis is the leading cause of blindness in horses in the world.
- Recurrent or persistent equine uveitis is immune mediated. The genetics of a horse’s immune system contributes to individual susceptibility. Pathogenesis of disease includes a Th-1 helper immune response. Etiology may involve autoantigens and/or microbial infections.
- Systemic leptospiral infection is associated with numerous cases of recurrent classic uveitis.
- Certain lines of Appaloosa and European warmbloods are at risk for persistent, insidious uveitis.
- New therapies aimed at controlling the aberrant immune response are under development. Recent research has focused on implanting devices that deliver drugs into the eye that modify the immune response, and mediating disease by removing ocular components that may provoke recurrence. Response to treatment is variable, and long-term visual prognosis is guarded.

KEY CLINICAL DIAGNOSTIC POINTS
By definition, uveitis is inflammation of the uvea of the eye (iris, ciliary body, and choroid). The complex of diseases known as “equine recurrent uveitis (ERU)” refers to intraocular inflammation that recurs or persists causing various degrees of inflammation, scarring, degeneration, and dysfunction of multiple components of the eye. Several classification schemes are used to differentiate subsets of clinically observed disease.

A. Classification by Observed Inflammation Over Time
22. Primary uveitis: Initial episode of inflammation of the uvea. May have inciting cause like a corneal ulcer or blunt trauma. May or may not go on to classic ERU.
23. Equine recurrent uveitis (ERU): Horses demonstrate repeated bouts of miosis, severe inflammation, and pain in one or both eyes. In between bouts, the eye(s) appear comfortable. As not all uveitis is recurrent, a case is not termed “recurrent” until two or more classic episodes have occurred.
24. Insidious uveitis: Horse appears normal to the owner and the animals do not exhibit overt ocular pain. Examination of the globe with a direct ophthalmoscope or slit lamp shows progressive deterioration of numerous ocular structures that is progressive over time. Pupils may be slightly miotic. Subtle signs include corneal haze, slight aqueous flare, muddy iris color, slight miosis, iris rim atrophy, corpora nigra atrophy, cataract, low-grade vitreous haze, and retinal scarring. Most often seen in Appaloosas and draft horses and European warmbloods.

****Miosis is a hallmark of acute ERU. Miosis is a component of persistent insidious uveitis as well, but usually to a milder degree.

B. Classification by Region of Ocular Involvement
25. Panuveitis: Cases where the entire uveal tract is inflamed. Most cases of ERU and insidious uveitis involve a panuveitis.
26. Posterior uveitis: Inflammation is predominantly observable in the posterior segment (vitreous, retina, and optic nerve). Most often seen in European horses, warmbloods, and draft horses.

C. Classification by Stage of Disease at Time of Examination
27. Active or acute cases are horses who are suffering from a flare-up of classic uveitis. Signs of an acute bout include pain, lacrimation, photophobia, chemosis, conjunctival hyperemia, corneal edema, corneal vascularization, dot-like keratic precipitates on the corneal endothelium, aqueous flare, hyphema or hypopyon, miosis, vitritis, and ocular hypotension (pressure of 10–12 mm Hg).
28. **Quiescent** cases are horses in a “calm” time of their cycle of repeat inflammation. Slit-lamp examination will show subtle flare in the anterior chamber, indicating low-grade, persistent inflammation. Ocular examination may reveal scarring or “footprints” of previous disease including: chronic corneal edema, iris atrophy, iris color change, synchiae (adherence of the iris to the lens or cornea), pigment rests on the lens, cataract, densities or haze in the vitreous, or scarring around the optic disc. Fundic scarring is seen as either a focal “bullet-hole” pattern of numerous tiny chorioretinal scars, or a “butterfly” pattern where wing-shaped islands of depigmentation flank either side of the optic disc.

29. **End-stage uveitis** cases are horses with eyes that have undergone severe degeneration and chronic scarring. They may show extensive corneal scarring, circumferential synechiae, dense cataract, lens luxation or subluxation, secondary glaucoma, retinal detachment, or phthisis bulbi. End-stage uveitis is associated with vision loss.

Diagnosis of uveitis is simplified by understanding that a horse may present anywhere along the spectrum from acute to end-stage, and as either a recurrent or insidious case. Horses that present with three or more signs of intraocular inflammation and a history that is suggestive of either recurrent disease or breed-associated insidious disease can be given a presumptive diagnosis of ERU or persistent uveitis. Note that some signs will reflect acute inflammation and other signs will reflect chronic ocular changes that are scars that have accumulated from previous inflammation. Inflammation is detectable on a cellular basis in both quiescent and insidious cases.

**Note:** Care must be taken to ensure that there is not another primary problem in the eye that is causing internal inflammation. The cornea must be examined closely for signs of corneal ulcer, stromal abscess, foreign body, neoplasia, and immune-mediated or idiopathic keratitis. The globe should be examined for neoplasia. Ruling out concurrent ocular disease that incites ocular inflammation is critical, as corticosteroid therapy is contraindicated in many of these conditions.

**KEY ETIOLOGIC AND PATHOPHYSIOLOGIC POINTS**

**Pathophysiology**

Uveitis begins with **compromise of the blood-ocular barrier.** This barrier normally functions to keep the aqueous and vitreous ocular media clear. Tight fenestrations between ocular capillary cell walls prevent circulating cells and large molecules from passing through the blood vessels of the iris, ciliary body, and choroid into the surrounding stroma. The blood-ocular barrier also serves to isolate intraocular structures from the normal traffic of immune surveillance, making the tissue of the inside of the eye an immune-privileged site.

In acute uveitis, the uveal blood vessels in the iris, ciliary body, and choroid thicken and become **congested.** Soon the vessels become “leaky,” and **cells and inflammatory mediators cross the compromised blood-ocular barrier** and enter the inside of the eye. Most of the initial cells that cross the barrier are **neutrophils.** This may be seen grossly as hypopyon, aqueous flare, and vitreous haze. Neutrophils that enter the eye are soon replaced by large numbers of **lymphocytes,** some of which infiltrate the connective tissue of the ciliary body and iris, forming large follicle-like clusters. **Antibodies** and **inflammatory cytokines** are detectable inside the eye and within ocular tissues. These substances probably react with host and (in some cases) infective factors to contribute to ongoing pathologic changes. Numerous heavy **exudates** appear on intraocular tissue surfaces, most notably on the epithelium of the iris and ciliary body, on the capsule of the lens, and in the layer between the retinal pigmented epithelium (RPE) and the photoreceptors of the retina. The exudates interfere with the function of adjacent ocular tissue. Cytokine activity mediates tissue destruction. With repeated or persistent inflammation, chronic changes occur within the ocular tissues, affecting variably the cornea, uvea, lens, and retina. Vision loss results when dense cataract and synchiae obscure acuity, when the retina detaches or degenerates and no longer can transmit processed light signals to the brain, or when glaucoma causes ischemic damage and degeneration of the retinal ganglion cells ± optic nerve.
Etiology
Decades of research have substantiated that recurrent uveitis is an immune-mediated disease. However, some bacterial, viral, and parasitic infections, as well as certain host conditions have been associated as triggering events for the syndrome.

These factors include:
- **Bacterial infections**: leptospirosis, *Borrelia burgdorferi* (Lyme disease), brucellosis, *Streptococcus*, *Rhodococcus equi* (foals), generalized septicemia
- **Viruses**: influenza, equine viral arteritis, parainfluenza
- **Parasites**: onchocerciasis, *Strongylus*, toxoplasmosis
- **Host conditions**: tooth root abscess, severe hoof abscess, septicemia, severe trauma

Of all the possible infectious triggers, **leptospirosis is the most significant worldwide**. *Leptospira*-associated cases account for at least 60% of the cases of ERU seen in the speaker’s practice, which is located in the temperate Genesee River Valley in western New York State, which is in the northeastern corner of the United States directly south of Toronto, Canada.

**Leptospirosis and Uveitis**
The most significant serovars associated with disease are *L. interrogans* serovar Pomona (seen often in the USA) and *L. interrogans* serovar Grippotyphosa (seen often in Germany and central Europe).

Factors that increase the risk of leptospirosis in horses include:
- Pasture access to cows, pigs, or deer
- Close proximity to streams or ponds frequented by the same
- Access to ponds or other non-flowing water sources
- Rat infestation in the stable
- Rainy season with persistence of ground water

Horses become infected when they drink water contaminated by the urine of a carrier animal (often a cow, deer, pig, or rat). The spirochete gains access to the horse’s bloodstream by mechanical penetration of mucous membranes. Bacteremia results in clinical illness, manifested by anemia, fever, and flu-like symptoms. Clinical disease is mild and self-limiting, and rarely diagnosed as a leptospiral infection when acute. Resolution of signs does not mean elimination of the bacteria from the body - the spiral organisms often colonize the kidneys of the horse and may persist for a few months, being shed in the urine. *Leptospira* Pomona has also been associated with numerous cases of abortion in mares.

Ocular signs of uveitis associated with leptospirosis rarely occur during acute, systemic, leptospiral infection. Rather they begin months later. Signs associated with the primary bout of ocular inflammation will subside with or without therapy, but usually recur at unpredictable intervals. Subsequent episodes of ocular inflammation may be more or less severe than the initial one. Inflammation and damage to ocular tissues associated with repeat episodes eventually compound and create visual deficits. Blindness is a common final outcome.

The pathogenesis of the “lepto link” with equine recurrent uveitis has been the subject of much research and debate. Key findings:
30. Antibodies to pathogenic serovars can be found in the sera, aqueous, and vitreous of horses with *Leptospira*-associated uveitis and studies have supported the hypothesis that intraocular synthesis of these antibodies is occurring.
31. Leptospiral organisms have occasionally been cultured from the ocular media of horses with uveitis.
32. Molecular homology has been demonstrated between the equine cornea and *Leptospira*.
33. Pineal inflammation has been shown to accompany *Leptospira*-associated uveitis in horses, similar to experimental models of uveitis in laboratory animals.
34. MHC II reactivity has been demonstrated on resident and infiltrating cells of horses with both natural and experimental *Leptospira*-associated uveitis.

35. Seroreactivity to equine retinal proteins has been found in horses with *Leptospira*-associated uveitis.

A unified theory is yet to appear to explain all the ocular events that accompany *Leptospira*-associated ERU, but three questions can be considered:

- Does ERU stem from a direct toxicity of intraocular infection with the spirochete?
- Is ERU an autoimmune disease triggered by molecular mimicry between *Leptospira* and host tissue?
- In ERU, are leptospiral organisms somehow modulating the immune response of the eye?

These questions continue to challenge researchers. Although systemic infection with pathogenic strains of *Leptospira* is clearly a common trigger for vision-threatening ERU, the genetic makeup of an affected horse, specifically the genes that determine the MHC complex and ELA (equine lymphocyte antigen) profile of that individual, probably play a major role in determining both susceptibility to leptospirosis as an inciting trigger, and severity of subsequent inflammatory episodes.

**Testing Horses for Exposure to Leptospirosis**

The speaker routinely submits serum from horses diagnosed with uveitis to the diagnostic laboratory at Cornell University for MAT analysis against a panel of leptospiral serovars (https://ahdc.vet.cornell.edu). Many non-uveitic horses will show low titers to the Bratislava, Autumnalis, Hardjo, or Canicola serovars; these are judged to be insignificant findings in the speaker’s practice geography. Positive titers above 1:400, to *L. interrogans* serovar Pomona or *L. interrogans* serovar Grippotyphosa in horses with ERU are judged to be significant and a likely indicator of *Leptospira*-associated etiology. The speaker has observed that seroreactivity to *L. interrogans* serovar Icterohemorrhagica is often paired with reactivity to *L. interrogans* serovar Pomona.

Research has shown that horses with uveitis can be occasionally seronegative for antibodies to *Leptospira* and still have leptospiral DNA or live organisms in the eye. Therefore, a negative titer does not fully rule out leptospirosis as an etiologic factor. A positive titer to serovars Pomona or Grippotyphosa is, however, strong cause for concern.

**Breed and Uveitis**

Recent work has also shown that certain breeds are at risk for uveitis, most notably Appaloosas, European warmbloods, and draft horses. A survey done by the speaker found the Appaloosa breed to be 8.3 x more than other breeds for uveitis. Appaloosas that have insidious disease often have overall roan or light coat colors rather than dark coats with a rump blanket. The skin around the lids of affected Appaloosas is often mottled or pink in pigmentation. Mane and tail hair may be sparse. It is theorized that these horses have genetic proclivity to uveitis due to aberrations in the major histocompatibility complex, specifically in their equine lymphocyte antigen subtype. Recent research from Germany has supported this concept in German warmbloods susceptible to disease.

**Unilateral vs. Bilateral Disease**

Little work has been done to document the incidence of ocular involvement in horses, but recurrent uveitis can be a unilateral or bilateral disease.

In a study of 160 cases reviewed by the speaker:

- Fifty percent of the lepto-associated horses had unilateral disease and 50% had bilateral disease.
- Over 80% of the Appaloosas had bilateral disease.
- Sixty-two percent of the non-Appaloosa horses that were seronegative to leptospirosis had unilateral disease.

Uveitis may begin in one eye and later occur in the fellow eye. However, if a case is unilateral and no attacks are seen in the other eye for two years after the initial attack, it is uncommon for uveitis to show up later in the contralateral eye.
**Key Therapeutic Points**

- **Mydriasis** is essential therapy for all cases of acute uveitis. Initial application of atropine should be frequent until pupil is fully dilated. Severe cases may show poor response to the action of mydriatics.

- **Topical corticosteroids** and **systemic NSAIDs** are the core elements of antiinflammatory field therapy for acute attacks. Therapy should be intense for about two weeks and may be tapered over another two weeks. **Subconjunctival and/or systemic corticosteroids** are indicated in severe cases.

- Corticosteroid topical therapy may induce **calcific band keratopathy**, especially in horses with *Leptospira*-associated uveitis. Treat with EDTA chelation/keratectomy.

- **Secondary corneal ulcers** are discovered in horses with ERU or persistent uveitis in about 25% of horses at some point in their disease course. This fact is not surprising giving the pain associated with uveitis and the propensity of horses to suffer self-trauma. Corticosteroids are contraindicated in these cases. Systemic NSAIDs can be used to treat pain, and the secondary ulcer should be treated topically with anti-infectives and anti-collagenases.

- **Acupuncture therapy** may help moderate the frequency or severity of episodes.

- **Insidious, persistent uveitis** is a challenging condition that is common in Appaloosas and some draft horses and warmbloods. Therapy does little to alter the progression of disease in affected horses.

- **Secondary glaucoma** is a complication seen in many horses, particularly Appaloosas suffering from insidious disease. Glaucoma therapy is often unrewarding in the long term, but timolol, dorzolamide, or a combination of these drugs (Cosopt) may be tried. Judicious topical steroids may help as well.

- **Suprachoroidal cyclosporine implant surgery** may reduce the frequency and/or severity of ERU and persistent, insidious uveitis. The best candidates for this referral procedure are early ERU cases that have only experienced a few “attacks” and show little or no permanent ocular scarring.

- **Pars plana vitrectomy** is frequently performed on ERU horses in central Europe with reported good results. The procedure is not often performed in the United States.

**Key Prognostic Points**

Visual prognosis for horses suffering from multiple acute attacks of uveitis or insidious, chronic disease is **always** guarded. Data on the incidence of blindness in uveitic horses are lacking, but it is clear that uveitis is the leading cause of blindness in horses worldwide. The speaker has observed ocular inflammation serious enough to threaten vision in at least 1–2% of her practice population.

Analysis of the visual outcome of 160 cases followed by the speaker over 11 years revealed the following trends:

- Fifty-six percent of the case series (89/160) lost vision in one or both eyes.
- Twenty percent of the cases (32/160) became completely blind.
- Thirty-six percent (57/160) lost vision in one eye.

Breaking the cases down further into those that were seropositive or seronegative to *L. interrogans* serovar Pomona, and those that were Appaloosas or “non-Appaloosas,” the following trends were seen:

- Horses that were seropositive to *L. Pomona* and also Appaloosas had a very poor visual prognosis: 100% lost vision in at least one eye and 50% went completely blind. (n = 14)

- Horses that were Appaloosas and seronegative had substantial occurrence of blindness: 72% lost vision in at least one eye and 29% went completely blind. (n = 28)

- Horses that were seropositive to *L. Pomona* and non-Appaloosas had a slightly lower rate of blindness: 50% lost vision in at least one eye and 17% went completely blind. (n = 86)

- Horses that were seronegative and non-Appaloosas had the best visual prognosis: 34% lost vision in at least one eye, and just 6% went completely blind. (n = 32)
Secondary complications and degeneration of ocular tissues are common sequelae of uveitis. Several interesting findings were noted in this series that are representative of sequelae seen in ERU horses in other geographic regions of the world:

- **Cornea**: Focal scars, folds, calcium deposits, and other corneal opacities were common. The seropositive horses experienced a high rate of calcific band keratopathy. Striae and dense corneal folds were common in Appaloosas and were highly correlated with blindness.

- **Iris**: Iris atrophy and color change were common, especially in Appaloosas and seropositive horses. Anterior synechiae were rare unless phthisis bulbi was present, but posterior synechia occurred in nearly one third of all cases and 40% of Appaloosas.

- **Lens**: Diffuse cataract(s) developed in 41% of all cases, and nearly 75% of the Appaloosas. These were a common cause of blindness. Lens luxation was common in Appaloosas (29%).

- **Posterior segment**: Severe vitritis was observed in nearly one third of the cases. Peripapillary scarring (focal or alar) was also present in about one third of the horses. Cataracts and synechiae often obstructed posterior segment evaluation, so inflammatory changes were probably underreported.

- **Glaucoma and phthisis bulbi**: Appaloosas had the highest rate of glaucoma (21%). Phthisical eyes developed most often in Appaloosas and seropositive horses.

Owners often are concerned that horses with ERU will need enucleation. In the above series, only 4% (6/160) were enucleated for complications from corneal infection or glaucoma. Of more concern is the fact that 43 of the 160 horses (27%) were treated for corneal ulcers over the observation period. Risk of corneal ulcers in ERU horses should be stressed, as owners often choose to medicate horses with painful eyes themselves, and they may potentiate serious infections by applying corticosteroids and delaying proper diagnosis. Ten horses suffered from calcific band keratopathy. This is a troublesome complication that limits therapeutic options for ERU.

**Additional Resources**
Website for owners of horses that are blind: www.blindhorses.org.

**References**

Coping Strategies: Use of Subpalpebral Lavage Systems and Management of Blind Horses
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**Subpalpebral Lavage Systems (SPLs)**

Subpalpebral lavage systems (SPLs) are essential devices used to deliver medication to the surface of the eye. They are most often used to treat horses with serious ulcerative keratitis, but may also be used to provide ocular therapy to fractious or painful horses suffering from uveitis, globe trauma, or other conditions that need frequent surface treatment. Mila International, Inc. (www.milainternational.com) sells SPL kits in two lengths (36” and 60”). The longer tubes are the most practical as they are long enough to use on warmbloods and draft horses and may simply be cut down to use on foals and ponies. The kits have a 12-ga trochar swedged onto the silicone tubing. Preparation for tube placement should include braiding the mane of the horse into 5–10 braids that can be used as tubing guides. To facilitate insertion, the horse should be heavily sedated prior to insertion and the mandible should be supported by a table made of bales.

The steps for insertion are as follows:
36. Perform auriculopalpebral and supraorbital blocks. Also block the skin over the target puncture site in the fornix, and the skin in the regions where the tape guides will be sewn.
37. Apply topical anesthetic to the globe with a syringe. Apply topical anesthetic to the fornix by holding a soaked cotton swab to the target site to be punctured.
38. Insert a gloved finger with the trochar laid along the finger length (but not beyond the tip) into the fornix. The desired trochar puncture site is deep in the fornix or ventral cul-de-sac adjacent to the orbital rim. Cautiously advance the trochar to the target puncture site, taking care that it is as deep into the fornix cleft as possible, and that the trochar pierces the eyelid skin without a lot of oblique tunneling through the dermis. Push the trochar through the lid and then pull the SPL tubing all the way through the eyelid until the footplate rests against the conjunctiva.
39. Take care that the footplate of the system is oriented so the oblique angle of the footplate is lined up with the angle of the fornix (upper lid) or cul-de-sac (lower lid).

The tubing can be put in the fornix of the upper or lower lid depending on lesion location and clinician preference. The tubing must be secured to the face. This can be done by sewing adhesive tape “wings” onto the face a few cm away from the exit hole, then at one or two other intervals as needed to guide the tubing towards the poll. The green, plastic, U-shaped guides that come with the SPL kit are used by some clinicians to secure the tubing to the face, but this author prefers to use adhesive or duct tape wings.

The tubing (with the trochar still attached) is then strung under the halter and woven through the mane braids. A suggested pattern is to insert the trochar under the portion of the braid closest to the poll, bring it through the middle of the braid twist, then direct the tubing over the base of the braid closest to the withers, then proceed the weaving pattern (under, through, over the braid) down the neck until a braid close to the withers is reached. The trochar and attached surplus tubing is then cut off and discarded and the exposed tubing end is ready to create the treatment port.

The treatment port should be made by inserting a 20-ga, 1-inch catheter (included in the kit) into the tubing and then inserting a standard male catheter cap into the lavage tubing. Tape the treatment port assembly to a half of a wooden tongue depressor, and then tape this handy platform to the associated mane braid to make a “handle” that will facilitate injection of medication. Sometimes it is advantageous to also sew the adhesive tape around the platform to both sides of the mane braid to prevent dislodgement.

Most specialists advise the injection of just one drug at a time through an SPL system. This is done using a tuberculin syringe and a 25-ga needle, injecting 0.1–0.2 ml of drug at a time, and then slowly
pushing the medication onto the globe with 1.0–1.5 ml of air. Subsequent medications are injected after a few-minute waiting period.

Some clinicians “stack” multiple medications in the tubing without air interface, and treatment is accomplished by pushing a few ml of air into the tube to discharge the stack of medication into the tear film. Other clinicians use infusion devices that pump a continuous cocktail of medication onto the globe at a fixed rate (Infu-disk, Mila International, www.milainternational.com). Some clinicians have found these methods practical and effective, but no research has substantiated the “cocktail” effect of drugs being mixed together and then applied to an infected cornea, so no recommendation can be made about any treatment methods that involve mixing of drugs in a stacked or reservoir system. The choice of solution application method should take into account the temperament of the horse, its reaction to the treatment process, and the possibility of washout of medication if large volumes are pushed through in succession. Another concern is the fact that some ophthalmic medications must be stored in the dark or at cold temperatures to retain potency, and medications that “sit” in an infusion device are exposed to both body and ambient environmental temperatures.

Horses that are being treated with SPLs may benefit from wearing protective hoods (Eyesaver, available from Jorgenson, Inc. www.jorvet.com in left and right models of various sizes). The cups of the hoods can be perforated with a drill bit to optimize air circulation. The terry cloth “tear catchers” that line the hoods must be changed daily and may be supplemented by gauze pads to wick tears and debris. Some horses have developed serious periocular dermatitis when hood management has been lax. This author rarely uses hoods for management of SPL tubes, but often uses fly mask coverings over the systems.

Occasionally, an SPL system may develop a hole or tear in the tubing. Repair can be done by cutting the tubing and “splinting” the severed ends with a 20-ga catheter. One end of the tubing can be slid around the catheter end, then the hub of the catheter can be cut off and the other end of the tubing coaxed to slide around the other end of the catheter. Most SPL systems are used to treat eyes for several weeks and some have been left in place for several months. The tubing near the footplate in the lid should be checked daily to ensure that the label that reads “Mila” is located about 2 cm away from the skin puncture. This will ensure that the footplate is seated securely in the tarsal conjunctiva and is not migrating towards the cornea.

Management of Blind Horses
Equine recurrent uveitis (ERU) is a common diagnosis, affecting over 1% of the population. A large number of horses lose sight in at least one eye as a result of ERU, trauma, corneal disease, or other condition and many of those horses become bilaterally blind. Recent changes in attitudes on animal welfare have changed the management of blind horses. In the past, most bilateral blind horses were subject to euthanasia either at slaughterhouses or at home farms. Now, many people choose to maintain blind horses as pets and companions. Some blind horses are ridden and a few compete in athletic events like dressage or reining.

Most horses lose vision gradually. As acuity decreases, handlers may notice progressive uncertainty, especially in low-light situations. Herd behavior may change. Riders may report frequent shying or balking. Certain low-light conditions may be accompanied by instances where the horse is observed bumping into obstacles.

When complete blindness occurs, some horses go through a period of fear or anxiety. Handlers may report episodes of rapid circling, freezing in place, prolonged neighing, and spooking. A previously tractable horse may become dangerous to be around if it crashes into a wall or runs over a handler. The horse may initially be observed to show a head tilt or walk slightly off balance. Other individuals show a calmer acceptance of blindness, but are still at risk for injuring themselves or others if they run into something.

Horses with reasonable temperaments adjust to blindness after an adaptation period of several weeks. Horses who are good candidates for adjusting to blindness are those with calm, easy-going temperaments and dedicated owners who are willing to make environmental and management changes that accommodate the disability. Owners of horses with vision loss should always allow the horse a
“transition period” of several days to weeks to adjust to blindness before judging what the long-term temperament will be.

Some horses with high-strung, nervous temperaments never adjust well to vision loss and pose a constant risk of injury to themselves or their handlers. Horses who have nervous temperaments and are owned by owners who are not dedicated to their long-term management may need to be euthanized.

Social Interactions of Blind Horses
Some horses benefit from the presence of a calm, sighted companion in their paddock or barn during the period where they are adjusting to blindness. Others fare better if they are kept alone.

Once the adjustment to blindness has been made, most blind horses enjoy the company of one gentle, compatible companion. This can be another horse, a pony, or a goat. The sighted horse may serve as a “seeing eye” for the blind one. Sometimes the sighted animal may even be observed to “lead” the blind horse around obstacles. Handlers should be aware that the bond between these pasture companions will be exceptionally strong. If one has to be separated from the other, both may display extreme anxiety. However, blind horses that lose their “pasture buddy” will generally adjust quickly to a replacement companion.

Blind horses generally do not fare well in herds. Herd interactions generally revolve around a social hierarchy and a blind horse will move to the bottom of the pecking order. Blind horses in herds are typically pushed around, treated as outcasts, and not allowed choice access to food, so they generally fail to thrive.

Environmental Considerations
Blind horses appear to construct a “mental map” of their environment and have a remarkable ability to sense the perimeters and pathways of their usual enclosures. Still, common sense dictates that their surroundings be made as safe as possible. Fencing should be a particular concern. Board fencing (wood or plastic), woven-wire or mesh-wire, diamond-weave fencing are good choices. Barbed wire should be avoided. The pasture should be free of holes, debris, equipment, and sharp objects. Trees or poles in the pasture should be fenced off or ringed with materials like sand-filled tires. Low-hanging branches or sharp elements on pasture vegetation pose a particular risk, and should be trimmed or cut down.

Some managers like to install environmental “cues” for their blind horses. Examples include a skirt of stone footing around pasture gates, and rubber mats in the pasture in the area where hay is fed. It is important not to “rearrange the furniture” - once the environment is established it should be kept constant. Hay and water stations should be established and kept constant.

Stalls should have solid walls and secure doors without sharp hardware projections. All “J”-shaped handles on buckets or feed tubs should be taped up to prevent eyelid lacerations. Windows should have safe casings and glass should be protected by wire mesh or another barrier. Signage should be posted stating that the stall occupant is blind to warn visitors that special handling of the horse is in order.

The remaining senses seem to be enhanced in blind horses. They appear to have acute hearing and a keen sense of smell. Their sense of touch is a key guide. The most richly innervated region of the face is the muzzle, and blind horses can be observed “reading” their environment with their muzzle much the same way a blind person uses their fingers to read Braille. The whiskers and facial hair of a blind horse should never be clipped as these hairs provide further sensory feedback and help the horse “map” its environment.

Training Blind Horses
Blind horses recognize their handlers by voice, smell, and touch. Handlers should use their voice, often both in the transition period of adjustment, and also once the horse has accepted blindness. A consistent set of voice signals will help reassure the horse and key it to certain obstacles like steps or trailer ramps.

Verbal cues for “whoa” should be taught early and should always be delivered in the same tone of voice. Similarly, cues for “walk” and “trot” can be taught while the horse is led. Other cues should be set to alert the horse to obstructions in their path. When leading horses towards obstacles, orientation may be improved if the handler raps on the obstacle loudly so the horse can gauge how far away it is. A voice cue of “Ahh….step” will be learned as a warning of an upcoming incline to be negotiated. Trailer loading and off-loading will be expedited if a specific set of cues are developed to help the horse navigate the
ramp or step into the trailer. The handler should always speak to the horse when approaching it in a field or stall.

Touch is an important training cue in addition to voice. A consistent approach when greeting the horse (say always approaching one shoulder, while speaking to the horse in a consistent voice) is a good idea. Horses that are anxious will usually settle down with a reassuring touch coupled with steady verbal cues from a familiar handler.

Habit and routine are important for all horses and are especially vital for blind horses. They will quickly learn the barn routines and anticipate where to go for feeding, turnout, etc. each day.

Blind horses respond well to natural horsemanship exercises on the ground. They can be taught all sorts of commands and reactions. Time spent with this kind of work will cement the bond of trust a blind horse has with its regular handler, and be mutually enjoyable for both the horse and the handler.

**Unilateral Loss of Sight**

Many horses lose sight in just one eye for a variety of reasons cited above. Horses that are unilaterally blind generally adapt very well and also perform well in a variety of disciplines. Horses that are sighted in one eye are permitted to race, compete in endurance events, and show in a variety of disciplines. Exceptions are the sport of polo, which does not allow unilateral blind horses to be used in official matches, and the hunter discipline, which also does not allow partially blind horses to show. Unilateral blind horses are acceptable in collegiate polo, so these strings often inherit horses from professional strings that have lost vision in one eye.

Personnel that work around horses that are unilaterally blind should be aware of the condition. Veterinarians who have to perform injections or other noxious procedures on unilateral blind horses are well advised to choose the sighted side for injection. It is best to approach a half-blind horse always on the sighted side.

**Enucleation of Blind Horses**

Most horses that become blind still have their globes when vision is lost. In some cases (dense cataract, some cases of detached retina, or cortical blindness) the globe remains a normal size and orientation. In other cases the globe becomes altered in size or orientation. Some blind eyes become chronically enlarged from glaucoma. Others, especially eyes afflicted with uveitis, shrink from destruction of the ciliary body and internal scarring to become phthisical. Some blind eyes show chronic strabismus with the globe usually being deviated in a ventral or nasal direction.

Blind eyes are at increased risk for trauma from the environment. It is common to diagnose corneal ulcers in blind eyes, but the ulcers may go undetected and untreated by the owner as the appearance of the globe was abnormal to begin with. Phthisical eyes often show chronic inflammation of the globe remnant, conjunctiva, and nictitans. Blind eyes with glaucoma appear comfortable in some horses, but other horses suffer chronic pain.

Horses that are blind and suffering chronic pain benefit from enucleation of the affected eye(s). Enucleation of a chronically painful, blind eye is a humane choice that benefits the overall welfare of the horse. Enucleation is a procedure that is relatively simple to perform and low risk in terms of postoperative complications. Many horses show an immediate improvement in temperament and demeanor after surgery, suggesting that the chronic ocular pain was affecting their quality of life. Recovery from enucleation is usually very fast and postoperative care is minimal.

Enucleation is a surgery that can be done under general anesthesia in 45–60 minutes. Recently, many clinics have been performing standing enucleations on horses in similar time spans. Standing enucleation has been performed by the author on over 50 horses with excellent results. Standing enucleation is a viable choice for horses who are anesthetic risks (i.e., horses who are very old, very large [draft breeds], very small [miniatures or small ponies] or suffering from diseases like Cushing’s disease or laminitis).

Silicone ocular prosthetics are available commercially as space-occupying devices that can be placed in the orbit. This author does not advocate their use, as the complication rate with implants is significant (reported rate of complications has been as high as 20%). The end cosmetic result after prosthetic insertion may not be significantly better than the appearance of an enucleated orbit without a prosthetic.
ADDITIONAL RESOURCES
Website for owners of horses that are blind: www.blindhorses.org.

REFERENCES
INTRODUCTION
In a recent study to define biomarkers of neurologic diseases of feedlot cattle, we evaluated 44 calves with a primary complaint of neurologic disease. We videotaped the animals, performed a physical and neurological examination, collected samples, and euthanized the calves to perform a necropsy. Our attempt for this presentation is to combine the clinical presentation with histopathological findings in selected cases.

POLIOENCEPHALOMALACIA
Polioencephalomalacia (PEM) and cortico-cerebral necrosis (CCN) are nonspecific terms associated with softening of the gray matter of the brain. These lesions can result from several causes. In cattle (and other ruminants) these lesions can result from alterations in the thiamine metabolism, sulfur intoxication, lead poisoning, salt toxicity, or water deprivation. Alteration of Thiamine Metabolism
Thiamine is an important coenzyme in several pathways of intermediate metabolism of carbohydrates and energy. It is essential for production of ATP and control of the neuronal intracellular osmotic milieu. Pre-ruminants are dependent on ingestion of thiamine. In ruminants, thiamine is produced by ruminal bacteria and protozoa under normal environment. Production is close to daily requirements and storage of thiamine is minimal. Disruption of the normal microbial environment can reduce thiamine production either by decreasing the number of bacteria synthetizing thiamine or increasing the population of thiaminase-producing bacteria, specifically thiaminase type II. Altered rumen environment is most common in feedlot cattle on high-carbohydrate diet associated with ruminal acidosis. Plants such as bracken fern, horsetail, and Nardo fern in Australia also produce a thiaminase resembling thiaminase type I. Drugs such as amprolium may act as thiamine analogs. Common clinical signs include central blindness, ataxia, muscle fasciculations, nystagmus, head pressing, seizures, and progress to recumbency, coma and death.

Definitive diagnosis is made on histopathological findings. Presumptive diagnosis can be made on history and clinical signs. There are increases in blood pyruvate and erythrocyte transketolase activity is reduced. Cerebrospinal fluid protein may be increased and cellularity varies from normal to mild pleocytosis (5–10 WBC/dl, ref: < 5 WBC/dl). The mainstay of treatment is thiamine (10 mg/kg) IV or IM every 6 hours for 1 to 3 days then daily. Prognosis is fair if the animals are still standing with some improvement in 24 hours and up to a week for complete recovery.

Sulfur-Induced Polioencephalomalacia (S-PEM)
In case of S-PEM, alteration of the thiamine metabolism is not as clear. Sulfur compounds in the rumen are metabolized into sulfide ions by sulfate-reducing bacteria. In the rumen, about half of the hydrosulfide is in its gaseous form: H2S. Hydrosulfide is eructated from the rumen and H2S can be aspirated in the lungs and cause PEM. The most common theory is that H2S may block the enzyme cytochrome c oxidase causing cellular energy deficiency, but some recent work suggests that cytotoxicity caused by H2S may involve reactive S species that depletes reduced glutathione and activates oxygen to form reactive oxygen species. Levels of thiamine do not appear to change significantly. Sources of excess sulfur are multiple: in many areas of Canada and the United States, surface and deep waters are high in sulfate. Distiller grains with solubles as a byproduct of ethanol production from corn, other co-products of corn, sugar cane, and sugar beet processing, as well as corn gluten feed, are usually high in sulfur.
Some cruciferous forages are important sources of sulfur. Some trace elements influence \( \text{H}_2\text{S} \) production by their presence or absence.\textsuperscript{7} Two clinical forms are recognized: an acute form is characterized by blindness, recumbency, seizures, and frequently death. In the subacute form, animals show blindness, twitching of the ears, facial muscles, and ataxia.\textsuperscript{7}

Acute changes of polioencephalomalacia: reactive endothelium, gliosis, and necrotic neurons

Subacute to more chronic changes of polioencephalomalacia: cavitation and severe gliosis

When suspecting S-PEM, both water source and feed should be tested. Ruminal hydrogen sulfide can be measured by aspirating rumen gas through percutaneous needle aspiration and using a precision gas sampler. Other methods such as thiosulfate concentration in the urine may prove useful.\textsuperscript{8} It is important to remember that concentration of \( \text{H}_2\text{S} \) in the rumen usually decreases when animals become anorectic. Treatment is supportive; administration of thiamine and corticosteroids may be helpful although the success rate is less than in cases of thiamine alteration. Removal of animals from sources high in sulfur is an important control measure. Regular analysis of feed and water for sulfur concentration can help making management decisions.\textsuperscript{7}
Acute Lead Toxicity
Lead has been a leading cause of accidental poisoning for a long time in ruminants. Main sources of lead include batteries, asphalt, discarded crankcase oil, old paint, and solder. Most ingested lead forms insoluble complex, excreted in the feces. Absorbed lead is distributed to blood (red blood cells and to a lesser extent albumin) and tissues (bone, teeth, liver, kidney, brain, and spleen). Lead interferes with multiple biochemical mechanisms. It binds sulfhydryl groups and inactivates enzymes involved in heme synthesis causing red blood cell abnormalities. Several mechanisms may be involved in neurotoxicity of lead: disruption of the blood-brain barrier by injuring endothelial, epithelial, and glial cells, as well as causing apoptosis of neurons. In the acute form of lead poisoning, animals may be found dead in pasture. Others may be staggering, showing muscle tremors with champing of the jaws, twitching of the eyelids and ears, and bellowing. Blindness and head pressing are common. The affected animals may fall and show tonic-clonic convulsions, hyperesthesia to the touch and sounds, and opisthotonos. In the subacute form, cattle show blindness, ataxia, circling, muscle tremors, and hyperesthesia.

Measurement of blood lead is a common means of diagnosis. Administration of calcium EDTA may increase urinary lead concentration. Normochromic, normocytic anemia and basophilic stippling of erythrocytes are more common with chronic exposure and are not pathognomonic. Management of lead toxicosis includes elimination of lead from the gastrointestinal tract (rumenotomy, administration of cathartic), chelation therapy (calcium EDTA), general supportive care, and removal of lead source from the animal’s environment. Administration of thiamine seems to reduce the neurologic signs.

Sodium Salt Poisoning and Water Intoxication
Range cattle, feeder calves have been reported to show neurologic signs associated with various combinations of excess dietary sodium salt, water deprivation or restriction followed by free access to water. Water can be restricted because of broken pumps, drought or frozen water supply. High-salt sources include sodium salt added to the diet, incorrect dosing of electrolytes, and use of feeds such as whey and bakery byproducts. Excess sodium results in inhibition of anaerobic glycolysis and decrease in the rate of sodium export. In cases of excessive drinking after water deprivation, there is a sharp drop in plasma sodium concentration due to hemodilution. This leads to increased shift in water into the cells, particularly in the brain causing edema. In the blood, hypotonicity leads to lysis of the cells and hemolysis causing a severe anemia and hemoglobinuria. In a group of calves, clinical signs can be manifested in all or a few animals. The condition is manifested by hemoglobinuria, diarrhea, and nervous signs such as hyperesthesia, muscular tremors, head pressing, blindness, nystagmus, and lethargy. Some cases may progress to depression, coma, and death within a day or two.

In conjunction with corroborating historical findings, supportive evidence for the diagnosis is an elevated serum osmolality or sodium concentration in salt intoxication, or decreased serum osmolality or sodium concentration in water intoxication with concomitant changes in the CSF. Low doses of furosemide with IV normal or hypertonic saline are indicated in hyponatremic cases. Mannitol may be indicated. Frequent administration of small amounts of water per os and IV 5% dextrose are recommended for hypernatremic animals. As a rule, therapeutic alterations of serum osmolality should be made slowly.

Postmortem Findings
All of the above-named diseases may result in changes in the brain, which are classified as polioencephalomalacia. Grossly, this lesion is characterized by swelling of the brain, particularly of the cerebral hemispheres. The brain is often pale, soft, and there may be gray discoulouration at the gray-white junction. The lesion is most severe in the dorsal/caudal regions of the cerebrum and is associated with the blood supply region of the middle and caudal cerebral arteries. In the classical polio lesion, the affected regions of the brain show autofluorescence when observed under a UV light. This is due to the accumulation of lipid metabolites in the damaged areas. The histologic lesion in this group of diseases is a laminar necrosis, usually in the deeper layers of the cerebrum. There is hypertrophy of endothelial cells, and the neuropil of affected regions is pale, may be spongiotic and contains dark-red, shrunken neurons which are necrotic. In severe cases, there may be full-thickness necrosis of the gray matter. If the animals survive for several days, grossly there may be a yellow/brown discoulouration at the gray/white matter.
junction and separation, or cavitation, may be observed. The brain shows severe edema, and there may be herniation in the caudal portions of the brain. Histology is similar, but larger areas are affected and there is a marked accumulation of gitter cells and gliosis may be severe. If animals are treated and survive beyond several weeks, healing by a glial scar may be seen. If the lesion was more extensive, gray matter may be lacking over the affected regions in chronic disease.

Differentiation of the causes of laminar cortical necrosis is difficult by pathological examination alone. History, epidemiology of the herd, and toxicology will be of assistance in arriving at a definitive diagnosis. In pigs which present with laminar cortical necrosis, the presence of a large number of eosinophils in the meninges of the brain is strongly suggestive of salt intoxication, but this is not often present in ruminants with this condition. Most cases of lead intoxication do not show significant lesions within the CNS, or only very mild vascular lesions are noted. Some cases of lead intoxication may also show vascular changes and gliosis in the cerebellum. Chronic lesions of polioencephalomalacia or very severe cases may also have softening and/or hemorrhagic foci in the thalamus and midbrain and anecdotally these may be more commonly associated with sulphur-induced polioencephalomalacia.

**BRAIN ABSCESS**

Brain abscesses are a relatively rare condition in cattle. Affected animals are commonly less than 1 year of age. Abscesses may be single or multiple and they are either hematogenous in origin, result from trauma, or from direct extension of a local suppurative process. The clinical signs are usually vague. The rectal temperature may be normal and the animal may be depressed. The neurologic signs depend on the location and extent of the abscess. Reported signs include head tilt, ventral strabismus, circling or hypermetria, intention tremors, and ataxia when the abscess is localized to the cerebellum. Signs are often asymmetrical.

The antemortem diagnosis of a cerebral abscess is often presumptive. Cerebrospinal fluid analysis may support the diagnosis, but the abnormal findings depend on the degree of encapsulation and amount of ependymal involvement. Reported changes include pleocytosis with increases in monocytes and lymphocytes. Computed tomography or magnetic resonance imaging allows antemortem diagnosis and localization. Treatment is usually not attempted.

**Postmortem Findings**

A brain abscess may form due to haematogenous spread of bacteria, and in these cases additional abscesses may be observed in other organs. Brain abscess as a result of extension of a suppurative process elsewhere in the head region such as otitis, or secondary to trauma/foreign body, is also reported. Abscess formation as a sequel to leptomeningitis is rare.

The gross appearance of the lesion is typical of abscesses elsewhere in the body. As they are slowly developing lesions, most present in the chronic state as one or more discrete areas of purulent inflammation, surrounded by a tough, fibrous tissue capsule of variable thickness. The capsular components of abscesses which form in the CNS are slightly different than elsewhere in the body. The capsule is formed both by condensation of vascular structures already present in the brain with a lesser contribution by fibroblasts. The masses are usually expansile, resulting in deformation or asymmetry of the surrounding brain. Depending on the location, they may obstruct the flow of CSF within the brain and secondary hydrocephalus is common.

Histologically, the lesions are also typical of abscesses. The center of the lesion contains abundant cell debris including many degenerate neutrophils and some bacterial colonies may be present. The capsule is composed of fibrous tissue containing histiocytes or gitter cells, and the surrounding neuropil is usually compressed. Moderate amounts of gliosis and edema may be seen in the surrounding tissue, and when near the meninges, suppurative inflammation may also be present on the surface of the brain. If secondary hydrocephalus is present, atrophy of brain matter is also present.

Brain abscess: clinical deficits may be caused by both the effect of a space-occupying mass as well as due to inflammation.
**THROMBOEMBOLIC MENINGOENCEPHALITIS**

*Histophilus somni* (formerly *Haemophilus somnus*) is a gram-negative bacterium that is a normal inhabitant of the bovine genital tract and in the bovine nasal cavity. Exposure may be by aerosol, and disease is often related to the presence of bovine upper respiratory tract disease, transportation, and other stressors. It causes a clinical syndrome including pneumonia, reproductive failure, mastitis, myocarditis, arthritis, and meningoencephalitis. Feedlot cattle are more often affected than pastured or dairy cattle. Most of the clinical syndromes are due to a septicemia. The relationship of *H. somni* to vascular endothelium is unclear. Recent studies suggest that *H. somni* induces apoptosis in bovine endothelial cells *in vitro* and that the bacterium forms biofilms in blood vessels. Sudden death may be the first sign of infection. In the early stages of the neurologic form, calves can have a fever and profound depression (sleeper syndrome). Blindness of central origin may be present, but that could also be due to ocular lesions such as retinal hemorrhages, edema, and necrosis. Some cattle may be in lateral recumbency with opisthotonos and show convulsions. Standing cattle show ataxia and weakness.

Etiologic diagnosis is usually confirmed at necropsy. Changes in the complete blood count are indicative of inflammation. Grossly, the CSF of animals with meningoencephalitis is cloudy and cytologic evaluation is consistent with bacterial infection and hemorrhage. *Histophilus somni* can be isolated from the CSF or other exudative body fluids. Treatment may be attempted in the early stage. Tetracycline is most commonly used, combined with nonsteroidal antiinflammatory drugs. In face of outbreaks, mass medication with antibiotics is often implemented. Immunization can reduce morbidity and mortality.

**Postmortem Findings**

Peracute death, often without lesions, may be seen. In this case, vessels plugged with bacteria are found throughout the body. A subacute syndrome is more common; meningoencephalitis and inflammatory lesions in a multitude of other organ systems are related to a vasculitis with secondary thrombosis.

Grossly, the meninges are cloudy and may appear multifocally thickened. Within the brain, multifocal areas of hemorrhage and necrosis are found. These are variable in size, up to several centimetres in diameter. They may appear randomly distributed, but are more commonly found in the thalamus and at the gray/white matter junction in the cerebral cortices.

Histologically, the lesion is identified as inflammation of small veins, with resulting endothelial damage and thrombus formation, thrombophlebitis. The areas of hemorrhage and necrosis are secondary to this vascular damage. Definitive diagnosis is by culture of the organism.

ITEME: multifocal areas of necrosis due to phlebitis and thrombosis
Listeriosis

Listeriosis affects cattle, sheep, goats, and camelids. It can cause a meningoencephalitis, septicemia, and late-term abortion. Listeria monocytogenes has unique characteristics as a pathogen: it is able to multiply in diverse environmental conditions such as a wide range of pH (5–9), temperature (4°C to 45°C). It is a bacterium capable of free-living growth and is ubiquitous in nature. It can be isolated from plant material, soil, surface water, and manure. It is often a contaminant of silage, which is an important risk factor for the development of listeriosis. The type of silage does not appear to be as important as the condition of it; silage where anaerobic conditions are not optimal allows Listeria monocytogenes (and other bacteria and molds) to proliferate. However, listeriosis has been observed in animals that do not have access to silage.

Pathogenesis of the encephalitic form of Listeria infection is still a matter of debate, but a proposed mechanism involves the penetration of bacteria through breaches in the epithelium of the oral mucosa of affected individuals and migration to the brainstem through roots of the trigeminal nerves.

In the neurologic form, it is the most common disease associated with presentation of ruminants with deficits of cranial nerves and lesion localization to the brain stem. Initially, animals can present with dullness, circling, and maybe a head tilt. As the disease progresses, other cranial nerves may be affected. Tetraparesis and ataxia are consistent findings. Later on, recumbency, coma, and death will occur. On physical examination, fever, anorexia, decreased rumen motility, bloat, and secondary exposure keratitis are often present.

CSF analysis reveals pleocytosis with increase in large and small mononuclear cells. Neutrophils may be present. Protein concentration is often slightly increased. Diagnosis is often confirmed with histopathology and immunohistochemistry. Administration of penicillin, sulfonamides, and tetracycline are effective treatment options. The treatment is often prolonged (2 to 4 weeks) and support of the animal is critical (fluids, electrolytes, and nutrition).

Postmortem Findings

Encephalitis due to Listeria infection may not show any obvious gross pathology, but mild thickening or cloudiness of the meninges of the brainstem and multifocal areas of dull-gray discolouration may be observed. This organism has a particular predilection for the caudal brainstem/medulla oblongata. Meningitis and encephalitis are found histologically and the typical histologic lesion of listeriosis includes gliosis and microabscessation.

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Cerebellar Hypoplasia
Cerebellar hypoplasia is a congenital disorder that can be hereditary, or due to an intrauterine viral infection. It is an inherited disorder in Shorthorn, Hereford, Angus, Ayrshire, and Holstein calves. In most cases the mode of inheritance is autosomal recessive. It is also commonly seen with pestiviral infections such as with bovine viral diarrhea virus. Experimentally, these congenital malformations are more likely to occur when susceptible heifers are inoculated between 100 and 170 days of gestation and, in field cases, between 75 and 150 days. Severely affected calves are unable to rise and show opisthotonos and extensor rigidity of all limbs. Moderately affected calves may require assistance to stand, and show a wide base stance, ataxia, hypermetria, and intention tremors. Other congenital defects such as retinal atrophy, cataract, and microphthalmia are sometimes present as well and result in blindness. Calves may also become persistently infected if the infection occurs early in that period of nervous system development. If infected later (after about 125 days), the calf will have antibodies at birth.

Postmortem Findings
Cerebellar hypoplasia is the most common, and often the most dramatic, lesion resulting from intrauterine viral infection (i.e., BVD virus), but other developmental lesions in the CNS such as microencephaly, hydrocephalus, and porencephaly are also reported. In addition, retinal dysplasia, optic neuritis, and microphthalmia may also be found in conjunction with CNS defects. When the fetus is exposed to BVD virus at the most susceptible time during gestation, there is direct virus-induced necrosis of the granular layer of the cerebellum and also a vasculitis is induced which results in edema, hemorrhage, and tissue necrosis in utero. This results in depletion of granule cells, swollen Purkinje cell axons within the granular layer, and loss or ectopia of Purkinje cells. At birth, the inflammatory lesion is resolved, and the virus is undetectable.

Hereditary syndromes of cerebellar hypoplasia show similar pathologic changes, but present without other developmental defects in the CNS. The cerebellum size may range from grossly normal, to visible only histologically. Thinning of cerebellar peduncles may be observed in some cases. Histologically, there is narrowing of the granular layer, and loss of Purkinje cells. The structure of the cerebellar cortex appears disorganized. Changes may be segmental, localized, or global throughout the cerebellum.

Cerebellar hypoplasia associated with in utero BVDV infection. Cerebellum is small and folia are thinned.
Cerebellar Abiotrophy
Cerebellar abiotrophy is an inherited disorder of Holstein and Angus cattle. Clinical signs may appear suddenly and include intention tremors of the head, lack of menace reflex, ataxia, base wide stance, and dysmetria. Animals with abiotrophy are usually clinically normal at birth and onset of signs usually starts between 3 and 24 months of age.5

Postmortem Findings6
Abiotrophy is defined as premature degeneration of normally formed tissues or cells, and is presumed to be due to an intrinsic, metabolic defect. Gross lesions of abiotrophy may be absent, the cerebellum is most often of normal size and structure in spite of severe, clinical disease observed. Histologic changes indicate ongoing degeneration of cells. Purkinje and Golgi cells may be shrunken and dark, or swollen and pale. Often there is degeneration and segmental loss of Purkinje cells resulting in ‘empty baskets’ at the junction between the granular and molecular layers of the cerebellum. Secondary astrogliosis and a spongy appearance in this region are common in later stages. Secondary loss of cells in the granular layer is also seen.

Cerebellar abiotrophy, immunohistochemistry GFAP. Left panel is normal cerebellum, with astrocyte processes surrounding normal Purkinje cells. Right panel: cerebellar abiotrophy; severe loss of Purkinje cells, astrocyte network is present.
Beta-Mannosidosis
Beta-mannosidosis is an inherited defect of glycoprotein metabolism. The disorder is associated with a deficiency of the lysosomal acidic β-mannosidase in calves of the Salers breed, resulting in the accumulation of mannose-based oligosaccharides in lysosomes in neural and extraneural tissues. Some affected neonatal calves are not able to rise. Others have intention tremors and nystagmus. The head may be moderately domed and some calves’ exhibit mild superior brachygnathism. Results of pedigree examination and breeding trials are consistent with an autosomal recessive trait. Decreased beta-mannosidase activity is present in the serum or plasma of affected calves. This test can be used to detect heterozygotes.

Postmortem Findings
Gross pathology lesions in the brains of calves presenting with beta-mannosidosis include dilation of ventricles, and pale and/or decreased amounts of white matter of the cerebrum and cerebellum. On histological examination, there is generalized fine cytoplasmic vacuolation of neurons. These vacuoles appear to be empty following normal histologic slide preparation procedures, but by ultrastructural examination they contain membrane-bound lysosomes containing lucent material. Many, sometimes large axonal spheroids are present and there is hypomyelination/dysmyelination which may assist in the differentiation of this disease from other storage diseases in the bovine. Multifocally, macrophages with vacuolated cytoplasm may be found perivascularly and mineralization may be found multifocally in the cerebrum and cerebellum. Similar cellular vacuolation may be found elsewhere in the body, especially in the epithelium of kidneys and thyroid gland, and reticuloendothelial cells.

Acquired Spinal Cord Diseases
Acquired spinal cord diseases result most commonly from infections, trauma, metabolic/nutritional or toxic causes and rarely, from neoplasia. Clinical signs depend on the location of the lesions.

Spinal Cord Trauma
Spinal cord trauma is often associated with vertebral fractures that compress the spinal cord. They may result from riding injury in heifers and cows, chute accidents in feedlot calves or forced delivery in neonatal calves. The lesions may be caused by disruption of the spinal cord tissue, compression from a hematoma or the fractured bone. Clinical signs appear soon after the injury as flaccid paralysis, caudal to the lesion and the animal will remain recumbent. Sensation may also be reduced caudal to the lesion. Infrequently, the neurologic signs can appear later if the spinal cord compression is associated with callus formation. Careful inspection and palpation of the vertebral column may reveal evidence of trauma or elicit pain or abnormal movement localized at the affected area. Rectal examination can help detect fractures with displacements. Diagnosis can be confirmed with radiography and/or myelography in young calves. The cerebrospinal fluid is usually normal unless there is bleeding in the CSF. Treatment consists in the administration of large doses of corticosteroids or nonsteroidal antiinflammatory drugs to limit or decrease the edema associated with the trauma. Prognosis is guarded at best.

Spinal/Vertebral Abscesses
Vertebral body abscesses/osteomyelitis or spinal abscesses usually occur by spread of bacteria hematogenously to the vertebrae. The origin of the infection may be the lungs, the liver, and the kidneys or associated with an omphalitis in young calves, a traumatic reticulitis in adult cattle. Bacteria most commonly isolated include Arcanobacterium pyogenes (mostly in calves) and Spherophorus necrophorus (more frequent in adults). In a study of 21 animals with vertebral abscesses, 12 were calves ranging in age from 2 weeks to seven months. Nine animals were adults from 1 to 5 years of age. The abscesses involve one vertebra in most cases, but sometimes up to 3 adjacent vertebrae or between vertebrae. Cervical, thoracic, and lumbar vertebrae can be involved. Clinical signs vary from ataxia, to reluctance to walk, adopting a dog-sitting position, weakness, and inability to stand without assistance. Hematologic findings include increased serum protein levels and plasma fibrinogen. Cerebrospinal fluid can be unremarkable or show a slight increase in protein and white blood cells if the abscess extends in the epidural space. Radiographs are helpful to determine the affected vertebrae. Treatment is rarely performed, but surgical drainage is reported.
Postmortem Findings

Compressive or traumatic lesions in the spinal cord show similar pathologic changes, with minor variations related to cause. In simple traumatic injuries, uncomplicated by inflammation, the initial reaction to trauma may be seen grossly as hemorrhage and edema surrounding and within the spinal cord. The spinal cord appears swollen and wet at the site of injury, more chronic lesions may show visible narrowing (if the compression was severe enough) and yellow discolouration. In compression related to abscesses and/or osteomyelitis, the meninges may appear cloudy or thickened and adhesions may be present. Areas of necrosis may be found, either as a result of the inflammatory process, or due to vascular compression or damage. This may occur directly from an acute traumatic injury or as a result of stretching/compression of vessels over bony prominences such as fracture calluses.

Histologic lesions show similar progression no matter the cause. Initial compression results in swelling of axons and dilation of myelin sheaths. The location, distribution, and severity depend very much on the insult severity and duration of compression. Reaction to this damage is also found in the neuronal cell bodies, visible as central chromatolysis and clumping of Nissl substance. Distal to the injury, Wallerian degeneration can be identified as swelling and fragmentation of axons. The anatomic location of these changes follows the axonal tracts and may be in a different region of the white matter than the initial injury. Removal of axonal fragments in the spinal cord is initially performed by microglia, and later by macrophages. Secondary demyelination and myelin fragmentation can be found. Chronically, healing is by astrogliosis and glial scars may be found in very longstanding lesions.

REFERENCES

An Update on BSE in Canada
Catherine Graham¹, DVM, MVSc, DACVP; Michel Levy², DVM, DACVIM; Ed Pajor², BSc, MSc, PhD; Stefanie Czub¹,², DVM, PhD
¹Canadian & OIE Reference Laboratories for BSE, NCAD-CFIA, Lethbridge Laboratory, Lethbridge, AB, Canada; ²Faculty of Veterinary Medicine, University of Calgary, AB, Canada

Bovine spongiform encephalopathy (BSE) is a chronic, fatal neurologic disease which affects cattle and other Bovidae and is known to also infect cats and humans. It was first reported in 1986 in the United Kingdom and is a result of feeding bovine meat and bone meal to bovines. Control measures implemented in the United Kingdom were focused on prevention of the recycling of the agent in feed material, including initially a ruminant-to-ruminant and subsequently an all-mammalian feed ban focused on tissues containing the highest levels of infectivity (termed specified-risk material, SRM).

The Canadian government identified BSE as a reportable disease in 1990 and implemented at the same time surveillance testing focused on animals with neurologic disease. These were mainly samples from animals to be tested for rabies virus. Also, as a proactive measure, Canada implemented in 1997 a feed ban which prohibited the use of ruminant material in any components of feed which was intended to be fed to other ruminants. This approach to surveillance was in accordance with the requirements of the OIE until 2001. In that year, the National TSE Surveillance Network was created, which includes the National and OIE Reference Laboratory for BSE at the National Centres for Animal Disease - Lethbridge Laboratory CFIA as well as provincial and university laboratories across the country. With the creation of this network, a greater number of samples could be evaluated across the country, and surveillance testing was expanded.

To date, Canada maintains a targeted surveillance program which focuses on animals that are greater than 30 months displaying clinical signs consistent with BSE or other evidence of disease. Targeted animals belong to one of four categories: dead, down/nonambulatory, diseased, or distressed (emergency slaughter). Animals in these categories are identified for testing by veterinarians, animal owners, or animal handlers; and samples are obtained by veterinarians or CFIA staff nationally for submission to an approved laboratory. In addition, an enhanced feed ban was enacted in 2007 to further restrict the possibility of transmission via feed. The current feed ban prohibits the inclusion of any material originating from ruminants in feed, and specified risk material is prohibited from being included in feed for any animal species as well as in fertilizers. SRM removed at slaughter also prevents any potentially infectious material from being introduced into food for humans.

In May of 2003, Canada detected its first indigenous case of BSE in a cow in Alberta, resulting in a dramatic increase in samples tested for BSE surveillance. Also in 2003, atypical BSE was reported for the first time in Europe. Compared to ‘classical’ BSE, this form typically occurs in older animals and can be differentiated in laboratory testing by its biochemical characteristics. These novel forms of BSE are termed either L- or H-BSE based on their characteristic behaviour in western blot testing. The origin of atypical BSE is unknown at this time, and its risk of transmission to other bovines via feeding is also unknown. It has been shown that atypical BSE is readily transmissible to nonhuman primates, and incubation period in this model is shorter than similar experiments using classical BSE. Due to these unknown factors, more data is needed to evaluate these diseases further to determine their risk to humans or other animals.

**Diagnosis of Bovine Spongiform Encephalopathy**

**Clinical Examination**

Clinical signs of BSE may be vague, and no pathognomonic signs exist for a confident clinical diagnosis. Clinical signs may include nervousness or anxiety; abnormalities in response to stimuli including auditory/visual or physical stimuli; abnormalities of locomotion; or changes in production (decreased milk yield or weight loss). Clinical disease due to BSE infection progresses over a long period (weeks to months), although clinical signs may not be identified or may be misinterpreted in the early stages. A description of clinical observation is important to include in each submission for BSE testing, as animals
with clinical disease consistent with BSE are more valuable to be presented to the OIE to demonstrate sufficient surveillance is being performed.

**Laboratory Diagnosis**

Surveillance testing in Canada for BSE is performed in a number of laboratories using commercially available tests which have been validated and approved. The most common test methods used are ELISA and lateral flow immunoassay. If a non-negative result is obtained, the test is repeated in duplicate at the surveillance laboratory, and the National BSE Reference Laboratory is contacted. If the sample is again non-negative, it is submitted to the reference laboratory for confirmation. At the reference laboratory, a panel of tests is performed to confirm the status of the sample and, if positive, to type the prion. Confirmatory testing includes immunohistochemistry with a panel of anti-prion antibodies and/or SAF immunoblot which is an extended western blot employing a more rigorous prion concentration to enhance the sensitivity of the method. In addition, a western blot is performed using two or more anti-prion antibodies to differentiate C-, H-, and L-type BSE. Below are examples of immunohistochemistry and western blot results which demonstrate the differences in C-, H-, and L-BSE.

![Immunohistochemistry](image)

**Immunohistochemistry**, monoclonal antibody F99 (VMRD), detection kit: Envision+ (DAKO), DAB chromogen. Brown staining is detection of PrPSc in neurons, glia, and neuropil.
Western blot. Detection of PrP<sub>c</sub> with monoclonal antibody 6H4 (Prionics). All types of BSE are detected with this antibody. CWD and scrapie have variable detection. Molecular weight and glycoform ratios are used to differentiate BSE types.

Western blot. Detection of PrP<sub>c</sub> using monoclonal antibody P4. C- and L-BSE are weakly reactive or negative with this antibody. H-BSE, CWD, and Scrapie are detected with this antibody.

**Research at the CFIA NCAD Lethbridge Laboratory**
Current research for BSE in the CFIA is focused on evaluation of the risk of contamination of feed, as well as to enhance or improve on current diagnostic test methodologies. To this end, projects involving
intracranial or oral transmission to bovines using atypical and classical BSE are underway. Intracranial transmission of atypical BSE to bovines was successful with a shorter incubation time and more rapidly progressive clinical disease when compared to classical BSE. Atypical BSE animals displayed clinical disease between 11 to 12 months post challenge; in contrast, in animals challenged with classical BSE clinical disease starts at around 18 to 20 months post inoculation. Once present, clinical disease in animals affected with atypical BSE progresses more rapidly and consistently as compared to classical BSE.

Oral challenge of calves with atypical BSE has not resulted in induction of clinical disease so far (~53 mpi), and oral challenge of adult animals with classical and atypical BSE has also not induced any clinical signs so far (~44 mpi). Should oral transmission be successful, evaluation of tissues from these animals will determine the distribution range of PrP$^{sc}$ and which tissues need to be excluded from animal feed or food destined for human consumption.

Sheep and goats are susceptible to infection with classical BSE and develop a disease which is clinically indistinguishable from scrapie, but ovine or caprine BSE can be differentiated from scrapie using western blot testing. In another experiment, we want to determine the potential for sheep to be infected with atypical BSE. Lambs will be challenged orally or intracranially with atypical BSE.
**IMPORANCE OF COW LONGEVITY, LAMENESS, AND COW COMFORT**

Longevity can be defined as the interval that a cow remains in the herd after first calving; in other words, it is the length of her productive life. The survival of a cow is influenced by many factors. Management-related factors include milk quota restrictions, availability of replacement heifers, nutrition, overstocking, management practices that influence the incidence of disease, and barn features such as stall size, bedding type, and flooring. Cow-related factors include, among others, metabolic and reproductive disorders, conception rates, milk production efficiency, body condition, and conformation of legs, feet, and udder.

The environmental impact of dairy production is reduced when the longevity of each cow and the efficiency of milk production are high. Lameness and injuries have a negative impact on the cow’s productivity and longevity, thereby reducing the overall efficiency of dairy production. Recent surveys in Canada have revealed an alarmingly high prevalence of lameness and injuries such as hock or neck lesions among dairy cows that can markedly affect production and longevity. Reduced cow comfort associated with poor flooring, lying stall, feeder design, and management are important risk factors for a high prevalence of lameness and injury. Changes in the duration of standing and lying can be used to detect problems with cow comfort that lead to lameness and injury. Recent technological developments have produced inexpensive automated methods of measuring lying time that are practical for on-farm use. Results from the first Dairy Cluster Project focused on ‘Improving cow comfort to increase longevity in tie-stalls and free-stalls in Quebec, Alberta, and Ontario dairy herds’ will be presented.

**LONGEVITY AND LAMENESS PROJECT**

One hundred and twelve free-stall farms (FS) were visited in Ontario (ON, n = 40), in Alberta (AB, n = 50), and in Quebec (n = 22). Additionally, 100 tie-stall farms (TS) and 25 automatic milking system farms (AMS) were visited in Quebec, Ontario, and Alberta, but will not be highlighted during the presentation.

**Herd Selection**

To be eligible for participation in this study, farms had to be enrolled within CanWest DHI (Guelph, ON). Farms were only included in the study if they had ≥ 40 Holstein-Friesian milking cows and if their milking cows had been housed in their current housing system for at least 1 year. To ensure study farms were representative of the majority of Canadian dairy farms, farms were excluded from the study if the milking cows had outdoor access for more than 2 hour/day (see Zaffino, et al. 2014 for details). On each farm, 40 cows were selected to represent the herd. As much as possible, these cows were between 10 and 120 DIM because this period has been shown to be associated with increased incidence of lameness and injuries. Cows < 10 DIM were not selected, to allow for habituation to their present environment and to account for the dry cows often being housed in separate facilities.

**Cow Measures**

**Lameness & Claw Lesions**

**Locomotion scoring:** All 40 cows were gait scored to determine the prevalence of lameness. The cows were recorded as they exited the parlor. At least two, complete walking strides of each cow had to be achieved in order to analyze the video. In free-stalls, we have used the scoring method developed by Flower and Weary, which has been simplified for on-farm use by Ito et al. Limping (L), asymmetric steps (A), and head bob (H) were the three clinical signs recorded when assessing locomotion. This
locomotion scoring method has been validated as a means of detecting hoof lesions,\textsuperscript{4} can identify problems of cow mobility in automated milking systems, and can be done with a high degree of agreement between trained observers.\textsuperscript{2,4}

**General description of gait behaviors**

<table>
<thead>
<tr>
<th>Behavior</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head bob</td>
<td>Up and down head movement when walking. The head moves evenly as an animal walks, but animals with injuries may have jerky head movements when walking.</td>
</tr>
<tr>
<td>Asymmetric steps</td>
<td>Abnormal rhythm of the foot placement. An animal free from injuries would be expected to place its hooves in an even “1, 2, 3, 4” fashion, but an animal with injuries may have an uneven rhythm of foot placement “1, 2….3, 4”. Not equal, a cow places her hooves in an uneven rhythm.</td>
</tr>
<tr>
<td>Limping</td>
<td>The animal is favoring one or more limbs. Animals free from injury bear weight evenly over the four limbs. An animal with an injury may not place all its weight on an affected limb.</td>
</tr>
</tbody>
</table>

**Description of the presence and absence of three important gait behaviors**

<table>
<thead>
<tr>
<th></th>
<th>Absence</th>
<th>Presence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head bob</td>
<td>Even, gradual up and down head movement when walking.</td>
<td>Jerky or exaggerated up and down head movements when walking.</td>
</tr>
<tr>
<td>Asymmetric steps</td>
<td>Hooves placement is in an even “1, 2, 3, 4” fashion.</td>
<td>Uneven rhythm of foot placement “1, 2….3, 4”. Foot placement is not equal on both sides; cow places her hooves in an uneven rhythm.</td>
</tr>
<tr>
<td>Limping</td>
<td>All legs bear weight equally.</td>
<td>Walk with an uneven, irregular, jerky, or awkward step as if favoring one leg.</td>
</tr>
</tbody>
</table>

**Example of scoring sheet**

<table>
<thead>
<tr>
<th>Date:______________________________</th>
<th>Scorer:_____________________________</th>
<th>Number of cows scored:_____________</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Asymmetry</th>
<th>Limping</th>
<th>Head bob</th>
</tr>
</thead>
<tbody>
<tr>
<td>uneven rhythm of foot placement</td>
<td>Walk with uneven weight bearing on one leg</td>
<td>Jerky or exaggerated up and down head movements</td>
</tr>
<tr>
<td>Cow 1</td>
<td>Etc.</td>
<td></td>
</tr>
</tbody>
</table>

**Lesion scoring:** Commercial hoof trimmers were trained for claw lesion scoring to guarantee a systematic and consistent lesion description. Lesions and location of the lesion were captured during regular trim sessions in an automated recording system (Hoofsupervisor®).

**Injuries**

Cows were scored for neck, knee, and hock injuries according to the criteria described by Gibbons et al.\textsuperscript{5} at exposed bony anatomical locations. Hock injuries were most commonly scored in the milking parlor, whereas knee and neck injuries were commonly scored in the free-stall area where cows were free to move.\textsuperscript{1}
## Injury assessment

<table>
<thead>
<tr>
<th>Scores 0 &amp; 1</th>
<th>Score 2</th>
<th>Score 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>No swelling</td>
<td>Medium swelling (1–2.5 cm) and/or lesion on bald area</td>
<td>Major swelling (&gt; 2.5 cm). May have bald area/lesion.</td>
</tr>
<tr>
<td>No hair is missing or bald area</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tally the number of cows scoring 0 &amp; 1</td>
<td>Note the IDs of the cows scoring 2</td>
<td>Note the IDs of the cows scoring 3</td>
</tr>
</tbody>
</table>

### Group 1

### Group 2

(etc.)

### Total # cows:

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>%:</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Condition of the hocks can be an important warning of the abrasiveness of stall bedding and cow comfort; prolonged exposure to an abrasive stall surface can cause injury. Knee health may be a sign of the hardness of the stall floor and cow comfort. Injury in hocks and knees is usually the result of prolonged exposure to a hard stall floor leading to swelling and skin breakage that provides an opportunity for infection to occur resulting in discomfort and possibly lameness. Neck injury is usually the result of prolonged exposure to rubbing, or hitting, against the neck rail/chain or feed-bunk rail/chain. The prevalence and severity of neck injuries can be an indicator of whether the neck rail/chain in the stalls and/or the feed bunk are at the correct height or length (chain), and that the feed is within easy reach.

### Cleanliness

Cow cleanliness is an important indicator of cow comfort. Frequent and strategic cleaning of the alleys and stalls will reduce the amount of manure on cows and the amount of manure tracked into the stalls. Conversely, dirty legs are often due to manure splashing in the alleys, whereas dirty flanks and udders are a result of animals lying in dirty and/or poorly bedded stalls. When the lying area is wet, cows reduce their lying time. Cows with large areas of dried caked manure indicate a long-term buildup of manure and highlight weaknesses in the cleaning routine of the alleys and/or stalls. Therefore udder, leg, and flank cleanliness was recorded, as follows:

<table>
<thead>
<tr>
<th>Score 0</th>
<th>Score 1</th>
<th>Score 2</th>
<th>Score 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Light</td>
<td>Moderate</td>
<td>Heavy</td>
<td>Very heavy</td>
</tr>
<tr>
<td>Contamination of fresh splashes of manure for &lt; 50% of the area</td>
<td>Contamination of fresh splashes of manure for &gt; 50% of the area</td>
<td>Contamination of dried caked and fresh manure for &lt; 50% of the area</td>
<td>Contamination of entire area with dried caked manure</td>
</tr>
</tbody>
</table>

### Lying Time

Accelerometers were attached to one rear leg of all cows and removed up to 10 d later. This provided us with measures of total daily duration of lying, frequency of bouts of standing and lying, and mean duration of bouts of standing and lying.

Other animal-based measures (somatic cell count, milk production, days in milk, and reproductive measure) were retrieved from the DHI/Valacta records. The size of cows was measured (e.g., length, height, width at hip).
The Relationship Between Lying Time and Lameness
The relationship between lameness and the time that cows spend lying down is complicated by the fact that cows can spend more time lying down once they are lame than healthy cows, but long standing times mean more exposure to concrete flooring, thereby increasing the chance of the development of claw lesions. There is also considerable evidence that poor stall design and management increase the risk of lameness by reducing the duration that cows spend lying down.\(^4,6,7\) Thus, a high proportion of lameness in a herd is likely to be associated with cows that spend either an unusually long, or an unusually short interval, lying down. In a study in British Columbia, Ito \textit{et al.} (2009) showed cows having extreme lying times (i.e., less than 9 or greater than 14 h/d) were 2.5 times more likely to be lame than cows with normal resting times.\(^3\)

Environment
Stall dimensions were measured as well as lunge space evaluated as ‘adequate’ if there was no obstruction 76 cm forward from the centre of the top of the brisket board and to a 45° angle to the left and right, and ‘inadequate’ if there was an obstruction in this space.\(^8\) Stall base and bedding type were recorded. Bedding depth was evaluated as none (unable to rake bedding), ≤ 2 cm (when raked evenly for organic bedding, or below the curb for sand bedding), or ≥ 2 cm (when raked evenly for organic bedding, or even with or above the curb for sand bedding). It has been documented that there were increased lying times for every additional 1 cm, or 1 kg, of bedding;\(^9,10\) furthermore, 2 cm would be the minimum required to completely cover the stall base so as not to have bare spots. Stall cleanliness was evaluated qualitatively after the cleaning routine; to get an indication of how well cleaning was being done. Stalls were considered clean if they had little or no manure or wet spots. For each pen, the stocking density was calculated as the number of cows divided by the number of lying stalls. The total daily time spent outside of the home pen for milking was calculated per pen as the time between the first cow leaving the home pen and the last cow returning back to the home pen, multiplied by the milking frequency per day. Cows’ slips and falls were observed for up to 30 min while they were being moved to the holding area for milking, or alternatively, in the pen on the way back from milking if observation in the holding area was not possible. Slips and falls were categorized as 1 (any number of cows slipped or fell) or 0 (none of the cows slipped or fell).\(^1\)

Management: The Final Piece of Data Collection
Each of the participating farmers in our project was interviewed to gain insight on topics related to stall and pen management, health, lameness, and welfare perception, as well as claw health, hoof trimming, and lameness monitoring.

Final Thoughts
The concept of cow comfort has been identified as key for preventing lameness and increasing longevity. Stall design and surface, bedding quality and quantity, flooring, space allowance, and management practices are all aspects of the cow’s life on the farm that play an important role in controlling lameness and enhancing the health of dairy cows, consequently increasing their longevity and ensuring their wellbeing. There was huge variability across Canadian dairy farms in our study in terms of management practices, barn design, cow comfort features, and prevalence of lameness.

Acknowledgements
We would like to acknowledge all participating producers, hoof trimmers, and collaborating partners at the different universities across Canada. Funders of this project were: Dairy Research Cluster Initiative (Dairy Farmers of Canada, Agriculture and Agri-Food Canada, the Canadian Dairy Network and the Canadian Dairy Commission), Valacta, Novalait, DHI, Alberta Milk, Alberta Livestock and Meat Agency (ALMA), and University of Calgary Faculty of Veterinary Medicine.
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Digital Dermatitis: The New Enemy of Cattle Welfare in Our Canadian Dairies
Karin Orsel, DVM, MSc, PhD, DECNHM; Team Members (Herman W. Barkema, Laura Solano, Casey Jacobs, Dörte Döpfer)
Department of Production Animal Health, Faculty of Veterinary Medicine, University of Calgary, AB, Canada

BACKGROUND
One of the most important foot disorders is digital dermatitis (DD; also known as “strawberry footrot,” “Mortellaro,” “hairy heel warts,” “raspberry heel,” and “papillomatous digital dermatitis”). Digital dermatitis is one of the most important causes of lameness in dairy cattle kept under intensive animal husbandry systems. This disorder, common on almost all cattle farms in North America, causes major economic losses due to decreased fertility and milk production, treatment costs, and an increased chance of early culling. These losses are a direct consequence of the pain associated with DD; therefore, DD is also a major animal welfare issue.

The pathogenesis of DD is not fully understood, perhaps due to the multifactorial nature. Multiple species of spirochetes and gram-negative bacteria have been isolated. However, DD lesions were recently reproduced experimentally by inoculation of Treponema spp., supporting an important pathogenic role of this agent. Lesions occur at various sites of the skin horn border of claws and change during disease development and regression. The lesions appear in various stages, namely M1-M2-M3-M4-M4, each representing a stage in the development of the disease from acute to more chronic and combined presentations. Risk factors associated with DD, at the cow level include, early stage of lactation and low parity, whereas at the herd level, poor biosecurity, wet and unhygienic flooring, and frequency of foot bathing were identified.

DIAGNOSIS, TREATMENT, AND CONTROL OPTIONS

Diagnosis of DD
Cows affected with DD are usually easily spotted by inspecting the skin horn border of the heels. Since the lesions can be very painful it can also cause the cow to walk lame. The lesions can also be identified by washing the heels in the parlour and using a bright light and mirror to identify abnormal heel skin, as validated by Relun et al. However, if the lesion is located around the coronary band, or high up in the interdigital space, diagnosis can be more difficult, as the observation of this part of the claw often requires a chute system to lift the leg for close inspection.

Treatment
The most commonly used options for treatment and control include topical antibiotics (e.g., oxytetracycline) and antibacterials or disinfectants (e.g., copper or zinc sulfate, or formaldehyde) administered to an individual cow (preferred for antimicrobials) or as herd treatment. In a recent clinical trial, it was determined that topical application of tetracycline hydrochloride, in a powdered form held in place with a bandage, or as a paste without bandage, were therapeutically effective for treatment of DD. Also, digital lesions can be painful during both active and healing stages indicating the need for treatment and husbandry interventions for pain mitigation. Digital dermatitis has long-term effects on heel horn formation, although most farmers are only aware of the short-term, active lesions and their consequences (e.g., lameness).

Due to the endemic nature of DD and increasing dairy herd size, the use of footbaths is the most common approach for prevention of DD. Chemicals used for footbaths to prevent DD are used in huge quantities in the field. Additionally, most products are used off-label, and several concerns have been raised, for example, the carcinogenic effect of formaldehyde, the environmental impacts of disposed solutions, and the risk of antibiotic residues in milk and meat.
**Control**

Digital dermatitis can spread within a herd as well as between herds. Therefore appropriate biosecurity measures are important to prevent new strains from entering a premise; biosecurity measures include quarantine for new additions to the herd, boot disinfection for farm visitors, the use of farm-specific equipment, and special attention should be paid to hoof-trimming equipment when it is used between farms. Trimming equipment should be disinfected between cows and before leaving the farm. For control of within-herd spread, special attention needs to be paid to introductions of new animals into the dairy herd (fresh cows as well as heifers). Therefore, lameness inspections and appropriate treatments should be done in the dry, or young, stock barn before these cattle are added to the herd.

**Canadian Situation**

Cramer et al. presented prevalence estimates for 11 foot lesions in Ontario dairy cattle as recorded by five hoof trimmers on 13,530 cows in 204 Ontario dairy herds. In free-stall housing systems, 46.4% of cows had a foot lesion; DD was the most common lesion, with 22.7% of cows and 96.7% of herds affected. They concluded that foot lesions diagnosed at hoof trimming are common in Ontario, and appropriate treatment for hoof horn lesions appeared to be low. In the same study, a questionnaire was used to identify herd-level risk factors. Important risk factors identified for seven of these foot lesions in free-stall housing included increased alley scraping frequency (2.2 to 2.4 fold for sole ulcers) and trimming in summer or fall (-0.2 fold vs. spring and winter for DD). Protective risk factors included intermediate bedding depth (0.4 fold for 2.5 to 7.5 cm vs. more or less bedding for interdigital fibroma) and trimming heifers before calving (0.1 fold for white line abscess).

Our research group recently conducted a study on lameness and cow comfort on 240 dairy, free-stall farms across Canada, of which 86 dairy farms in AB. Using a questionnaire, we identified DD as one of the issues of greatest concern to farmers. This observation is supported by data obtained through our collaborative study with the AB Dairy Hoof Health Project (www.hoofhealth.ca) overseen by Alberta Milk. In the 33,000 cows included in that study, DD accounted for 44% of all claw lesions. A similar project in Ontario with 24,045 cows resulted in 35% of the cows with a lesion being affected by DD. In both provinces, the within-herd prevalence ranged widely among farms (0 to 81%).

**Ongoing Digital Dermatitis Related Research**

At the University of Calgary, Faculty of Veterinary Medicine, two clinical trials are ongoing. The first one is evaluating early detection and preventative treatment methods for control of DD in Alberta dairy cows. The objective is to determine whether an experimental hoof treatment is comparable to a positive control (5% copper sulfate solution) for prevention of DD and lameness. The other study is focusing on adaptation of a standardized footbath protocol using an automated footbath system. The ultimate goal is to provide the dairy industry with a practical and effective control measure for infectious foot disorder like DD.

**Acknowledgements**

The team thanks all the hoof trimmers and producers involved in our field trials. Also, a huge thanks to funders and supporters of our study, including: Alberta Milk, Alberta Livestock and Meat Agency, and the University of Calgary Faculty of Veterinary Medicine.

**Recommended Sources**

41. http://vancepublishing.adobeconnect.com/p9e7hvdez9c/. (VIN editor: Link no longer accessible as of 4-3-14.)
42. [www.dairyherd.com](http://www.dairyherd.com)
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When Species Mingle, So Do Diseases!
Karin Orsel, DVM, MSc, PhD, DECNMH; Team Members (Susan Kutz, Mathieu Pruvot, and others)
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INTRODUCTION
In southwestern Alberta, cow-calf operations extensively use large pastures in the foothills of the Canadian Rocky Mountains. These foothills are covered with a mosaic of forest and grassland, characteristic of the montane ecosystem which is also a primary habitat for North American elk (Cervus elaphus). Cattle and elk not only share the same ecosystem, but also have similarities in their foraging behavior, which tends to increase the likelihood of their interactions compared to other sympatric ungulates.1-3 The opportunity for pathogen transmission has been described at the interface between livestock and wildlife, and is now gaining increased attention. For example, in Riding Mountain National Park, the high elk density, expanding on the ranchland surrounding the park, enabled bovine tuberculosis to be transmitted between elk and cattle.4 Similarly, the shared use of feeding grounds by elk and cattle resulted in transmission of brucellosis in the Great Yellowstone area.5,6 One of the key aspects in understanding how pathogen transfer can occur between host species is the spatio-temporal, contact patterns between these species. In other words, where are cattle located relative to elk at various times of the year and does that allow pathogen transmission between species?7

The cattle industry has identified diseases that have a high economic impact on production, known as production-limiting diseases.8 Several of these diseases have also been suspected to circulate in wildlife.9-12 When multiple host species are susceptible to a disease, it is important to consider the respective role of each host and appropriately define the populations of interest. The ‘target population’ is the population of concern or interest to us. All other potentially susceptible host populations that are epidemiologically connected, directly or indirectly, to the target population are non-target populations and could potentially constitute all, or part of, the reservoir. A reservoir can be defined as one or more epidemiologically connected populations or environments in which the pathogen can be permanently maintained and from which infection is transmitted to the defined target population.13 However, populations in a reservoir may be the same or a different species as the target. Populations large enough to maintain an infection are called ‘maintenance populations’, whereas populations in which the infection naturally fades out are called non-maintenance populations.13 Finally, a population that occasionally gets infected from the target population, but cannot maintain the infection is often called a spillover host. An infection transmitted from a spillover host to an individual of the target population is sometime called a spillback event. The respective role of cattle as our target population and wildlife species in the same reservoir for production-limiting diseases depends, for example, on the susceptibility of these hosts to the pathogens. We can use the prevalence estimations of pathogens in each hosts and the contact structure to better understand disease maintenance in the populations.

STUDY OBJECTIVES
45. Determine the infection status of Mycobacterium avium subspecies paratuberculosis (MAP), BVD, IBR, and Neospora caninum in free-ranging elk herds in southwestern Alberta.
46. Assess the influence of interspecies comingling associated with occurrence of these pathogens in cattle and elk.

DATA COLLECTION IN SW ALBERTA
We selected four production-limiting pathogens known to be present in cattle in southwestern Alberta: bovine viral diarrhea virus, bovine herpesvirus 1, MAP, and Neospora caninum. For each of these four pathogens, previous reports have highlighted their significance for the beef producers in this area.14-17 Elk were also reported to be susceptible to these pathogens and to present signs of infections.18-22 In addition, local veterinarians indicated that Fascioloides magna was of interest for producers in this area.23,28 F. magna,
or giant liver fluke, is a trematode of elk and deer that can accidentally infect cattle and cause clinical signs and mortality in young stock. Cattle are susceptible, but non-competent hosts, meaning that eggs of the parasite are not released in the environment and cannot be detected in the feces.24

Having the opportunity to collaborate with the Montane Elk Research Program, we focused on 7 elk herds included in this project: Castle-Carbondale, Beauvais Lake, Porcupine Hills, Livingstone, Whaleback, Waterton National Park (NP), and Crowsnest Pass (www.montaneelk.com). Another 3 elk herds were added to the study (Banff NP, Jasper NP, and Yaha Tinda) where elk forage in areas without cattle presence. Blood samples were collected during captures for GPS collaring; elk location data were available from GPS collars and fecal samples were collected during additional field sampling. In the next phase of the project, we recruited 30 ranches of which 15 grazed cows on pastures used by elk, and 15 ranches in areas were no elk were observed. From these cow-calf herds, we collected blood samples, fecal samples, and management information through an extensive questionnaire and interview.

**RESULTS AND CONCLUSIONS**

**Pathogen Circulation**

Initial laboratory projects were to adapt and validate diagnostic tools, since commercially available diagnostics are not always suitable for wildlife species.25

In elk, there was an overall seroprevalence of 63.8% (178/279) for herpes virus, whereas in cattle the overall seroprevalence was 96.5% seropositive (844/875), which can be explained by vaccination status of the herds. Based on results from RT-PCR for alpha-herpesvirus, we identified infections in elk were due to an elk-specific rhadinovirus and not to BHV1.

Regarding BVD, 92.6% of all cattle in our project were seropositive (810/875), with many of those attributed to vaccination. However, in elk, overall seropositivity was 2.9% (8/279), with all 8 positive samples from elk herds exposed to beef cattle. It was noteworthy that all cattle and elk samples were negative on PCR for viral antigen.

Six (of 28) ranches had at least one seropositive cow for MAP, although only one ranch was found to have cows actively shedding the bacterium. Further analysis indicated that up to 20% of cow-calf herds in southwest Alberta may have at least one positive cow. Applying the modified ELISA to 284 elk sera, 1.3% (95% CI: 0.03–2.6%) tested positive with the original kit cutoff value, and 3% were positive based on the newly defined, elk-specific cutoff value. The occurrence of MAP in elk was not associated with their exposure to cattle, which may suggest circulation in elk independently of contact with cattle (maintenance population).

The overall prevalence of *Neospora caninum* in cattle was 6%, with at least one positive cow on 53% of the ranches. Exposure to *N. caninum* was also detected in 5% of sampled elk. Elk in contact with cattle had a higher risk of being seropositive for *N. caninum*. We also identified the importance of proper carcass disposal to mitigate circulation of this pathogen in cattle and we speculated there may be a sylvatic cycle of this parasite, involving wild carnivores and elk. In contrast, the occurrence of *N. caninum* in cattle was not related to the presence of elk. Perhaps vertical transmission in beef cattle (from dam to calf) is most important, and little influenced by the density of susceptible host, whereas horizontal transmission among elk, cattle, and carnivores may increase the risk for elk in areas frequented by infected cattle.

Finally, the prevalence of *F. magna* was significantly lower in elk herds comingling with cattle, as elk without contact averaged a prevalence of 40% fecal egg positive and herds with cattle contact averaged 0%. Our team developed an ELISA and Western Blot for detection of exposure to *F. magna* in cattle; overall, 5 of the 15 ranches with exposure to elk had seropositive cows, whereas there were no seropositive cattle on ranches that were not exposed to elk. Since cattle are dead-end hosts for this parasite, if cattle and elk are grazing the same pastures, perhaps cattle decrease the risk of elk becoming infected.

We concluded that indirectly transmitted pathogens were more likely to co-occur in cattle and elk, indicating the potential importance of this transmission route in assessing the risk of pathogen transmission in multispecies grazing systems.26
This project gave us new insights regarding risk of disease transmission between cattle and elk. We are currently using the information gained on factors attracting elk on cattle pasture to quantify the potential for interspecies transmission by various routes of transmission, and by measuring direct and indirect contact rates between cattle and elk.

**ACKNOWLEDGEMENTS**

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We acknowledge other collaborators without who’s support this project would not have been successful: participating ranchers, and veterinary clinics: Pickard Veterinary Services Ltd. and Fort MacLeod Veterinary Clinic; collaborators: Mark Hebblewhite (University of Montana), Evelyn Merrill (University of Alberta), Blair Fyten (Banff National Park), Geoff Skinner (Jasper National Park), Barb Johnston (Waterton National Park), and Mike Alexander (Alberta Sustainable Resource Development).

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Determinants of Animal Welfare in Practice - A Case-Based Approach
Michelle Lem, DVM, MSc
Community Veterinary Outreach, Ottawa, ON, Canada

Traditional determinants of animal welfare have been based on Brambell’s five freedoms. Initially developed for food and working animals, they are: 1) the freedom from hunger/thirst, 2) the freedom from discomfort, 3) the freedom from pain/injury/disease, 4) the freedom from fear/distress, and 5) the freedom to express normal behaviour. While still applied in many contexts, these freedoms are often assumed in day-to-day, companion-animal practice. Less commonly discussed determinants that directly impact the health and welfare of animals in a practice setting include veterinarian- and team/clinic-based factors such as values, cohesiveness, knowledge, communication, expectation, and experience. Client-based factors include compliance and pet attachment. Through a series of case studies, these determinants, as well as solutions to common barriers, will be demonstrated and discussed.

VALUES
The Veterinarian Oath (CVMA 2004) describes the values which we professionally share and swear to adhere to, including promoting animal welfare and “relieve animal suffering.” However, in practice our actions are sometimes inconsistent with those values. When the team shares common goals and values that are in line with our oath for all animals, the outcome is improved animal welfare in the community. As veterinarians, we are responsible to not only promote the health and welfare of our own patients, but as our oath decrees, we are to use our, “knowledge and skills for the benefit of society.” Clearly understanding what our professional obligations are in this context will be discussed through case studies.

VETERINARY TEAM/Clinic-based Factor
Impacting the veterinary team’s ability to act together for improved animal welfare in practice are a number of factors including the clinic’s mission and values, team leadership, conflict resolution, and communication. Of these, and less commonly discussed in our profession, is workplace bullying and/or harassment, yet not only does this negatively affect us personally, emotionally, and physically, but also significantly and detrimentally impacts animal welfare in practice. While not well researched in veterinary practice, it has been demonstrated in the nursing profession that workplace bullying and harassment negatively impacts patient care and outcomes (Vessey, DeMarco, DiFazio 2010). With growing concern for the emotional health of veterinarians, workplace bullying and harassment is one area that needs to be further discussed and assessed in veterinary practice.

CLIENT-based Factor
Client compliance is of significance to animal welfare in veterinary practice similar to compliance in a human health care setting and includes a host of factors including socioeconomic status, the healthcare system itself, therapeutic related, patient centred, and disease factors also play significant roles in overall compliance (Jin et al. 2008). One aspect of socioeconomic status is social support and for those clients with few human attachments pets confer social capital and the people are highly attached to their pets (Adamelli et al. 2005; Marinelli et al. 2007). Pet attachment can be a profound predictor of animal welfare with the potential to both positively and negatively impact animal welfare. While high pet attachment in practice more commonly predicts better compliance and outcomes, it has also been associated with hoarding (Patronek, Nathanson 2009), inability to appropriately elect euthanasia, and overfeeding of animals, and may contribute to problem behaviours and anthropomorphism (Wensley 2008). For the practitioner, understanding the role of the pet in the family can aid in assessing compliance. Hodgson and Darling (2011) developed a way of documenting in the medical record the household members and their attachment to the pet through diagrammatic genograms (Hodgson, Darling 2011). These genograms can assist the veterinary team understand who in the family helps take care of the pet, and therefore,
ability to follow through with recommendations, improving overall compliance, outcomes, and animal welfare.

**Patient-Based Factor**
A significant patient-centred factor to welfare is the animal’s behaviour, and yet veterinarians are minimally trained in this profound predictor of animal relinquishment and welfare (Sherman, Serpell 2008). Salman (2000) found that in the UK, behavioural reasons were the most common reason for relinquishment of dogs and the second most frequently given reasons for relinquishment of cats to shelters (Salman et al. 2000). In private practice, it is unknown how many “healthy euthanasias” are performed for behavioral reasons, and how many of those could be prevented. Behavioural problems are complex, time consuming, and may be expensive to treat (Reisner 2013). However, the veterinary team has the opportunity to educate and aid owners in understanding animal behaviour, and provide recommendations for common and uncomplicated behavior problems.

**References**
152. Reisner I. Behavioral euthanasia: how and when to face a difficult decision. In: Proceedings from the Western Veterinary Conference; February 17–21,2013; Las Vegas: NV.
Myths & Truths About Animal Welfare in Practice - An Economic Argument
Michelle Lem, DVM, MSc
Community Veterinary Outreach, Ottawa, ON, Canada

Pervasive public beliefs exist around companion animal ownership such as, “People who can’t afford an animal should not be allowed to own an animal.” Similar belief systems exist in the economics of veterinary practice such as, “If I reduce the cost of veterinary services in my practice, it will devalue veterinary medicine and the profession.” These belief systems are not only a detriment to animal health and welfare, but are also not evidence based. Drawing on 10 years of experience working with homeless and marginalized (low and no income) pet owners, these and other myths along with some truths discovered along the way, will be presented and discussed.

**Myth #1: People who cannot afford a pet should not own a pet.**
The logical question to this logical statement is then “Who can afford a pet?” Looking at the economic factors and trends including wealth distribution by income, consumer spending, and poverty in Canada, the increasing divergence between the wealthy and the poor affects not only retail spending habits, but also ability to afford and access veterinary care in our practices, affecting the welfare of animals in our communities.

**Myth #2: The welfare of pets of the homeless is poor.**
There has been little research to date assessing the animals of those who are homeless, street involved, and vulnerably housed. Through my MSc research, we sought to assess aspects of the welfare of these animals that included amount of time owners spent playing and exercising with their pet, the time spent alone, as well as level of pet attachment. We found that in many circumstances, animals of those who are homeless may have a better state of welfare than animals of those who are housed, and revealed that main welfare concerns arose through lack of access to veterinary care and education.

**Myth #3: If I reduce the cost of veterinary services in my practice it will devalue veterinary medicine and the profession.**
Similar, false belief systems existed in the value of animals through adoption. Most shelters believed that free adoptions devalued the animal resulting in lower attachment as well as a poorer perception of the shelter through which the free adoption was facilitated. This was challenged by a study conducted by researchers with the ASPCA (Weiss, Gramann 2009) where they found no difference in level of attachment nor perception of the shelter through which the adoption took place. Since the debunking of this myth, many shelters across North America have challenged their perceived bias towards lower cost, or free adoptions, resulting in increased adoption of adult cats and decreased euthanasia of healthy animals. Perceptions of such value should be challenged as animals and their health contribute to significant social capital to individuals and communities (Wood 2011) and are not comparable to material objects. More research is needed to assess outcomes with regards to human-animal interactions.

**References**
The Social Impact of Animal Welfare in Practice and in the Community: The Bigger Picture
Michelle Lem, DVM, MSc
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The veterinary profession today is facing some critical challenges. Although veterinarians have never been more scrutinized and criticized by the public and the media, there has never been a greater opportunity for the profession to play a vital role in One Health. Private practitioners have the potential to positively impact not only animal health and welfare, but also human health and welfare, thereby contributing to the improvement of community health. Through the lens of community outreach, the role of the companion-animal veterinarian and veterinary services as a vehicle for social change in the community will be explored.

For many pet owners, their animal companions are considered to be part of the family. This strong emotional tie holds across cultures (Risley-Curtiss, Holley, Wolf 2006) and economic circumstances (Staats, Miller, Carnot, Rada, Turnes 1996). Among the estimated 12–19% of the homeless and vulnerably housed in Canada that own pets (Stephen Hwang, Centre for Research on Inner City Health, St. Michael's Hospital, Toronto 2011; Bill O'Grady Professor, Department of Sociology and Anthropology, University of Guelph 2012, personal communication), research has demonstrated not only a universally high level of pet attachment among this population, but also that homeless pet owners will often put the needs of their pets before their own. The result of this is that pets act as a motivator for positive behavior change of the person. For example, among street-involved youth, pet ownership has been described as a motivator to decrease youths’ use of alcohol or drugs, avoid arrest, and develop responsibility, a positive sense of self, structure, and routine (Lem 2012).

While One Health initiatives have traditionally focused on threats to human and animal health (such as diseases transmissible between humans and animals and a secure food supply), it has been slow to adopt an understanding of the many beneficial physical and psychosocial impacts of human-animal relationships and how these can be leveraged to improve both human and animal health around the world. Additionally, current One Health initiatives are undertaken at international, federal, and provincial levels yet fail to have impact at a community level where over 80% of physicians and veterinarians provide primary care. Community Veterinary Outreach’s One Health engagement initiatives includes the integration and community-level collaboration of veterinary teams with human healthcare providers to cooperatively improve the health and welfare of humans and animals. Through services provided to marginalized homeless populations in communities, we are demonstrating that veterinarians and veterinary care can act as a direct avenue to improve health service delivery for underserved populations. We believe that our model of health-service delivery for both humans and animals is both reproducible and applicable to other in-need populations.

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Using the Dairy Code of Practice to Improve Animal Welfare
Trevor Lawson, DVM, MSc, BSc (Agr)
Fundy Veterinarians Ltd., Murrah Siding, NS, Canada

INTRODUCTION
Private practice is full of challenges, perhaps none more than making meaningful change in areas of tremendous societal importance while growing your practice and improving the lives of animals in our care. I am keenly aware of the risk associated with upsetting a client, and the potential financial risk associated with this outcome. However, in the past several years there have been many examples where the veterinary profession has shown leadership in challenging areas, for example, cosmetic surgery and elective procedures in companion and food animals. Many of the changes we face in practice are directed by legislation and regulation and the NFACC codes of practice for livestock, some of which is new and some which is already in place. One of the pillars of our profession is self-regulation. Key to self-regulation is ensuring we meet our societal responsibilities, as these societal expectations change, or become increasingly clear, so must our view of what we do.

Animal welfare science dictates our professions direction in many cases and will be used to inform society, regulators, and clients. There are times when our personal beliefs will be challenged, but we have a responsibility to be aware and engage our clients to meet present day standards. One of the most significant challenges we face is recognizing opportunity when faced with change! Society expects our profession to be leaders in animal welfare; animal welfare in practice is not standing still and it is about looking forward and making continual improvements for animals.

THE DAIRY CODE
The NFACC Dairy Code of Practice was published in 2009. The code provides a wealth of science based practical guidance which is outcome driven to improve animal welfare in the dairy industry. All sectors of animal agriculture will continue to face greater public scrutiny, often driven by the retail market to satisfy a societal demand for humanely raised food. There are many challenges and opportunities for veterinarians and dairy producers in implementing the code when we accept our responsibility. In some instances legislation will also play a role in farm animal welfare and veterinarians should be equally aware of their legal responsibilities. Veterinarians and dairy farmers should be equal advocates for dairy cows, working to improve on-farm outcomes and enhance public trust in the veterinary profession and the Canadian dairy industry.

Since publication, the dairy code has received little practical use and attention on farm. This is disappointing given the fact that it is a thorough and well-written document that has considerable potential to improve dairy cattle welfare throughout the cycle of dairy production. There are several participants in the process who are responsible for implementing the code, and to date, all have failed to some degree to see it used in a meaningful way since its publication. The dairy industry has not yet appreciated the value and overarching purpose of the document at a farm level, veterinarians engaged at the farm level have not yet picked it up as a guiding document to any significant degree, and the national dairy industry has yet to implement a method of on-farm assessment. Many of the current animal welfare challenges faced by the Canadian dairy sector (lameness, mastitis, fitness for transport, euthanasia) could be addressed to significant degree if the code was examined closely and implemented at the farm level.

The purpose of this session is to provide one individual’s perspective in attempting to achieve some increase in NFACC Dairy Code of Practice awareness and recognition of on-farm welfare challenges.

ON-FARM, WELFARE ASSESSMENT OUTLINE
Designing a system for on-farm assessment use should be practical and easy to use. In order for the assessment to be meaningful, I have tried to use the model of S.M.A.R.T objectives in my approach (specific/measurable/attainable/realistic/timely).
I have made an attempt to develop a relatively straightforward approach for my own use to consistently evaluate areas of known risk (much like the Atlantic Johne’s Initiative herd assessment). The assessment should evaluate all the major areas on a dairy farm, including pre-weaned calves, heifers, bred/pregnant heifers, lactating cows, and dry cows and should consider a multitude of important factors including herd-health management planning, housing and environment, feeding, common husbandry practices, identification of sick and injured animals, culling and euthanasia decisions to name a few. Specific areas of measurement should include hygiene, hock scoring, lameness evaluation, body condition scoring, SCC, and morbidity and mortality in each group of livestock.

There are many valuable resources in the area of dairy cattle welfare; our challenge is to actively use them with our clients. Some examples are included in this introduction. I encourage you to examine them and perhaps find new ones which will work for you.

**APPENDIX 1 - COW HYGIENE**

Several options exist for evaluating cow hygiene. With this scoring system, Schreiner and Ruegg (2003) have demonstrated a significant association between poor udder hygiene and increasing, individual, cow linear score and intramammary infection with an environmental pathogen. Other scoring systems using leg and hind-end contamination may also be useful (Nigel Cook, University of Wisconsin).

Udder hygiene scoring chart available from UW Extension

The chart is available at: [http://milkquality.wisc.edu](http://milkquality.wisc.edu).
APPENDIX 2 - BODY CONDITION SCORING (OMAFRA)

Plot individual cows on the chart according to stage of lactation. The chart can be used to profile a herd at one point in time or to monitor changes over a lactation for an individual cow.
## APPENDIX 3 - HOCK SCORING

### QUICK-LOOK RESOURCES
49. [www.nationaldairyfarm.com](http://www.nationaldairyfarm.com) - National Dairy FARM Program.

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 - Emaciated</td>
<td>The individual vertebrae of the spine are prominent. The short ribs are sharp to the touch and give a shelf-like appearance to the loin. The hook and pin bones of the pelvis are well defined. The anal area of the cow is receded displaying a prominent vulva. Considered unfit to travel.</td>
</tr>
<tr>
<td>2 - Thin</td>
<td>The short ribs can be felt but are less outstanding. The hook and pin bones are still well-defined, though the area around the anus is less sunken and the vulva is prominent.</td>
</tr>
<tr>
<td>3 - Average</td>
<td>The short ribs are palpable with slight pressure. There is no shelf-like appearance to this area. The spine and hook and pin bones are all rounded and smoothed over. The anal area is filled out, and there is no evidence of fat deposits.</td>
</tr>
<tr>
<td>4 - Heavy</td>
<td>The short ribs are rounded over with no evidence of a shelf-like appearance and may only be felt with firm palpation. The ridge of the backbone is flattened over the loin and rump areas. The hook bones are smoothed over and the area around the pin bones shows some fat deposits.</td>
</tr>
<tr>
<td>5 - Fat</td>
<td>The bone structures of the spine, hook and pin bones, and short ribs are not discernible. There are fat deposits around the tailhead and over ribs. The thighs curve out and the brisket and flanks appear to be very full and heavy.</td>
</tr>
</tbody>
</table>
2014 CVMA ANNUAL CONVENTION PROCEEDINGS

Welcome to the 2014 CVMA Convention in St. John’s, Newfoundland. The following papers are compiled to accompany the presentations scheduled in the continuing education sessions at the convention. The proceedings are organized by day and by stream as follows:

FRIDAY, JULY 11, 2014

Companion Animal – Clinical Pathology
Companion Animal - Neurology
Companion Animal - Dermatology
Equine - Field Radiology for the Equine Practitioner
Equine - Field Surgery for the Equine Practitioner
Bovine - Dairy Calf Health
Bovine - The Transition Dairy Cow Shelter Medicine
Other - Shelter Medicine

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In collaboration with the Canadian Association of Animal Health Technologists and Technicians
Intervertebral Disc Disease (IVDD) or Why I Love Dachshunds: Part I
Romain Béraud, DV, DES, MSc, DACVS
Centre Vétérinaire Laval, Laval, QC, Canada

Intervertebral disc disease (IVDD) is a frequent disorder in dogs. The related neurological signs usually lead to a high level of stress from the owner… and also from the veterinarian! Dogs can be presented with a wide variety of signs. Therefore, after having performed a full physical exam, along with a cranial nerves exam (in order to exclude an intracranial disease), following the same routine for your neurological exam is the best way to easily localize the lesion. The goal is to get to our differential and then confirm the IVDD in order to provide the owner with precise information regarding the best treatment and prognosis.

An easy diagnostic approach can be narrowed down to four questions:

**Is There a Neurologic Problem?**
That can sound like a stupid question, but some dogs with an orthopedic problem (bilateral patellar luxation, cranial cruciate disease, hip osteoarthritis, etc.) may look like they are paretic/paralyzed…and vice versa: dogs with a lameness may have a neurological problem.

Evaluating proprioception is an easy way to confirm or not spinal cord involvement.

**How Severe Is It?**
When a herniated disc is compressing the spinal cord, major functions will be lost in a predictable way, based on the location and the size of the corresponding fibers in the white matter (from first to last to be lost):

- **Proprioception** (ability to recognize the location of its body in space): Large superficial myelinated fibers. Proprioception deficits can be evaluated by analyzing the animal’s gait (ataxia) and different tests (positioning reactions [knuckling], hopping, wheel barrowing, hemiwalking, placing reactions…).
- **Motor function** (ability to voluntarily move its limbs with appropriate strength): Smaller and deeper myelinated fibers. Motor function deficits will be demonstrated by paresis (present but weak voluntary movements) or paralysis/paraplegia (completely absent movements).
- **Pain sensation** (ability to ‘centrally’ feel pain when a noxious stimulus is applied): Small and deep nonmyelinated fibers. Pain sensation is tested by pinching the dog’s toes; the expected reaction should be ‘central’ (in opposition to just pulling the leg, which is the withdrawal reflex): barking, trying to bite, moving away from you, tachycardia, tachypnea, myosis, etc.
- **Grade of severity**: You should then be able to grade the severity of the lesion. A standardized grading system is interesting when different persons may be involved in the followup of the dog: everyone is on the same page and can therefore accurately follow the dog’s progression. Different systems exist; the one I use is:
  - Grade 1 - Pain (no neurological deficits)
  - Grade 2 - Ataxia or ambulatory paresis
  - Grade 3 - Nonambulatory paresis
  - Grade 4 - Paralysis with pain perception
  - Grade 5 - Paralysis without pain perception

**Where Is It Located?**
- Spinal cord is divided in 4 functional segments: C1–C5, C6–T2, T3–L3, L4–S3. Remember that spinal cord segments are different from vertebral segments, due to the presence of 8 cervical segments vs. 7 cervical vertebrae and to the retraction of the caudal spinal cord in the vertebral canal during embryogenesis!
Localizing the problem to one of these segments is done by testing reflexes on the legs. Is the leg normal? Or does it show signs of an upper motor neuron (UMN) or lower motor neuron (LMN) lesion?

- **LMN lesion**: Decreased to absent reflexes, decreased muscle tone, neurogenic atrophy (early, severe)
- **UMN lesion**: Normal to increased reflexes, normal to increased muscle tone, disuse atrophy (late, mild)

**Reflexes tested (see table below)**

Other localizing tools exist: posture, Horner’s syndrome, panniculus reflex, spinal palpation, etc.

<table>
<thead>
<tr>
<th>Reflexes</th>
<th>Implied nerve(s)</th>
<th>Spinal segment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triceps</td>
<td>Radial</td>
<td>C7–C8–T1</td>
</tr>
<tr>
<td>Extensor carpi radialis</td>
<td>Radial</td>
<td>C7–C8–T1</td>
</tr>
<tr>
<td>Biceps</td>
<td>Musculocutaneous</td>
<td>C7–C8</td>
</tr>
<tr>
<td>Flexor/withdrawal Thoracic limb</td>
<td>Median</td>
<td>C6–C7–C8–T1–T2</td>
</tr>
<tr>
<td>Patellar</td>
<td>Femoral</td>
<td>L4–L5–L6</td>
</tr>
<tr>
<td>Cranial tibial</td>
<td>Fibular/peroneal</td>
<td>L6–L7</td>
</tr>
<tr>
<td>Flexor/withdrawal Pelvic limb</td>
<td>Sciatic</td>
<td>L6–L7–S1</td>
</tr>
<tr>
<td>Perineal</td>
<td>Pudendal</td>
<td>S1–S2–S3</td>
</tr>
</tbody>
</table>

**Clinical signs - Summary**

**C1–C5**
- Neck pain, low head carriage, decreased neck motion
- Ataxia (4 legs) to tetraparesis
- Forelimbs: UMN lesion (normal to increased reflexes and muscle tone)
- Hindlimbs: UMN lesion (normal to increased reflexes and muscle tone)

**C6–T2 (cervical intumescence)**
- Neck pain, low head carriage
- Ataxia (4 legs) to tetraparesis
- Forelimbs: LMN lesion (decreased/absent reflexes and muscle tone: flaccidity ± neurogenic atrophy)
- Hindlimbs: UMN lesion (normal to increased reflexes and muscle tone)
  ± Horner’s syndrome, nerve root signature

**T3–L3**
<table>
<thead>
<tr>
<th>Back pain, hunched back</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hindlimbs ataxia, paresis or paralysis</td>
</tr>
<tr>
<td>Forelimbs: Normal (normal reflexes and tone)</td>
</tr>
<tr>
<td>Hindlimbs: UMN lesion (normal to increased reflexes and muscle tone)</td>
</tr>
<tr>
<td>± UMN bladder (large, spastic, overinflated!)</td>
</tr>
<tr>
<td>± Panniculus reflex abnormality</td>
</tr>
</tbody>
</table>

**L4–S3 (lumbar intumescence)**

<table>
<thead>
<tr>
<th>Low back pain, hunched back</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hindlimbs ataxia, paresis or paralysis</td>
</tr>
<tr>
<td>Forelimbs: Normal (normal reflexes and tone)</td>
</tr>
<tr>
<td>Hindlimbs: LMN lesion (decreased/absent reflexes and muscle tone: flaccidity ± neurogenic atrophy)</td>
</tr>
<tr>
<td>± LMN bladder (flaccid, incontinence)</td>
</tr>
</tbody>
</table>

**What is it due to?**

- The dog’s history (breed, age, clinical onset, etc.) and your neurological exam should help you narrow down your differential to a few diseases.
- **Cerebrospinal fluid tap and analysis** can be performed if you are unsure. Remember that myelogram is absolutely contraindicated in case of meningomyelitis!
- **Diagnostic imaging** is, however, necessary at that point to confirm an IVDD:
  - Plain radiographs and myelogram. Radiographs can show narrowing of the intervertebral space, calcified disc(s), ‘clouding’/distortion of a foramen, spondylosis, etc., but they are just hints! You cannot diagnose an IVDD based only on radiographs. A myelogram is needed to confirm and show the side and location of the extradural compression of the spinal cord as demonstrated by the deviation, thinning or loss of the contrast columns.
  - CT scan ± myelogram and, most interesting, the MRI when available.

You now have your diagnosis: it is an IVDD! You will then have to face a few questions from the owners!
‘Doctor, why did my dog get a herniated disc?’

Quick anatomy review: There is an intervertebral disc between each pair of vertebrae except C1–C2. The disc is formed by an outer annulus fibrosus (mainly composed of fibrocytes and type I collagen) and an inner nucleus pulposus (composed of 80–88% water, proteoglycans and type II collagen). Its functions are to allow motion and absorb/transmit load.

When the nucleus pulposus degenerates, IVDD occurs:

- **Hansen I IVDD**: The nucleus pulposus undergoes chondroid degeneration with progressive loss of water and mucopolysaccharides and calcification. This happens in chondrodystrophic dogs (Dachshunds, Poodles, Shih Tzu, Lhasa Apso, Cocker Spaniels, Beagles, etc.) starting early in their life (around six months of age). Breed and family predisposition have been shown. This therefore affects young to middle-aged dogs, and the signs are typically acute to peracute, as there is a sudden extrusion of the nucleus. Injury to the spinal cord is primarily due to the concussion as well as the compression from the herniated disc, and secondarily due to the biochemical cascade following cellular death (release of superoxide radicals, etc.). Most type I IVDD occur in the cranial cervical segment (C2–C3–C4) and thoracolumbar junction (T12–T13–L1).

- **Hansen II IVDD**: The nucleus pulposus undergoes fibrinoid degeneration with progressive loss of water and collagen deposition, along with partial/complete rupture of the annulus fibrosus. This happens in older dogs and reflects a standard ‘aging’ process of the disc that can happen in any non-chondrodystrophic dog (German Shepherds, Rottweilers, Labradors, etc.). Onset of signs is typically chronic with a progressive protrusion of the disc. Injury to the spinal cord is mainly due to the compression from the herniated disc, which chronically leads to spinal cord atrophy and gliosis.

- **Hansen III IVDD** (aka traumatic disc herniation, high-velocity low-volume IVDD, non-compressive IVDD): It occurs in healthy dogs consecutively to a strenuous exercise or trauma. This can happen in any breed and at any age. A rent in the annulus acutely forms, and the nucleus pulposus ‘explodes,’ shooting very small fragments of disc on the spinal cord. Most of the damage is due to the concussion, as there usually is no compressive component. Signs are typically nonprogressive after 24 hours.

<table>
<thead>
<tr>
<th>Breed</th>
<th>Type I IVDD</th>
<th>Type II IVDD</th>
<th>Type III IVDD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Young-middle</td>
<td>Older</td>
<td>Any</td>
</tr>
<tr>
<td>Onset</td>
<td>Acute</td>
<td>Chronic</td>
<td>Peracute</td>
</tr>
<tr>
<td>Disc degeneration</td>
<td>Chondroid</td>
<td>Fibroid</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>Disc extrusion</td>
<td>Disc protrusion</td>
<td>‘Explosion’</td>
</tr>
<tr>
<td>Type of injury</td>
<td>Concussion &amp; compression</td>
<td>Compression</td>
<td>Concussion</td>
</tr>
</tbody>
</table>

‘What is the best treatment ... and what are the chances of my dog recovering?’

Medical or surgical management: that is the question! Recommendations vary between different neurologists/surgeons, but most agree on:

- **Type I IVDD**: Medical management can be tried for single/occasional episodes of mild to moderate pain (grade I), but surgery is recommended as soon as there are neurological signs!
  Prognosis/success rate is:
  - With surgery: Grade 1 to 4: more than 90% success; Grade 5: 50-60% during the first 24 h and then quickly drops
With medical management: ~ 50% success, ~ 30% recurrence and ~ 20% failure

- Type II IVDD: Surgery is recommended; however, as the spinal cord compression is chronic, deficits may be more or less reversible...but surgery will at least prevent further worsening! Therefore, a precise prognosis is hard to give with surgery. Medical management will at best slow the progression...but for how long?
- Type III IVDD: As there is no spinal cord compression, surgery has no benefit effects. Medical management is all we can do! Recovery is expected in two-thirds of cases.

Medical management involves: strict rest for 1–2 months, physical therapy and analgesia (NSAIDs or steroids, muscle relaxants, opioids, etc.).

Surgical management involves a ventral corpectomy for cervical IVDD and hemilaminectomy or mini-hemilaminectomy/pediculectomy for thoracolumbar type I IVDD. Fenestration at the site of the herniation and possibly at other sites is recommended. Postoperative care involves good nursing care (bedding, fluids, etc.), analgesia, bladder management if needed, and physical therapy. Physical therapy is of uppermost importance in nonambulatory dogs, but even ambulatory ones will benefit from it. Bladder management is critical if bladder function has been lost. Manual expression or catheterization (intermittent or indwelling) may be indicated.

Medications can also be used to ease urination:

<table>
<thead>
<tr>
<th>Medication</th>
<th>Action/Target</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenoxybenzamine</td>
<td>Alpha-antagonist (smooth muscle)</td>
<td>0.25–0.5 mg/kg q12h</td>
</tr>
<tr>
<td>Prazosin</td>
<td>Alpha-antagonist (smooth muscle)</td>
<td>1 mg/15 kg q12–24h</td>
</tr>
<tr>
<td>Diazepam</td>
<td>Relaxant (skeletal muscle)</td>
<td>2–10 mg/dog q8h</td>
</tr>
<tr>
<td>Bethanechol</td>
<td>Cholinergic</td>
<td>5–25 mg/dog q8h</td>
</tr>
</tbody>
</table>

'When should we proceed? Is it an emergency?'

An IVDD is a surgical emergency when deep sensation has been lost, as illustrated by the success rate for grade 1–4 vs. grade 5. Main prognostic indicators for recovery are presence of pain sensation and rate of onset of clinical signs. Prognostic indicators for time to recovery are more numerous: pre- and postoperative voluntary motor function, time from onset of nonambulation to surgery, duration of clinical signs, etc.

'Is there something I can do to prevent recurrences?'

This is a legitimate question, as occurrence of an IVDD at another site has been reported in 2.6 to 26.5% of cases operated! Dachshunds have been shown to have a higher risk of recurrence than all other breeds. Calcified discs are known to have 1.4 more chances to herniate than a non-calcified one. Although many studies have focused on ways to screen for at-risk patients, very few can be done for prevention. Use of a harness instead of a collar, regain/keep an adequate body condition score, try to limit high-impact activities ... these may help, but nothing will truly prevent recurrence. Surgically speaking, however, disc fenestration has been shown to prevent occurrence of disc disease, so it is recommended.
What’s New in Lumbosacral Disease?
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Lumbosacral disease (LSD), also known as cauda equina syndrome or degenerative lumbosacral stenosis, is the most common abnormality of the canine lumbosacral junction, resulting in a wide variety of clinical signs.

ETIOPATHOGENESIS
Cauda equina syndrome is a collective term used to describe the clinical signs resulting from a variety of compressive, inflammatory, malformative or vascular diseases affecting the L7–S1 space and corresponding nerve roots of the cauda equina (not the spinal cord!). A number of abnormalities may combine to cause compression of the cauda equina: disc protrusion (Hansen type II IVDD), soft-tissue proliferation/hypertrophy (joint capsules, ligamentum flavum, dorsal longitudinal ligament), subluxation/misalignment of L7–S1, epidural fibrosis, articular facets osteophytosis, congenital osseous stenosis of the vertebral canal, synovial cysts and sacral osteochondrosis. Given that motion in the normal lumbar spine is greatest at the lumbosacral space, it is postulated that instability of this region plays a role in the pathogenesis of numerous degenerative processes that may occur in the LSD. These degenerative conditions may perpetuate the progression of the disease by further altering the biomechanics of the vertebral column in affected dogs. However, due to the complexity of the lumbosacral junction, the true etiology of the LSD is still poorly understood.

PRESENTATION
Young adult, male, large-breed dogs (especially German Shepherds) are predisposed to the development of LSD, although dogs of any age or breed may be affected. Lumbosacral disease has also been described in cats. The use of dogs in activities that involve heavy work or training (police or military dogs) is a known risk factor.

CLINICAL SIGNS
Clinical signs of LSD are heterogeneous and can be static or intermittent and vague or nonspecific. Lower lumbar or pelvic limb pain is a common historical complaint. Manifestations of pain or paresthesia range from crouched pelvic limb posture, pelvic limb lameness, and pelvic limb nerve root signature to self-mutilation of the limbs, genitals or tail. Working dogs may be reluctant to jump, climb or work, and signs may be exacerbated by activity. Pain can be elicited during the examination by various manipulations. Pelvic limb neurologic dysfunction can vary from mild proprioceptive deficits observed without an associated gait disturbance to monoparesis/plegia or paraparesis/plegia. The specific nerves that form the cauda equina are the L7, S1, S2, S3 and caudal nerves 1–5, each of which may be affected in animals with LSD. The sciatic, cranial tibial, gastrocnemius, anal and flexor withdrawal reflexes may be normal, depressed, or absent. Neurogenic muscle atrophy may be present. The tail may be carried abnormally as the result of pain, hypotonia or paralysis. Occasionally, urinary and fecal incontinence may be the predominant or only clinical sign.

DIAGNOSIS
Given some of the pitfalls associated with the diagnostic examinations commonly used in the management of LSD, a thorough history and performance of complete physical, orthopedic, and neurologic examinations are crucial for the comprehensive interpretation of any abnormalities identified by ancillary diagnostic investigations:

- **Radiographs:** Survey radiographs may demonstrate L7–S1 spondylosis deformans or malformation, narrowing of the disc space and endplate sclerosis; however, these signs can be common in normal geriatric dogs and are not in themselves indicative of LSD. Conversely, dogs with clinically significant LSD may have normal radiographs. Discography/epidurography can be quite sensitive for the detection of lumbosacral lesions, but require some experience.
Electrophysiologic studies: Motor and sensory nerve conduction studies may be normal or abnormal depending on the severity of the disease.

CT scan: Common CT abnormalities include loss of normal epidural fat, abnormal soft-tissue density within the intervertebral foramen or vertebral canal, bulging of the L7–S1 disc, displacement of the thecal sac, and subluxation and osteophytosis. CT examinations performed in flexion and extension can also increase the sensitivity of detection of LSD.

MRI: The soft-tissue resolution provided by MRI being superior to that of CT, MRI is a tool of choice for evaluation of LSD. Abnormalities observed are displacement or loss of epidural fat, disc protrusion, soft-tissue foraminal stenosis, ligamentous hypertrophy, and soft-tissue proliferation of the joint. Flexion and extension MRI can also be performed.

TREATMENT
‘The current best evidence for clinical decisionmaking for dogs with LSD consisted of reports of lower evidentiary value and the clinical experiences of the supervising clinician.’

Conservative Management
Medical management, consisting of strict cage rest and analgesia (NSAIDs or steroids, tramadol, gabapentin, etc.) is often recommended for dogs experiencing their first episode of lumbosacral pain, or for nonworking dogs whose signs are limited to episodic lumbosacral pain. Approximately 50% of dogs managed this way can be expected to have a good outcome; however, recurrences are common upon resumption of normal activity. The authors of one study reported that 79% of 38 dogs with disc-related LSD treated with epidural administration of 1 mg/kg methylprednisolone acetate demonstrated clinical improvement with this therapy. Treatment consisted of three injections given approximately 2 and 6 weeks following the first injection and appears like an excellent medical option.

Surgical Management
Most neurologists and surgeons agree that surgical management is indicated for LSD when there is moderate to severe pain and when dogs display neurologic deficits. However, great controversy is ongoing about the best surgical management of LSD in dogs. The most common approach is a dorsal L7–S1 laminectomy followed by a discectomy. Distraction and fusion of L7–S1 may also be recommended; many techniques have been described. If foraminal stenosis is evident on diagnostic imaging, foraminotomy can be performed (either endoscopically assisted through a mini-laminectomy or a transiliac lateral approach).

OUTCOME/PROGNOSIS
Several studies have evaluated the long-term outcomes of dogs following a dorsal laminectomy and discectomy, but unfortunately no studies have compared distraction/fusion to dorsal decompression. In a large retrospective study involving 131 dogs with a mean followup time of 26 months, 79% of dogs returned to normal function and 93% showed clinical improvement. The greatest percentage of dogs that improved after surgery included dogs that had mild clinical signs (mild pain, mild locomotive disturbances, no neurologic deficits), whereas only 50% of dogs that had the most severe clinical signs returned to normal function. However, recurrences of clinical signs occurred in 18% of the dogs between 6 and 36 months. In another study, the presence of urinary or fecal incontinence and the duration of urinary incontinence were associated with a poor prognosis.

Several reasons can be hypothesized for the high rates of recurrence: new bone formation or scar tissue formation over the laminectomy site, continued mobility/instability.
**Canine Wobbler Syndrome**  
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Hôpital Vétérinaire Rive-Sud, Brossard, QC, Canada

Cervical spondylomyelopathy (CSM) is a relatively common condition in large-breed dogs. However, its etiology is not completely understood. Like the majority of congenital joint diseases, a combination of factors seems to be involved, including nutritional, genetic and congenital implications. Some authors suspect a possible relationship with the type of conformation of the skeleton and the development of clinical signs. For example, bigger, taller patients with more rapid growth might present with clinical signs earlier in their life and be more severely affected. Although this may be observed clinically, it has never been proven.

We do know that the changes identified are secondary to both static and dynamic insults to the spinal cord and its components. The fact that some of these animals have evidence of static changes through advanced imaging but remain asymptomatic supports the idea that there is a dynamic component to the disease. These static changes include degeneration of intervertebral discs with or without a certain degree of protrusion, osteoarthritis of cervical vertebral articular facets, the presence of synovial cysts and so on. However, the most important component in determining the development of clinical signs appears to be the degree of the vertebral canal stenosis. In fact, diameter of the spinal canal is smaller in symptomatic patients. This stenosis leads to degeneration of the spinal cord, but it is also responsible for the development of new sources of insults and compression.

**Two Types, Two Syndromes**

Two specific types of CSM are recognized. Both syndromes affect the spinal cord and related neurological structures; however, their pathophysiology differs. The first type is disc-associated, and the second type is mostly associated with joint (bone and soft tissue) changes.

Historically, the first type was mostly studied in the Doberman pinscher breed but is now recognized in most medium- and large-breed dogs. Clinical signs typically develop in middle-age adults and are particularly linked to changes to the caudal cervical intervertebral discs (C4–C7). Affected patients demonstrate primary stenosis of the vertebral canal secondary to a conformational defect, which is aggravated by a component of type II herniated disc. The spinal cord compression is mostly ventral, and sometimes lateralized, depending on the location of the disc protrusion. The nerve roots are often involved, leading to the development of cervical pain. While stenosis is also observed in asymptomatic patients, it is suspected that a component of dynamic compression, exacerbated by certain movements or neck positions, is involved. However, as mentioned earlier, the dynamic component remains very difficult to prove.

The second type is encountered mainly in large- and giant-breed dogs. Patients are presented young, often during adolescence. This CSM is associated with bony changes such as proliferation of the dorsal vertebral arch, of the vertebral articular facets dorsolaterally and sometimes of the pedicles laterally, as well as vertebral deformities leading to the development of osteoarthritis. The dynamic component is therefore very important in this second type of CSM. In other words, the degree of compression on neurological structures varies significantly depending on the position of the joint. Although this type is suspected to be congenital in nature, to date there is no genetic link recognized between affected patients.

**Clinical Presentation**

While there are 2 different syndromes, the typical presentation of each is different with some similarities. Both patients generally have a slowly progressive history, but can also (and frequently) be presented following a sudden deterioration in their condition while the owners had previously not noted anything abnormal. Neck pain, although revealed in the majority of cases during the physical and neurological examinations, is rarely the primary reason for presentation.

Predisposed breeds include the Great Dane, Basset Hound, Doberman Pinscher, Mastiff, Rottweiler, Bernese Mountain Dog, German Shepherd Dog and Borzoi. Mixed breeds as well as most large breeds
can also be affected. Disc-associated cases are mostly presented over 3 years of age, whereas giant breeds with joint-associated syndrome are typically presented around the age of 12 to 18 months.

Patients often present with muscular atrophy of the shoulder muscles (innervated by the brachial plexus nerve roots), cervical rigidity, 4-limb proprioceptive ataxia that can be difficult to appreciate on the thoracic limbs. The latter frequently are spastic and show short amplitude of movement. They are also often thrown forward which gives an impression of floating motion. Occasionally, a lameness of a thoracic limb may be observed secondary to the compression of a nerve root composing the brachial plexus. This is known as a root-signature. Pelvic limbs are typically more severely affected and exhibit more convincing proprioceptive ataxia, paresis and, in more chronic cases, slow-motion long (dragging) strides (“cross-country skiing motion”).

The gait of affected dogs is described as a “2-motor gait”: long, slow strides on pelvic limbs and short, rapid, stiff strides on thoracic limbs. Visualisation of gait anomalies is easier when the patient walks slowly. Additionally, elevating the head during the gait can exacerbate deficits and allow better evaluation. The examination of the gait should be systematic and done very carefully in order to detect every anomaly.

Proprioceptive positioning (knuckling) and hopping responses vary depending on the severity of the condition. Deficits are generally more severe on pelvic limbs and may be asymmetric. Spinal reflexes may be normal to increased on pelvic limbs (UMN involvement) and normal to decreased on thoracic limbs (signs LMN involvement if the brachial plexus is involved). Even though the patient may not clearly demonstrate neck pain in its day-to-day activities, some degree of reduced cervical range of motion is usually noted upon neurological examination.

Once the evaluation is complete with a detailed history, physical and neurological exam, and lesion localisation has been made, differential diagnosis should include: CSM, IVDD, meningomyelitis, discospondylitis or chronic osteomyelitis (causing spinal cord compression), vertebral fracture (if presentation is acute and very painful), fibrocartilaginous embolism myelopathy (FCEM) (if presentation is acute and nonpainful) and local neoplasm.

**Diagnostic Procedures**

According to the established differential diagnosis, investigations should include: complete blood analyses including thyroid function in older patients, cervical radiographs (may require sedation or even anesthesia, especially in very large patients to ensure a perfect positioning) and, ideally, advanced imaging (computed axial tomography or magnetic resonance imaging).

Cervical radiographs are very difficult to perform, and optimal positioning is necessary for the evaluation of all structures. In larger patients, the use of sedation or, in some cases, general anesthesia is often required. Staff should be well trained, and images should be repeated if their quality or the patient’s positioning does not allow evaluation of all intervertebral spaces, vertebral articular facets and vertebral alignment.

While most patients with CSM have soft-tissue involvement as well as vertebral canal stenosis, assessment of neurological structures can only be achieved by advanced imaging techniques such as computed axial tomodensitometry/graphy (CT scan) and, ideally, magnetic resonance imaging (MRI) of the cervical vertebral column. Axial tomodensitometry is more accurate than conventional radiographs as well as faster and less expensive than MRI. It is more easily accessible, presents good resolution and allows transverse imaging of the vertebral column. This leads to good assessment of the diameter of the vertebral canal as well as the degree of stenosis. It may even be useful to evaluate dynamic components of the condition as images can be obtained after varying the patient’s positioning on the exam table.

MRI, on the other hand, provides a much better assessment of the soft tissues compared with axial tomodensitometry and is, therefore, the only modality allowing the evaluation of the spinal cord itself. While some patients show irreversible changes to the spinal cord, this information may be paramount when the time comes to determine the treatment recommendations. The dynamic component of the condition is, however, difficult to assess by MRI, as changing the position of the patient within the machine is very challenging.
**Medical vs. Surgical Management**

Medical management is recommended if severe, irreversible changes to the spinal cord are identified on the MRI, if multiple intervertebral spaces are affected, if the patient is not a good surgical candidate (other concomitant diseases) or if the owner has financial constraints. A combination of analgesics (gabapentin, tramadol) and anti-inflammatory medications (cortisone or NSAIDs) will be prescribed as well as changes to the environment to reduce impacts on the vertebral column leading to injury of the spinal cord and other neurological structures. Ideally, a physical therapy plan will be implemented in order to maintain muscle mass as much as possible.

Surgical intervention is recommended if spinal cord compression is secondary to intervertebral disc protrusion. On the other hand, while the ventral compression can be minimized surgically, the dynamic component and compression of the spinal cord by the other components of the condition remain, which can still cause irreversible changes to the spinal cord.

New surgical techniques are being developed and focus is being placed on the dynamic effects of the condition. Therefore, efforts are being made to immobilise affected intervertebral junctions while enlarging intervertebral space. These new techniques are promising, but further studies are needed not only to assess short-term clinical improvement of affected patients, but mostly long-term quality of life. Indeed, based on the available studies, the medium- to long-term prognosis does not differ whether the therapeutic approach is surgical or medical.

At this time, prognosis of this condition remains guarded secondarily to the irreversible damages to the spinal cord. The size and weight of affected patients often complicate the care. On the other hand, as awareness of the condition increases, more interest is now being placed on research to find better treatment options.
Canine Epilepsy 101
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Canine epilepsy is a complicated condition and its understanding remains, unfortunately, limited. However, as we continue to make an effort to better define and classify seizures, the condition may become clearer and easier to diagnose, work up and treat.

The International League Against Epilepsy (ILAE) was created in 1909. It is a multinational organization aiming at better understanding epilepsy. Subcommittees and task forces have been nominated to outline terminology and classification themes for human patients. There is no equivalent association in veterinary medicine, but numerous authors have attempted to use the ILAE classification and adapt it to our small animal patients.

**Definitions and Classifications in Veterinary Medicine**

Seizures are episodes thought to be secondary to excessive neuronal activity in the brain (thalamocortex). They can originate from the cortex itself (epileptic seizure) or be secondary to a transient metabolic/toxic insult to the neurons (reactive seizure). The latter is not considered epilepsy and will not lead to recurrent seizures as long as the cause is removed and there is no permanent damage to the neurons.

Seizures can be focal or generalized. Focal seizures result in activation of only a localized area of one cerebral hemisphere without implication of the entire cortex. This type of seizure often indicates a specific cerebral lesion and generally does not lead to loss of consciousness. Generalized seizures, on the other hand, involve both cerebral hemispheres and lead to loss of consciousness.

Epileptic seizures can present as isolated, in clusters or as status epilepticus. The latter is defined as more than 30 minutes of seizure activity or incomplete recovery of consciousness between clustered seizures. A status can be generalized or focal, and both forms require emergency intervention from the veterinarian.

Epilepsy is a chronic, neurological condition characterized by recurrent seizures originating within the brain. Therefore, an epileptic patient is simply one that has had more than 1 epileptic seizure in its lifetime.

There are 3 large subcategories of epilepsy in small animal veterinary medicine:
1. Genetic epilepsy (previously known as “idiopathic epilepsy”)
2. Structural epilepsy (previously known as “symptomatic epilepsy”)
3. Epilepsy of unknown cause (previously known as “probably symptomatic epilepsy”)

**1. Genetic Epilepsy**

Genetic epilepsy suggests an inherited mode of transmission, and its onset is age-dependant. Seizures are therefore secondary to a known or strongly suspected genetic defect. There is no identifiable structural lesion within the brain parenchyma, and the condition is recognized in specific breeds. Previously, it was believed that seizures should always be generalized with genetic epilepsy. However, studies have now shown that genetic epilepsy can lead to both generalized and focal seizures in different breeds. As the mode of inheritance varies from one breed to another, mixed-breed patients might also be affected (autosomal recessive).

As a guide for the veterinarian, a patient with genetic epilepsy should fill these 5 conditions (with the exception of some rare cases):
1. Be a breed in which genetic epilepsy is proven or strongly suspected;
2. Have the first seizure between the age of 6 months and 5 years (often closer to 10 months to 3 years of age);
3. Present with a specific seizure pattern (most often generalized except for specific breeds: Vizsla, English Springer Spaniel, Standard Poodle);
4. Have a progressively increasing frequency of seizures;
5. Lack any detectable anomaly on the neurological examination, including normal mental status and behaviour, between seizures.

The term “genetic” is now preferred to “idiopathic,” as the latter did not seem to have a similar meaning between neurologists. In fact, in some reports, authors would classify the epilepsy as being idiopathic if no structural lesion could be identified on advanced imaging, regardless of the recognition of an inherited trait in the breed. As a result, there was confusion as to what was the actual cause of the epilepsy. Therefore, the term “genetic” appears more appropriate and more accurate to describe epilepsy secondary to an inherited trait. To date, this condition has never been recognized in cats.

2. Structural Epilepsy
Animals diagnosed with structural epilepsy have a specific lesion identified within the thalamo-cortex that is causing recurrent epileptic seizures. The term “structural epilepsy” is now preferred to “symptomatic epilepsy,” as animals are considered to have clinical signs and not symptoms - a term reserved for human patients.

Seizures in this group can be focal or generalized and often do not follow a specific pattern. The neurological examination will often reveal anomalies, and owners will occasionally report, although sometimes very subtle, changes in behaviour at home. Structural lesions can be secondary to brain tumours, cerebrovascular accidents, congenital malformations (hydrocephalous and other), degenerative disorders such as storage diseases, traumatic injuries, etc.

3. Epilepsy of Unknown Cause
To classify a patient within this category, an underlying cause must be suspected without detectable lesion within the thalamo-cortex. This could be because the lesion is too subtle to be detected by current available diagnostic technologies or because a genetic defect is not yet recognized.

DIAGNOSTIC PROCEDURES
Once seizures are classified as epileptic, further investigations should be pursued to better classify the epilepsy. This classification will dictate not only the specific treatment to initiate, but also the prognosis of the condition. As epilepsy is a chronic disease involving the brain, pet owners are often very uncomfortable regarding the topic. The more the owner understands the condition and knows about prognosis, the better the compliance will be in following the treatment plan.

A detailed patient history is mandatory with any seizing patient. Once the history is obtained, complete physical and neurological examinations are performed. Close attention should be given to the specific tests evaluating the thalamo-cortex. These include: evaluation of mental status, menace and nasal septum stimulation responses, as well as proprioception. All patients should have complete bloodwork and urinalysis performed to rule out metabolic anomalies and serve as a baseline once antiepileptic treatment is initiated. More specific analyses should be performed if a specific disease is suspected.

If structural epilepsy is suspected, thoracic radiographs and abdominal ultrasound should be performed to eliminate possible concomitant diseases. If possible, all epileptic patients should undergo advanced imaging, ideally MRI, of the brain. However, the cost of these procedures often limits their use.

TREATMENT
Treatment efficacy may vary for a number of reasons. The “3 Ds rule” can be followed: Diagnosis (adequate recognition and classification), Drug (selected medication based on type of seizures and specific patient), and Dose.

Proactive intervention is recommended to prevent development of kindling effect (increased frequency of seizures secondary to uncontrolled epilepsy) and to prevent further damage to neurons. The goals of treatment are: decrease the frequency of seizures, decrease risks of cluster or status, decrease postictal effects and improve the patient/owner’s quality of life.
Treatment options have unfortunately not changed much over the past few years. Phenobarbital remains by most as the first-line choice, followed by potassium bromide, levetiracetam, zonisamide (not available in Canada) and gabapentin.
Neurology and Diagnostic Imaging: What Modality Should I Choose?
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Imaging the central nervous system is challenging. Newer modalities are now more easily available, but their strength and limits are not always well understood. First and foremost, the imaging exam should complete and confirm the neurological exam. Imaging should therefore support the findings and not be used as a screening test. For this reason, a very close relationship between neurologists and radiologists is often the key.

**Imaging the CNS: The Challenges**

From the neurologist point of view, the challenges are choosing the appropriate modality based on the localisation of the lesion(s) and differential diagnosis; the patient having to undergo general anesthesia or deep sedation for most modalities; considering financial dispositions or constraints of the owners; and the accessibility of the different modalities.

Imaging the entire patient is, for most modalities, not a valid option considering the time needed to obtain images, the need for general anesthesia (or deep sedation), not to mention the extent of the financial aspect for the owner. Therefore, the neurological and physical examination must be performed with great care to determine the exact lesion localisation and differential diagnosis. In fact, the established differential diagnosis should dictate which modality to use and must, therefore, be as precise as possible. On the other hand, lesion localisation will determine which anomaly visualized on the images obtained is significant and explains the clinical presentation. This is usually not a problem in a younger and healthy patient where only 1 lesion will be seen. Yet, in some cases, anomalies may be present on the images obtained that may represent incidental findings. It becomes the neurologist task to determine what is clinically significant and what is not.

From the radiologist point of view, the central nervous system is challenging as it is well protected by a bone barrier that limits the use of certain modalities such as ultrasound and radiology for most CNS structures. There are also different types of soft tissues in a very limited area such as, for example: fat, cerebrospinal fluid, grey matter, white matter, nerve roots, and blood vessels within the very small vertebral canal. With some modalities, very little difference exists between different pathologies when it comes to the images they provide. The use of contrast agents is, most of the time, required which may lead to increase in time, cost, and risks for the patient and the owner. Again, a very close relationship between the neurologist and the radiologist is often the key to a successful imaging.

**Radiography: The Bone Modality**

Radiographs are mostly used, in veterinary neurology, to image the vertebral column. Its use is excellent to screen for mineralized intervertebral disks, osteoarthritis, vertebral malformation, fracture or luxation, discospondylitis, osteomyelitis, and bone tumour. It can also be used to assess the bone component of the middle ear, tympanic bullae.

It is extremely important, however, to have excellent radiographs to ensure adequate visualisation of all structures. The patient must be perfectly positioned and contrast should be optimal. Even a very mild rotation may change the perception of the reader. Every structure should be carefully evaluated. Again, adequate clinical localisation of the lesion is important, as incidental findings are possible.

**Axial Tomodensitometry: The “Super” Bone Modality**

The axial tomodensitometry or CT scan uses X-rays to obtain images of sections of the body. A circular tube emitting X-rays and a ring of aligned detectors circle around the patient simultaneously, but in opposite direction. X-rays are released as the patient, lying on a table, moves forward through the emitter-detector tube. This allows 3D imaging of the patient that may then be viewed as “slices.” The entire process is very fast and images may be obtained in less than 2 minutes depending on the area imaged and the strength of the machine used.
This modality is used mostly for the vertebral column and skull (including tympanic bullae) as bone definition is excellent. It is, therefore, sensitive and excellent to detect IVDD in chondrodystrophic dogs, discospondylitis/osteomyelitis, bone tumour, fracture/luxation, vertebral malformation, middle ear diseases, and may help detecting anomalies of the pituitary gland as well as most cases of meningioma. Its limits are that the patient must be anesthetised, or at least deeply sedated, to undergo the exam; it administers higher doses of radiation to the patient compared with radiographs; it gives much less soft-tissue details and definition when compared with magnetic resonance imaging (MRI) and gives high-resolution images on transverse planes only.

Another major limit to axial tomodensitometry is that numerous artefacts are known to occur in specific areas of the body (caudal fossa for example) making its use in these areas very difficult. Artefacts secondary to the presence of metallic objects within the body are also possible, not to mention artefacts related to the technique itself. Because of the existence of these artefacts, a certain level of experience is necessary to adequately read images obtained by axial tomodensitometry. This modality is, however, now easily available in most reference facilities, is less expensive than MRI, allows exams of patients without general anesthesia (in most cases) and is very quick which makes it a very good choice for numerous conditions.

**Magnetic Resonance Imaging (MRI): The Soft-Tissue Modality**

As its use has become more and more available, MRI is now considered the goal standard imaging technique for most intracranial conditions. It also remains the only modality allowing the evaluation of the spinal cord parenchyma, evaluation that may be very important not only to diagnose specific conditions, but also to determine their prognosis. MRI is, therefore, recommended not only to image the brain (for all suspected conditions involving it), but also the vertebral column in cases of: IVDD in non-chondrodystrophic breeds, cervical spondylomyelopathy, lumbosacral disease, and diseases affecting the spinal cord parenchyma such as myelitis, fibrocartilaginous embolic myelopathy (FCEM), spinal cord and nerve root tumour, syringomyelia, and spinal cord malformations.

MRI is cartography of mobile hydrogen protons. These are present in all cells of the body, but their concentration is higher in water and fat. Differences in hydrogen protons content of different tissues will provide a specific image on the computer. The machine used is a giant and very powerful magnet that sends different radio waves of specific frequency and energy stimulating the hydrogen protons within the tissues. A special antenna is used to capture and transmit the waves released by different tissues to the computer where the images are formed. The diagnosis can be made by comparing different sequences (frequency and energy specific) developed to detect more specifically distinctive tissues, again based on their hydrogen content. An exam may take up to 2 hours depending on the area imaged and the suspected condition.

With this modality more than with any other, it is extremely important to know exactly what to look for as the patient enters the machine. In fact, the MRI exam will be adjusted based on the sequences needed to image specific suspected conditions. If the lesion localisation and differential diagnosis is not precise, the exam may not allow determination of the diagnosis.
What’s New in Atopic Dermatitis in Dogs?
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Always rule out other potential or confounding causes of pruritus such as parasitic infestation (*Otodectes, Trombicula, Cheyletiella, Sarcoptes, Demodex*, lice); microbial infections/overgrowth (*Staphylococcus*, other bacteria, *Malassezia*, other yeasts); other hypersensitivity disorders (flea bite hypersensitivity, dietary hypersensitivity, allergic contact dermatitis, *Malassezia* or bacterial hypersensitivity); cornification disorders (metabolic diseases, zinc/vitamin A-responsive dermatoses); neoplastic diseases (epitheliotropic lymphoma, mast cell tumour); other skin diseases (irritant contact dermatitis, cutaneous drug reaction).

**Conceptual Dermatologic Pearls**

**The T Helper-1:T Helper-2 Cell Paradigm**
- CD4 + cells, balance between TH1:TH2 direct the immune system.
- T helper-1 cells - local immune defense system (IFN).
- T helper-2 cells - humoral immune defense system (IL4, IL5).
- Allergies result in TH2 stimulation and TH1 suppression.
  - Secondary bacterial, yeast, fungal, parasitic, & viral infections.

**The Allergic/Pruritic Threshold**
- Level above which a pet will start to exhibit clinical signs.
- Allergies are a spectrum of disease, not separate entities.
- Food, flea, contact, and environmental allergies add up to push a patient above its allergic threshold (summation of effect).

**Calendar Updates for Followup**
- Record events on a daily basis for easy review at recheck/email/telecom.
- Visualize patterns of response + minimizes “husband/wife” discrepancies.
- Make efficient use of appointment time.

**Vaccinating the Allergic Patient**
Vaccines can cause sensitization to food antigens, stimulate the production of lymphocytic thyroiditis in dogs, and stimulate allergen specific reactions in allergy-prone dogs.

I therefore recommend the following in my allergic breeds:
6. Vaccinate outside of the pet’s allergy season (e.g., midsummer or winter time = lowest allergen load).
7. Move to a 3-year, rotating vaccination protocol with labeled and licensed 3-year vaccines.
8. If not able to move to 3-year rotation, split up vaccine injections by 2- to 4-week intervals.
9. Consider titers in dog with severe reactions to vaccine.
10. Consider pre-/post-vaccination use of antiinflammatory medication to prevent reactions.

**Overview of My Approach to Treatment of Allergies in Dogs**

11. Immunotherapy based on serologic and intradermal allergy testing.
12. Immunotherapy based on intradermal allergy testing only.
13. Immunotherapy based on serologic allergy testing only.
14. Long-term symptomatic therapy using nonsteroidal immunomodulators such as cyclosporine.

All of the above may require adjunctive therapy such as topical antiinflammatory agents, antihistamines, pentoxifylline, essential fatty acids, ectoparasite control, barrier repair products, and shampoo therapy.

Also, dietary restriction may be used long term or during peak allergy seasons to minimize CAFR’s contribution to the allergen load.

**Tacrolimus (ProTopic® 0.1% & 0.03%) for Local Immunomodulation**

- Macrolide lactone produced by fungus *Streptomyces tsukubaensis*.
- **MOA:**
  - Inhibits T-lymphocyte response to antigens; production of IL2 → decreased T-cell proliferation; IL3, IL4, IFN-γ, and TNF-α important in allergies and inflammation.
  - Downregulates cytokine expression in Langerhans, mast cells, basophils, eosinophils, keratinocytes.
- **Uses:**
  - Localized DLE, SLE, PF, PE, allergic dermatitis, vasculitis.
- **SE:**
  - ± irritation at site of application, erythema.
- **Study:**

**Hydrocortisone Aceponate (Cortavance®, Virbac) Spray**

- Topical esterified hydrocortisone 0.584 mg/mL in propylene glycol methyl ether.
- 2 pump spray activations over a 10 cm × 10 cm.
- **MOA:**
  - Typical steroidal antiinflammatory mechanism of action; however, steroid diesters are lipophilic substances of low-plasma availability that effectively penetrate the skin to accumulate at site of inflammation without significant systemic absorption.
- **Uses:**
  - Localized DLE, SLE, PF, PE, allergic dermatitis, vasculitis.
- **Dose:**
  - From 10 cm away, dose rate = two sprays/100 cm².
- **SE:**
  - Erythema and/or pruritus can occur in very rare cases; suppress HPA axis with high-dose or long-term, off-label use.
- **Study:**
  - **Part 1: Results at day 28:**
    - 11/15 HCA vs. 3/13 placebo = 50% reductions in CADESI.
    - 7/15 HCA vs. 1/13 placebo = 50% reductions in pruritus.
    - Owner global efficacy score: HCA 3.1 vs. placebo 2.4 (p = 0.0001).
  - **Part 2: Maintenance from day 28 to day 70.**
- 12/15 HCA + 9/13 placebo continued treatment for 42 days.
- 5/21 additional therapy required (5 placebo).
- 3/21 daily (2 HCA/1 placebo).
- 7/21 q 48 h (4 HCA/3 placebo).
- 6/21 2 X/week (6 HCA).
- No effect of coat length.
- No adverse effects or changes to blood parameters.

**Mometasone (Elocon®, Mometamax®, Merck)**
- Potent topical steroid with minimal to no systemic absorption.
- Mometamax also contains gentamicin (antibiotic) and clotrimazole (antiyeast).
- **MOA:**
  - Inhibit the activation of inflammatory proteins.
  - Activate the secretion of anti-inflammatory proteins.
  - Stabilizing inflammatory cell membranes.
  - Decreasing the influx of inflammatory cells.
- **Uses:**
  - Localized DLE, SLE, PF, PE, allergic dermatitis, vasculitis, *Malassezia*/bacterial fold dermatitis (cheilitis, peri-vulvitis or pododermatitis).
- **SE:**
  - Erythema and/or pruritus can occur in very rare cases.
  - Suppress HPA axis with high-dose or long-term, off-label use.

**Cyclosporine (Atopica™ 10-mg, 25-mg, 50-mg, 100-mg Capsules, Novartis)**
- Cyclosporine is a macrolide derived from a fungus.
- **MOA:**
  - Targeting T lymphocytes binding immunophilin (cyclosporin binds cyclophilin, tacrolimus binds macrophilin) → inhibiting calcineurin → decreasing stimulation of nuclear factors of activation (NF-ATp) → decreased production of inflammatory cytokines (IL-2, others).
  - As T lymphocytes serve as the brains of the entire immune system, the entire inflammatory cascade is suppressed.
  - Affects Langerhans cells by decreasing migration & ability to process antigen.
  - Inhibits the release of preformed mediators from mast cells and basophils.
  - Decrease synthesis of TGF-α and -β, IFN-γ, GM-CSF, IL-2, IL-3, IL-4, and IL-5.
  - Cytostatic effect on keratinocytes of the epidermis.
  - As cyclosporine only affects inflammatory cells (unlike systemic steroids), the number and severity of adverse reactions is limited.
  - Also has anti-neurogenic activity by calcineurin inhibition on nerves binding to capsaicin receptor TRPV1 resulting in a burn then cool sensation.
- **Study:**
    - Multicentre, blinded, parallel, randomized, placebo controlled trial.
    - N = 32 severe and moderate nonseasonal AD, topical CsA (17), or placebo (15)
      - BID 6 weeks.
    - 87.5% and 28.6% in the CsA and placebo groups respectively.
  - **Study:**
      - N = 6 research hounds; randomized crossover study.
      - Daily for 7 d w/14-d washout; sampling on d 1, 4, 7 - blood 1.4 h/24 h, skin 4 h/24 h.
Treatment groups:
- T1 = 5 mg/kg Atopica.
- T2 = 2.5 mg/kg Atopica.
- T3 = 2.5 mg/kg Atopica + 5 mg/kg ketoconazole.
- T4 = 2.5 mg/kg Atopica + 2.5 mg/kg ketoconazole.

Results:
- Blood: T3 (644 ng/ml) >> T4 (417 ng/ml), T1 (307 ng/ml) > T2 (169 ng/ml).
- Skin: T3 (1.2 ng/mg) >> T4 (0.7 ng/mg), T4 (0.6 ng/mg) > T2 (0.26 ng/mg).

Conclusion:
- 5 mg/kg Atopica = 2.5 mg/kg Atopica + 2.5 mg/kg ketoconazole.

**Pentoxifylline’s Multiple Uses**
- Synthetic xanthine derivative related to caffeine and theophylline.
- Phosphodiesterase inhibitor in RBC and immune system.
- Rheologic agent, immunomodulator, wound healing.

**Uses:**
- Vasculopathies, ischemic folliculopathies, allergic dermatitis, autoimmune disorders.

**Dose:**
- 10–35 mg/kg PO BID to TID.

**SE:**
- Rare; nausea, vomiting, hyperexcitability; minimized using sustained-release tablets.

**Interaction:**
- Cimetidine and fluoroquinolones decreases clearance.
- Do not use with warfarin, anticoagulants, or in patients with hemorrhagic conditions.

**Studies:**

**Cetirizine - My New Favourite Antihistamine**
- Second-generation member of the piperazine family.
- Active metabolite of hydroxyzine with mild sedative effects.

**Uses:**
- Antihistaminic activity and also decreases influx of eosinophils into skin, which is great for treatment of eosinophilic granuloma complex patients and allergies.

**Onset of action:**
- 1–3 hours following oral administration.

**Peak concentrations:**
- At 10 hours.

**Duration of action:**
- Ranging between 12–24 hours.
- Cetirizine is excreted largely unchanged in urine.
- A minimum of 2 weeks of continuous dosing is necessary to fully assess any antihistamine.

**Dose:**
- 0.5–1.0 mg/kg q 12–24 h.

**SE:**
- Same as other antihistamines except for sedation.
Study:

**Oclacitinib (Apoquel®, Zoetis)**
- **MOA:**
  - Selectively inhibits Janus kinase 1-dependent cytokines of which many are proinflammatory, pro-allergic, & pruritogenic including interleukin (IL)-2, IL-4, IL-6, and IL-13. Oclacitinib also inhibits the pruritogenic cytokine IL-31 involved in the neurogenic itch pathway.
- **Uses:**
  - Humans: psoriasis, rheumatoid arthritis, neoplasia, polycythemia vera, etc.
  - Dogs: atopic dermatitis and other pruritic conditions such as flea allergy dermatitis, *Sarcoptes*.
- **Dose for dogs:**
  - 0.4 mg/kg BID
- **Studies:**
  - Cosgrove S, et al. Multicentre clinical trial to evaluate the efficacy and field safety of oclacitinib.
    - N = 341 dogs, placebo matched study, significant improvement 14 d, some vomiting and diarrhea noted.
  - Fleck T, et al. Comparison of Janus kinase inhibitor oclacitinib, and prednisolone in canine models of pruritus:
    - Oclacitinib greater effect and faster than prednisolone.
  - Wheeler DW, et al. Oclacitinib for the treatment of pruritus and lesions associated with canine flea-allergic dermatitis:
    - N = 36 FAD, sig improvement in VAS 0.4, 0.8 mg/kg >> placebo.

**MULTIMODAL ANTIINFLAMMATORY THERAPY - THE “FOREST FIRE” ANALOGY**
Even with the introduction of nonsteroidal alternatives such as cyclosporine with long-lasting effects equivalent to those of steroids, glucocorticoids remain the only treatment option that will give the patient immediate relief or “put out a fire” within 24–48 hours. If the analogy of a forest fire is used, the “fire retardant” dropped from airplanes is the steroid, while the other therapeutic options such as omega-3 fatty acids, antihistamines, dietary restriction, flea control, epidermal barrier repair, cyclosporine, and allergen-specific immunotherapy are the “trench diggers” controlling the fire from spreading. When oclacitinib reaches the market, it will be positioned in the “fire retardant” group. The incorporation of this multimodal approach to control the allergic reactions, prevents discomfort and damage to the epidermal barrier, as well as calms the inflammation and microenvironment, hence minimizing recurrence of secondary infections, so much so, that I typically have clients err on the side of giving a little extra steroid to prevent a secondary infection rather than to give too little steroid and have to return to 4 weeks’ worth of antimicrobial therapy to treat a *Malassezia* or bacterial infection.

**PREVENTATIVE USE OF ANTIINFLAMMATORY MEDICATIONS**
Lastly, once a pattern of seasonal reactions has been established and appears quite predictable, use of cyclosporine, steroids, or other antiinflammatory medications in advance of the allergy season may allow the owner to start at maintenance control doses as opposed to using high loading doses to put out the fire first before moving to maintenance therapy. Ultimately, this approach will help minimize cost and side effects associated with high daily doses of allergy medications as well as prevent costly secondary infections such as *Staphylococcus* and *Malassezia* dermatitis.
SUGGESTED READING
What’s New in Atopic Dermatitis in Cats?
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Before tackling treatment of feline atopic dermatitis, always be certain to rule out other common causes of itching, licking, hair loss, ulceration, and eosinophilic granulomatous diseases in the cat. In particular, address flea allergy dermatitis, Cheyletiella mites (“walking dandruff”), Notoedres (feline scabies), cutaneous adverse food reactions, dermatophytosis, Demodex (D. cati and D. gatoi), pemphigus foliaceus, Malassezia dermatitis, and paraneoplastic pruritus as primary or concurrent etiologies. Flea combing, ear swabs, acetate-tape preparations, skin scraping, and hair plucking in mineral oil are used to evaluate for fleas, Demodex gatoi, Otodectes, and Cheyletiella. Ideal sampling sites include those with inflammation or easily epilated hairs. If no lesions exist, sample broadly across several regions, especially areas the cat cannot reach. If other “unaffected” cats are present in the household, consider sampling these individuals since they are less likely to groom excessively and remove the mites. Fecal examinations for ingested hair or ectoparasites (e.g., Demodex gatoi) may aid in differentiation of pruritic dermatoses. Regardless of a lack positive identification of ectoparasites on dermatologic or fecal examination, aggressive ectoparasiticidal trials for lice, flea, and mite control is an essential part of a diagnostic and therapeutic approach to any pruritic cat. If D. gatoi is identified or strongly suspected, use lime sulfur dip (8 oz/1 gallon) once weekly for 4 to 6 weeks. Treat all in-contact pets or isolate the affected individual.

Avoidance
If minimal to no response is noted to the above diagnostic and therapeutic interventions, then consider environmental ± combined with food allergies (CATopy) as one of your primary underlying etiologies. A seasonal dietary trial may help lower the pet’s allergen load below its threshold, such that you may not even need to address the environmental component, or at least be able to minimize the need for symptomatic therapy. Environmental modifications such as moving the cat outdoors while vacuuming or indoors while mowing the lawn are always worth considering when attempting to minimize allergen load. Other avoidance measures include minimize exposure to mulch; dry carpet cleaning to decrease survival of indoor dust mites and mold spores; changing vacuum bags and filters for the central air system frequently; using a HEPA filter (Honeywell QuietCare®) for the area where your patient spends most of their time; installing vent register filters and changing them every 3–4 weeks, along with duct cleaning to remove accumulated dust mites and mold spores; purchasing hypoallergenic mattress/pillow covers, remove cedar bedding; using T-shirts or ThunderShirts (www.thundershirt.com) as a barrier to the environment, to prevent further damage from scratching, and to help minimize anxiety that may contribute to pushing a pet over their allergic threshold; and pheromones (Feliway™) to eliminate other stresses from the environment. For more information, pollen & mold spore counts, and other useful information go to the American Academy of Allergy and Immunology website (www.aaaai.org/home.aspx).

Treatment of CATopy
More often, the veterinarian is dealing with an allergic etiology whereby the offending allergen cannot be avoided or eliminated from the patient’s environment. In this situation, treatment of an allergic dermatitis often involves a multimodal approach. I often equate my approach to allergies to a forest fire where we have: a) trench diggers, who control and prevent the fire from spreading; and b) fire retardants - the planes/helicopters that drop the fire retardant and quickly put out the fire so that the trench diggers can continue to do their job.

A) The Trench Diggers
Dietary restrictions, ectoparasites control, epidermal repair products, shampoo therapies, essential fatty acids, antihistamines, cyclosporine, and immunotherapy all fall under the “trench digger” category as they work slowly and methodically to control and prevent allergy flares with minimal side effects. A
combination of therapeutic approaches will often lead to minimization of the need for more potent medications such as steroids.

**Epidermal Barrier Repair**
Epidermal barrier repair products that contain ceramides, free fatty acids, essential fatty acids, phytosphingosine, and essential oils are used to repair seborrheic skin conditions and maintain an intact epidermal barrier to minimize percutaneous absorption of allergens and adherence of microbes. If shampoo therapy is possible, appropriate selection of shampoos based on active ingredients used with cool water on a weekly basis will help to minimize surface microbes, wash off accumulated allergens, provide anti-inflammatory relief, and help repair the epidermal barrier. These products should be considered for use as a preventative as well as a topical adjunct to treatment of active clinical signs.

**Omega-3 Fatty Acids**
Products such as omega-3 fatty acids (EPA - 180 to 200 mg/day) may provide relief for the pruritic cat during the infancy of a pruritic event and while trying to taper the use of steroids in the latter phases of a pruritic condition. Reliable veterinary sources should be recommended to achieve optimal responses and avoid adverse reactions from rancid oils and contaminants. Oral fatty acids should not be used during a food trial.

**Antihistamines**
Antihistamines are most useful in a cat with environmental allergies. My current favourite is cetirizine (0.5–2.0 mg/kg q 24 h) based on the availability of pharmacokinetic data in cats and its action of decreasing influx of eosinophils into affected sites ideal for cats with eosinophilic granulomas. If minimal response is noted to cetirizine in 14 days, I then consider chlorpheniramine (2 mg/cat q 12 h) or amitriptyline (5 mg/cat q 12–24 h). If appetite stimulation is a desirable side effect, then I reach for cyproheptadine 2 mg q 12 h. Trials are continued for 14 days until an effective option is found. Antihistamines have minimal side effects or contraindications for long-term use.

**Cyclosporine**
Micro-emulsified cyclosporine provides long-term symptomatic treatment alternative to corticosteroids for owners who cannot afford or who do not wish to pursue allergy testing and immunotherapy for their cat. Cyclosporine is a potent inhibitor of interleukin-2 (IL-2) and hence T lymphocyte-dependent immune responses. As well, cyclosporine decreases IL-3, IL-4, IL-5, tumour necrosis factor (TNF)-α, and interferon (IFN)-α production. It also serves to inhibit antigen presentation, histamine release from mast cells, neutrophil adherence and growth, and differentiation of B lymphocytes. Cyclosporine at 7.5 mg/kg daily is as effective as steroids for controlling pruritus and treating eosinophilic granuloma complex. Cyclosporine’s effects however can take 4–8 weeks before optimal response is noted. In patients with seasonal or chronic allergies, prophylactic use of cyclosporine before the allergy season may negate the need for daily induction dosing. Diarrhea, anorexia, and vomiting are the most common side effects and are often transient. Be certain to test for retroviruses before beginning therapy. Fatal systemic toxoplasmosis has been reported hence caution should be exercised if considering use in cats at high risk for toxoplasmosis, such as outdoor hunters. Cats on cyclosporine should not be allowed to hunt and should be fed cooked or processed food.

**Allergen-Specific Immunotherapy (ASIT)**
Allergen-specific immunotherapy (ASIT) modulates response to allergens and diminishes the severity of hypersensitivity reactions by isotype switching away from production of paranoid antibodies (IgE) and shifting toward generation of tolerant or blocking antibodies (IgG) as well as modulating the T-regulatory cell and IL-10 production in treated patients. The safety record is very good and there is excellent evidence of efficacy. Maximum doses of 0.5 ml weekly of a 20,000 PNU concentration of allergen extract, subcutaneous immunotherapy (SCIT) make this a compliance enhancing and economically attractive long-term treatment option. For those clients that find it difficult to administer subcutaneous injections (SCIT) to their cat, RUSH immunotherapy can be used to accomplish a 30-day induction in one day, thus allowing the owner to return to your clinic for weekly injections thereafter. Another alternative is to
consider sublingual immunotherapy (SLIT), a daily, oral, low-dose administration of the immunotherapy in a glycerin base. Response rates as high as 60–90% have been reported for ASIT in feline patients and with quicker responses than dogs or humans. As ASIT is not a “light switch” type treatment, cats may require adjunctive therapy, such as antihistamines, cyclosporine, or intermittent corticosteroids, especially during the induction phase of ASIT and at time during ASIT maintenance at peak pollen seasons. Note that it is the only therapy that may cure atopy or at a minimum prevent the development or progression of allergies. All other treatments for allergies are “band aids” or symptomatic at best.

**Chlorambucil**

In refractory cases or cats that develop side effects associated with steroid or other antiinflammatory medications, chlorambucil at 0.1–0.2 mg/kg q 24 h PO initially then tapered to every other day or less has also been shown to be an effective therapy for CATopy. Adverse reactions include anorexia, vomiting, diarrhea, bone marrow suppression, and hepatotoxicity. Regular monitoring of CBC, chemistry profile, and urinalysis is recommended.

**B) The Fire Retardant**

**Glucocorticoids**

Glucocorticoids are currently the **only** effective method of providing immediate relief from symptoms. Due to the prednisone to prednisolone conversion defect identified in cats, I tend to use dexamethasone (0.05–0.1 mg/kg/d) or prednisolone or methylprednisolone (1-2 mg/kg/d) daily for 7 days then taper to the lowest effective dose. Steroids are often used in combination with one or more of the above “trench diggers.” Once the clinical signs have abated, steroids may also be used once daily in 3- to 7-day bursts to “put out fires” as they arise. Methylprednisolone acetate given by subcutaneous injection is recommended primarily for management of cats that do not tolerate administration of oral medications. Each injection should last for 8–12 weeks if treating environmental allergies. If shorter intervals are necessary, consider ruling out other differentials including adverse food reactions. Long-term inappropriate steroid use can result in recurrent secondary infections, diabetes, and feline cutaneous hyper-fragility syndrome.

**CONCLUSION**

Feline pruritus can be frustrating for both owners and veterinarians. There are many underlying etiologies that often present with similar clinical findings. As cats do not follow “dermatologic rules,” it is up to the veterinarian to not become complacent with injectables such as methylprednisolone acetate, rather carefully and consistently approach each cat eliminating ectoparasites and infectious disorders before moving to immunomodulators of itch.

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**SUGGESTED READING**


What’s New in Food Allergies in Veterinary Dermatology?
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Adverse food reactions (AFR) account for up to 15–46% and 10–23% of cutaneous inflammatory lesions in dogs and cats respectively. AFRs are also often noted concurrently with environmental and flea allergy dermatitis. Selection of the appropriate diet will either significantly improve clinical signs of allergies or completely eliminate them.

Strategies involving diet manipulation include:
33. The dietary trial to decrease the patient’s allergen load below the allergic threshold
34. Integration of diet as part of the multimodal approach to allergic dermatitis
35. The use of dietary restrictions to prevent the allergic response

Dietary Trial to Address Adverse Food Reactions
Diagnosis of adverse food reactions is performed by the evaluation of history and clinical signs, elimination of other differentials, and the dietary trial.

Signalment
Signalment may in some cases be helpful to at least help increase your index of suspicion of AFR in pets. Top breeds associated with food allergy in the author’s experience include Labrador Retrievers and Cocker Spaniels, along with others described in literature including, but not exclusively, the Soft-Coated Wheaton Terrier, Dalmatian, West-Highland White Terrier, Bichon Frise, Collie, Chinese Shar Pei, Lhasa apso, Dachshund, Miniature Schnauzer, Boxer, Springer Spaniel, Cairn Terrier, Irish/English Setter, Golden Retriever, German Shepherd dogs, Hungarian Vizsla, along with Siamese and Birman cats. Age at presentation is typically less than one year (33–52%; 2 months to 16 years) in dogs and before 2 years (38.5%; 4 months to 15 years) in cats. In general, food allergy should be moved to the top of the differential list in any pet that has an onset of pruritus or other clinical signs prior to 6 months and after 6 years of age. In all the studies thus far, there appears to be no sex predilection.

History
Historical presence of nonseasonal pruritus is the most common clinical manifestation of food allergy and the reason for seeking veterinary attention in most cases. Food allergy is often associated with other pruritic dermatopathies including atopy, flea allergy, Malassezia dermatitis, and superficial staphylococcal pyoderma. Simultaneous occurrence of food allergy with atopy or flea allergy may present as a nonseasonal condition with seasonal peaks. Food allergic patients’ response to conventional antiinflammatory doses of steroids is incomplete in most cases. The association of sudden dietary change with onset of clinical signs is more the exception than the rule. As pets can be allergic to one or more antigens in a diet, generating an inventory of current and previous commercial pet foods, table foods, snacks, treats, drive-thru rewards, chewable medications, and digestible chew toys (e.g., rawhides) should be part of the historical database. Specific questions regarding gastrointestinal disturbance should likewise be addressed in the acquisition of a proper history. Episodic food hypersensitivity may occur following intermittent exposure to flavored medication, the offending food antigen from the table, from predation, or eating garbage. Coprophagic dogs may obtain undigested material that could affect a dietary trial.

Clinical Signs
Cutaneous
Cutaneous findings typically include pruritus, erythema, epidermal hyperplasia, papular eruption, urticaria and angioedema, secondary scale and crusts, malodor are common findings in patients with AFR. Seborrhea with recurrent secondary bacterial or Malassezia dermatitis is often present in chronic cases.
The ear region was most consistently involved (80%), followed by the feet (61%), and the inguinal/perineal region (53%) in a study evaluating AFR in dogs. The axillary, anterior foreleg, and periorbital regions were nearly equal in occurrence (31–37%). The ear was the only area affected in 12 dogs (24%). Another pattern of clinical signs is similar to flea allergy dermatitis affecting the lower back, tail head region and caudolateral thighs. When the dorsal lesions extend beyond the thoracolumbar junction over the chest area, this also increases my index of suspicion for food allergy, as not many other diseases affect this region. AFR has to be considered among the top differentials for acral lick dermatoses, along with neurologic root-based conditions, behavioral obsessive compulsive disorders, arthritic pain if lesion is noted over a joint, hypothyroidism, environmental allergies, demodicosis, and secondary fungal, bacterial or yeast infections. Lastly, perianal pruritus is almost exclusively due to food allergy when considering allergic etiologies.

CAFR in cats can present with similar distribution to those in dogs; however, the head and neck (ring around the collar) appears more commonly affected than the ears, feet, and rears.

Other cutaneous conditions that may be attributable to or triggered by AFR include Cocker Spaniels idiopathic seborrhea, symmetric lupoid onychodystrophy, sterile interdigital cysts, feline eosinophilic granuloma complex, feline symmetric (fka psychogenic) alopecia, feline miliary dermatitis, chin acne, pemphigus complex, perianal fistulas, pinnal vasculitis, recurrent generalized demodicosis, and food-induced cutaneous vasculitis.

Gastrointestinal
Gastrointestinal signs are present in up to 32% of food allergic patients. These include vomiting, changes in the stool consistency, increased frequency of bowel movements, eructation, halitosis, borborygmus, flatulence, tenesmus, eosinophilic or lymphocytic-plasmacytic colitis/IBD, anal gland impaction & scooting, pica and/or coprophagia.

Neurologic/Behavioral
Neurologic/behavioral signs such as malaise, seizures, behavior changes, dominance aggression, attention deficit disorders, and difficulty in training have either been proposed, documented, or are currently being researched.

Respiratory
Respiratory signs associated with AFR include asthma, rhinitis, and sinusitis.

Musculoskeletal
Musculoskeletal conditions potentially attributable to food allergies include sterile polyarthritis and masticatory muscle myositis. Concurrent conditions noted with frequency include environmental, flea, intestinal parasite, and insulin hypersensitivities; secondary recurrent pyoderma and Malassezia pachydermatis infection (may be the only sign); and Sarcoptes infestation.

Diagnosis
It is important to eliminate/address concurrent conditions, especially atopy and scabies, before or while on dietary trial. I find that voicing the “treat list” aloud to owner will help to accurately catalog dietary ingredients, as some owners do not feel that the monthly heartworm preventative is a “treat.” I usually start with an open-ended question such as, “what crosses your pet’s mouth during the course of a week?” and follow it with my checklist including: treats and rawhide chews, toys, drive-thru treats, goodies from the neighbor/service person, popcorn, tuna juice, end-o-cereal/ice cream bowl, access to other pet’s food or stool, pillling vehicles/devices (pill pockets, cheese, etc.), supplements (chondroitin/GAG), alternative medications (Echinacea), flavored toothpaste, and/or chewable medications. I also include fruits and vegetables on my checklist, as a condition called oral allergy syndrome has been documented in humans whereby ingestion of oral antigens from various food items may have similar antigenic protein (e.g., Bet v1 in birch and apple; cedar pollen and raw tomato) or may crossreact with environmental allergens to escalate clinical signs of the consumer’s atopic condition.

The use of either a RAST (radioallergosorbent test) or an ELISA (enzyme linked immunosorbent assay) technique for evaluating circulating IgE levels is commercially available. Although the advantage
of these tests is obvious, their correlation to actual food hypersensitivity is not known. In fact, serum IgG may correlate better with clinical disease. There have been reports in humans with good correlation between *in vitro* test procedures, skin testing and food allergy. This has not been documented in veterinary medicine. In contrast, studies performed suggest the lack of correlation between food allergy, RAST, or ELISA results, gastroscopic food sensitivity testing, colonoscopic allergen provocation studies (COLAP) and *in vivo* intradermal allergy test results. Positive predictive values were recently determined to be 40% whereas the negative predictive values were 60.9%. A small study by Bethlehem *et al.* evaluating the new chicken hydrolysate and cornstarch diet using patch testing identified 100% of previously challenged chicken and cornstarch-allergic dogs. Interestingly, 1 out of 5 dogs reacted to the hydrolysate feather diet on patch testing and upon dietary challenge, again questioning the effect of non-hydrolyzed components within the diet and/or our knowledge of the molecular weight to which dogs can react. The overall consensus of most dermatologists is that the dietary trial followed by dietary provocation is the most effective method of diagnosis. Standardizing a diet based upon *in vitro* test results provides minimal advantage over empirical decision without prior testing.

The **dietary trial with 100% restriction** is currently the best way to confirm food allergy. The diet is restricted to specific food determined by the animal’s previous exposure and known reactions. The top offending food items include beef, milk, lamb, wheat, corn, chicken egg, soy, chicken in dogs, adding tuna and salmon to the list in cats. The preferred diet is home prepared and simplified to include a protein source and a carbohydrate source (1:2 in dogs; 1:1 in cats). Selection of a novel protein and carbohydrate may require reviewing the list of ingredients of commercial dog/cat foods and treats. Although lamb and rice diets have been in vogue for dietary trials for some time, the prevalence of these substances in commercial feed is limiting their usefulness as novel proteins and in fact, an increase in dietary related skin disease has been associated with lamb & rice based foods. Novel protein sources currently available for home cooking include kangaroo, camel, ostrich, emu, bison, elk, venison, rabbit, duck, squid, and fish. Old world grains such as spelt along with oatmeal, quinoa, rutabaga, butternut squash, parsnips, peas, sweet potato, and white potatoes have taken the place of rice as a result of its gluten and popularity in commercially prepared diets. Tofu has been used successfully in home-prepared foods, but has two major disadvantages. The first is the poor palatability and the second is the low caloric density failing to provide adequate satiety. Tofu is composed of soybean, which restricts its use if the pet’s previous commercial food contained soy. Cats may pose difficulties in performing dietary trials as they have finicky appetites and easily become bored with the diet following initiation of the trial. Keeping cats indoors may prove difficult for some cat owners, as well as eliminating the access of house foods from the table or garbage due to cat’s ability to leap onto various surfaces. Supplementation with a multivitamin is not necessary for the length of the trial. If concerns arise about young growing dogs or cats placed on a home-prepared elimination diet, an option is to select one of the commercial foods approved for puppy/kitten growth. Consulting with nutritionists for advice about specific breed or life-stage requirements may be helpful.

Although it is advantageous to utilize home-prepared diets, the 8- to 12-week duration of the trial along with sufficient availability of commercial foods to conduct a good trial often leads owners to abandon home-cooked diets. The selection of the single protein and carbohydrate source should ultimately be based on the dietary history of the animal. The selection of a food with limited ingredients is the next priority. A large variety of limited-ingredient foods exist based upon different novel and hydrolyzed protein sources. The use of a commercial food provides a conservative way of evaluating food allergy although it has some compromise compared to a home-prepared fresh food because of multiple ingredients and additives present. Owners will often wish to provide treats to their pets during the elimination diet. Using the same or similar ingredients as the main diet provides an option that does not compromise the trial. The most difficult part of the trial is avoidance of any other food. Pitfalls include multiple dog/cat households where access to another food occurs. Table foods fed by children is a concern since it would invalidate the dietary restriction.

**Monthly evaluation** of response to the dietary elimination trial is mandatory. I often preface the start of a dietary trial with, “It should only get better, it should not get worse.” If clinical signs deteriorate any time before the initial 4-week recheck, the owner should discontinue the diet and other ancillary...
medications, allow the reactions to calm and switch to a different diet. During the time of the dietary trial, it is also important to control coexistent factors that may potentiate the pruritus and obscure the results such as flea allergy dermatitis, Malassezia dermatitis, and superficial pruritic pyoderma. I also often include glucocorticoid therapy during the first 30–45 days of the dietary trial to control intense pruritus and self-mutilation. After this time, the steroids are terminated, the restricted diet is continued, and the patient continues to be evaluated.

The **dietary challenge** is the only means of confirming food allergy following optimal improvement of the case post-trial. Challenge most often uses the single most common diet fed prior to the elimination trial. Some owners may be reluctant to institute a dietary challenge particularly if the improvement has been dramatic. It is important to follow through with careful monitoring of the dietary challenge. Observing the recurrence of the pyoderma, gastrointestinal, neurologic, behavioral, musculoskeletal, and/or respiratory symptoms upon food challenge is evidence of the relationship. Relapses typically occur within 15 minutes to 14 days (12–48 h most commonly). In the event of relapse during the challenge, the pet should be returned to the elimination diet for a period of time to allow the patient to reach the pre-challenge clinical state before adding another test food. Return to pre-challenge level of control takes much less time than the original response noted during the original trial.

**INTEGRATION OF DIET AS PART OF THE MULTIMODAL APPROACH TO ALLERGIC DERMATITIS**

Even with the introduction of nonsteroidal alternatives such as cyclosporine (Atopica®) with long-lasting effects equivalent to those of steroids, glucocorticoids remain the only treatment option that will give the patient immediate relief or “put out a fire” within 24–48 hours. If the analogy of a forest fire is used, the “fire retardant” dropped from airplanes is the steroid, while the other therapeutic options such as omega-3 fatty acids, antihistamines, dietary restriction, flea control, epidermal barrier repair, cyclosporine, and allergen specific immunotherapy are the “trench diggers” controlling the fire from spreading.

The incorporation of this **multimodal approach** to control the allergic reactions, prevents discomfort and damage to the epidermal barrier, and calms the microenvironment minimizing recurrence of secondary infections. The decision to implement the use of various therapeutic modalities to address atopic dermatitis is also based on the duration of clinical signs. Allergic symptoms lasting less than 30 days are best treated with glucocorticoids, as the nonsteroidal options take too long to exert their beneficial effects. Pruritus lasting 4–6 months is amenable to the “trench diggers” (e.g., dietary restriction, Atopica®, antihistamines, pentoxifylline, and shampoo therapy), although a short burst of the “fire retardant” (e.g., steroids) given concurrently will hasten control of the clinical signs. Allergen-specific immunotherapy (ASIT) is the best long-term treatment option with the fewest systemic side effects, in that it works primarily by modulating the immune system (e.g., IgG isotype switching, T-regulatory cell and IL-10 shifting toward a T-helper 1 response) and does not tax the body’s organ systems. However, ASIT is not a “fire retardant” and hence requires additional ancillary support to control clinical signs, especially through the initial induction phases of immunotherapy. The dietary trial is an option that also does not tax the body’s organ systems and can be used consistently or even periodically as adjunctive therapy during peak pollen seasons to help minimize need for ancillary medications. When the allergen/pollen load subsides, owners may relax their dietary restrictions.

**THE USE OF DIETARY RESTRICTIONS TO PREVENT THE ALLERGIC RESPONSE**

Tater et al. have demonstrated that allergen-specific IgE levels increase to food antigens being fed in allergy-prone dogs immunostimulated by prophylactic vaccines for at least three (3) weeks post-vaccination and subside to normal pre-vaccination levels in 8–9 weeks. Imani et al. proposed that human vaccines induce the expression of IgE mRNA through the activation of an antiviral protein kinase. Based on the potential for sensitization to foods being ingested at the time of booster vaccinations during puppyhood and kittenhood, I typically advise that a “sacrificial protein” be fed during this stage of a dog or cat’s life especially in breeds with a predisposition toward allergies. Once the pet reaches adulthood and vaccines occur annually or less frequently, then switch to a different protein source; for example, start with a lamb-based diet in puppy-/kittenhood, then move to a chicken-based diet into
adulthood. Avoid remaining on the same protein source through the entire pet’s life to **prevent** development of food allergies.

Another avenue of dietary intervention/**prevention** would include using a novel protein dietary and treat restriction in allergic patients that have a consistent/predictable pattern of seasonal reactions to help minimize allergen load. By restarting the dietary restriction 1 month prior to the typical onset of clinical signs, the restricted diet will prevent exacerbation of environmental allergies by minimizing allergen load due to oral allergy syndrome.

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**Table 1. Oral allergy syndrome: cross-reactions between food and environmental allergens**

- Birch (Bet v 1):
  - Apple, carrot, cherry, pear, peach, plum, prune, nectarine, apricot, kiwi fruit, honey, potato, tomato, spinach, celery, parsnips, green pepper, lentils, peas, beans, peanut, parsley, anise, dill, fennel, caraway, coriander, cumin, wheat, buckwheat, hazelnut, walnut, almond
- Mugwort sage (Art v 1):
  - Celery, carrot, spices, melon, watermelon, apple, chamomile, hazelnut, anise, fennel, coriander, cumin
- Grass(Phl p 1–6)
  - Potato, melon, tomato, watermelon, orange, cherry, peanut, kiwi
- Ragweed (Amb a 1)
  - Watermelon, cantaloupe, honeydew, chamomile, honey, banana, sunflower seeds, zucchini, cucumber
- Latex (Hev b 5)
  - Avocado, potato, banana, tomato, chestnut, kiwi fruit, herbs, carrot
- Peanuts (Ara h 1)
  - Legumes, grass, wheat, corn
- Plantain (Pla l 1)
  - Melon

The seasonal dietary restriction may then allow the veterinarian and owner to diminish their need for cyclosporine, Temaril/Vanectyl-P, or other antiinflammatory medications. At a minimum, the restricted diet will permit the use of maintenance control doses as opposed to using induction high loading doses of medications. Ultimately, this approach will help decrease cost and side effects associated with high daily doses of allergy medications as well as prevent costly secondary infections such as *Staphylococcus* and *Malassezia* dermatitis.

**CONCLUSION**

Omission of food allergy as an important differential is an infraction on the veterinarian’s oath to, “**Above all, do no harm!**” Food allergy is one of the easier immune-mediated diseases to control by means of avoidance. In fact, AFR should always be considered a potential trigger or underlying etiology, because the alternative of surgeries or lifelong immunomodulatory therapies in a dog or cat may pose a significant health risk.

What have you got to lose by considering a diet trial for:

- Inflammatory bowel disease vs. lifelong steroids and immunosuppressive agents
- Anal gland disease vs. surgery and its complications
- Asthma vs. chronic antiinflammatory medications
- Chronic recurrent otitis externa vs. total ear canal ablation-bulla osteotomy
- Idiopathic epilepsy vs. lifelong phenobarbital, potassium bromide, or levetiracetam
- Behavioral disorders (ADD, aggression) vs. lifelong behavior modifying agents or euthanasia
- Immune-mediated disease vs. lifelong immunosuppressive therapies and repeated blood work to monitor for adverse effects

Again, I ask you, what have you got to lose by performing a dietary trial?

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**Recommended Reading**


What’s New in the Treatment of Alopecia in Veterinary Medicine?
Anthony A. Yu, DVM, MS, DACVD
Yu of Guelph Veterinary Dermatology, Guelph, ON, Canada

Canine alopecia is a common presenting complaint in veterinary dermatology. Hair loss can be associated with self-trauma (pruritus due to allergies, ectoparasites, behavioural), inflammation of the hair follicle (folliculitis - Demodex, dermatophyte, bacteria), immune-mediated attack of a follicular component (alopecia areata, pemphigus, other immune-mediated), or clinically noninflammatory, non-pruritic alopecias of the dog.

The latter group can be subdivided into several groups:

- **Endocrine alopecias** - hyperadrenocorticism, hypothyroidism, sex hormone imbalances (Sertoli cell tumor, testicular and ovarian neoplasia, cystic ovaries), and pituitary dwarfism
- **Hair cycle arrest** - alopecia X, post-clipping alopecia, telogen defluxion, nutritional deficiencies
- **Follicular dysplasia** - cyclical flank alopecia, color-dilution alopecia, black-hair follicular dysplasia
- **Other** - pattern baldness, sebaceous adenitis, alopecia areata, congenital alopecia, breed-specific alopecias, dermatomyositis

Although all alopecias may look alike, they are not alike in many other ways. Subtle historical and dermatologic findings can help distinguish between the various noninflammatory alopecias.

### Table 1. Alopecias are not alike - some clues that help

<table>
<thead>
<tr>
<th>Loss of telogenized hairs in frictional areas, dorsal muzzle, tail, back</th>
<th>Hypo T4, Cushing’s, sex hormone, alopecia X</th>
</tr>
</thead>
<tbody>
<tr>
<td>Telogen phase when hair is shaved; vasoconstriction due to cold temp at shaved area</td>
<td>Post-clipping alopecia</td>
</tr>
<tr>
<td>Hair growth at sites of trauma</td>
<td>Alopecia X - due to release of platelet derived growth factor</td>
</tr>
<tr>
<td>Hair loss affecting one colour</td>
<td>Follicular dysplasia - genetic</td>
</tr>
<tr>
<td>Hair damage due to abnormal clumping of melanin in hair shafts</td>
<td>Color dilution alopecia</td>
</tr>
</tbody>
</table>

**Approach to Alopecia in Dogs**

**Canine Cushing’s Syndrome**

This condition is a diagnostic dilemma. Classic clinical signs are most helpful and include bilateral, symmetric alopecia; recurrent, secondary infections; atrophic skin (abdominal blood vessels visible through skin); pot-bellied appearance due to fat redistribution and ventral abdominal muscle atrophy; the poly complex (polyuria, polydipsia, polyphagia, and poly-panting); and calcinosis cutis. Some of the more unusual clinical findings that suggest Cushing’s syndrome include loss of muscle mass (temporal muscle loss noted by prominent occipital process), “split ends,” coat color change, collagen breakdown (especially noticeable at spay incision), poor healing wounds, anterior cruciate rupture, reproduction failure (FSH - failure to cycle; LH - testicular atrophy), and secondary hypothyroidism (downregulation of TSH receptors - reversible hypothyroidism).

Encompassing clinical signs with a combination of screening and differentiating tests (beyond the scope of this lecture) will help to provide a definitive diagnosis of Cushing’s syndrome. When discussing the benefits of treatment with your client, one must also weigh the potential risks/negative effects of treatment including cost of monitoring, relapse of inflammatory conditions such as allergies and arthritis when endogenous steroids are returned to normal levels, Addisonian crisis, and potential for adrenal necrosis. Below are key points to consider when selecting a treatment for Cushing’s syndrome.

**Treatment Options**

50. Mitotane (o,p-DDD):
a. Adrenolytic agent causing progressive necrosis of the zona fasciculata and reticularis, but usually spares the zona glomerulosa
b. Most frequently used in the treatment of dogs with PDH before trilostane
c. Should be dosed between 12.5–25 mg/kg BID, given with food for induction, then decreased to 1–3 X weekly when appetite and water intake have decreased by 50% or normalized
d. Start treatment on Friday as most notable effects seen within 7–10 days of the induction regimen
e. Dispense prednisolone (0.5 mg/kg) to mitigate any adverse effects (lethargy, anorexia, V/D)
f. Control achieved in well over 80% of cases noted by ACTH stimulation tests that remain within normal pre-stim levels post-ACTH stimulation
g. Relapses in 50% of dogs on maintenance therapy during the first year of treatment
h. Most common cause for relapse is being too conservative with the initial weekly maintenance dose
i. Should try to achieve 50 mg/kg weekly for maintenance
j. Addison’s disease develops in about 5%; Nelson’s syndrome may occur in 10% of patients

51. Trilostane:
   a. Synthetic steroid analogue competitively inhibiting 3β-hydroxysteroid dehydrogenase → reduces synthesis of cortisol and aldosterone; reversible and dose related
   b. 1-2.5 mg/kg q 12 h with food
c. Improvement of PU/PD/Pphagia within 4 weeks; hair regrowth within 2–3 months
d. Monitoring ACTH stim test at 0, and between 4 to 6 hours post-ACTH on day 10, 30, and 90 then every 4 to 6 months thereafter with the goal post-ACTH = 50–150 nmol/L 4 to 6 h post-trilostane. If post-ACTH > 250 nmol/L double the dose; if post-ACTH < 20 nmol/L, discontinue trilostane for 2 days, then return to the next lowest pill size.
e. Adverse effects: lethargy, decreased appetite, mild electrolyte changes, adrenal cortical necrosis, Addison’s disease even after a single dose, Nelson’s syndrome
f. Consider using prior to adrenalectomy and/or to treat adrenal tumours

52. L-deprenyl/Selegiline HCl (Anipryl®):
   a. An irreversible inhibitor of monoamine oxidase-B of the brain causes decreased ACTH production
   b. Initially reported to produce partial to complete improvement in signs in 83%
   c. Others have reported only 20% success rates most likely because this is the incidence of pars intermedia-based Cushing’s syndrome
d. May have suppressive effects on the growth of pituitary masses
e. Minimal side effects therefore indicated for milder cases of Cushing’s syndrome
f. Monitoring is solely based on clinical signs

53. Ketoconazole:
   a. Interferes with steroidogenesis by preventing cholesterol side chain cleavage/17 α-hydroxylase activity
   b. Helpful in 50–67% of either PDH or AT
c. Side effects are uncommon (anorexia, diarrhea, elevated liver enzymes)
d. High cost + risk of hepatotoxicity + daily administration

54. Retinoid therapy:
   a. Retinoic acid (isotretinoin) decreases corticotrophin secretion in vitro and in vivo in rodents
   b. Recently used to treat pituitary dependent Cushing’s @ 2 mg/kg q 24 h per os
c. Clinical signs, plasma ACTH levels, and UCCR improved + size of pituitary adenomas decreased
d. No adverse events or signs of hepatotoxicity
55. **Gefitinib:**
   a. Tyrosine kinase inhibitor targets the epidermal growth factor receptors (EGFR)
   b. EGFR is overexpress by some pituitary adenoma

56. **Radiotherapy:**
   a. Pituitary macro-tumors (> 1 cm in diameter) ± micro-tumors
   b. With or without concurrent neurological
   c. ACTH production may take many months to decrease

57. **Transsphenoidal hypophysectomy:**
   a. Effective if CT or MRI ± an experienced surgeon
   b. One-year survival was 84%; 2-year estimated survival 80%; recurrence rate was 11.6%
   c. Treat with desmopressin transiently and thyroid hormone indefinitely
   d. Offers a potential cure for the disease or at least debulk for radiotherapy

58. **Bilateral adrenalectomy:**
   a. High morbidity and mortality
   b. Currently therapy of choice in cats if minimal response to trilostane

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**Hypothyroidism**

Hypothyroidism has been described as one of the most common endocrinopathies in veterinary medicine. This in part, is most likely due to the over interpretation of baseline thyroxine (T4) concentrations. As well, clinical signs of hypothyroidism may resemble nonthyroidal illnesses (NTI), which in turn may suppress serum thyroxine levels, leading to misdiagnosis of the disease process. The ability of diagnostic thyroid evaluations to differentiate between the hypothyroid and euthyroid-sick individual in veterinary medicine is becoming more refined. One must, however, always remember to combine results of thyroid tests with clinical impressions to obtain a diagnosis of canine hypothyroidism.

**Clinical Signs**

The most common reason for presentation is the classic clinical sign of alopecia that tends to be bilaterally symmetric, from localized to generalized, predominantly involving the ventrolateral thorax and abdomen, the dorsal muzzle proximal to the planum nasale. Not always present, but when observed a hyperpigmented and alopecic “rat’s tail” that is typically noted in the latter stages helps support a diagnosis of hypothyroidism.

Hyperpigmentation with macular lentigines involving the trunk, orthokeratotic hyperkeratosis (including of the peri-pinnal margin) resulting in adherent crusts, seborrhea oleosa, or seborrhea sicca with lightening of coat color are other cutaneous findings. Often chronic recurrent infections including bacterial pyoderma, ceruminous otitis externa, and adult-onset demodicosis can be attributed to hypothyroidism. Myxedema, comprising of increased deposition of dermal mucin, results in a swollen skin appearance, which is typically cool to the touch, and provides the classic “tragic facial expression.” Acral lick granulomas are often associated with hypothyroidism as a result of the myxedema causing a “phantom limb” sensation along with a compromised local immune defense system resulting in protracted secondary infections.

Other noncutaneous clinical signs that suggest hypothyroidism include:

59. GI signs - constipation or diarrhea
60. Cardiovascular signs - bradycardia, decreased blood pressure, decreased contractility, atherosclerosis, cardiomyopathy
61. Reproductive changes - anestrus, irregular cycles, stillborn puppies, azoospermia, and decreased libido
62. Neurologic signs - megaesophagus, bilateral laryngeal paralysis (voice changes), muscular atrophy & weakness, polyneuropathy (i.e., facial and vestibular nerve involvem as depicted by a droopy eye lid and lip, along with a head tilt, myxedema coma, and hypertrophy of slow twitch/atrophy of fast twitch fibers)
63. Ocular - corneal lipid deposits
64. Anterior cruciate ligament ruptures as a result of ligament laxity
A high index of suspicion for hypothyroidism is based on signalment (middle-aged [6 years]; medium- to large-breed dogs), historical findings including heat seeking, weight gain, lethargy, and weakness, consistent dermatologic lesions, and physical exam findings of a decreased heart rate and decreased rectal temperature. Unlike Cushing’s syndrome, a definitive diagnosis can be achieved by routine blood work. Clinicopathologic changes may include a normocytic/normochromic anemia, elevated cholesterol & triglyceride, elevated creatine phosphokinase, urinalysis with increased WBC due to secondary to a bacterial cystitis, and increased activated clotting time and partial thromboplastin time. Thyroid tests of value are the total T4, free T4, and TSH assay as primary diagnostic tools. A low T4, low free T4, and elevated TSH is a classic hypothyroid profile and provides 99.9% certainty. The thyroglobulin autoantibodies may be used as an adjunctive test to help support an early-onset hypothyroid condition.

Treatment of concurrent diseases such as bacterial pyoderma, demodicosis, Malassezia dermatitis, and seborrhea is advocated as any residual concurrent diseases may alter the response to thyroid supplementation. Thyroid supplementation products on the market can vary in their bioavailability, formulation, and consistency. Therefore, use of products with proven pharmacokinetic research is recommended to start with before considering a generic version. The brand names I tend to rely upon include Synthroid®, Soloxine®, and Thyro-Tabs®. Dosing is easily calculated as 0.1 mg per 10 lb twice daily. I typically do not exceed 0.8 mg BID in a large-breed dog, as our dose calculations should ideally be based on body surface area (0.5 mg/M²). Twice daily (versus once daily) administration is key to elevating the local immune response, increasing metabolic activity that will help with energy levels (within 7–14 days), hair regrowth/skin integrity (within 4–6 weeks), muscle development and weight loss, and improve ligament strength to minimize anterior cruciate ligament rupture (within 3–6 months). In more mature animals with heart conditions, I will compensate by gradually tapering my dose upwards to avoid cardiac decompensation.

Monitoring is achieved by serologic evaluation of a 6-hour post-pill T4 at 4 to 6 weeks after the initiation of therapy. Ideal peak supplementation levels should fall within 10% of the upper-normal reference range. Once ideal post-pill supplementation levels have been achieved, I recommend rechecking 6-hour post-pill T4 levels every 6 to 12 months throughout the patient’s life. If suboptimal thyroid supplementation is noted on appropriate doses, consider one or more of the following: improper administration, incorrect dosage, poor bioavailability of the brand of synthetic thyroxine, improper diagnosis, or concurrent condition exists (euthyroid sick syndrome; ESS). ESS is a condition whereby the thyroid is still functional, but its activity is decreased due to nonthyroidal illnesses such as chronic renal failure, Cushing’s, Addison’s, diabetes, hepatopathies, and chronic skin disease. The severity of the illness is directly correlated to severity of serum T4 suppression. Medications may also cause falsely lowered thyroid values and result in misinterpretation of the results. Drugs such as glucocorticoids, phenylbutazone, anticonvulsants, anesthetics, long-term sulfonamides, and radiocontrast dyes all may either inhibit TSH secretion or serum thyroid hormone synthesis, release, binding or conversion, or they may impair thyroglobulin iodination and coupling of tyrosinases. Therefore ESS must always be taken into consideration when a complete response to thyroid supplementation has not been achieved within 3 months.

Alopecia X of the Nordic Breeds

Also known as pseudo-Cushing’s, adrenal sex hormone imbalance, congenital adrenal hyperplasia-like syndrome, Lysodren-responsive dermatosis, follicular dysplasia of Nordic breeds, growth hormone deficiency, hyposomatotropism, growth hormone-responsive alopecia, biopsy-responsive alopecia, post-clipping alopecia, castration-responsive dermatosis, gonadal sex-hormone alopecia, sex-hormone/growth-hormone dermatoses, Siberian husky follicular dysplasia, and follicular growth dysfunction of the plush-coated breeds, the term alopecia X encompasses multiple dysplastic follicular conditions that potentially have many underlying pathogenic mechanisms that are not completely understood with an end result of hair cycle arrest.

In general, there does appear to be a genetic predisposition to a hormonal/growth-factor imbalance systemically, or perhaps more likely at the level of the hair follicle receptors. Similar to congenital adrenal
hyperplasia-like syndrome in humans, Lothrop et al. (1990) proposed that a partial 21-hydroxylase adrenal enzyme deficiency resulting in overproduction of progesterone, 17-OH progesterone, androstenedione, estradiol, and other sex hormones was responsible for the changes seen especially in Nordic-breed dogs. Unfortunately, both affected and unaffected individuals may have similar changes in circulating levels pre- and post-ACTH stimulation. These post-ACTH findings are not unlike atypical Cushing’s syndrome noted in some dogs with normal cortisol results on screening tests for hyperadrenocorticism. Although polyuria and polydipsia are not typically noted in alopecia-X patients, it has been proposed that the bilateral symmetric alopecia may categorize these patients as atypical Cushing’s syndrome; after all, they do respond favourably to medications used to treat Cushing’s syndrome (trilostane, mitotane). Interestingly, treatment does not appear to alter the sex hormone profiles and in some patients, discontinuation of therapy without a relapse of clinical signs is possible. Lastly, several alopecia-X patients will respond to neutering, while others may resolve spontaneously, casting doubt as to whether this condition is mediated strictly by a circulating hormone imbalance and rather emphasizing the need for more focus on the effects of local hormones and growth factor levels on hair follicular arrest.

Clinical Signs
Typically, a nonpruritic, bilaterally symmetric alopecia with marked hyperpigmentation in later stages, alopecia X involves the neck, trunk, and tail, while sparing the head and distal limbs. Hair regrowth at areas of trauma is common. The mean age of onset is 2 years (9 months to 11 years) and is more frequently seen in intact or neutered male dogs. Miniature poodles and Nordic breeds with a plush coat are predisposed, in particular Pomeranians, Chow Chows, Keeshonden, Samoyeds, Malamutes, and Huskies. Diagnosis is made by ruling out other endocrinopathies and histopathologic findings consistent with catagen arrest/flame follicles. Evaluation of gonadal and adrenal sex hormones pre- and post-ACTH stimulation has questionable value, but is typically recommended if Lysodren or trilostane are being considered for treatment.

Treatment Options
65. Neuter and assess response within 3–6 months:
   a. Regrowth in up to 50 to 75% of cases
   b. Expected resolution for months to years post-neutering

66. Trial melatonin therapy:
   a. Oral melatonin - 1 mg/kg divided TID
   b. Injectable melatonin - 20 mg SQ q 2 weeks for 3 treatments then as needed
   c. Implantable melatonin - 3 constant-release implants of 12 mg each at one time
   d. A product of the multistep conversion of L-tryptophan → serotonin → melatonin
   e. Production is directly proportional to the length of the dark period
   f. Centrally stimulates the pulsatile LHRH activity from the hypothalamus
   g. Controls photoperiod-dependent molting and/or coat color via direct effects on hair follicles or within the pars tuberalis to alter secretion of melanocyte-stimulating hormone and/or prolactin
   h. Fifty- to sixty-percent response rates are noted within 3–6 months
   i. Discontinue once hair regrowth is noted so that the treatment can be used again in the future if needed

67. Trilostane:
   a. Inhibitor of 3-beta hydroxysteroid dehydrogenase
   b. Complete hair regrowth within 6 months
   c. Study: 14/16 Pomeranians and 8/8 Miniatures Poodles (@ 11 mg/kg)
   d. 3/3 Malamutes (@ 3.0–3.6 mg/kg)
   e. Hypoadrenocorticism, expense, death due to adrenal necrosis reported

68. Low-dose Lysodren therapy:
   a. Necrosis of zona reticularis & fasciculata of adrenal gland and usually spares zona glomerulosa
b. 15–25 mg/kg PO daily for 5 to 7 days’ induction
c. Maintain between normal resting cortisol levels both pre- & post-ACTH stimulation
d. May require 1 to 5 times per week dosing
e. Gradually lower the dosage once a full coat has returned
f. 50–100% response rates reported; good response documented in Chow Chows

69. Others:
   a. Leuprolide acetate - expensive anti-gonadotropic drug used if elevated estradiols
   b. Deslorelin implant (GnRH-agonist) - 4.7 mg/dog regrowth of hair within 2 to 4 months
      Adverse effects were not noted, other than a decreased testicular size in intact males
      (Cerundulo 2013 ESVD/ECVD).
   c. Dutasteride, osaterone - anti-androgenic that decreases estradiol and progesterone
   d. Cyclosporine prolongs the anagen phase of the hair cycle by inhibiting expression of
      protein kinase C thus stimulating hair follicle growth and hair fiber production.
   e. Low-level laser therapy - 5 min/administration twice weekly for a maximum of 2 months
      with a therapeutic laser with a cluster probe (470 nm/685 nm/830 nm). 4/7 received 16
      laser treatments and 3/7 only 10. Complete hair regrowth in 5/7 animals; 2/7 had an
      improvement in hair density/length. Biopsies revealed an increase in anagen hairs at
      treated sites (Olivieri 2013 ESVD/ECVD).

Canine Recurrent Flank Alopecia (CRFA)
Based on the breed predispositions (Airedale, Boxer, and Bulldog), CRFA is considered to be a genetically
influenced photoperiod-related melatonin deficiency resulting in the recurrent alopecia directly or
indirectly by its effects on prolactin, androgens, estrogens, and/or growth hormone.

Clinical Signs
CRFA is a non-scarring, initially cyclical alopecia of the thoracolumbar region that is usually bilaterally
symmetric, but can favour or affect one side only. Lesions are well demarcated often with marked
hyperpigmentation and most commonly seen in Boxers, Airedales, English Bulldogs, and Schnauzers of
either sex. Although it can affect any breed, CRFA seems rare in plush-coated Nordic breeds, German
Shepherds, and Cockers. As the condition may also involve the dorsum of the nose, base of the ears, base
tail, and perineum in Airedales, Golden Retrievers, Doberman, and Giant Schnauzer, hypothyroidism
and Cushing’s syndrome must be considered strong differentials, especially on nonseasonal patients.
Most often, however, both the early mean age onset of 4 years (8 months to 11 years) and the seasonality
of the hair loss (November or March north of the 45th parallel) with spontaneous complete regrowth,
move the progressive endocrinopathies lower on the rule-out list. As well, 20% of cases may have only
one isolated episode, and some may skip a year. The alopecia may worsen from year to year, eventually
becoming permanent. When regrowth is noted, it may vary from a normal return of hair to coat color
changes (darker to aurotrichia). A classic history of recurrent hair loss is typically sufficient to diagnose
this condition. Histopathologic findings of a follicular dystrophy (“witch’s feet”) will help support the
diagnosis.

Treatment Options
70. Benign neglect
71. Trial melatonin therapy:
   a. Oral melatonin - 1 mg/kg divided TID
   b. Injectable melatonin - 20 mg SQ q 2 weeks for 3 treatments, then as needed
   c. Implantable melatonin - 3 constant-release implants of 12 mg each at one time
   d. On subsequent years, treatment should be initiated one to two months before expected
      onset of alopecia
   e. Apparent success rate ~ 50–75%

Canine Pattern Alopecia (CPA)
CPA is a relatively common disorder resulting in alopecia of the postauricular regions, ventral neck,
thorax, abdomen, and caudal medial thighs. The age of onset is typically at 6 months with gradual
progression over months to years. Although primarily a condition of the Dachshund, CPA may also be seen in Chihuahuas, Miniature Pinschers, Whippets, Greyhounds, Boston Terriers, and Boxers. Diagnosis is based on history, dermatologic findings, ruling out other endocrinopathies (HAC, estrogen-responsive dermatosis, congenital hypothyroidism) and dermato-histopathologic evidence of miniaturized hair follicles.

**Treatment**

72. Trial melatonin therapy
   a. Oral melatonin - 1 mg/kg divided TID
   b. Injectable melatonin - 20 mg SQ q 2 weeks for 3 treatments then as needed
   c. Implantable melatonin - 3 constant-release implants of 12 mg each at one time
   d. Hair growth noted within 6 weeks; maximum growth in 3 to 4 months
   e. 60–70% response rate

**Post-Clipping Alopecia**

Protracted alopecia is sometimes noted after preparation for a surgical or epidural procedure. The lower back and dorsal pelvic region tend to be predisposed and often present with hyperpigmentation pending the duration. This syndrome is seen most commonly in long-coated breeds (e.g., “plush-coated” breeds such as Samoyed and Chow Chows, German Shepherds). Similar to alopecia-X patients, tufts of hair may be seen to regrow at sites of trauma. It is thought that the alopecia results from decreased vascular perfusion/vasoconstriction in response to decreased skin temperatures at the sites of hair clipping.

Another theory is that the hair that was clipped happened during a normal catagen stage of hair growth, and that the time to regrow hair is dependent on the proximity of the anagen phase of the hair cycle. Therefore, with benign neglect, it may take 6 to 24 months for hair to regrow.

**Treatment Options**

73. Pentoxifylline:
   a. Phosphodiesterase inhibitor that promotes increased rheologic activity, hence increased blood flow and nourishment of hair follicles
   b. 10–30 mg/kg TID for a minimum of 3 months
   c. Caution with natural or chemically induced blood dyscrasias

74. Cyclosporine:
   a. Prolongs the anagen phase of the hair cycle by inhibiting expression of protein kinase C thus stimulating hair follicle growth and hair fiber production
   b. 5 mg/kg daily until hair regrowth, then discontinue

75. Thyroxine supplementation:
   a. Stimulate hairs to nonspecifically move into the anagen (growth) phase of the hair cycle
   b. Acquire baseline thyroid values, then administer 0.05 to 0.1 mg/10 lb once to twice daily to stimulate anagen growth
   c. Supplementation for 3 months will not result in permanent thyroid shutdown

76. Low-level laser therapy - see under alopecia X above (Olivieri 2013)

**Bald Belly Bald Thigh Syndrome (BBBTS)**

BBBTS is a nonpruritic, noninflammatory alopecia that affects the caudal aspect of the pelvic limbs and can progress to involve the abdomen, often noted in breeds with very little subcutaneous fat such as greyhounds and whippets. The hair loss may be caused by decreased oxygen tension at the skin as a result of muscle to skin compression of superficial blood vessels with very little subcutaneous fat to act as padding, in essence, and ischemic folliculopathy. Ruling out other endocrinopathies, in particular hypothyroidism, is important in these patients to help confirm a diagnosis.

**Treatment Options**

77. Pentoxifylline:
   a. 10–30 mg/kg TID for a minimum of 3 months before tapering the dose
b. Other ischemic folliculopathies that might be considered pentoxifylline candidates include: traction alopecia, elbow/hock calluses, and deep scarring pyoderma/pododermatitis

78. Melatonin:
   a. 1 mg/kg divided TID for a minimum of 3 months before adjusting the dose

79. Thyroxine:
   a. 0.05 to 0.1 mg/10 lb BID as a nonspecific anagen hair growth stimulator
   b. Discontinue at 3 months regardless of whether or not hair growth is noted

80. Cyclosporine:
   a. Prolongs the anagen phase of the hair cycle by inhibiting expression of protein kinase C thus stimulating hair follicle growth and hair fiber production
   b. 5 mg/kg daily until hair regrowth, then discontinue

81. Soft, warm bedding, and LLLT:
   a. To minimize compression of the superficial blood vessels and promote increased circulation

Color-Dilution Alopecia (CDA)
CDA is the result of the dilute mutation causing abnormal clumping of melanin within the hair shaft with subsequent fracturing and alopecia. Age of onset is typically between 3 months to 3 years. Predisposed breeds include blue/fawn/red-coated Doberman Pinschers, Great Danes, Whippets, Dachshunds, Standard Poodles, Chow Chows, and fawn Irish Setters. The patients are normal at birth and then progress gradually to develop a dry and dull coat with partial to complete alopecia on the dorsum of the trunk. Seborrhea, comedones, and secondary bacterial folliculitis often present in affected areas. Aggressive bathing may hasten the presentation of affected individuals. Diagnosis is based on signalment, trichoscopy, and dermato-histopathologic findings of melanin clumping within the epidermis of the hair follicle, increased melanophagic activity, and varying degrees of follicular dysplasia.

Treatment Options
82. To help minimize scale and seborrhea - oral retinoids (vitamin A, isotretinoin, acitretin), omega-3 and -6 fatty acids, epidermal barrier repair products (Allerderm Spot-On, Douxo Antiseborrheic pipettes)
83. To help address secondary infections - topical and/or oral antibiotics
84. To help improve hair regrowth - melatonin

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Suggested Reading

General Hair Loss

Cushing’s Syndrome

**Alopecia X**

**Canine Recurrent Flank Alopecia**

**Canine Pattern Alopecia**
What’s New in the Treatment of Otitis Externa in Veterinary Dermatology?
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Overview of the Issue
Otitis externa (OE) is defined as inflammation of the external ear canal and is a common condition in small animals (4.5–20% of all cases).1,2 OE is due to primary, predisposing and perpetuating factors. Primary factors account for the underlying etiology; predisposing factors are present prior to the development of the otitis; and perpetuating factors occur as a result of the inflammation and include fungal and bacterial infections.1 The most commonly isolated pathogens in cases of infectious canine OE are Staphylococcus pseudintermedius, Pseudomonas aeruginosa, and Malassezia pachydermatis.1

Objectives of the Presentation
107. To review the latest treatment options available for otitis externa
108. To review tips to help manage patients with otitis externa

Diagnosis of Otitis Externa
Diagnosis of OE is based on history, clinical signs, otoscopic examination, cytology and possibly cultures from the external ear canal. In any case of OE, cytology must be performed to diagnose any secondary infections.1 There is discrepancy between studies as to how many organisms constitute an infection versus normal flora. For gram-negative rods, even small numbers can constitute an infection as they are not usually seen within the ear canal.3,4 When inflammatory cells are noted on cytology, this is a significant finding and the number of organisms is irrelevant.1 In cases where gram-negative rods are visualized, a bacterial culture may be recommended, as well as in cases where previous antibiotics have been ineffective.3,5 If an oral antibiotic is selected, especially for cases of otitis media, these culture results will guide the choice of oral antibiotic. With OE, there is debate as to whether systemically administered antibiotics will reach the desired concentration within the external ear canal. Unless the ear canal epithelium is ulcerated, systemic antimicrobials are unlikely to reach therapeutic concentrations and should be reserved for cases of suspected otitis media.6 For most cases of otitis externa, topical therapy will be used. Response to topical medication does not often correlate with susceptibility testing results due to the fact that topical medications will reach much higher concentrations within the ear. Therefore, the choice of topical antimicrobial should not be based solely on the culture results.6 There is some discordance noted between otic cytology and culture results, so always interpret findings in light of cytological findings and clinical signs.7 A thorough otoscopic examination is also an important diagnostic tool.

Treatment of Otitis Externa
If medications are to be given topically, they must be able to reach the surface of the ear canal. In cases where excessive debris is present, flushing of the ear canal to remove exudate and allow administration of topical products is warranted.8 At-home ear cleaners can be prescribed for patients, with instructions to properly clean the ear canal. There are many products available, including those with ceruminolytics, antiinflammatories, and antimicrobials - some of which may be the only treatment required.9

Multiple ear medications are available, and many contain antibiotics, antifungals and antiinflammatory agents simultaneously. Antimicrobials for canine OE are most often selected empirically based on otic cytology.6 Current debate over the application of first- and second-line antimicrobials is ongoing. When cocci are noted on cytology, antimicrobials with action against coccoid bacteria are appropriate. When rods are noted on cytology, otic medications including gentamicin, polymyxin B and enrofloxacin can be considered for treatment.5 A topical ticarcillin preparation was found to successfully treat Pseudomonas otitis in a small numbers of cases.10 Tromethamine edetate disodium dihydrate (TrizEDTA® Aqueous Flush, Dechra Veterinary Products, KS, USA) is commonly used as an adjunct therapy for dogs with Pseudomonas otitis.11 It has been documented that flushing the
canal with TrizEDTA® 15 minutes prior to the application of a topical antimicrobial agent is beneficial in resolving gram-negative infections. Studies also suggest that a chelating agent may be beneficial in cases of Malassezia OE. Neomycin and gentamicin may not always be successful in clearing OE, as they are inactivated in purulent material, so may be best selected when there is scarce pus within the ear canal. For acute Pseudomonas OE, polymixin B is an appropriate choice. For chronic cases, topical fluoroquinolones or aminoglycosides may be required.

For treatment of Malassezia otitis, clotrimazole, miconazole and nystatin are commonly found in otic medications. Resistance to antifungals has only rarely been reported, and there is little evidence for in vitro antifungal resistance. Antifungal medications can be compounded to contain solely an antifungal if the need arises.

For inflammatory OE, topical glucocorticoids such as fluocinolone can aid in decreasing inflammation (Synotic®, 0.01% fluocinolone, 60% DMSO, Zoetis). In one study, a 0.1% Tacrolimus solution was instilled into the ears of dogs without otitis and was well tolerated. With further research, this could be an option for inflammatory otitis.

For cases of chronic proliferative OE, consider Triamcinolone injections as a salvage procedure prior to total ear canal ablation.

Bacterial Biofilms and Otitis
A bacterial biofilm is a community of sessile bacteria that form layers of planktonic bacterial cells and then become irreversibly attached to a surface. The biofilm produces a matrix (extra-polymeric substance, EPS) made of carbohydrates, proteins and DNA. This protects the bacteria from desiccation, the host immune response and antimicrobials serving to increase antimicrobial resistance and immune-system evasion. A recent study shows 40% of Pseudomonas otic isolates do form biofilms and once a biofilm has formed, this increases the MIC against certain antimicrobials. As with planktonic cells, studies have shown that TrizEDTA® used in combination with an antimicrobial can reduce the MIC and minimum bactericidal concentration (MBC) for certain antibiotics against biofilm-embedded bacteria.

Treatment Tips
109. In cases of OE with stenotic ear canals, antiinflammatory therapy with oral prednisone or dexamethasone may be needed for 7-14 days before otoscopic examination or treatment with topical medication is successful. Most cases of OE will benefit from either topical or oral antiinflammatory treatment with glucocorticoids.

110. Ceruminolytic ear cleaners should be used for cases with excessive cerumen accumulation.

111. Demonstrate to clients how to clean the ear and apply medication. Rechecks every 2-4 weeks depending on length of otitis and treatment selected. At these rechecks, repeat cytology should be performed to determine whether treatment is working. Continue treatment until clinical and cytological resolution.

112. Often owners apply 1-2 drops into ears; must be stressed that this volume is not enough to coat ear canal. Consider using ml volume instead of drops, e.g., 0.5 ml for medium dog, 1.0 ml for large dogs.

113. Always address underlying etiology.

REFERENCES


What’s New in the Treatment of Bacterial Pyoderma in Veterinary Dermatology?
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OVERVIEW OF THE ISSUE
Bacterial pyodermas can be frustrating to treat due to underlying etiologies causing inflammation and increased susceptibility to infection, as well as the inherent defense mechanisms of bacteria including toxin production and encapsulation. The majority of bacterial pyodermas in dogs and cats are due to Staphylococcus pseudintermedius. Therefore, when considering antibiotic therapy one must select an antibiotic, either topical or systemic, with a mechanism of action suited to this bacterial species. S. pseudintermedius is a normal resident of the skin in dogs and cats and is likely acquired from the mother in the neonatal period. Bacterial infections may also be due to transient organisms such as Pseudomonas aeruginosa, E. coli, etc.

OBJECTIVES OF THE PRESENTATION
114. To review topical and systemic treatment options for both susceptible and resistant bacterial pyoderma
115. To review treatment options for recurrent bacterial pyoderma

DIAGNOSIS OF BACTERIAL PYODERMA
Clinically a superficial bacterial pyoderma will present with papules, pustules, epidermal collarettes and crusting. Bacteria invade the hair follicle causing a folliculitis, clinically apparent as circular lesions of alopecia. A deep bacterial pyoderma will present as erythematous nodules with potential draining tracts. In any animal suspected of having a bacterial pyoderma, cytology (skin swabs) must be performed. Cytology helps the clinician determine whether the infection is due to cocci (most likely Staphylococcus) or rod-shaped bacteria. Without knowing which bacteria are causing the pyoderma, we are unable to select appropriate antibiotic therapy. Staphylococci do not cause infection in the skin of healthy individuals unless there has been trauma to the skin. Therefore, any animal with a bacterial infection likely has an underlying metabolic or immunologic abnormality, such as allergic skin disease, endocrinopathies, predisposing to development of the bacterial pyoderma. With any case of secondary bacterial pyoderma, the underlying etiology should be addressed to prevent recurrence of the infection.

When to Perform a Bacterial Culture and Susceptibility
A bacterial culture and susceptibility should be performed: If rods are noted on the skin in large numbers
116. If there is a deep pyoderma (furunculosis)
117. If there is a mixed infection
118. If a resistant bacterial population is suspected

TREATMENT DURATION
Treatment of a superficial bacterial pyoderma should be a minimum of 3–4 weeks and at least 7–10 days past clinical resolution. Treatment of a deep bacterial pyoderma should last for 4–12 weeks.
TREATMENT OPTIONS FOR SUSCEPTIBLE BACTERIAL PYODERMAS

Topical Treatment
122. Antibacterial ointments/creams (applied daily)
   a. Mupirocin - poor activity against gram negative
   b. Polymixin B, bacitracin - inactivated by purulent material
   c. Neomycin - potential for contact dermatitis
   d. Fusidic acid, gentamicin, thioestrepton, silver sulfadiazine

123. Antibacterial shampoo (5- to 10-minute contact time q 2–7 days)
   a. Chlorhexidine 2–4%
   b. Benzoyl peroxide
   c. Acetic acid and boric acid - questionable efficacy
   d. Accelerated hydrogen peroxide

Systemic Treatment
S. pseudintermedius is a β-lactamase producer; therefore, antibiotics resistant to this enzyme must be selected. Cephalosporins (cephalexin, cefpodoxime, cefovecin), amoxicillin-clavulanate, and clindamycin are often considered as “first-line” antimicrobials and can be used as empirical therapy. Tetracyclines, fluoroquinolones, potentiated sulfonamides are also options for treating susceptible Staphylococcal pyoderma; however, issues with inherent resistance to tetracyclines and developed resistance to other antibiotics must be considered. Furunculosis or pyoderma due to gram-negative rods; therapy will be based on culture results. Any case of bacterial furunculosis must be treated with systemic antibiotics.

TREATMENT OPTIONS FOR RESISTANT BACTERIAL PYODERMAS
The most common resistant bacteria causing skin infections is methicillin-resistant Staphylococcus pseudintermedius (MRSP). Oral antibiotic selection is dependent on bacterial culture and susceptibility testing. Topical therapy may also be used and may be preferable in certain cases where the MRSP is resistant to multiple antibiotics. Standard bacterial susceptibility testing results do not apply to topical products, as they can reach concentrations of 100–1000 times that of their systemic counterparts. Chlorhexidine and antibiotic ointments can be efficacious. One study showed that acetic acid/boric acid was not efficacious in treating MRSP in vitro. Another study noted that topical therapy with miconazole was efficacious in treating MRSP pyoderma. For systemic treatment, the clinician may need to reach for antibiotics such as rifampin, chloramphenicol. Appropriate bloodwork must be run prior to and during therapy with these antimicrobials.

TREATMENT OPTIONS FOR RECURRENT BACTERIAL PYODERMAS
124. Human Interferon alfa-2b - given orally at 1000 IU/m/day
125. Staphage Lysate® (Delmont Laboratories) - Autogenous staphylococcal bacterin, S. aureus phage lysate
   a. Combat potential bacterial hypersensitivity
   b. 0.5 ml, SQ, twice weekly for 10–12 weeks (induction)
   c. 0.5 ml, SQ q 7–14 days (maintenance)

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How to Improve Your Equine Radiographic Technique in the Field: Parts I and II
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INTRODUCTION
Excellent radiographic technique is at least half the battle in obtaining an accurate radiographic diagnosis in our equine patients and providing clients with the highest-quality prognostic and treatment information. We are all familiar with the expression “no foot, no horse”… indeed the same principle applies to radiography: “lame image, lame diagnosis.”

With the advent of digital radiography, high-quality radiographs are much easier and faster to obtain. Practitioners are less hesitant to take a greater number of images of more patients, and the hassle of repeat visits for retakes has fortunately been eliminated for both the practitioner and the horse owner. Nonetheless, many basic radiographic principles still apply to digital radiography, and the potential for a greater number of projections can in fact increase radiation safety concerns. Because images are now more easily shared between practitioners, owners and potential sellers and buyers, medicolegal and image quality concerns must be better understood and more rigorously applied than ever.

Beyond technical errors, making a radiographic interpretation has other inherent sources of error in both perception of abnormalities and analysis of what those abnormalities mean.¹ Practitioners must first perceive or become aware of a variation in the radiographic image in order to consider it as a potential abnormality. Once a variation is seen, the practitioner must use all available knowledge and experience in order to analyse or interpret whether that variation truly represents an abnormality and what impact that abnormality can have.

MEDICOLEGAL CONSIDERATIONS
Patient identification standards are established by provincial/state veterinary licensing bodies. Typically each radiographic image must contain the following information: patient/owner name or other identification, patient case number, examination date and name of the practice/practitioner. Limb laterality (left/right) must be identified and, below the carpus/tarsus, lateral must be differentiated from medial and hindlimbs from forelimbs. Rigorous patient and limb identification has become much more important, as it can easily be lost during electronic transfer of images between practitioners and to owners.

Patient/limb identification must be included in the image in a non-modifiable manner. With conventional film/screen radiographs, this typically means using leaded tape or another type of imprinting mechanism prior to exposure/development. With digital radiographs, DICOM (Digital Imaging and Communications in Medicine) format is the legal standard developed in association with the American College of Radiology in humans and adopted/recommended by the American College of Veterinary Radiology. From a medicolegal standpoint, this image format ensures that patient identification is embedded within the image information and that any modification of that information is recorded. From an image quality standpoint, DICOM images are uncompressed and ensure the highest possible image resolution. Although many licensing bodies are lagging on highlighting the importance of this, when transferring images to another practitioner (including a specialist) for potential interpretation, the DICOM format should be used to ensure optimal image quality. It is not advisable to submit jpegs or digital photographs for interpretation. First, image resolution will likely be suboptimal. Second, these formats do not permit the viewer to easily modify image contrast and brightness, to zoom, invert or pan the images, thus limiting visualisation of more subtle abnormalities.

Using a standard patient identification system is essential for efficient image archiving, particularly when there are multiple veterinarians in the same practice. It can be very frustrating to try to locate
previous images on a patient when they are not stored in a central and easily accessible electronic location. An archival system should ensure that any subsequent examinations from the same patient be linked to a common form of patient identification (typically a patient identification number unique to that individual patient). It goes without saying that an archival backup is a must. Many companies now offer Web-based “cloud” image archiving and management systems. An additional consideration is the ability to store ultrasound images in the same location and patient file as radiographs.

**Radiation Safety**

Radiation safety principles have not changed substantially in the past 15–20 years. The ALARA (“as low as reasonably achievable”) principle still stands, and the practitioner must consider how best to reduce time (number of retakes and people involved) and increase both distance (cassette-holders, exposure switch) and shielding (collimation, thick walls and other barriers, lead aprons, thyroid-protectors and gloves) in order to protect him/herself and perhaps more importantly the personnel he/she works with. Each employee involved in taking radiographs is obliged to wear a dosimeter, and exposure rates must not exceed those authorised by Health Canada (typically 1 mSv per year, unless special permission is sought). Detailed radiation safety recommendations are available at Health Canada’s website under Safety Code 28: Radiation Protection in Veterinary Medicine at www.hc-sc.gc.ca/ewh- semt/pubs/radiation/91ehd-dhm151/index-eng.php. Practice owners are ultimately responsible for the radiation safety of anyone involved with the practice.

Where radiation safety concerns have changed in recent years is the fact that digital radiography can in some cases increase a practitioner’s exposure. Practitioners who use digital radiography tend to take more views on more patients than with film-screen radiography. This can potentially cancel out the benefit of the reduced number of retakes that comes with switching to digital radiography. Also, in order to avoid the poor signal quality that occurs with digital radiography underexposure (see below), it is sometimes recommended to slightly overexpose radiographs. This overexposure is not apparent on the image and is called “dosage creep” when it is abused. Specifically, it should be noted that computed radiography (CR) plates require a greater amount of radiation (up to 2 times more) to generate an equivalent quality image when compared with film-screen radiography. However, the opposite is true for the direct digital radiography (DR) plates used by most equine practitioners.

A final suggestion is to consider alternatives whenever possible to holding both the X-ray machine and the cassettes. In fact, the National Council on Radiation Protection and Measurements report on Radiation Protection in Veterinary Medicine recommends that “when practicable” the operator should stand at least 6 feet from the X-ray tube, in addition to wearing a lead apron. A significant amount of radiation leakage occurs from a typical portable X-ray machine, particularly the top and cathode sides of the machine; therefore, the parts closest to the operator’s hand and neck. Placing a lead glove on top of the machine, placing it on a block to elevate it to the required height or using an adjustable X-ray machine stand (several models are commercially available) are some alternatives. In addition, these suggestions also help stabilise the X-ray machine and reduce motion artifacts. Cassette-holders can take many forms, as long as they increase the distance between the holder and the cassette...even a long-handled pair of pliers is better than holding the cassette directly, and they are compact enough to carry in the ambulatory practitioner’s vehicle.

**Technical Considerations**

The environment in which an ambulatory equine practitioner may be required to take radiographs can vary greatly from one patient to the next. Adaptability, creativity and, above all, planning and organisation are a must! When planning to take radiographs, consider what personnel or other alternatives are available to hold the horse and the cassette. Taking radiographs in bright sunlight (not to mention driving rain or snow!) is made more challenging by the fact that the collimation cannot be seen. Judicious use of sedation can be a valuable timesaver. It is usually easier to move the cassette and machine rather than the horse, particularly if the latter is sedated, so plan to have enough room to manoeuvre and the order of the projections you want to take (i.e., start at dorsomedial and move to lateral). Also, fewer labelling errors are made by completing one region/limb before starting the next.
Finally, appropriate focal-film distance should be respected, and the cassette/panel should be held as closely as possible to the limb and aligned with the radiographic beam to avoid magnification and distortion.

Poor positioning is one of the most common sources of poor radiographic quality. The patient should be standing as squarely as possible with weight evenly distributed on all four limbs for most projections, in order to avoid distortion of the point spaces. The X-ray beam should be centered on the specific joints of interest. For example, when evaluating for distal tarsal osteoarthrosis (bone spavin), centering the beam on the distal tarsus rather than the talus or calcaneus will minimise joint space distortion. In more extreme cases, the horse’s conformation may need to be adapted to. For example, if a horse is particularly “cow-hocked,” the X-ray tube may need to be angled slightly distally (downward) in order to properly project the intertarsal and tarsometatarsal joint spaces. In very “toed-out” horses, the heel bulbs can be used to direct the X-ray beam. As a result, the angles required for DP and LM views of the foot will appear quite different from those for the fetlock or carpus/tarsus, and may seem oblique relative to the horse’s upper limb.

Other aspects of patient preparation are also important. Ensure that the horse is clean of any dirt or other debris. Mineral debris, the ergot (fetlock) and chestnuts (carpus/tarsus) can all mimic osseous fragments. Wet hair, bandage material and iodinated cleansers create visible soft-tissue or mineral opacity lines on the radiograph that can mimic or hide abnormalities. Not only should the hoof be packed for radiographs of P3 and the navicular bone, but sometimes the frog must be trimmed and cleaned first in order to remove deeply embedded debris. Although it can be a hard sell, shoes should be removed for P3 views, especially oblique projections. If a known foreign body (i.e., a nail) can be safely left in place, it is better to take radiographs prior to removal, such as to better determine what underlying structures may be affected.

It is beyond the scope of this presentation to enumerate which views should be made for which region and many texts list standard radiographic projections. That being said, it is helpful to have a preliminary idea of potential differential diagnoses. For example, oblique projections for detecting medial malleolar OCD in a young horse are not taken at quite the same angles as for an older horse in which osteoarthritis is suspected (D30°L-PM oblique vs D45°L-PM oblique). Oblique projections of the fetlock need to be selected according to whether dorsal or plantar/palmar fragments are suspected (D10°L-PM oblique vs 30°D45°L-PM oblique). In order to detect certain fractures, projections of various obliquities must be taken. In addition to the typical 4-view, radiographic examination, many alternative projections exist for specific pathologies. Finally, injecting iodinated contrast medium into a wound or draining tract (fistulogram) can help detect foreign bodies or rule out/in osseous involvement.

With conventional film-screen radiography, over and underexposure are major sources of technical errors. Overexposure darkens the image such as to “burn out” the valuable information that can be gleaned from the soft tissues or thinner osseous structures (i.e., mineral fragments, gas, distal splint bone fractures, hoof wall vs. P3). Excessive image contrast (a very “black and white” image) can have the same effect. Underexposure lightens the image such that abnormalities within the thicker osseous structures (i.e., fracture lines, osteolysis, cysts) can be hard to differentiate from normal surrounding bone. Inadequate image contrast has a similar effect.

Although digital radiography is more forgiving, over and underexposure have a similar impact on image quality. Overexposure (also called saturation) appears similar to conventional radiographs with soft tissues and thinner osseous structures being completely blackened. Underexposure (also called quantum mottle or inadequate signal-to-noise) appears a little differently: the radiograph will have a particularly grainy appearance such that even the edges of bony structures become ill-defined. In digital radiography, the exposure index can be helpful to determine if exposure was adequate, but nonetheless has limitations. Digital radiography systems must be properly calibrated and equipped with specific algorithms (or look-up tables, LUTs) that have been developed for equine patients. The algorithm essentially determines how the image will appear: whether or not the osseous structures are highlighted vs. the soft-tissue structures, etc. For this reason, the appropriate image reconstruction algorithm must be used for the appropriate anatomical region. Finally, even in digital radiography a low kVp relative to
mAs will provide better bone detail than a relatively high kVp. Tighter collimation also improves radiographic detail by limiting scatter radiation, even in digital radiographs.

A final source of poor image quality and potential diagnostic error is poor image resolution. Digital radiographic images have very high inherent spatial resolution, usually being upwards of 16 megapixels. Image resolution is therefore primarily limited by monitor resolution. In an ambulatory practice environment, images are immediately available and evaluated on a laptop computer screen, usually the one provided by the digital radiography vendor. Not only are these screens small, but they are often of both poor resolution and brightness. In addition, image viewing conditions can be suboptimal due to excess ambient lighting. It is advisable to reevaluate radiographic images using a larger, high-resolution monitor adjusted to the highest possible brightness.

PERCEPTION ERRORS
Perception errors are of 2 types: 1) a variation in the typical radiographic appearance of a structure is present, but is not identified (false negative) or 2) normal radiographic appearance is perceived as being an atypical variation (false positive).

Certain types of perception errors arise from the fact that a 3-dimensional structure is being projected as a 2-dimensional image. Classic examples are summation, silhouetting, magnification and distortion and Mach lines.

Perception errors can also arise from poor or incomplete search habits and include “satisfaction of search” (the natural tendency to stop looking for other abnormalities once a lesion is found), lack of a systematic approach and searches guided by excess clinical bias. Although experienced practitioners appropriately orient their search for radiographic lesions according to a certain amount of clinical bias (i.e., looking for OCD in a young racehorse with tarsocrural effusion), it helps to not completely abandon the systematic approach that we were taught as students so as not to miss the occasional “zebra” diagnosis.

Our visual system is easily overloaded, which can also cause perception errors. Examples are trying to interpret too large a number of radiographic images simultaneously (i.e., the prepurchase exam), an inconsistent “hanging protocol” (orientation in which each radiographic image is projected), or evaluating an unfamiliar radiographic area or one with a lot of anatomic “structured noise” (i.e., skull, teeth). Taking the radiographs in a logical and organized order (i.e., region by region and each limb separately, in separate exams) usually means that they are available for viewing in that order, which helps in evaluating one region at a time and reducing visual overload. Orienting each view consistently helps the brain develop familiarity with normal radiographic appearance. Beyond these good habits, using zoom/pan features, modifying window width and level (i.e., contrast and brightness), or modifying viewing distance and using a hot lamp also help detect subtle lesions. Backlighting or glare on the computer monitor should be avoided, and ambient lighting should be dimmed but not completely turned off.

ANALYSIS ERRORS
Once a radiographic “variation” has been perceived, the practitioner must analyse what that variation means. Is it normal or abnormal? If it is abnormal, what does it mean clinically for the patient? Similar to perception errors, false negative and positive analysis errors occur.

Common errors are over- or under-interpretation, which can be influenced by various biases, including a desire to obtain a diagnosis and meet an owner’s expectations. Inexperienced practitioners can be particularly prone to over-interpretation, as can experienced practitioners when evaluating an unfamiliar anatomic region or new radiographic projection. Being conscious of potential clinical biases is important. Revising the clinical history with the owner/trainer relative to new or unexpected findings can sometimes reveal details that were not initially reported. Finally, obtaining a colleague’s second opinion (made much easier with digital radiography) is often invaluable.

Knowledge of radiographic anatomy and the plethora of normal (i.e., crena on P3, various nutrient foramina, osseous spur dorsoproximal 3rd metatarsus) and age-related (i.e., physis of the styloid process in foals) variations are fundamental. Radiographs of the contralateral limb or a nonclinical individual can
be helpful. Also, comparison of lesion evolution from previous exams is important in assessing clinical relevance of a radiographic abnormality.

**Radiographs and the Prepurchase Exam**

A question that often arises is the role of radiographs and other imaging modalities in the prepurchase exam. As mentioned above, any imaging examination needs to be appropriately identified as to patient and region (limb, laterality). Also, images should be made available in DICOM format. Imaging, even radiographs, is not mandatory to the prepurchase examination. In fact, when imaging examinations are made without clinical abnormalities, interpretation of their clinical relevance can be difficult. An increasing volume of literature is available addressing the wide variety of normal variations (i.e., number of distal navicular synovial invaginations) and nonclinical abnormalities (i.e., mild distal phalanx pedal osteitis) that can be seen on radiographs and various other imaging modalities. Assessing future impact of certain abnormalities (i.e., distal tarsal osteoarthrosis, mild navicular degenerative changes) can be impossible without a crystal ball, particularly in young horses that have not been worked to their intended level. In addition, individual variation can be large: two different horses with radiographically similar findings can appear and evolve very differently from a clinical standpoint. This reality can be hard for potential buyers to understand.

Should radiographic evaluation be elected, specific regions and projections should be selected following a thorough discussion regarding the horse’s age, prior use, intended use (discipline and level) and, perhaps most importantly, the potential for resale. No matter the region evaluated, complete radiographic examination (typically 4 views) should be made. Radiographic evaluation can also be limited to regions where a clinical indication is present (i.e., positive hindlimb flexion, tarsocrural effusion). As a final consideration, permission needs to be obtained from the owner/seller to sedate the horse as needed.

Obviously the prepurchase examination report should include a description of the specific imaging examinations/regions that were requested or declined and a thorough radiographic interpretation of each region.

**References**

Radiographic Diagnosis of Equine OCD
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WHAT’S IN A NAME? OSTEochondROSIS VS. OCD VS. DOD
Osteochondrosis is a disease that primarily affects juvenile animals and is characterised by a disruption of the normal stages of endochondral ossification. Endochondral ossification is the process of long and cuboidal bone formation, whereby the bones go through an initial cartilage scaffold before becoming ossified. By the time an animal is born, the process is still active at the growth plates (physes) and epiphyses/apophyses, where it permits the bones to lengthen and thicken according to the animal’s growth and maturation. It is also to some extent still active in the cuboidal bones of the tarsus/carpus.

In osteochondrosis, the process of cartilage ossification is interrupted, either at the growth plate or at the epiphyseal level (at the interface between epiphyseal bone and articular cartilage), and growth cartilage is abnormally retained. Osteochondrosis is therefore in some ways a fairly general term. However, because of the limited number of ways that bone and cartilage respond to a variety of pathological insults, other types of osteochondral lesions involving growing cartilage can potentially resemble osteochondrosis. Some authors consider that the term “dyschondroplasia” would more accurately describe what occurs during the disease. Other authors believe that only a “primary” failure of endochondral ossification should truly be considered to be osteochondrosis. Even more confusing is that histological definitions of osteochondrosis lesions vary and the underlying predisposing factors are numerous. Among the factors that have been shown to be associated with osteochondrosis are genetics, nutritional, biomechanical/conformational, exercise-level, traumatic, vascular, etc.

Osteochondritis dissecans (OCD) is a specific form of osteochondrosis within the epiphysis/articular cartilage whereby a cartilage “core” is retained in the subchondral bone during epiphyseal ossification, undergoes necrosis, and creates a fissure within the articular surface. These lesions are typically found in high-motion areas of the joint, and it is thought that they become apparent through repeated sliding of rotational motion of the joint (i.e., femoral trochlear ridges). OCD lesions also seem to develop in areas where cartilage ossification occurs last (i.e., medial malleolus). There are specific sites of predilection for these lesions, and they are often bilateral, which supports a genetic predisposition, particularly in the tarsus. In the horse, the tarsus, fetlock and stifle are most common, with the shoulder and vertebral articular processes occasionally being affected.

Subchondral bone cysts are widely considered to be another form of osteochondrosis that also occur within the epiphysis/articular cartilage, but in areas of high loadbearing. The medial femoral condyle is the most common example in the horse. Subchondral bone cysts and osseous cyst-like lesions also occur in the phalanges, tarsus, fetlock and elbow. Some of these may be manifestations of osteochondrosis, while others may be mechanical or traumatic in origin.

Developmental orthopedic disease (DOD) is a broad umbrella that includes osteochondrosis/OCD and other bone/cartilage lesions that have not or not yet been shown to represent a failure of endochondral ossification.

WHY RADIOGRAPHS?
Radiographs remain the most widely available and cost-effective way to detect osteochondrosis/OCD. In most sites, the subchondral bone defect and surrounding sclerosis caused by focal failure of endochondral ossification is readily apparent, in addition to the overlying, partially mineralised osteochondral fragment. Some exceptions exist, such as the shoulder and the medial malleolus of the tibia, where radiographic detection is more challenging.

OCD lesions can be seen radiographically in foals as young as 3 (tarsus) to 5 (stifle) months of age. Prior to that, incomplete ossification or the appearance of active mineralisation of the epiphysis at the junction with the articular cartilage can mimic or hide OCD lesions. This is particularly true of the femoral trochlear ridges, which are highly irregular and ill-defined on radiographs around 4–5 months of
age. Because of the chronology of articular endochondral ossification, OCD lesions should already be present by 7–8 months of age. Spontaneous resolution of OCD lesions can occur, but typically not after 8–10 months of age. An exception to this may be sagittal ridge OCD, which can resolve at a later age when managed conservatively.

Because OCD lesions are often bilateral, radiographs of the contralateral limb are recommended; clinical signs are not always present, particularly in young horses that have not yet begun training. In the fetlock, OCD of the sagittal ridge can affect all four limbs.

**Tarsus**

OCD in the tarsus occurs in 3 classic locations: distal intermediate ridge of the tibia, lateral trochlear ridge of the talus, and medial malleolus of the tibia. Rarely, OCD lesions have been reported involving the medial trochlear ridge of the tibia and lateral malleolus. Caution should be made when evaluating the medial trochlear ridge: focal dorsal flattening and small distal smoothly marginated “teardrop fragments” are considered to be incidental findings. At the lateral malleolus, more commonly a sharply marginated and angular fragment is seen and represents a traumatic fracture rather than OCD. A recently defined type of fragmentation involving the proximal plantar tubercle of the talus may also represent OCD.¹

A 4-view radiographic series is recommended to fully evaluate fragment number, size and possible displacement. The most “effective“ view to detect tarsal OCD is the D55°M-PIL oblique, which highlights both the lateral trochlear ridge and the distal intermediate ridge.² The lateromedial projection further helps define these two types of lesions and permits identification of plantar tubercle fragments. Most challenging is detection of medial malleolus fragments because of their axial location. Although the dorsoplantar projection can be diagnostic, the D30°L-PIM oblique more effectively highlights the joint space between the medial malleolus and medial aspect of the trochlea of the talus. These lesions can also be detected ultrasonographically.²

All three of these sites have the typical appearance of a well-defined, rounded, smoothly marginated osteochondral fragment with an underlying subchondral bone defect. Medial malleolus fragments are often very small, whereas lateral trochlear ridge fragments can be large, multiple and involve a significant proportion of the ridge, particularly in draft horses. Fragments from the distal intermediate ridge of the tibia and lateral trochlear ridge may become displaced and be seen distally, cranially to the talus or even the central tarsal bone. Atypically, distal intermediate ridge fragments are non-mineralised and the lesion appears only as a subchondral defect.

**Fetlock**

OCD in the fetlock occurs at the sagittal ridge of the 3rd metatarsal/carpal bone, and all four limbs can be affected. The most useful radiographic projection is a well-positioned flexed lateromedial, which removes superimposition from the greatest possible extent of the sagittal ridge.³ With very distal or even plantar/palmar lesions, flattening of the sagittal ridge can be seen on a dorsoplantar/palmar projection.

A partially mineralised fragment is not usually present, with the subchondral bone defect and flattening of the sagittal ridge often being the only radiographic sign. Flattening may occur anywhere along the ridge: usually dorsoproximal, but distal and plantar/palmar lesions are possible.

A form of osteochondral fragmentation occurs at the subchondral articular bone of the axial aspect of the proximal plantar (usually) or palmar (rarely) eminence of the proximal phalanx. Because these fragments can be seen in young horses prior to training and because there is evidence that it has a hereditary component in Standardbreds, they may be a form of OCD. However, a traumatic etiology has also been proposed.

Nonetheless, the most useful radiographic projection for detecting these fragments is a D35°L45°P-PlMDi oblique, which “opens up” the plantar joint space by removing superimposition with the proximal sesamoid bones.³ Fragments are usually mineralised, well-defined, small and rounded or triangular and are seen adjacent to a small semilunar subchondral bone defect. Occasionally, only the subchondral defect is seen.
Abaxial, nonarticular proximal plantar (usually) or palmar (rarely) osteochondral fragmentation can also occur. It is also unclear whether or not this is a form of ODC, versus chronic fracture or nonunion. These fragments can be large and multiple, are rounded and well-defined, but irregular and associated with an irregular underlying bone defect in the plantar eminence. These fragments are readily seen on standard oblique projections of the fetlock, or on the D35°L45°Pr-PIMDi oblique used to see the smaller axial articular fragments.

**Stifle**

Femoropatellar OCD is quite common and most often affects the lateral trochlear ridge of the femur, but also affects the medial trochlear ridge, both trochlear ridges, or, rarely, the trochlear groove. A well-positioned lateromedial projection is most useful, although a Cd35°L-CrM oblique view can sometimes show smaller lesions on the lateral trochlear ridge, in addition to highlighting medial femoral condyle cystic lesions. Ultrasound can be useful for diagnosis and characterisation of OCD lesions, particularly when they are not clearly visible on radiographs.

In the least severe cases, focal subchondral bone flattening is the only radiographic abnormality. Defects can be seen in the proximal, middle or distal third of the ridge, but most often in the proximal/middle third. When more severe, a significant length and depth of the trochlear ridge subchondral bone may be affected, with irregularity and underlying sclerosis. Lateral trochlear ridge lesions tend to be longer than medial trochlear ridge ones, making medial trochlear ridge lesions more challenging to diagnose. The fossa of the lateral digital extensor tendon should not be confused with a subchondral defect. Also, a small indentation is often present at the junction between the trochlea and femoral condyles and should also not be mistaken for a lesion. Fragmentation is only sometimes visible and can be multiple. The mineralised size of the fragment is not always proportional to the size of the underlying subchondral bone defect. As for the tarsus, fragments can migrate away from the subchondral defect and be found in other locations in the joint. The joint should be evaluated for osteoarthritis, particularly in horses that are older on presentation, as this seems to be a more frequent secondary occurrence than with tarsal or fetlock OCD. This is best seen on a Cd10°Pr-CrD projection.

Subchondral cysts/cyst-like lesions occur at the medial femoral condyle, and very rarely the lateral femoral condyle. They range in appearance from a focal semilunar indentation of the articular surface with underlying sclerosis to a well-defined and regularly margined large circular area of decreased subchondral bone opacity. It is important to assess for the presence of articular involvement. As with femoropatellar OCD, the joint should be evaluated for the presence of osteoarthritis. Typically, they are seen on a Cd10°Pr-CrD projection, and it is of the utmost importance not to underexpose this projection, regardless of whether digital or conventional radiographic equipment is used, as underexposure can hide the contrast between the cyst and the surrounding bone. Smaller lesions may require a Cd35°L-CrM oblique view. Performing this oblique view with flexion may be even more useful.

**Shoulder**

Because of its poor prognosis, fortunately, OCD lesions of the shoulder are uncommon. They typically involve the humeral head with or without secondary involvement of the articular surface of the glenoid cavity. Lesions are notoriously hard to diagnose because of the thick overlying soft tissues and often dramatically underestimated on a flexed mediolateral radiograph. Using a grid helps radiographic detail, but requires equipment capable of high exposures with short exposure time. Radiographs can be made under general anaesthesia in order to improve patient comfort and radiographic definition. A mineralised fragment is not typically seen, with the lesion appearing instead as an irregular subchondral bone defect of variable length and underlying sclerosis. Osteoarthritis is a common complication, and the joint margins should be closely assessed for this.

**Other Sites of OCD, Cysts and Osteochondrosis**

Other than the stifle, subchondral bone cysts/osseous cyst-like lesions occur most often in the distal 3rd metacarpal/tarsal condyles, proximal or distal subchondral surfaces of the proximal and middle phalanges and the proximal subchondral surface of the distal phalanx. They are also rarely seen in the
elbow, shoulder, proximal and distal tibia and tarsal bones. When associated with the pastern joint, they can be particularly predisposed to subsequent osteoarthrosis.

Radiographically, cysts appear as a round/oval radiolucent defect that is regularly margined and well-defined and often surrounded by a thin sclerotic rim. Standard orthogonal radiographic projections are often sufficient for detection, but specific projection angles can be used to determine if there is communication with the articular surface. For distal limb cysts, CT and MRI are very helpful to determine articular involvement.

OCD can also affect the cervical articular processes. In these cases, OCD can or not be associated with neurological signs of cervical stenotic myelopathy (Wobbler syndrome). Not only are the cervical articular processes enlarged and rounded, but they also often have a heterogenous and well-defined but irregular contour because of the presence of a subchondral bone defect and osteochondral fragments. These lesions can be mistaken for chronic fractures. In addition to laterolateral projections, oblique projections are very helpful for characterising the lesion and determining its laterality.

A debate remains as to whether noninfectious, developmental (“acquired”) physisis should be considered as a form of osteochondrosis as it involves a disturbance of endochondral ossification. Many of the same factors that predispose to the development of osteochondrosis also contribute to physisitis. The term “physeal dysplasia” may in fact be more appropriate for this disease. The distal radius, distal metacarpus and distal tibia are predisposed, and the age of presentation corresponds with periods of active endochondral ossification (up to 6 months for the metacarpus and 18 months for the distal radius/tibia).

Radiographically, lesions are located at the side of the growth plate that receives higher loadbearing, typically dorsomedially. The physis becomes widened and more irregular, with adjacent sclerosis. Metaphyseal and epiphyseal “flaring” at the physeal margins also occurs. Taking radiographs on long cassettes/plates is helpful in order to evaluate any underlying or secondary angular limb deformity.

**REFERENCES**

Castration of male horses is the most common surgical procedure performed in the field in equine practice. Because castration is such a common procedure (and done at relatively low cost to the owner), it is often perceived by equine owners to be ‘risk free,’ when in fact castration has one of the highest morbidity rates of all equine surgical practices. Many complications are life-threatening if not dealt with immediately. Clients should be educated on the associated risks and complications prior to surgery, and veterinarians performing the procedure must be aware of how to deal with them should they occur. This presentation will cover dealing with castration-associated hemorrhage, evisceration/eventration, penile trauma, swelling and infection in the field.

Surgical Techniques
Routine castration in horses can be done as a standing procedure or under short-acting IV anesthesia and as either an open or closed procedure in reference to whether or not the vaginal tunic covering the testis is incised. Any of these methods are acceptable and all come with advantages and disadvantages. These are described below in reference to associated complications. Local and specific circumstances, personal preference and experience will likely dictate which method is used. Castrations are most commonly performed through a surgical site that is left open to heal by 2nd intention. Primary closure of the surgical site can also be used, but it requires strict adherence to aseptic technique and may not be appropriate under field conditions.

Complications
Hemorrhage
There are many vessels that may be cut or torn during dissection to expose the testis that may create a small amount of postoperative bleeding but are generally of little concern. This type of bleeding can be avoided by using blunt dissection as much as possible during the procedure. However, hemorrhage from the testicular artery or pampiniform plexus can be significant and potentially life-threatening if not addressed. This hemorrhage is best avoided by leaving the emasculators in place long enough (2–3 minutes) to ensure cord vessels are crushed. In a particularly large cord, it may be advantageous to crush the cremaster muscle separately from the vessels, or perform a ‘modified open’ technique to allow less tissue bulk within the instrument and ensuring an effective crush. With this theory in mind, performing an open castration is thought by some to have the advantage of better hemostasis over a closed castration, as there is less tissue bulk within the crush of the emasculators. Ligation of individual vessels at the time of surgery can also be effective against postoperative hemorrhage, but circumferential ligation of the entire cord has little effect on controlling hemorrhage.

If there is a steady stream of blood from the castration wound 30–60 minutes postoperatively, or the site continues a steady drip for several hours postoperatively, the hemorrhage should be addressed. Packing the surgery site with a roll of gauze may stop mild bleeding; however, it generally does not stop significant hemorrhage. The pressure from continued bleeding separates tissue planes and creates a large hematoma adjacent to the pack in the inguinal area. The most direct, effective, and fastest way to resolve the bleeding is to reanesthetize the horse, explore the surgery site, and ligate the offending vessel. Positioning the horse into dorsal recumbency, balanced between 2 hay bales, will markedly improve visualization and access to the inguinal area versus attempting to address the hemorrhage with the horse standing or in lateral recumbency. If an obvious source of hemorrhage is not found, reapply the emasculators and/or double ligate the spermatic cord and cremaster (0 or #1 suture material), as these are the likely sources of bleeding. Once ligated, tightly accordion several gauze rolls (tied to each other)
into the space that was created in the inguinal area. The packing should not be forced into the inguinal canal, as it can potentially enter the abdominal cavity. Once it gets there, it is likely to form an adhesion to small intestine, which creates an even bigger problem when the packing is pulled 24–48 hours later (evisceration). Suture the wound opening closed with a few simple interrupted sutures, tacking the end of the gauze to the skin.

The sutures and gauze packing are removed 24–48 hours later. I generally place these horses on antibiotics and phenylbutazone as the resolving hematoma and trauma associated with 2 surgeries create a great environment for bacterial growth. Once the packing is pulled, forced exercise and cold hosing therapy can begin to help encourage drainage and decrease postoperative swelling.

**Evisceration**

Evisceration following castration occurs infrequently (0.2–2.6% of castrations) but is likely fatal if not addressed immediately. Standardbreds and draft horses have been shown to be at increased risk. Closed castration technique is thought to help prevent this complication. However, even though the vaginal tunic is crushed and sealed in a closed castration, pressure of the small intestine can easily open this seal. To prevent evisceration with a closed castration technique the vaginal tunic must be physically occluded. This is done by a well-placed, secure, transfixation ligature of 0 or #1 absorbable suture material. Open castration does not allow for closing of the vaginal tunic and may increase the risk of evisceration.

Evisceration following castration usually occurs within several hours postoperatively, often on the initial effort to stand up from anesthesia. If this occurs, prompt emergency care is essential for a successful outcome. Initial therapy is aimed at keeping the intestines from hitting the ground, becoming contaminated or traumatized by the horse stepping on them. It is easier to work with gravity, not against it. Therefore, if the risk of stepping on the bowel is minimal, the horse should be reanesthetized and placed on its back. The exposed intestine is lavaged with sterile saline to rinse off gross contamination and placed back in the scrotum, which is then sutured closed or closed with several towel clamps. If a large amount of bowel is exposed, it may be impossible to get it into the scrotum. In this case, a moist towel or drape should be made into a sling to support the bowel. Alternatively, a moistened hand towel can be sutured to the inguinal area under sedation and local anesthesia. Parenteral antibiotics, sedation and flunixin meglumine should be administered prior to immediate transport to a surgical facility for definitive repair. At the referral centre, the horse will be reanesthetized, the bowel examined, lavaged and returned to the abdomen through a ventral midline incision. Resection and anastomosis may be necessary. The prognosis is related to the amount of bowel eviscerated and the duration and degree of exposure and strangulation, with reported survival rates ranging from 36–87%.

**Penile Trauma**

The penis may be encountered during castration while searching for an inguinal testis or may be mistaken for a testis, incised or even amputated before its true identity is recognized. This is most common in castration of young animals with a standing procedure where visualization is poor and the diameter of the testis and penis is similar. For this reason, I recommend castration of colts < 1 year of age be performed under general anesthesia and care be taken to identify the shaft of the penis as well as 2 descended testicles before incision. Excessive stripping of the fascia surrounding penile shaft (as one would strip a testis) can result in paraphimosis and excessive edema. This should be treated with penile support, hydrotherapy and antiinflammatories until the penis can be returned to the prepuce. An incision into the urethra of the penis will result in extravasation of urine into surrounding tissues, resulting in severe necrosis. The colt should be transferred to a referral centre as soon as urethral damage is recognized for definitive repair. Should the penis be amputated in error, severe hemorrhage is likely to occur. A tourniquet can be placed on the distal end of the penile stump and the horse transported to a referral center for surgical repair.

**Postoperative Swelling and Infection**

This is the most common complication encountered after castrations where the surgery site is left open. The primary causes of swelling include surgical trauma, hemorrhage, infection, lack of drainage, and postoperative management. Surgical technique plays a role in the development of postoperative swelling.
Knowing exactly what is planned and proceeding quickly through surgery minimizes trauma. Minimizing bleeding decreases fluid in the surgery site and improves speed due to better visualization. The incision left open must be large enough to allow adequate drainage and not heal closed too soon. Postoperative management can help lessen the amount of swelling that develops. Exercise causes movement of these tissues and encourages ventral drainage and lymphatic activity. Nonsteroidal antiinflammatory drugs also help reduce swelling postoperatively.

All castration sites left open become contaminated and may develop a low-grade infection. Good drainage lessens the amount of serum that accumulates in the dead space and lessens the risk of persistent infection. Perioperative antibiotics should also help reduce the risk of infection. Antibiotic use is largely a personal preference. Most veterinarians administer a single preoperative dose of antibiotic but do not carry out administration much longer than this.

Persistent infection may develop within the spermatic cord. This is referred to as ‘schirrous cord.’ This condition may be predisposed by leaving the vaginal tunic behind during open castration. Early recognition and treatment with antibiotics and drainage may resolve the infection, but surgical excision is often necessary. Horses with schirrous cord usually present weeks after castration with palpably thickened spermatic cords, and there is usually drainage from the previous castration site. Surgery is performed to dissect the infection back to normal cord, which is then re-emasculated. The surgery site is left open to heal by 2nd intention.

REFERENCES
Cryptorchidism is the most prevalent, nonlethal developmental defect in the horse and occurs when one or both testes fail to descend into the scrotum. The undescended testis can reside anywhere within the inguinal canal (high flanker) to within the abdominal cavity (true abdominal cryptorchid). Although there are conflicting studies as to whether inguinal or abdominal cryptorchidism is more common, an older study of 350 cryptorchid horses found that 75% of left undescended testes were located within the abdomen, whereas only 42% of right undescended testes were retained abdominally. In bilaterally affected horses, 60% were abdominal testes.

The cryptorchid horse provides a challenge to the equine field practitioner. A testis that is nonpalpable within the scrotum may present itself under heavy sedation or once the horse is anesthetized and placed on its back. Should this occur, routine castration can proceed as planned. However, there are instances where a testis was palpated before surgery, only to find that it was the penis, a lymph node, or a wad of inguinal fat once surgery has begun. Or, the ‘missing’ testis does not show itself once the horse is on its back...now what? The following presentation explains approaches to dealing with cryptorchids in the field.

**EXAMINATION OF THE POTENTIAL CRYPTORCHID**
I find the most reliable way to determine if the retained testis is inguinal or abdominal is to examine the inguinal ring/canal with deep palpation with the horse anesthetized and on its back. In dorsal recumbency, it is rare not to be able to feel an inguinal testis. Standing palpation, even under heavy sedation, can be difficult with young, unhandled colts. Rectal ultrasound examination can be reliable in finding abdominally retained testicles, but again, who really wants to do this in a 2-year-old colt? For these reasons, I usually advise owners of all the different scenarios that may play out once the horse is in dorsal recumbency and deep palpation of the inguinal canal has occurred and proceed with anesthesia.

**SURGICAL APPROACHES IN THE FIELD**
If palpable through the inguinal ring, the retained testis can be easily retrieved with an 8- to 15-cm incision placed directly over the external inguinal ring. Once a skin incision has been made, blunt finger dissection should be used to access the superficial inguinal ring to avoid transection of the large vessels traversing this area. The inguinal testis is readily encountered once the superficial ring is exposed and can then be grabbed with tissue forceps or a towel clamp and exteriorized from the canal. From this point the colt is castrated as usual. The inguinal incision can be closed or left open to heal by second intention.

If the testis is not palpable within the canal after induction of anesthesia, chances are it’s in the abdomen. At this time a decision should be made whether to proceed with surgery in the field or to recover the horse and refer to a surgical centre. Under field conditions, it is advisable to start with the retained testicle. If the testis is not found, do not proceed to castrate the descended testicle. This can make it very difficult for a referral centre to determine what side the descended testis is on and may necessitate a more expensive laparoscopic procedure.

Abdominal cryptorchids can also be approached through the inguinal approach discussed above and can be attempted in the field. It is advisable to have an assistant handy to scrub in, as retraction of the superficial inguinal ring can be extremely helpful. If the testis is not immediately encountered after exposure of the superficial inguinal ring, it can be located by placing tension on the ligamentous remnant of gubernaculum testis. With an assistant retracting the edges of the superficial inguinal ring, this ligament can be identified by carefully examining the margin of the superficial ring for a fibrous band that descends into the canal. It can be found on either the medial or lateral aspect of the ring, usually at the junction of the middle to cranial third of the ring. Tension placed on this ligament will evert the end of the vaginal process into the inguinal canal. The vaginal process is a glistening, white structure about...
the size of a fingertip. Once exposed, it can be grasped with sponge or Ochsner forceps and incised longitudinally to expose the epididymis, which is tensed to allow exteriorization of the small, deformable testis through the canal. The cryptorchid testicle can be removed with emasculators or ligature placement. It is not necessary to close the external inguinal ring, but the subcutaneous tissue and skin are closed with absorbable interrupted sutures. Following removal of the retained testis, the descended testis can also be removed in routine fashion.

In teaching this technique to interns and residents, I have found the most common mistake is for the veterinarian to get impatient when attempting to locate the remnant of the gubernaculum testis. The tendency is to want to get the hand through the inguinal canal, into the abdomen to start blindly fishing for the testis. This is rarely successful and can potentiate complications such as adhesions, peritonitis and need for a more complicated approach to remove the testis. Trust the gubernaculum…it will always lead you to the testis without having to put your hand in the abdomen.

Also, it is important to remember that a retained testicle still looks like a testicle. If you are unsure that you have removed the testicle in its entirety, you probably haven’t.

**Complications**

Complications of cryptorchid castration are similar to that of regular castration, with the addition of failure to locate the testicle. Hemorrhage from the retained testis is generally not a concern, as the structures are much smaller than that of a normal descended testis. Infection will be a greater concern if this procedure is performed on farm. Pre- and postoperative antibiotics are warranted, especially in cases where the abdomen has been entered. Evisceration becomes a bigger risk if the internal ring is significantly enlarged (blindly fishing for the testis with a hand through the inguinal canal). In these cases, I do attempt to close with inguinal ring (internal if possible), or pack the inguinal area for 24–48 hrs to occlude the space until soft-tissue swelling occludes the opening into the abdomen.

**References**

Trauma and laceration to the distal limb (below the fetlock) in horses is very common. Due to the anatomy of the horse in this region, limited soft-tissue coverage and poor digital perfusion, complications and poor wound healing are common. Synovial structure involvement greatly affects prognosis unless early diagnosis and aggressive treatment is instituted soon after wounding. Also, constant movement of healing tissue planes in this region commonly leads to delayed healing and production of excessive granulation tissue. In this lecture, we will discuss the diagnostic approach and treatment options for distal limb wounds.

Depending on depth and location, wounds in the pastern region of the horse can involve the coffin or pastern joint, the navicular bursa and/or the flexor tendon sheath; all synovial structures that, when infected, greatly affect the prognosis for soundness. However, when diagnosed early and treated appropriately, can still have an excellent prognosis.

**EXAMINATION & DIAGNOSIS OF SYNOVIAL CONTAMINATION**

All acute wounds to the distal limb should be investigated by deep palpation with a sterile glove after thorough cleaning of the wound. Based on depth and direction of the wound, any synovial structure that may be involved should be investigated. In horses with chronic wounds with established synovial sepsis, lameness may be apparent. However, if the synovial structure is still able to decompress through an open wound, lameness may be minimal until the wound seals.

In horses with wounds in close proximity to a synovial structure, it is necessary to determine if the synovial structure has been penetrated and contaminated. It is also important not to initiate iatrogenic sepsis, so centesis should be performed at a site as remote as possible to the traumatized area. After synovial samples have been obtained, up to 100 ml of sterile saline can be infused and the wound examined for leakage. This is a very affective diagnostic technique in acute wounds where cytologic changes to synovial fluid may not yet be present. If there is suspicion of synovial sepsis but centesis and/or infusion cannot be performed, the horse should be referred. A ‘wait and see’ approach will greatly decrease the prognosis for soundness in horses where communication does actually occur.

Gross examination of the synovial fluid is often suggestive of sepsis in subacute or chronic cases. Typically the fluid will be turbid and flocculent. Fluid is collected into an enriched medium for culture and sensitivity testing, and into EDTA or heparin tubes for cytology. A smear should be made and Gram stained. If the synovial fluid cell count is > 30 x 10⁹/L (normal: < 5 x 10⁹/L) and total protein > 4 g/dL (normal: < 2 g/dL), the joint should be considered infected. White cell counts > 50 x 10⁹/L are common in cases of septic arthritis with neutrophils being the predominant cell type.

**TREATMENT**

Distal limb wounds with synovial penetration/involvement require aggressive therapy. Chronic wounds should be referred. However, acute wounds that are diagnosed within 24 hours can be successfully treated on farm. Beyond 24 hours clearing infection is very difficult with on-farm methods due to the inability to remove fibrin from the synovial structure. Fibrin provides a place for bacteria to be harbored and contributes to the inflammatory and adhesion formation process.

Horses with acute wounds with synovial penetration should be treated with parenteral antibiotics (penicillin/gentamicin combo), thorough wound cleaning, through-and-through lavage of the synovial structure and local antibiotic therapy via either intrasynovial injection or regional limb perfusion. Through-and-through lavage can usually be accomplished in the standing, sedated horse with 16- to 18-gauge needles placed in several areas of the joint. At least 1 L of saline (preferably 3 L) is infused through a single site and out the additional needles. Following lavage, all but 1 needle are removed, and that
needle is used to instill intrasynovial antibiotic. The limb is then placed in a sterile bandage. The lavage may need to be repeated 1–2 times. Should the horse become lame or cytologic examination of subsequent synovial fluid samples become worse despite appropriate therapy, referral should not be delayed.

As an alternative to intrasynovial injection, regional limb perfusion can be performed. The technique involves placement of a tourniquet proximal to the infected joint or tendon sheath, and the administration of antibiotic via a catheterized vein. For IV distal limb perfusion, the animal is sedated and suitably restrained. The vein (e.g., metacarpal) is catheterized with a 20-gauge over-the-needle catheter, and 500-mg amikacin in up to 60 ml of saline is administered. The tourniquet is maintained for 20 minutes and then removed.

**FOOT CAST PLACEMENT**

Once synovial involvement is eliminated or under control, placement of a ‘foot’ cast can greatly enhance healing of wounds to the pastern/heel bulb area. Numerous benefits are derived from the rigid support of wounds, particularly in the distal limb of the horse. Counter pressure applied to a wound reduces swelling, thereby decreasing tension on a suture line. Decreased edema, reduced postop hemorrhage, and obliteration of dead space will aid in preventing excessive fibrous tissue formation. Immobilization protects suture lines from tension and eliminates movement between planes. In addition, immobilized wounds have superior resistance to bacterial growth, and immobilization of contaminated wounds reduces the spread of bacteria within the wound.

A foot cast can be placed on the forelimb in a standing, sedated horse, but short-acting IV anesthesia will be needed for the hind limb due to limb anatomy. The cast should incorporate the entire foot and end just below the fetlock. A double layer of stockinette is applied to the limb to a point above where the cast will end, and a strip of orthopedic felt is applied at the level the cast will end. A single layer of custom support foam or cotton cast padding is applied to the pastern area but is not necessary over the hoof. At least 3 layers of 3- to 4-inch casting tape should be applied to ensure adequate strength, and once the casting material begins to cure, polymethylmethacrylate is placed on the solar surface of the cast to increase its wear life. The horse is kept stalled while in the cast and monitored closely for complications such as cast sores (rare with foot casts). The cast is worn for 2–3 weeks and then removed standing. By this time, the wound is generally healed. A 2nd cast can be placed if necessary or, alternatively, a bandage.
The most important step in reducing or eliminating calf diarrhea is to make sure the farm has a good colostrum management program. Failure of passive transfer is the most common problem I encounter on farms associated with calf health problems. These calves may appear normal but are at increased risk for illness. They also shed pathogens into the environment at a much greater rate than do calves that have had adequate colostrum. Dairy producers should set a goal of 100% of heifers achieving adequate colostrum absorption.

**INTRODUCTION AND PATHOPHYSIOLOGY**

It is well known that ruminants are born essentially agammaglobulinemic because of the syndesmochorial placentation that prevents the maternal blood supply from contacting the fetal trophoblast. However, the enterocyte of the neonate has the unique ability to absorb protein macromolecules. During the first 24 hours or so of life, the intestinal enterocytes will nonselectively absorb macromolecules, including immunoglobulins by pinocytosis. Immunoglobulins are transported across the cell to the lymphatics where they drain into the bloodstream. This ability to absorb macromolecules is time limited, and within 24 hours the enterocytes cease to absorb macromolecules, which is termed “gut closure.” Gut closure is not an “all or none” phenomenon, as the calf’s ability to absorb macromolecules begins to decline almost immediately following birth. The process of pinocytosis is less efficient after the first 6 hours of life and really begins to decline after 12 hours. Thus the timing of colostrum administration is crucial.

It should be emphasized that a good colostrum management program - ensuring that all calves get adequate volume of high-quality colostrum within the first 6 to 12 hours of life - is the single most important aspect of neonatal calf management on dairy farms.

**COLOSTRUM MANAGEMENT**

There are several myths about colostrum management amongst producers and veterinarians.

**First Myth**

“Colostrum from heifers is of poor quality and should be discarded.”

Traditionally it has been recommended to discard colostrum from heifers and use only the colostrum from multiparous cows. Although there is an effect of lactation number on IgG concentration, there is no significant difference in the colostrum IgG concentrations from 1st or 2nd lactation cows. Only cows in their 3rd and greater lactations have higher IgG concentrations. Since the average lifespan of a cow in a commercial dairy herd is three lactations, only a small percentage of the animals in a herd are usually ≥ 3 lactations. Therefore, these cows cannot be relied upon to feed all the calves. Colostrum from many first-lactation cattle contains adequate IgG concentrations, and the practice of routinely discarding colostrum from these animals is not recommended.

**Second Myth**

“Pooling of colostrum from different cows improves the quality.”

This theory is based on the principle that by pooling colostrum, this minimizes the effect of low-quality samples. However, this has been shown not to be the case, because cows that produce larger volumes of colostrum consistently have lower IgG concentrations. For example: Cow A produces 15 kg of colostrum containing 20 g/L of IgG, and cow B produces 5 kg of colostrum containing 40 g/L of IgG. If we mix the colostrum from these 2, the pooled colostrum will not have 30 g/L of IgG. This pooled colostrum will contain only 25 g/L of IgG ([((20) (15) + (40) (15)) / 20]). Low-IgG, high-volume colostrum generally becomes overrepresented in pooled colostrum, and the risk of disease transmission (i.e., Johne’s,
Salmonella, etc.) is also increased. Therefore, the practice of pooling colostrum should be strongly discouraged.

**Third Myth**

“The thicker the colostrum, the better” or “I can tell good-quality colostrum when I see it.”

It is a common misconception amongst dairy producers (and some veterinarians) that visual examination of colostrum can identify samples of low and high quality. However, research has found this not to be the case. Actually the concentration of IgG in colostrum between cows on the same farm has been shown to vary significantly, and the only way to accurately differentiate poor-quality from good colostrum is to test.

Calves need to absorb about 40 grams of IgG for adequate passive transfer, but because intestinal absorption is not 100% efficient, they need to ingest between 150 to 200 grams of IgG or approximately 1 gallon (4 liters) of good-quality colostrum. Most dairy farms feed colostrum either by bottle or esophageal feeder. Some dairies still leave the calf with the cow during the initial 24 hours and let the calf nurse colostrum (this should be strongly discouraged). Colostrum should be given within the first 6 hours after birth and **must** be given by the time the calf is 12 hours old. Research suggests that bottle-fed calves may have slightly better IgG levels than calves fed via esophageal feeders; however, the difference is insignificant. Since the current recommendations indicate that a significant volume of colostrum should be fed, esophageal feeders are gaining popularity since many calves are unwilling or unable to drink this volume in a single feeding.

The most important aspect of feeding colostrum is volume. Each calf should receive 3 liters (Jerseys, Guernseys, or Ayrshires) or 4 liters (Holsteins and Brown Swiss) of good-quality colostrum. This can be given in one or two feedings but should be completed by the time the calf is 12 hours old. If there is any delay in colostrum administration, the entire 4 quarts should be given at the first feeding.

**Testing for Passive Transfer**

The only way to know there is a colostrum problem is to test the calves for passive transfer. Regular (monthly) testing provides good feedback to the producer on how he is doing with his program. To get an adequate assessment of passive transfer status, at least 12 calves should be tested. Testing can be done as soon as 6 hours after feeding colostrum but no later than 1 week of age. Usually serum total protein concentration is the test of choice, and if you use a concentration of 5.5 g/dl as the cut point, there should be no more than 3 of the 12 calves that fall below this value unless there is a herd problem with FPT. For smaller herds, accumulate test results until 12 calves have been sampled.

When a failure of passive transfer problem is identified, the following should be examined as potential causes:

126. Unobserved calvings occurring on a regular basis
127. Calves remain with dam for more than 90 minutes
128. Colostrum administration occurs more than 4 hours after calving
129. Calves do not routinely receive 4 liters of colostrum or not within the first 12 hours
130. There is a shortage of colostrum from appropriate donors without a backup supply
131. There is more than a 2-hour lapse between colostrum milking and either feeding or refrigeration of colostrum
132. Refrigerated colostrum is > 7 days old or frozen colostrum that is > 1 year old or frozen colostrum that has been through more than 1 freeze-thaw cycle
133. Bacterial contamination of colostrum is excessive (total bacterial count > 1,000,000 CFU/ml and/or fecal coliform count > 10,000 CFU/ml)
134. Colostrum is routinely pooled
135. Fresh cow health is poor or
136. Transition cow management (nutrition, group changes, bedding, density, vaccinations, medications) is a concern
Contamination of colostrum and/or milk/milk replacers can also be a problem leading to diarrhea in dairy herds. Common sources of contamination include infected quarters, inadequate cow preparation at the time of milking, poorly sanitized milking equipment, or storage without adequate cooling. Milk and milk replacers can also be contaminated by people or from the feeding equipment (bottles, buckets, delivery equipment, etc). Colostrum and/or milk/milk replacers can be cultured when suspected problems exist. Dr. Sheila McGuirk at the University of Wisconsin has recommended the following goals for milk and milk replacers fed to calves: total bacteria count less than 10,000 CFU/ml, fecal coliform count of 0 CFU/ml, coagulase-negative \textit{Staphylococcus} counts less than 5,000 CFU/ml, and gram-negative bacterial counts (other than coliforms) less than 5,000 CFU/ml. Although the importance of bacterial contamination hasn’t been emphasized much in the past, we are beginning to realize what an important cause of calf diarrhea this has become.
The two primary nutritional diseases of dairy calves are abomasal bloat and hypernatremia. Both syndromes are most often related to problems or errors in feeding milk, milk replacer, and/or oral electrolytes to calves. Risk factors include improper mixing of milk replacer or oral electrolyte products, feeding a large volume of milk in a single daily feeding, feeding cold milk (or milk replacer), not offering water to calves, erratic feeding schedules, and failure of passive transfer.

**Abomasal Bloat**

Abomasal bloat is a syndrome in young calves characterized by anorexia, abdominal distension, bloat and often death in 6 to 48 hours. This condition occurs most commonly in dairy calves and seems to have a sporadic occurrence with some farms having multiple outbreaks at times. Clinical signs are often mild and may inconsistently include diarrhea, mild abdominal distention with fluid and gas, splashing on abdominal succession, and mild depression. Hyperglycemia (190–500 mg/dl or 10.5–28 mmol/l) with an accompanying glucosuria (1,000–2,000 mg/dl or 55–110 mmol/l) consistently develops. In severe cases, the calves are usually dehydrated, show signs of colic, have prominent abdominal distention and diarrhea, and become recumbent. At necropsy, most of these calves will have abomasal tympany, forestomach and abomasal edema, hemorrhage, mucosal necrosis, and occasionally mural emphysema. Gross lesions include hemorrhage, edema, and necrosis of abomasal and ruminal mucosa. Occasional emphysematous bullae may be present in the stomach wall. Pathogens isolated include alpha streptococci, other *Streptococcus* spp., and *E. coli, Clostridium, Sarcina*, and *Candida* spp.

Recently the abomasal bloat syndrome was experimentally reproduced by drenching young Holstein calves with a carbohydrate mixture containing milk replacer, corn starch, and glucose mixed in water. The authors of this study proposed that the pathophysiology of abomasal bloat is primarily excess fermentation of high-energy gastrointestinal contents. Gas-producing bacteria such as *Clostridium perfringens, Sarcina ventriculi*, or *Lactobacillus* species have also been thought to play a role in this syndrome. Although the exact pathogenesis of abomasal bloat is not completely understood, the disease is likely to be multifactorial in origin. Having large amounts of fermentable carbohydrate present in the abomasum (from milk, milk replacer, or high-energy oral electrolyte solutions) along with the presence of fermentative enzymes (produced by bacteria) would likely lead to gas production and bloat. This process would be exacerbated by anything that slowed abomasal emptying or caused gastrointestinal ileus. In fact, feeding high-osmolality electrolyte products and/or milk replacers has been noted to be a risk factor on some farms for the development of abomasal bloat in calves.

Treatment generally involves placing the calf in dorsal recumbency and inserting a needle or catheter into the abomasum to relieve the gas. It is important to remember that passing a stomach tube to remove the gas from the stomach, similar to what is done to relieve bloat in adult cows, is not effective. Since the gas is trapped in the abomasum, passing a tube into the rumen will not evacuate the gas, since it is impossible to pass a tube through the mouth and into the abomasum. Attempting to deflate the bloat in a standing calf is generally unrewarding. This does not fully evacuate the gas in the abomasum, and there is a significant risk of stomach fluid leaking into the abdomen. This can result in other serious complications such as peritonitis or vagal indigestion. Antibiotic therapy is also indicated in these calves (most likely parenteral procaine penicillin or oral β-lactam antibiotics to target *Clostridium* bacteria).

Prevention of abomasal bloat focuses on establishing a consistent feeding program. Specifically, maintaining a consistent feeding schedule and being careful with the use of very high osmolality milk replacer or electrolyte products that could slow abomasal emptying and facilitate bacterial fermentation in the abomasum. As a goal, farms should try to avoid feeding either milk replacers or oral electrolyte products that have an osmolality greater than 600 (mOsm/L), as the risk of bloat is likely to increase. The author has measured the osmolality of milk replacers on over 20 dairies with bloat problems, and has found values well above 1,000 mOsm/L. However, I have not found any milk replacer whose osmolality
would be greater than 600 mOsm/L when mixed according to label directions. Therefore, mixing errors are largely responsible for very high values. These errors might be unintentional or can be done on purpose, such as concentrating powder to increase growth rates, for additional energy during cold weather, or adding electrolyte salts to milk without adding additional water.

Other risk factors for abomasal bloat include feeding a large volume of milk in a single daily feeding, cold milk (or milk replacer), not offering water to calves, erratic feeding schedules, and failure of passive transfer. Therefore, for farms that are having problems with bloat in young calves, the recommendation is first to look at the feeding program. Make sure milk replacers are being mixed correctly and fed at a proper temperature. It is also important to establish a consistent feeding schedule. Therefore, it is important to work with your veterinarian to develop protocols and how to treat cases of bloat when they happen on the farm.

In summary, abomasal bloat in young calves is a syndrome whose prevalence seems to be increasing in the dairy industry. It is caused by a number of factors, and there is not one particular thing that can be done to effectively control the disease. The most factors for prevention are to establish a consistent feeding program with milk or milk replacer that has been mixed according to the label directions. Also work with your producers to establish treatment programs for these calves, so when cases do occur, they can be managed quickly and effectively.

**Hypernatremia**

Hypernatremia is defined as a serum (blood) sodium concentration ≥ 160 mEq/L (or mmol/L) and is another problem that can be occasionally encountered in calves. Hypernatremia in food animals is generally caused by one of the following: 1) excessive loss of free water (i.e., water deprivation, heat stress); 2) iatrogenic administration of IV crystalloid solutions to animals that don’t have access to water; and 3) excessive intake of sodium without an adequate volume of free water. This last cause is by far the most common syndrome in calves. Most cases of hypernatremia result from mixing oral electrolyte solutions improperly, or in some circumstances hypernatremia has been seen with very high osmolality milk replacers.

During hypernatremic states, water will follow concentration gradients and move into the relatively hyperosmolar CSF and plasma. This results in cellular dehydration and brain shrinkage (neurons shrink when the water moves out). Neuronal cells will increase their intracellular osmolality in an attempt to minimize the water efflux - however, this adaptation can only compensate for mild increases in sodium concentrations. Severe hypernatremia leads to neurologic disease in calves.

Early clinical signs of hypernatremia include lethargy and depression which, without blood work, cannot be differentiated from many other possible diseases such as acidemia, dehydration, hypoglycemia, or hypothermia. More advanced clinical signs of hypernatremia include twitching of facial muscles, muscle rigidity, tremors, and myoclonus. Calves will demonstrate seizure and/or coma activity near death. Prognosis in these calves is generally guarded to poor even with aggressive treatment, so therapy is reserved only for valuable animals in most cases.

Treating hypernatremia is very difficult. If the sodium concentration is lowered too quickly, water follows a concentration gradient and moves into neurons, resulting in cerebral edema. When sodium concentrations are higher than 160 mEq/L, it is very easy to produce cerebral edema with any isotonic fluid, even those that contain sodium. This is because of the large difference between the calf’s extracellular fluid sodium concentration and the sodium concentration in the fluids. Therefore, it becomes necessary to add supplemental sodium to standard fluid types. The fluids must be formulated so that the sodium concentration is equal to or slightly lower than the calf’s sodium concentration. The calf’s sodium concentration should be reassessed at least 1–2 times per day and fluids reformulated. The goal would be to return the sodium concentration to normal over a period of several days - decreasing it by 3–4 mEq/L per day. Obviously this is expensive (requires frequent blood chemistry analysis) and difficult (requires a lot of labor to constantly reassess fluid therapy) and is not always successful. Many calves will still develop cerebral edema even with careful fluid therapy.

Hypertonic saline (7.2%) contains sodium at a concentration of 1.2 mEq/mL. For example, if a calf had a sodium concentration of 177 mEq/L, the goal would be to give fluids with a sodium concentration
somewhere in this range (175–177 mEq/L). Since isotonic (0.9%) saline has a sodium concentration of 154 mEq/L, the clinician would need to add an additional 22–23 mEq/L of additional sodium to the liter of fluids. This would be equal to 19 ml of hypertonic saline per liter of isotonic saline (1.2 mEq/ml x 19 = 23) or 6 ml of 23.4% saline. After about 12 hours of fluid therapy, the clinician should then reassess the calf’s sodium concentration and reformulate fluids. The use of 5% dextrose in any calf with suspected hypernatremia should absolutely be avoided. This fluid type has a very large amount of free water (basically no sodium) and will result in a rapid lowering of extracellular fluid sodium concentration and cerebral edema. If the calf is hypoglycemic, a better option is to add a bolus of 50% dextrose to the liter of fluids already supplemented with sodium. This will increase the overall osmolality of the fluids to be administered; however, the sodium concentration will still be approximately equal to the calf’s.

If the calf also has a metabolic acidosis, isotonic sodium (1.3%) bicarbonate can be used. In practice, this is usually made by adding 13 grams of baking soda (NaHCO₃) to a liter of sterile water. This contains a sodium concentration of 156 mEq/L. Supplemental sodium (i.e., hypertonic saline) can be added to each liter of sodium bicarbonate as described above for isotonic saline.

If the calf’s neurologic signs get significantly worse after fluid therapy has started, cerebral edema should be suspected. Treating cerebral edema is difficult but imperative to prevent further brain damage and/or death. Corticosteroids are the easiest choice normally; however, they are only marginally effective in treating moderate to severe cases of cerebral edema. Mannitol (25%) and glycerin have also been recommended for severe cases of cerebral edema but can be more difficult to obtain.

In summary, abomasal bloat and hypernatremia are both syndromes that are becoming more frequent in the dairy industry. They are both primarily related to problems or errors in feeding milk replacer or oral electrolytes to calves, and thus are largely avoidable with proper management. The most important control factors are to establish a consistent feeding program with milk or milk replacer that has been mixed according to the label directions.
Troubleshooting Calf Diarrhea
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Calf health should be a priority on both beef and dairy farms. Despite this importance, the USDA Dairy 2007 study shows a preweaned heifer calf mortality rate of 8.7% and reports that only 40% of farms can supply an adequate number of replacements from their own herd. Although calf mortality is slightly less in beef calves, 4 to 5% still die prior to weaning. The major diseases of both beef and dairy calves continue to be diarrhea, pneumonia, and septicemia. Colostrum management continues to be a significant area of concern on many modern dairies.

The most important step in reducing or eliminating calfhood diseases is to make sure the farm has a good colostrum management program. Failure of passive transfer is the most common problem I encounter on farms associated with calf health problems. These calves may appear normal but are at increased risk for illness. They also shed pathogens into the environment at a much greater rate than do calves that have had adequate colostrum. Dairy producers should set a goal of 100% of heifers achieving adequate colostrum absorption.

DIARRHEA
This is the most common cause of death in young calves and is almost entirely avoidable by good management. The highest risk for diarrhea in dairy calves is from birth until about 1 month of age. Clinical signs begin with loose feces and can progress through dehydration to metabolic acidosis, circulatory shock, and death. Although there are many different pathogens that can cause diarrhea in calves (including *E. coli*, rotavirus, coronavirus, *Cryptosporidium*, *Salmonella* species, *Clostridium perfringens* type C, and coccidia), veterinarians rarely try to identify the etiology. This is because calves are generally infected with more than one pathogen, and the goals of treatment are generally the same regardless of the etiology. In problem herds, a diagnosis might be pursued with fecal and/or intestinal exams in order to institute control programs. Knowing the potential pathogen provides insight into the infection source as well as knowledge on what vaccination, treatment, or disinfection protocols might be effective in reducing the problem.

The most important step in reducing or eliminating calf diarrhea is to make sure the farm has a good colostrum management program. Failure of passive transfer is probably the most common problem I encounter on farms associated with calf health problems. These calves may appear normal but are at increased risk for illness. They also shed pathogens into the environment at a much greater rate than do calves that have had adequate colostrum. Dairy producers should set a goal of 100% of heifers achieving adequate colostrum absorption.

The approach to infectious disease control described by Radostits over 3 decades ago includes: 1) removing the source of infection from the calf’s environment; 2) removing the calf from the contaminated environment; 3) increasing the nonspecific immunity of the calf; 4) increasing the specific immunity of the calf; and 5) reducing stress. These approaches are still applicable today and represent the core of calf diarrhea control programs. While on the farm, practitioners should be looking for likely sources of infection and carefully evaluating hygiene and cleanliness.

Pathogens that cause calf diarrhea may be introduced onto a farm by contaminated feedstuffs, water, fertilizor, livestock, wildlife, insects, people or equipment. Management is directed at reducing the risk of adverse health and production consequences through minimizing the challenge dose and promoting host immunity. Maternity pen risks and management also represent a significant opportunity for pathogen transmission. Calves may be exposed to *Cryptosporidium*, *Salmonella*, etc., by fecal material from the dam during birth when the calf contacts the environment, or when calves contact the underside of the cow attempting to nurse. Pathogens may also proliferate in organic bedding material used in calving pens. It is important to appreciate that significant pathogen exposure can occur within the first few hours of life. Control points in maternity pens are directed at minimizing contamination (time cows
spend in the pen, choice of bedding material, and frequency of bedding changes) and exposure risk (time calves spend in the maternity pen).

Control points for reducing the risk of disease transmission associated with the feeding of colostrum include: 1) effective cleaning of equipment used in the harvest and storage of colostrum; 2) avoid pooling of colostrum; 3) verify refrigeration units used for storing colostrum are working; 4) make sure colostrum is cooled rapidly if not fed right away; 5) record date of collection on refrigerated colostrum and discard after 2 days; and 6) maintain dedicated equipment for administering colostrum. Fresh colostrum fed to calves should contain less than 100,000 CFU/ml total bacteria count and less than 10,000 coliforms/ml.

To evaluate the risk of calf exposure to common diarrheal pathogens, it is necessary to trace the path from the place calves are born to the location where they are housed. Bottlenecks that have the potential to concentrate pathogen exposure (such as calf warmers, temporary holding pens, and trailers used for transporting calves) should be avoided when possible and regularly disinfected when they must be used. Primary removal of organic material reflects 99% of the cleaning process and is important to avoid neutralization of the disinfectant. Scheduling cleaning activities to specific days and times of the week helps to establish a consistent routine that ultimately gets done. Many pathogens are susceptible to ultraviolet radiation, so it is a good idea to leave hutches upside down between calves during summer months to help reduce pathogen load. Ideally calves should be placed in a clean, dry, comfortable environment in an area that has good drainage and is not exposed to manure from adult cattle or dairy effluent. Individual calf hutches provide an effective means of minimizing calf-to-calf pathogen transfer. Hutches should be moved to clean ground before they are reused, to avoid pathogen buildup. Calf sheds and crates reflect alternate housing systems. Group housing provides efficient utilization of space and when calves are healthy reduces labor requirements. The age range of calves in group housing should be kept as tight as possible. Mixing age groups increases the risk of pathogen exposure in young calves and can compromise their access to milk at feed time. In regard to calf comfort, if calves get wet and are exposed to cold and windy conditions, energy reserves are rapidly depleted and sick calves rapidly succumb to disease. Placing straw in the hutches of newborn calves helps to keep them warm and dry. In hot climates, calves can be subjected to relatively extreme temperatures, and it becomes increasingly important to provide continuous access to fresh water.

Adequate calf nutrition is critical for host immunity, and energy-deprived calves are more likely to succumb to disease. When assessing nutritional requirements, it is important to factor in seasonal conditions that increase the calf’s energy demands. Lack of refrigeration carries significant risk when residual milk from one feeding is kept for subsequent feedings. This is particularly important during hot weather, when proliferation of bacteria is increased. Designing calf milk storage and delivery systems to conform to safe food standards reduces the risk of disease transmission. Pasteurization is also effective at eliminating Salmonella and other bacterial contamination of milk, and when implemented properly, provides significant risk management. Inclusion of quality control monitors is important to detect failures in the pasteurization process. Pasteurizers can also be difficult to clean, so it is appropriate to implement routine bacterial (total plate) counts for calf milk (after pasteurization) to verify that the system is functioning according to expectations. Feeding utensils and personnel often play a significant role in transmitting pathogens between calves, and adequate cleaning is essential.

**Vaccination to Minimize Diarrhea**

Although there is very little published science in this area, in my opinion every pregnant dairy cow should be given a killed rotavirus/coronavirus vaccine containing a K99 E. coli toxoid prior to calving. These products are designed to be administered prior to calving and will boost colostral antibody titers to these enteric pathogens. Although this certainly does not guarantee complete protection from diarrhea, colostral antibodies have been shown to be the most effective way of producing immunity for these pathogens. Colostral antibodies provide local (mucosal) protection in the small intestine, and absorbed serum IgG1 can be re-secreted from blood back across mucosal surfaces for an extended period of time to help prevent enteric diseases.
There is very little published data available on oral vaccines that are designed to be given to calves at birth to boost immunity to enterotoxigenic *E. coli*, rotavirus and/or coronavirus. Oral vaccines such as First Defense® and Calf-Guard® are intended to be given to calves immediately following birth to prevent diarrhea caused by rotavirus and/or coronavirus. Several field studies using these products have shown them to be ineffective in controlling calf diarrhea due to either virus on farms. In challenge studies, oral vaccines did offer some protection against viral diarrhea when given to specific pathogen-free (colostrum-deprived) calves. However, when the vaccine was given to calves that also got colostrum, it failed to offer any protection against the development of diarrhea. These studies demonstrated that the vaccine virus present in these products is inactivated by antibodies in the colostrum causing failure of these products under most field conditions. I do not recommend using oral vaccines to prevent rotavirus or coronavirus diarrhea in calves and believe vaccination of the dry cow and ingestion of colostral antibodies to be a much better approach.

In conclusion, calf diarrhea is largely a management disease that can be prevented by having a good colostrum program and maintaining good cleanliness on the farm. Since fecal-oral is the main route of contamination for most pathogens and infected calves are the reservoir for enteric pathogens, limiting contact between young calves is critical. Risk factors for calf diarrhea include dirty calving pens, inadequate colostrum ingestion, nursing dirty teats, unsanitary feeding utensils (nipples, bottles), and group housing, particularly with continuous rearing (not all in, all out), overcrowding, and failure to isolate sick calves.
This paper provides an overview of the key elements of metabolism and inflammatory and immune response in peripartum dairy cows. Dairy production is challenged by the fact that 30 to 50% of dairy cows are affected by some form of metabolic or infectious disease around the time of calving. Essentially all peripartum dairy cattle experience a period of insulin resistance (IR), reduced feed intake, negative energy balance, lipolysis and weight loss in early lactation; reduced immune function for 1 to 2 weeks before, and 2 to 3 weeks after calving; and bacterial contamination of the uterus for 2 to 3 weeks after calving. These factors, as well as dramatic changes in circulating progesterone, estrogen, and cortisol concentrations contribute to a substantial reduction of immune function, in particular of neutrophils, at this time (Kehrli et al. 1989; Goff et al. 1997). Innate immunity from neutrophils is a primary means of immune response in the udder and uterus, and neutrophil migration and phagocytic and oxidative activity are associated with the risk of retained placenta (RP) (Kimura et al. 2002), metritis, and endometritis (Hammon et al. 2006). Yet, while metabolic disease is excessively common, only a minority of cows experience these problems, even with a herd in which cows apparently have similar nutritional and management experiences.

Adipose tissue is metabolically active and secretes proinflammatory signals (tumor necrosis factor (TNF)α and interleukin (IL)-6; Tilg, Moschen 2005). Many inflammatory mediators block the intracellular signalling of insulin (Hotamisligil 2006) and so contribute substantially to IR. Oxidative stress is also known to contribute to this process. Many of the same inflammatory mediators are implicated in uterine disease. IL-1, IL-6, and IL-10 mRNA are differentially expressed in cows that have endometritis (Herath et al. 2009). An excessive proinflammatory state appears to be a key feature of cows with endometritis (Sheldon et al. 2009), but it is unclear what initiates or sustains this.

**Growth Hormone, Insulin, IGF-1 and Insulin Resistance**

The mechanisms of partitioning and energy and fat metabolism in dairy cows have been summarized by Lucy (2008). Many of the changes are in support of making glucose available to the mammary gland for milk production. Briefly, growth hormone (GH) increases before calving, causing increased gluconeogenesis in the liver and increased lipolysis of adipose. Blood insulin levels are low, and the liver, muscles, and adipose are insulin resistant, sparing (partitioning) glucose for the udder where glucose uptake is insulin independent. Blood glucose levels are low despite increased gluconeogenesis because of the massive drain of glucose to the udder. In simple terms, higher production is associated with higher blood GH and lower insulin concentrations. Growth hormone causes secretion of insulin-like growth factor (IGF)-1 from the liver. A unique adaptation in dairy cattle is that about 2 days before calving, GH receptor expression in the liver is reduced and remains low for approximately 2 weeks. During this time, with low IGF-1 to feed back against GH secretion from the hypothalamus, circulating GH levels are high, favouring lipolysis and driving characteristic NEFA increases. NEFA may be used as an alternative fuel source to glucose in peripheral tissues or incorporated directly into milk fat. Elevated levels of GH and NEFA contribute to IR. The temporary decrease in GH receptor with increased GH secretion is referred to as uncoupling of the somatotrophic axis. The mechanism for decreased GH receptor expression is not clear, but increased GH receptor expression and ‘recoupling’ appear to involve or depend on insulin, apparently as an indicator of energy balance. The nadir of negative energy balance (NEB) may occur around 10 DIM (Butler 2000), although NEB typically lasts for 6 weeks (Grummer 2008). Eventually glucose supply exceeds demand, at which time increased circulating glucose triggers increased insulin, which increases liver GH receptor expression, in turn increased IGF-1 levels and feedback to lower GH secretion, ending the homeorhetic changes to support peak lactation.

The associations of these processes with return to ovulation have been well studied, and reviews are available (e.g., Butler 2000; Lucy 2008). Glucose, NEFA, insulin, and IGF-1 all provide signals to influence...
secretion of LH, which appears to be the determining factor in progression to the first postpartum ovulation. Much less has been described about the involvement of these signals and processes with development of metritis or endometritis. Cows with ketosis throughout the first week postpartum, and apparently more so in those that also had metritis, had higher NEFA starting 2 days before calving, and lower IGF-1 and insulin 1 day before calving, continuing through the first week postpartum (Kerestes et al. 2009). Cows with chronic ketosis with or without metritis had lower peak insulin response to glucose at 7 DIM (i.e., indication of reduced pancreatic insulin secretion). All cows had reduced glucose response to injection of insulin at 7 DIM, but cows with chronic ketosis (with or without metritis) had a smaller drop in glucose in response to injection of insulin, i.e., were more insulin resistant (Kerestes et al. 2009).

**Immune Function**

The mechanisms of impairment of immune defence in the mammary gland in the transition period have been described (Burvenich et al. 2007). Less is known about the determinants of uterine health or how resistance to uterine disease may be enhanced through animal management. Uterine immunity in dairy cows has been reviewed (Sheldon et al. 2009; LeBlanc et al. 2011; LeBlanc 2012). It is known that cows with severe metritis ate less (2 to 6 kg of dry matter per day) than healthy cows in the 2 to 3 weeks preceding the clinical signs of metritis (Huzzey et al. 2007). Lower feed intake is associated with increased circulating concentrations of nonesterified fatty acids (NEFA), which may directly (Scalia et al. 2006; Ster et al. 2012) or indirectly (Zerbe et al. 2000; Hammon et al. 2006) inhibit neutrophil function. Because of both high metabolic demands and pathogen challenges, cattle also routinely experience substantial oxidative stress in early lactation (Sordillo, Aitken 2009), which also contributes to a proinflammatory state that may not be effective for immune defence (Hotamisligil, Erbay 2008).

Retained placenta is a disease of immune function, with changes in neutrophil function and IL-8 levels two weeks before calving (Kimura et al. 2002). Cows in a greater degree of negative energy balance prepartum and those with lower circulating vitamin E were at greater risk of RP (LeBlanc et al. 2004). Recruitment and function of an adequate flux of neutrophils to the uterus are also important in the days after calving for clearance of bacteria and lochia and prevention of subsequent endometritis (Gilbert et al. 2007). However, there is evidence that substantially higher, apparently excessive, inflammatory status in the first (Herath et al. 2009) and perhaps second (Chapwanya et al. 2009) week postpartum is associated with endometritis.

Measurable changes were noted in phagocytosis, TNFα and IL-6 prepartum in cows with postpartum endometritis (Kim et al. 2005), weeks before disease becomes manifest, coincident with the onset of insulin resistance and lipolysis (at least in cows at higher risk of disease). Worse postpartum negative energy balance is associated with more severe or prolonged uterine inflammation and impaired tissue repair capacity (both measured by gene expression; Wathes et al. 2009). Hammon et al. (2006) showed that cows with metritis or cytological endometritis had worse neutrophil killing capacity than did unaffected cows, and these changes preceded disease by several weeks. They also reported associations between increasing NEFA concentration, especially in the last week before calving, and lower neutrophil oxidative activity, which has been replicated by others (Ster et al. 2012). Additionally, Hammon et al. reported an association between lower feed intake in the 3 weeks before calving and lower neutrophil killing capacity from the week before until 3 weeks after calving.

Neutrophils rely primarily on glucose uptake or glycolysis for chemotaxis; however, glycogen stores are necessary for phagocytosis and oxidative burst, even in the presence of glucose (Galvão et al. 2010). Intra-neutrophil glycogen was lower at calving in cows that had metritis than in healthy cows, and lower at weeks 1, 4 and 6 postpartum in cows with endometritis than in healthy cows (Galvão et al. 2010).

**Fat Metabolism and Fatty Liver**

There is a rapidly growing body of information in human medicine, based on studies in rodents and in people, on interactions among metabolism (specifically related to insulin and fat), inflammation, and immune function (e.g., Osborn, Olefsky 2012). Fat has been described as the “master regulator in the development of systemic insulin resistance” (Osborn, Olefsky 2012). Fat releases NEFA but also glycerol and proinflammatory cytokines (TNF, IL-6). These phenomena are being investigated in dairy cows
where they appear to be central to health in the transition period (Bradford 2011). Antiinflammatory treatments may represent an ‘upstream’ approach to prevention or control of IR (Bertoni et al. 2009).

Increased intracellular accumulation or decreased oxidation of NEFA leads to buildup of FA metabolites which block the action of insulin. Therefore, NEFA mobilization and fatty liver lead to insulin resistance, especially in the liver (Kahn et al. 2006).

Circulating numbers of leukocytes increase to peak at or just after calving, and these counts were similar between cows with and without fatty liver (> 40 mg fat/g liver in the first 2 weeks postpartum) (Zerbe et al. 2000). However, neutrophil function surface markers declined after calving in cows with fatty liver whereas they generally recovered or increased above prepartum levels by 10 DIM. Phagocytosis did not change through the transition period (also confirmed by Ster et al. 2012) and was not associated with fatty liver. There were trends or significant differences (particularly at 7 to 10 DIM) for reduced generation of reactive oxygen species and decreased lytic capacity of blood neutrophils from cows with fatty liver. Generally, neutrophils flushed from the uterus in the first week postpartum had lower functional capacity compared to circulating neutrophils at the same time (Zerbe et al. 2000). Similar tendencies to the case of blood PMN were observed in the uterus; i.e., apparently lower function in cows with fatty liver, especially in the first 10 days after calving.

**OXIDATIVE STRESS**

Inflammation (phagocytosis and intracellular digestion) inherently produces reactive oxygen species (ROS) and creates a burden of oxidative stress, which increases proinflammatory output from these cells. LPS interacts with TLR4 to result in increased production of proinflammatory cytokines TNFα, IL-1, and IL-8, which form part of the response to gram-negative bacteria. However, at least the TNFα response is heightened when antioxidant status is lower or oxidative stress is greater, e.g., in the peripartum period (Sordillo et al. 2009). It is not clear if this results in a more effective response or just the possibility of increased bystander tissue injury or unintended consequences such as increased IR. Optimization of antioxidant status (e.g., with supplementation with selenium, vitamin E, retinol, or polyunsaturated fatty acids) may help to keep immune responses effective and prevent excessive inflammation or its side effects.

**REFERENCES**


OBJECTIVES OF MONITORING PROGRAMS
Routine and proactive actions, observations, or analysis are intended to accurately and efficiently provide early detection of problems, to provide an opportunity for investigation and intervention in order to limit the consequences and costs of health problems and reduced animal performance or welfare. There are two main reasons for monitoring transition dairy cows in general and running metabolic tests in particular. The objectives overlap, but are distinct and should be clear before embarking on a program. The objectives are: herd or group level - to monitor the success of current management with the goal of early detection of problems or deviation from the management program; individual level - to identify cows at high risk for disease with the goal of intervention for these individuals to prevent or mitigate clinical disease.

METHODS OF MONITORING
The principles and practice of screening programs for fresh cows have been well described (Guterbock 2004).

Clinical Disease Records
A starting point for assessment of peripartum health is to have accurate records of the farm-specific incidence of the clinical diseases of importance to the herd. This would typically include the number of cattle that had dystocia, RP, milk fever, metritis, and DA, or that were culled or died in early lactation divided by the number of cows that calved in a defined time period. The incidence of clinical mastitis and lameness per month or other time period is also useful although complicated by the risk period extending throughout lactation and the possibility for multiple occurrences in the same cow, which may not be independent events. For all diseases, it is important that the case definitions be clear, mutually exclusive, and consistently recorded. The records should allow for measurement of the incidence of the condition of interest, not the treatment rate (e.g., if some cases of RP are not treated, the disease event should still be recorded; conversely, if a condition is treated for 3 days, there should only be 1 recorded occurrence of the disease (as opposed to 3 treatment events). Investigation of the pattern of affected animals and the risk factors for disease is suggested if the following crude lactational incidence risks are exceeded: dystocia > 20%; RP > 10%; milk fever > 2%; metritis > 10%; DA > 5%. However, herd size, demographics, and management influence the expected incidence, so reference to herd-specific goals and recent history are more useful than broad benchmarks. Also, tracking the rates of clinical disease is necessarily retrospective and therefore at best allows for reaction to problems rather than early warning. Finally, clinical disease is typically only the “tip of the iceberg” with respect to health problems, and therefore these records underestimate the prevalence of potentially performance-limiting health conditions. For example, the incidence of clinical ketosis is typically reported at 5 to 10% (Kelton et al. 1998; Zwald et al. 2004), but if measured systematically, the incidence of subclinical ketosis in the first 3 weeks of lactation is approximately 40% (Duffield et al. 1998). Trends in the prevalence of culling in early lactation may also provide an additional element of herd-level information (Nordlund, Cook 2004).

Measurement of Feed Intake
Adequate feed intake by all peripartum cattle is crucial for health and production. It is therefore desirable to measure feed dry matter intake (DMI) in prepartum and early postpartum cows. Although measurement of only group average intake may be feasible in commercial free stall barns, that is still likely to be useful information. For example, if there is < 2% of feed remaining prior to the first feeding of the next day, then ad libitum intake is likely not to be achieved by all animals in the group. Assessment of individual feed intake and lack of competition for feed access are advantages of tie stall housing.
Milk Production
Milk production is expected to increase rapidly in early lactation, and a consistent rise should result from good health and feed intake. Therefore, automated daily measurement of milk production in the first few weeks of lactation offers promise as a means to identify cows with clinical or subclinical health problems. The variability of daily milk yield is high, especially in early lactation, and is influenced by many factors beyond health (e.g., weather, changes in diet, movement of cattle to new groups, etc.). However, decreased milk production often precedes clinical disease, and daily yield coupled with activity monitoring may be useful for screening of cows for earlier disease detection (Edwards, Tozer 2004). Trends in projected production from early lactation provide herd-level information on the success of transition into lactation (Nordlund, Cook 2004). New tools to compare expected to actual production in early lactation (Transition Cow Index®; Nordlund 2006) may also help to quantify the impact of suboptimal peripartum health and management.

Body Condition Scoring
Body condition scoring provides a rapid, simple, and acceptably precise estimate of body fatness (Edmonson et al. 1989; Ferguson et al. 1994). It reflects the nutritional, metabolic, and to some extent health history of a cow in the preceding weeks. While cows that calve in fat body condition, or moreover cows that lose 1 point or more of BCS in early lactation, are often reported to be at higher risk of adverse outcomes, BCS alone (other than extremes, i.e., > 4 or < 2.5 at calving) is not a sensitive or specific tool for prediction of disease or reproductive performance. Recent research has suggested that the target BCS at calving should likely be lower (≤ 3.0) than previously advocated to optimize health and production (Garnsworthy 2008).

Screening Cows for Uterine Disease
The pathophysiology (Sheldon 2008), diagnostic criteria and treatment for metritis have been reviewed elsewhere (LeBlanc 2008). Briefly, metritis may practically be identified based on at least 2 of fetid discharge, fever, and signs of systemic illness (dullness, inappetence, or decreased milk production). Daily monitoring of rectal temperature for 7 to 10 days after calving may increase the rate of diagnosis of metritis, and if this practice is implemented it should not be the sole basis for treatment with antibiotics. Routine, systematic screening of fresh cows is likely useful to increase early detection of health problems, especially in large herds; but it is likely most useful if training and experience of personnel and facilities allow for assessment of the cows’ attitude, appetite, ketosis status (once or twice weekly), rumination, and abomasal displacement.

Accurate diagnosis of purulent vaginal discharge (“clinical endometritis”) requires examination of discharge in the vagina after a minimum of 3 weeks postpartum (LeBlanc et al. 2002), which may be done with a vaginoscope, clean gloved hand, or a Metricheck device (Pleticha et al. 2009). Subclinical endometritis is common and has substantial impacts on reproductive performance (Gilbert et al. 2005). Subclinical endometritis is diagnosed by endometrial cytology obtained transcervically either by uterine lavage or cytobrush (Barlund et al. 2008). Neither technique is sufficiently rapid or practical for widespread use in clinical practice, although rapid cow-side tests have been explored.

Metabolites to Measure Energy Status in Transition Cows
Circulating concentrations of NEFA and β-hydroxybutyrate (BHB) measure aspects of the success of adaptation to negative energy balance. The concentration of NEFA reflects the magnitude of mobilization of fat from storage and mirrors DMI (Adewuyi et al. 2005), while BHB reflects the completeness of oxidation of fat in the liver. Ketone bodies (BHB, acetone and acetoacetate) are the intermediate metabolites of oxidation of fatty acids, specifically resulting from the incomplete oxidation of fatty acids. As the supply of NEFA to the liver exceeds the ability of liver to completely oxidize the fatty acids to supply energy, the amount of ketone production increases. Ketone bodies can be used by muscle as an alternative fuel source to glucose, sparing glucose for milk production (Herdt 2000). However, ketone production does not result in as much net energy release as complete oxidation of fatty acids. Additionally, increasing concentrations of ketones are thought to suppress feed intake (Allen et al. 2009).
Glucose is the primary metabolic fuel and is absolutely required for vital organ function, fetal growth, and milk production. In dairy cows, the massive energy demand to support milk production is largely met through gluconeogenesis. Glucose concentrations are under tight homeostatic control. Therefore, although glucose has a central role in metabolism, it is a poor analyte for monitoring or investigating herd problems (Herdt 2000).

**NEFA**

In a large multi-region field study, NEFA ≥ 0.3 mmol/L was associated with increased incidence of RP (Chapinal et al. 2011). Similarly, as NEFA in the week before calving increased by 0.1 mmol/L, the odds of RP increased by 5% (Quiroz-Rocha et al. 2009). Cows with NEFA ≥ 0.3 (0.2 in one study region) mmol/L in the week before calving were more likely to develop metritis (OR = 1.8) (Chapinal et al. 2011). Similar large field studies (Ospina et al. 2010a, b) confirm that NEFA > 0.3 mmol/L in the 1 to 2 weeks before expected calving is associated with increased risk of RP, metritis, or displaced abomasum (DA), decreased milk production (1.6 kg/day [Chapinal et al. 2012] or 683 kg 305 d mature equivalent) and increased time to pregnancy. Similarly, in the 2 weeks after calving NEFA > 0.6 mmol/L was associated with increased risk of metritis or DA, and NEFA > 0.7 mmol/l was associated with longer time to pregnancy and with 650 kg less milk in multiparous cows (Ospina et al. 2010a, b). Dubuc et al. (2010) found that NEFA ≥ 0.6 mmol/L in the week before calving was associated with increased odds of metritis (OR = 1.6) but not with purulent vaginal discharge (PVD) or endometritis.

There was substantial and dose-dependent decrease of proliferation of blood mononuclear cells and their production of IFN\(\gamma\) in vitro as well as decreased neutrophil oxidative burst activity with addition of NEFA to reflect levels in the first week postpartum (Ster et al. 2012). The effects on monocytes were present as low as 0.013 mmol/L NEFA and started at 0.5 mmol/L for neutrophil oxidative burst.

**Ketosis**

Cows with milk BHB > 100 µmol/L in the first week postpartum were 1.5 times more likely to be anovular at 9 weeks postpartum (Walsh et al. 2007a). Cows that experienced ketosis in the first two weeks of lactation had reduced probability of pregnancy at the first insemination. Furthermore, cows that had ketosis in one or both of the first two weeks after calving had a lower pregnancy rate until 140 DIM. The median interval to pregnancy was approximately 108 days in cows without ketosis, was significantly longer (124 days) in cows with ketosis in the first or second week postpartum, and tended to be longer still (130 days) in cows that had subclinical ketosis in both of the first weeks of lactation (Walsh et al. 2007b).

Subclinical ketosis (BHB > 1.2 to 1.4 mmol/L) in the first or second week after calving was associated with 3 times greater risk of metritis (Duffield et al. 2009). Milk yield at first test was reduced by 1.9 kg/d when BHB was > 1.4 mmol/L in week 1 and by 3.3 kg/d when BHB was > 2.0 mmol/L in week 2. Cows with serum BHB > 1.8 mmol/L in week 1 had > 300 kg lower projected production for the whole lactation. A herd prevalence of > 15% of cows with prepartum NEFA > 0.3 mmol/L, postpartum NEFA > 0.7 mmol/L, or BHB > 1.15 mmol/L was associated with increased herd risks of DA or clinical ketosis, lower pregnancy rate, and decreased herd average milk production (Ospina et al. 2010c).

In a large field study in NY (778 cows in 38 herds), Cheong et al. (2011) reported that producer-recorded clinical ketosis (incidence = 5%) was a risk factor for SCE (OR = 3.8), particularly in multiparous cows. However, in an even larger study, Chapinal et al. (2011) found no association of producer-reported clinical ketosis or serum BHB measured systematically in week 1 postpartum with metritis. In a study with 1295 cows, Dubuc et al. (2010) found that ketosis (BHB > 1.1 mmol/L) in week 1 postpartum was a risk factor for endometritis (OR = 1.4) but not for PVD or for metritis. Plasma BHB was higher at calving that developed metritis, and similar to Dubuc et al. (2010), higher at week 1 postpartum in cows that later had endometritis (Galvão et al. 2010). Likewise, cows with metritis or endometritis had higher BHB from 1 until 4 weeks after calving, although there was no association of BHB with neutrophil killing ability (Hammon et al. 2006). In vitro titration of BHB did not affect proliferation of blood mononuclear cells or their production of IFN\(\gamma\), or oxidative burst activity of neutrophils (Ster et al. 2012). Therefore, the effect of ketones per se on immune function is at best inconsistent. It is not clear if the mechanism of fatty liver/ketosis association with diminished neutrophil function is direct (and if so whether it is on mature
PMN in circulation, or whether NEFA, ketones or other signals or metabolites affect PMN in the bone marrow, or through effects on mononuclear cells that are responsible for antigen presentation and initial chemokine signalling/stimulation of neutrophils (Zerbe et al. 2000).

**Hypocalcaemia**

Essentially all cows experience some degree of hypocalcaemia at calving and for 1–3 days after. There are conflicting data about thresholds of circulating calcium concentrations that may be associated with undesirable outcomes. Recently, we have shown that serum calcium concentrations < approximately 2.2 mmol/L in the week after calving, despite being within the range for healthy cows, was associated with increased odds of displaced abomasum, approximately 3 kg/d lower milk yield in early lactation, and slightly decreased odds of pregnancy at first insemination (Chapinal et al. 2011; Chapinal et al. 2012).

In large field studies, no association of milk fever was found with metritis, PVD, or endometritis (Dubuc et al. 2010; Cheong et al. 2011). Chapinal et al. (2011) also found no association of serum calcium measured in week 1 (but before disease diagnosis) with the odds of metritis. Similarly, in pastured cows, Burke et al. (2010) found no association of plasma calcium through the peripartum period with endometritis at week 6, but did find that plasma magnesium was significantly lower (at 2 and 4 weeks postpartum) in cows with endometritis. However, Martinez et al. (2012) studied 110 cows in one herd in Florida, USA. Cows with Ca < 2.14 mmol/L at least once between 0 and 3 DIM had 4.5-fold increased odds of developing metritis. Attributable risk of metritis for hypocalcemia was 75%. Se = 89; Sp = 55%. Hypocalcaemia was also associated with decreased neutrophil oxidative burst and decreased circulating neutrophil counts at 1 and 3 DIM.

**Haptoglobin**

Haptoglobin (Hp) is an acute-phase protein produced by the liver and associated with several inflammatory and disease conditions in cattle. Huzzey et al. (2009) found that Hp > 1.0 g/L at 3 DIM was preceded and increased the incidence of metritis (OR = 7). Haptoglobin ≥ 0.8 g/L in week 1 postpartum was associated with increased risk of metritis (OR = 2.2), PVD (OR = 2), and endometritis (OR = 1.6) (Dubuc et al. 2010). Consistent with that, Galvão et al. (2010) also found slightly higher Hp at week 1 postpartum in cows that later had endometritis.

**TESTING STRATEGIES AND INTERPRETATION**

**Test Selection and Sampling**

Serum or plasma NEFA concentrations measured in the week before calving (samples taken 4 to 10 days before expected calving) provide a uniquely useful component of assessment of peripartum health. Unfortunately, there are presently no on-farm diagnostic tests for measurement of NEFA, which implies the cost and delay of submission of samples to a diagnostic laboratory. The concentration of NEFA typically begins to rise 2 to 4 days before and peaks approximately 3 days after calving, but the magnitude of increase is greater, and the increase starts earlier in cows that subsequently experience metabolic disease (LeBlanc et al. 2005). NEFA concentrations peak just before feeding (Herdt 2000). In cows fed TMR, there is a greater prevalence of elevated serum NEFA 1 h before first feeding than at 4 or 10 h after feeding, but serum BHB concentrations are quite stable across these time points. For monitoring, samples should be collected at approximately the same time of day to avoid confounding of the results by diurnal or postprandial variations.

In a field study of 1010 cows in 25 herds in Ontario, the peak incidence (first diagnosis of new cases) of subclinical ketosis was 30% and occurred in the first week after calving, with few new cases beyond the third week postpartum (Duffield 2000). The cumulative incidence to 9 weeks postpartum was 43%. The mean incidence varied among the 25 herds from 8 to 80%. Therefore, the first two weeks postpartum are the optimal time to test for subclinical ketosis. The median overall prevalence in the first two weeks was approximately 20%. Diagnosis rates of clinical ketosis are commonly a reflection of the diagnostic criteria used (which may not be valid) and the intensity and consistency of efforts to apply these criteria. Accordingly, treatment rates for clinical ketosis often do not reflect the true incidence of ketosis (Duffield 2000; Oetzel 2004).
Ketosis is associated with management in the pre-fresh, maternity, and early post-fresh periods. Practically, recognition of when ketosis is occurring should give direction to preventive efforts. When ketosis is detected primarily in the first two weeks postpartum, emphasis should be placed on bringing cows to the dry period in moderate body condition (BCS = 3 to 3.5), avoidance of excess energy intake between dry-off and 3 weeks prepartum (Dann et al. 2006; Drackley 2007), and particularly on measures to enhance feed intake in the last few weeks before and through the calving period. Further investigation of an elevated prevalence of ketosis in early lactation may be aided by NEFA testing of cows in the 10 days before expected calving. If there is little evidence of ketosis in the first two weeks postpartum, but an increased incidence 3 to 6 weeks postpartum, that suggests that preventive measures should emphasize enhancing feed intake in post-fresh period. Ketosis that occurs later than the first two to three weeks of lactation may also be associated with failure to meet the nutritional needs of cows with high production, or with poorly fermented wet grass or legume silage with high levels (> 0.5 to 1% of dry matter) of butyric acid. Further research to describe the occurrence of subclinical ketosis under different management conditions is warranted. Until such data are available, present evidence indicates that most subclinical ketosis occurs in the first two weeks after calving. Therefore, testing programs with the objective of monitoring the prevalence of subclinical ketosis should focus on the first two weeks after calving.

Used with knowledge of their test characteristics to inform interpretation, serum BHB, whole blood BHB measured with Precision XTRA®, milk BHB measured with Keto-Test®, or Ketostix® in urine (Table 1) are valid diagnostic tests for subclinical ketosis. These 3 cow-side tests are economical, practical and sufficiently accurate relative to laboratory analysis of serum for use in the field. Selection of the 100 or 200 µmol/L cut-point for classification of the Keto-Test® will depend on the objective of the testing. If the objective is group-level monitoring for early detection of increased prevalence of ketosis (as a reflection of the general success of transition management), then greater sensitivity is desirable and the 100 µmol/L should be used. If the objective is to select individual cows for treatment with the goal of preventing clinical disease, then fewer false positives may be desirable and the 200 µmol/L cut-point would be appropriate.

Table 1. Performance of cow-side tests for detection of subclinical ketosis

<table>
<thead>
<tr>
<th>Test subset</th>
<th>Blood</th>
<th>Milk</th>
<th>Urine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preferred test</td>
<td>Precision XTRA (MediSense, Abbott)</td>
<td>Keto-Test (KetoLac BHB) Sanwa Kagaku Kenkyusho Co.</td>
<td>Ketostix (Bayer)</td>
</tr>
<tr>
<td>Sensitivity*</td>
<td>87–93%</td>
<td>At 100 µmol/L on the strip: 83% At 200 µmol/L: 82%</td>
<td>At “small” level, when read after 5 seconds 79%</td>
</tr>
<tr>
<td>Specificity*</td>
<td>93–100%</td>
<td>At 100 µmol/L on the strip: 54% At 200 µmol/L: 94%</td>
<td>96%</td>
</tr>
<tr>
<td>Approximate cost</td>
<td>$2/test; $40 for the meter</td>
<td>$2/test</td>
<td>$0.25/test</td>
</tr>
<tr>
<td>Validation</td>
<td>Iwerson et al. 2009</td>
<td>Summarized by Oetzel 2004</td>
<td>Carrier et al. 2004</td>
</tr>
</tbody>
</table>
| Comments | Glucose tests available for this meter are less accurate in cattle than the BHB strips. | Bioketone® powder (relative to serum BHB ≥ 1200 µmol/L): sensitivity = 28% and specificity = 100% (Geishauser et al. 2000). The lack of sensitivity (too many false negatives) makes this test unsuitable for monitoring programs. | Typically only able to induce 50% of cows to urinate when sampling. Acetest® tablet sensitivity = 100% but specificity = 59% (Nielen et al. 1994). The lack of specificity (too many false positives) makes this
* Relative to serum BHB ≥ 1400 μmol/L measured in a diagnostic laboratory

Test characteristics varied somewhat among studies, apparently largely as a function of the prevalence of subclinical ketosis among the cows being tested. As the prevalence increases, the sensitivity is generally greater and the specificity lower. For confirmation of a diagnosis of clinical ketosis, all of these tests would be acceptable, and the performance of milk powders is also adequate.

**Sample Handling**

Serum or plasma is acceptable for BHB and NEFA testing. BHB may be falsely elevated by hemolysis in the sample (Duffield 2000), and NEFA results may not be accurate if there is more than mild hemolysis. NEFA concentrations could be slightly falsely elevated if serum were not separated within 12–24 h of blood collection, or if samples were not kept chilled (Stokol, Nydam 2005). Serum can be kept frozen for at least 1 month without affecting NEFA results. Samples should be collected from the tail vein (not the milk vein) and ideally chilled, separated within a few hours, and then frozen or shipped chilled for receipt at the laboratory within 1 to 2 days. However, delay of up to 24 hours for separation, and maintenance at room temperature for 1 day or refrigerated for < 3 days does not substantially affect results (Stokol, Nydam 2005).

**Number of Samples and Interpretation**

The number of samples required for group or herd-level interpretation depends on the prevalence of affected animals that is judged important to detect, the certainty of detection that is desired, and the size of the group of interest (Dohoo et al. 2003). Fortunately, the latter criterion has the least influence. Examples are given in Table 2. Practically, the minimum number of samples is 5, and 10 to 12 samples will allow for interpretation in most situations. Typically, 18 to 35% of cows have NEFA > 0.4 mmol/L in the last week before calving (Oetzel 2004; LeBlanc 2005). Published reports indicate a typical prevalence of subclinical ketosis of around 15% (Oetzel 2004); studies in Canada have found average prevalence of 20% (Duffield et al. 1998; Duffield et al. 2003). Adjusting for cow-side test performance, a threshold of 10% true prevalence of subclinical ketosis corresponds to an apparent prevalence (proportion of tests that are positive) of 25% when using the Keto-Test® with a 100 μmol/L cut-point, or 11% at the 200 μmol/L cut-point (Oetzel 2004).

**Table 2. Examples of the number of cows that need to be sampled as part of a routine monitoring program or to investigate a suspected problem of ketosis in a dairy herd**

<table>
<thead>
<tr>
<th>Number of animals in the group of interest (for ketosis, typically the first 2, or at most 5, weeks of lactation)</th>
<th>Estimated prevalence of the condition (What prevalence is it important to detect?)</th>
<th>Confidence (How certain do you wish to be of detecting that the prevalence of interest has been reached?)</th>
<th>Number of samples required</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>10</td>
<td>75</td>
<td>8</td>
</tr>
<tr>
<td>10</td>
<td>10</td>
<td>95</td>
<td>10</td>
</tr>
<tr>
<td>10</td>
<td>20</td>
<td>75</td>
<td>5</td>
</tr>
<tr>
<td>10</td>
<td>20</td>
<td>95</td>
<td>8</td>
</tr>
<tr>
<td>50 to 1000</td>
<td>10</td>
<td>75</td>
<td>12</td>
</tr>
<tr>
<td>50 to 1000</td>
<td>10</td>
<td>95</td>
<td>14</td>
</tr>
<tr>
<td>50 to 1000</td>
<td>20</td>
<td>75</td>
<td>6–7</td>
</tr>
<tr>
<td>50 to 1000</td>
<td>20</td>
<td>95</td>
<td>12–14</td>
</tr>
<tr>
<td>50 to 1000</td>
<td>30</td>
<td>95</td>
<td>9</td>
</tr>
</tbody>
</table>
It is important to interpret both NEFA and ketosis tests as the proportion of animals above a meaningful threshold, because this best describes the biology of the condition. It is misleading to calculate the average BHB or NEFA from a group of samples, and similarly most information is lost if samples from multiple animals are pooled together.

**CONCLUSIONS**

Undesirably elevated concentrations of NEFA and subclinical ketosis are prevalent and important conditions associated with increased risk of metabolic and uterine disease, decreased milk production, and decreased reproductive performance. Measurement of the prevalence of subclinical ketosis in the first 2 weeks of lactation is useful for investigation of herd problems of transition cow health and performance, and for routine monitoring. The timing, magnitude, and duration of peripartum increases in circulating concentrations of NEFA and BHBA are associated with the risk of abomasal displacement, uterine disease, and reproductive performance from 1 through 20 weeks later. Programs to monitor management of the transition period may usefully include NEFA concentrations in the week before expected calving and BHB concentration in the first week after calving. A key link among diseases is feed intake. Peripartum energy metabolism and immune function will plausibly be favoured when cows have unrestricted access to diets formulated to meet nutrient requirements and to water in the transition period. Proactive management and investigation of problems should focus on minimizing nutritional, housing, social, and environmental factors that may impair feed and resting access for all or some members of the groups of peripartum cows.

**REFERENCES**


INFLAMMATION IN THE REPRODUCTIVE TRACT

Endometrial inflammation appears to be an inevitable and necessary part of involution, but down-regulation of the immune response within a few weeks after calving appears to be important, and apparently excessive inflammation even in the first week postpartum is associated with persistent and deleterious inflammation one month later (Herath et al. 2009). It is not clear if excessive or persistent inflammation is provoked by the type (species, strain or virulence factors) or quantity of bacterial infection (LeBlanc et al. 2011), by genetic or metabolic influences on immune function and regulation, or both. While the risk factors and pathophysiology of PVD and cytological endometritis are at least partly shared, uterine and cervical tissue trauma and bacterial infection appear to have a greater role in PVD, while regulation of the immune response appears to have a greater role in cytological endometritis.

Recruitment and function of an adequate flux of neutrophils to the uterus is also important in the days after calving for clearance of bacteria and lochia and prevention of subsequent endometritis (Gilbert et al. 2007). An excessive proinflammatory state appears to be a key feature of cows with endometritis. Proinflammatory cytokines (IL-1, IL-6, and IL-10 mRNA) are more expressed in cows that have endometritis (Chapwanya et al. 2009; Sheldon et al. 2009; Herath et al. 2009), particularly in the first and second weeks after calving in cows diagnosed with endometritis at week 4 or 5 (reviewed in LeBlanc 2012). What is not known is what sets up this excessive inflammatory status.

An important concept is that reproductive tract disease represents a failure of the local (and perhaps systemic) immune system to switch fast enough or far enough from the down-regulated state necessary for maintenance of pregnancy to a heightened state of function for postpartum clearance of bacteria and tissue debris, and back away from active inflammation 3 to 4 weeks later. A desirable response appears to be a prompt, substantial (and presumably effective) flux of neutrophils into the uterus after calving.

ASSOCIATIONS OF BACTERIA WITH REPRODUCTIVE TRACT DISEASE

Most cows have bacterial infection of the uterus for several weeks after calving, but the relative importance of infection (the stimulus side of the inflammation equation) versus immune response (effectiveness and regulation of inflammation) is in question. Escherichia coli (E. coli) are particularly prevalent in the first week postpartum and are associated with metritis, with increased risk of infection with Arcanobacterium pyogenes in weeks 2 and 3, and with endometritis (Dohmen et al. 1995; Gilbert et al. 2007; Williams et al. 2005). Metritis and endometritis are commonly associated with mixed bacterial infection of the uterus, often including anaerobes, notably Fusobacterium and Prevotella species. Until recently, these pathogens have been assumed to be ‘generic’ or not specifically adapted to or associated with metritis or endometritis. Recent studies have explored the potential for specific virulence factors or strains of bacteria to be associated with uterine disease, and these data have recently been summarized (LeBlanc et al. 2011). Briefly, there are strains of E. coli that appear to be adapted uterine pathogens, particularly expressing virulence factors related to adhesion (Bicalho et al. 2010; Sheldon et al. 2010). New data (Bicalho et al. 2012) build the case that specific virulence factors in E. coli, A. pyogenes, and F. necrophorum are associated with metritis and PVD. It is generally considered that bacterial infection of the uterus initiates inflammation of the endometrium and perhaps deeper layers of the uterus. This inflammation is a normal adaptive response, but it may be inadequate for the task (i.e., the balance tips in favor of bacterial growth, adhesion, inflammation, and tissue damage rather than clearance and healing - insufficient response), or inflammation may be disproportionate in degree or duration (excessive response). It is not clear if excessive or persistent inflammation is provoked by the type (species, strain or virulence factors) or quantity of bacterial infection (LeBlanc et al. 2011), by genetic or metabolic influences on immune function and regulation, or both.
**Prevention of Reproductive Tract Disease**

Presently, there are few management practices or interventions that can be supported specifically to prevent metritis or endometritis. Based on current understanding of these diseases, the general objective is to support and maintain innate immune function and so reduce the risk that the inevitable inflammation and bacterial contamination after calving progress to metritis, endometritis, or cervicitis. Excessive negative energy balance and circulating free fatty-acid concentrations, and excessive insulin resistance contribute to a state of (metabolic) ‘meta-inflammation’ that may actually impair neutrophil function. While there is a great deal still to be learned about the determinants of immune function in dairy cattle in the transition period, and in particular about specific means to prevent uterine disease, Table 1 proposes management practices generally recommended for peripartum dairy cows that are likely to contribute to reducing the incidence of reproductive disease in the early postpartum period.

**Table 1. Summary of management practices and monitoring targets to reduce the risks of reproductive tract disease in dairy cows**

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevent consumption of dietary energy above requirement in the ‘far-off’ dry period (weeks 8 to 3 before calving)</td>
<td>Dann <em>et al.</em> 2006; Janovik <em>et al.</em> 2011</td>
</tr>
<tr>
<td>Provide for unrestricted feed bunk access (i.e., all animals able to eat at the time of fresh feed delivery) i.e., 75 cm of linear bunk space per cow or no more than 4 cows per 5 headlocks</td>
<td>Cook, Nordlund 2004; Nordlund 2010</td>
</tr>
<tr>
<td>Provide space to allow for lying 11 to 12 h per day ≥ 1 free stall per cow or 100 ft² (10 m²) of bedded pack per cow</td>
<td>Nordlund 2010; Cook 2007</td>
</tr>
<tr>
<td>Minimize pen moves and social group changes</td>
<td>Nordlund 2010</td>
</tr>
<tr>
<td>Build dry cow and fresh pens for approximately 130–140% of the expected average number of calvings per month</td>
<td>Nordlund 2010</td>
</tr>
<tr>
<td>Provide heat abatement (fans and sprinklers) when the temperature-humidity index exceeds 68</td>
<td>Smith 2012</td>
</tr>
<tr>
<td>Manage nutrition so that cows calve at BCS of 3.0 or 3.25 (on the 5-point scale) and maintain a minimum BCS of 2.5</td>
<td>Roche <em>et al.</em> 2009</td>
</tr>
</tbody>
</table>

**Monitoring methods and targets (serum or plasma tests)**

- NEFA < 0.4 mmol/L in the week before expected calving | Dubuc *et al.* 2010
- BHB < 1.1 mmol/L in week 1 and < 1.4 in week 2 after calving | Dubuc *et al.* 2010
- Haptoglobin < 0.8 g/L in week 1 after calving | Dubuc *et al.* 2010

Updated from LeBlanc *et al.* 2011

**Treatment of Reproductive Tract Disease**

Treatment of reproductive tract disease has been reviewed (LeBlanc 2008; Lefebvre, Stock 2012). There is consistent evidence that cows with PVD have improved reproductive performance when treated with a single intrauterine (IU) infusion of cephapirin approximately one month before first insemination, relative to receiving no treatment (McDougall 2001; LeBlanc *et al.* 2002; Runciman *et al.* 2009). Intrauterine infusion of ceftiofur at approximately 6 weeks postpartum between two injections of PGF two weeks apart reduced the prevalence of uterine bacterial infection with *E. coli* from 10 to 2% and with *A. pyogenes* from 6 to 1% among cows with PVD but did not improve the probability of pregnancy in a ‘Presynch’ timed AI protocol (Galvão *et al.* 2009). In the same study, it is notable that only 41% of cows with PVD had any bacteria cultured from the uterus at the time of diagnosis. These data support the lack of association between cytological endometritis and concurrent uterine bacterial infection.
Numerous older studies reported that one or two injections of prostaglandin F\(_{2\alpha}\) (PGF) improved reproductive performance or produced clinical outcomes similar to IU antibiotics. However, in studies of cows with risk factors for, or with endometritis, PGF consistently did not improve reproductive performance, but many of these studies lacked valid case definitions, statistical power, or both (LeBlanc 2008). In a clinical trial in over 2000 cows - including over 600 with PVD, cytological endometritis, or both - cows were randomly assigned to receive PGF at weeks 5 and 7 postpartum, or not (Dubuc et al. 2011). Overall, or among cows with reproductive tract disease, there was no difference in time to pregnancy between PGF-treated and control cows, which is similar to the findings of Galvão et al. (2009) for cytological endometritis. However, the data from Dubuc et al. (2011) were reanalyzed to examine cows with PVD specifically and without regard to endometritis status (i.e., to address the clinical question of treatment of cows examined only for PVD (which is practical) but without diagnosis of endometritis by cytology (which is well validated, but impractical for routine clinical application). Among 323 cows with PVD at 5 weeks postpartum, clinical resolution (absence of PVD) at 8 weeks postpartum was 72% in cows that received PGF at weeks 5 and 7 and 58% in untreated controls (bivariable \(\chi^2 p = 0.01\)). Among these cows with PVD, 43% had a corpus luteum (CL) (serum progesterone > 1 ng/ml) at week 5, and 63% had a CL at week 7; 69% had a CL at least one of the times of administration of PGF. Accounting for parity, BCS at calving, occurrence of dystocia, RP or twins, and herd, cows with PVD that received 2 injections of PGF tended (\(p = 0.07\)) to become pregnant sooner than untreated cases (hazard ratio = 1.2, 95% confidence interval 0.95 to 1.6). There was no interaction of the effect of PGF with the presence of a CL. Therefore, these results join others (LeBlanc et al. 2002) pointing to an equivocal effect of PGF for treatment of PVD. Different strategies for PGF as therapy for reproductive tract inflammatory disease merit further investigation.

Taken together, it appears that IU cephapirin is beneficial for reproductive performance in cases of PVD (which may be associated with cervicitis or endometritis) but that the benefit of PGF as commonly employed as therapy for PVD is unclear. While there is one study (Kasimanickam et al. 2005) that reported a benefit to reproductive performance of either PGF or IU cephapirin relative to no treatment, further investigation of rapid ‘cow-side’ diagnostic tests and treatment for cytological endometritis are needed. Development of more effective treatments for reproductive tract inflammatory disease will require a better understanding of the factors that initiate and sustain endometrial inflammation, but investigation of antiinflammatory approaches to treatment are of interest.

**INVESTIGATION OF UNDERLYING CAUSES AND TREATMENT OF KETOSIS**

Several practical guidance documents for investigation of health problems in transition dairy cows by veterinary practitioners or other advisors have been put forward (Mulligan et al. 2006; O’Boyle 2008; Cook, Nordlund 2004; Nordlund et al. 2006; Nordlund 2008). The critical principles are to investigate and ensure that all cattle have unrestricted access to feed at the time of fresh feed delivery, to clean water, and to a comfortable resting place. Unfortunately, there is presently little evidence to inform choices of intervention in individual cows in response to elevated NEFA or BHBA. Administration of propylene glycol, insulin, or corticosteroids might be beneficial, but further research is needed on treatment regimes that might be effective at reducing the risk of disease or reduced performance among cows identified at high risk of these problems. Based on currently available data (Nielsen, Ingvartsen 2004; McArt et al. 2011; McArt et al. 2012), drenching with 300–500 ml of propylene glycol once a day for 5 days may be a reasonable treatment for cows with elevated NEFA or BHB.

**REFERENCES**


INTRODUCTION
There is a homeless pet crisis in most countries that is a direct result of animals left unaltered. The ASPCA and HSUS estimate that in the United States alone, 6–8 million animals end up in animal shelters each year and that 3–4 million of these are euthanized. That equates to one animal euthanized every 2 seconds every working day each year. There are no reliable estimates of the number of stray dogs and feral cats that are killed on highways, die of disease, or die of starvation each year. It doesn’t, however, have to be that way.

There are logical approaches to solving the overpopulation problem. These include, but are not limited to:

- Pediatric spay/neuter
- Shelter spay/neuter programs
- Low-cost, high-volume spay-neuter clinics/programs
- The spay-by-5 campaign
- Nonsurgical sterilization
- Transport programs
- Education about overpopulation and the importance of spay/neuter

Each of these approaches will be addressed, briefly, in this presentation.

PEDIATRIC SPAY/NEUTER
For years veterinary schools have taught that the most appropriate age to spay or neuter a dog or cat is six months. But why? What is the scientific evidence that confirms that 6 months is the best age? What is the scientific evidence that dictates that we should not spay or neuter at an earlier age? The reality is that the 6 months recommendation is based on clinical sentiments, but not on scientific studies. And the clinical sentiments have changed over the decades. Several decades ago the recommendation was that dogs and cats should have one litter first. Then that recommendation changed to let them have one heat cycle. And now the recommendation is 6 months. None of these has strong scientific support.

Historically veterinarians have expressed two major concerns regarding pediatric spay/neuter. One concern is anesthetic risk. The second concern relates to suspected adverse physiological effects of spay or neuter prior to six months of age. But anesthetic agents used today are safe in puppies and kittens as young as 6 to 8 weeks of age, and numerous research projects over the past twenty years have dispelled most of the concerns related to adverse physiologic effects.

Pediatric spay/neuter is safe, the surgical procedures are easier and quicker, and there are virtually no long-term adverse physiological effects associated with pediatric spay and neuter. Accepting and teaching that pediatric spay/neuter is appropriate is an important element in the reduction of the overpopulation of dogs and cats and is an essential feature of the next approach: shelter spay/neuter programs.

SHELTER SPAY/NEUTER PROGRAMS
The Association of Shelter Veterinarians’ Guidelines for Standards of Care in Animal Shelters states “animal shelters should require that cats and dogs who are adopted into homes be spayed or neutered.” Voucher programs and spay-neuter agreements/contracts just don’t consistently work. Some studies have shown the compliance rate of spay/neuter programs after adoption to be as low as 40%. To ensure that adopted animals are sterilized, the sterilization surgery should be performed prior to the animal leaving the shelter. Given the numbers of and ages of puppies and kittens adopted from shelters, this means that
spay or neuter in the shelter environment should be performed as young as 6 to 8 weeks of age; whatever age the animal is put out on the adoption floor.

The Mississippi State University College of Veterinary Medicine Shelter program currently goes to 20 different animal shelters in north Mississippi, and since January 2007 has performed over 45,000 sterilization surgeries. Our program services shelters in the northern 40% of the state. Prior to starting to work with a shelter, we hold discussions with local veterinarians to make sure they are supportive. We now have 2 mobile units, both of which are on the road 3 or 4 days a week and average 30 surgeries a day (each), with students performing the majority of the surgeries. Every third-year veterinary student in our curriculum will make two trips to shelters with us and average 7 or 8 surgeries per trip. Either a faculty member or resident is always scrubbed in with the junior students. Our senior veterinary students can take a shelter medicine spay/neuter elective clinical rotation. The rotation runs year-round, and we can take 5 senior students at a time for two weeks each. On the first day of the senior rotation, a faculty member or a resident scrubs in with the senior students. After that we are available to scrub in, but only do so if the student needs our assistance. Senior students average nearly 80 surgeries each during this elective. The shelters pick the animals they wish for us to sterilize, and we find that just over 50% of our patients are pediatric.

Our program is funded completely by grants and donations, so we are able to offer our services to shelters at no cost. It is truly a win-win-win situation. Win for the shelters, as they get animals spayed and neutered at no cost. Win for the animals, as the shelters we go to collectively have over a 70% euthanasia rate, but over an 80% adoption rate of the animals we sterilize. Win for the students, as they get extensive spay/neuter experience and learn about the realities of the overpopulation problem.

Unfortunately, not all shelters have access to a program like ours. Many shelters have difficulty finding veterinarians who will perform spays and neuters at a price the shelters can afford. And even more shelters have difficulty finding veterinarians who will perform pediatric spays and neuters. This has led to the trend of establishing low-cost, high-volume spay/neuter clinics.

Low-cost, high-volume spay/neuter clinics are being established all over the United States. Many of these clinics are modeled after the very successful program in Asheville, NC, Humane Alliance. In fact, Humane Alliance has established the National Spay/Neuter Response Team, which assists programs attempting to establish low-cost, high-volume spay/neuter clinics. To date, they have assisted over 125 such clinics across the United States. These programs provide a very needed service, but many practicing veterinarians express concerns about such clinics. Generally these concerns fall into two categories: concern about quality of care and concern about losing clients.

Veterinarians express sentiments such as “if they can do it for such a low cost, they must be cutting corners.” But such statements ignore a couple of critical points. Most veterinary practices are not designed for efficient spay/neuter - because they do everything else as well. Most veterinary practices are equipped - and their staffs are trained - to do hundreds, perhaps thousands, of different things. But low-cost, high-volume clinics are specifically focused on just a few things: spay and castration. Protocols are geared for efficient spay/neuter. Facility design is geared directly toward efficient spay/neuter. Equipment is focused specifically toward spay/neuter. Such clinics achieve high volume not by concentrating on speed of surgeries but on efficiency in every aspect of the program; not by taking shortcuts, but by being efficient. Part of good surgical technique is eliminating inefficiency. Efficiency in surgery means the elimination of all unnecessary patient manipulation, unnecessary tissue handling, and any wasted motion. While improvements in technique that increase efficiency will also reduce surgical times, the goal is not to be “fast” per se - rather the goal is an efficient, minimally traumatic procedure that will produce the best patient outcomes. This efficiency allows the clinics to handle more patients - thus “high volume” and lower costs.

In July 2008, the JAVMA published “The Association of Shelter veterinarians veterinary medical care guidelines for spay-neuter programs.” These guidelines prescribe presurgical, anesthetic, surgical, and post-surgical protocols and standards and are designed to maintain quality of patient care and
surgical standards. Most low-cost, high-volume spay/neuter clinics meet or exceed the standards outlined in that document.

For the most part, fear of losing clients to low-cost, high-volume clinics is unfounded. While many practitioners fear the “competition,” the reality is that these clinics service a different clientele than that served by private veterinary clinics. In many respects, these clinics may benefit the private practitioner by: taking pressure to perform reduced-rate surgeries off the private practice, by providing a referral outlet for low-income pet owners, and by increasing adoption from local shelters and, thereby, potentially increasing the number of potential clients for the private practitioners.

The advances in veterinary medical care over the past several decades have been amazing. We can now practice human-level medical care on dogs and cats. But these advances have come at a price. Increasing costs of veterinary equipment, increasing cost of veterinary facilities, increasing cost of veterinary education have resulted in significantly increased cost of veterinary medical care, leaving many individuals unable to afford even basic veterinary care for their pets. Low-cost, high-volume clinics attract such individuals. Put simply, pet owners can be placed into three different groups: those who provide full veterinary care for their pets, those that shop around searching for the least expensive care, and those who won’t or can’t afford to provide veterinary care. Private practitioners make their living, pay their bills, primarily from the first group; they only see the second group if they offer discounts or specials, and they rarely ever see the third group. It is the second and third groups that are targeted by low-cost, high-volume clinics.

Humane Alliance is one example of a successful low-cost, high-volume spay/neuter clinic. Humane Alliance started in 1994 as a small high-volume spay/neuter clinic in Asheville, NC. They now have a large clinic, a national training center, and are home of the National Spay/Neuter Response Team that assists others to start spay/neuter clinics. But they still serve the needs of Ashville and surrounding areas. And since 1994, with a rapidly growing human population, intake and euthanasia at the local shelters are down by 75%. Veterinarians in the area are supportive of and refer clients to Humane Alliance. And Humane Alliance has an exceptional safety record with a mortality rate of 0.05%. A mortality rate of 0.05% is 1 animal out of every 2000. This low mortality rate is a testament to the quality of care and the level of surgery performed in clinics such as this.

So high-volume clinics are a critical component in reducing overpopulation of unwanted pets. But not everyone wants to devote their career to spay/neuter. Not everyone wants to work in a high-volume clinic. So how can the private practitioner assist in stopping the overpopulation?

**Spay-By-5 Campaign**

Spay-By-5 is a national campaign to reduce the routine age of spay and neuter of dogs and cats to 4½ to 5 months. If a practice currently recommends spay/neuter at 6 months, this campaign is simply requesting that the surgeries be done 4 to 6 weeks earlier. The reality is that cats and small-breed dogs can go into heat and become pregnant prior to 6 months of age, but generally not prior to 5 months of age. Most veterinarians have routine puppy and kitten health maintenance programs that involve veterinary visits, vaccinations and other routine care every 2 to 3 weeks starting at 6 weeks of age. Spay-By-5 promotes adding one more appointment to those programs, resulting in most pets being spayed or neutered between 4½ and 5 months of age. This would prevent those “oops” litters, and veterinarians would discover that the surgeries are easier and faster, recoveries are quicker, they have fewer complications, and their clients’ pets are spayed before the females ever become pregnant and before the males develop sexual behaviors.

But surgery isn’t the only answer.

**Nonsurgical Sterilization**

Nonsurgical sterilization, in time, could play a significant role in reducing pet overpopulation. Surgical sterilization requires special expertise, special equipment, special facilities. Nonsurgical sterilization could be easier, faster, less expensive, and, therefore, be assessable to more of the population.
The mission of the ACCD (The Alliance for Contraception in Cats and Dogs) is to expedite the successful introduction of methods to nonsurgically sterilize dogs and cats and to support the distribution and promotion of these products to humanely control cat and dog populations worldwide.

The Found Animal Foundation has introduced the Michelson Prize. This is an effort to produce a single-dose, safe and effective, permanent sterilant for dogs and cats. They are devoting up to 25 million dollars to support research into the development of such a sterilant, 25 million to bring such a product to market, and 25 million as a prize to the individual, institution or organization that develops such a sterilant. When such a product is produced, if it is at a reasonable cost, it will fundamentally change the approach to pet overpopulation. Until then, the primary tools will continue to be surgery, plus transport programs and education.

TRANSPORT PROGRAMS
Not all areas have serious overpopulation problems. In some areas, the problem animal shelters face is they have more people wanting to adopt pets than they have pets available. Transport programs move adoptable dogs from areas of extreme overpopulation to areas where there is a shortage. A very successful transport program is the Rescue Waggin’ funded by PetSmart Charities. The Rescue Waggin’ defines source shelters and destination shelters and routinely transports healthy, adoptable animals from source to destination shelters. This program is fully funded by PetSmart Charities and since its inception in 2004 has saved the lives of over 50,000 dogs and cats.

Another example is Homeward Bound. Organized and run by veterinary students at Mississippi State University College of Veterinary Medicine, Homeward Bound transports approximately 50 adoptable pets (mostly puppies) from overcrowded shelters in Mississippi to destination shelters in the northeast. Since its inception in May 2007, Homeward Bound has transported over 3500 adoptable animals. Every transported animal is fully vaccinated, dewormed, treated for external parasites, tested and (if necessary) treated for heartworm disease, spayed or neutered, and placed in a foster home for isolation and socialization prior to transport.

But spay, neuter, and transport alone won’t fully solve the problem. Educating the public on the severity of the overpopulation problem and the benefits and need for spay/neuter is an essential component.

EDUCATION PROGRAMS
All of the spay/neuter programs combined won’t work if people don’t know to spay or neuter their pets or if people are opposed to sterilization of pets. It is essential that people understand the overpopulation problem, essential that people understand the numbers of dogs and cats that are euthanized in shelters each year due to homelessness, and essential that people recognize the importance of spay/neuter and adoption. Solving the overpopulation problem requires more than spay/neuter and transport. It requires changing peoples’ attitudes. Increasing peoples’ understanding of responsible pet care, including spay/neuter.

The PAWS program (Pet Awareness of Students) is one example of a program directed toward changing peoples’ attitudes on pet care. Veterinary students go into schools to discuss pet care and responsible pet ownership, addressing such issues as appropriate care of pets, pet overpopulation, importance of spay/neuter and adoption as an alternative to purchase of pets.

CONCLUSIONS
Overpopulation of unwanted dogs and cats is a serious problem. At present, the primary approach to address overpopulation is spay/neuter, including pediatric spay/neuter; shelter spay/neuter programs; low-cost, high-volume spay/neuter clinics; the Spay-By-5 campaign; transport programs; and education. Nonsurgical sterilization promises to be a major factor in the future. If all of these approaches are used, if all of these approaches are accepted, if practitioners get involved by spaying and neutering before 5 months and by not opposing high-volume clinics, shelter spay/neuter programs, and pediatric spay/neuter, we can end the problem of overpopulation of unwanted dogs and cats. We can significantly reduce shelter intake and end euthanasia of adoptable pets.
**Efficient Dog and Cat Spay/Neuter Techniques**
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**INTRODUCTION**
Most veterinary schools teach students how to perform spays and neuters at a point in their education when they are very inexperienced surgeons. Therefore, students are taught many techniques that are simply designed to compensate for poor surgical skills. Students are taught to double ligate everything because we don’t trust the students’ ligatures. They are taught interrupted patterns because we don’t trust their knots. They are taught long incisions and extensive exposure because we believe students don’t fully understand abdominal anatomy. As veterinarians gain experience in surgery, they become much more efficient, but often veterinarians fail to abandon those techniques that were simply designed to compensate for lack of experience. Many of those techniques can be replaced by ones that are much more efficient.

**PATIENT POSITIONING**
Where does the surgeon stand while performing a spay? What factors influence where you stand during a spay? Do you stand with the patient’s head to your right or to your left? Most right-handed veterinarians stand with the patient’s head to their left, and most left-handed veterinarians stand with the patient’s head to their right. But why is this? Try standing with the patient’s head to the side of your dominant hand. There is a very valid reason for this. If you strum the suspensory ligament of the ovary, this allows you to strum it with your stronger hand. If you cut the suspensory ligament, it allows you to cut the ligament easily with your dominant hand. While I am not necessarily recommending that you change sides of the table if you have been doing surgery for years, I am recommending that you always ask why you are doing a particular technique a particular way and consider if there is a better, more efficient approach.

In a spay, position the patient with the front legs along its side rather than pulled forward past its head. Pulling the legs forward, which is commonly done, tightens the muscles of the back and tightens the suspensory ligaments of the ovaries. Pulling the limbs alongside the patient’s thorax will relax the suspensory ligaments and make delivery of the ovaries through an abdominal incision easier. A simple restraint device allows this positioning of the patient and helps prevent tilting of the patient to one side or the other.

**SURGICAL TECHNIQUES**
**Placement of Incisions**
One key to efficient ovariohysterectomies is making appropriately placed small incisions. While most surgery instructors promote long incisions and maximum exposure, lengthy incisions are considerably more time consuming to close. Small incisions, obviously, can be closed much more rapidly than long incisions. The proper location of the incision varies with species and with age of the patient. In a cat spay, the tissue that is more difficult to exteriorize is the uterine body. In the adult dog, it is more difficult to exteriorize the ovaries. Puppies are intermediate. Vary the location of your incisions accordingly. In the cat spay, the skin incision should be located on the ventral abdominal midline at the midpoint between the umbilicus and the anterior brim of the pubis. In the adult dog, the skin incision is on the ventral abdominal midline just caudal to the umbilicus. In the puppy spay (6 months or younger), the skin incision is on the ventral abdominal midline a little cranial to the location of the cat spay incision and a little caudal to the location of the incision in an adult dog.

In the adult dog, the right kidney and the right ovary are located further cranial in the abdomen than the left kidney and left ovary. It is, therefore, more difficult to exteriorize the right ovary than the left ovary. To equalize the difficulty of exteriorizing the two ovaries, make the entry into the abdomen through a right paramedian incision. Incise the skin on the ventral abdominal midline, undermine only...
on the right side of the linea alba, and, depending on the size of the dog, incise the rectus sheath 1/2 to 2

cm to the right of the linea alba. To prevent hemorrhage, incise only the fascia. Enter the abdomen by

bluntly separating the fibers of the rectus abdominis muscle and cutting the peritoneum.

Castration incisions in the cat, the puppy, and in the adult dog can be made through the scrotum.

**Ligation Techniques**

Most of you were probably taught to double ligate ovarian pedicles and uterine stumps and to ligate

before transecting the tissue - but why? As stated above, you were, most likely, taught how to perform

spays when you were very inexperienced at surgery. Accordingly, at that stage of development, it was

not wise to trust your tissue handling and your ligations. Both of these techniques, however, can slow

you down considerably. It is much more efficient to transect the ovarian pedicles prior to ligation and to

single ligate each pedicle. The most efficient technique is to place 3 hemostats - the first most proximal,

the second several millimeters distal to the first (but still proximal to the ovary), and the third between

the ovary and the uterine horn. Close the first hemostat one click, the second two clicks, and the third

three clicks. The purpose of the 1, 2, 3 clicks is to avoid completely crushing the tissue at the most

proximal clamp. Complete crushing would predispose the pedicle to tearing. Before ligating, transect the

ovarian pedicle just distal to the second hemostat. Ligate with a square, surgeon’s, or Miller’s knot. If you

are skilled at hand ties, that, too, will improve your efficiency.

**Hand Ties**

Becoming skilled at hand ties - square knot, surgeon’s knot, and Miller’s knots - will improve efficiency in

both dog and cat spays. To be efficient, this skill must be practiced. But once you are skilled at hand ties,

it increases your speed significantly.

**Pedicle Ties**

The pedicle tie is a method of ligation in which the structure is tied to itself around a hemostat. The

pedicle tie can be used in cat castrations, puppy castrations, and in ligating the ovarian pedicles in cat

spays. There are several variations of the pedicle tie in the cat spay. In the technique I use, deliver the

ovary through the abdominal incision, cut the suspensory ligament, and tear a hole in the broad ligament

just caudal to the ovarian vessels. Hold the ovary in your non-dominant hand and gently pull the ovary

toward you. Using the dominant hand, a curved hemostat is crossed over the ovarian vessels into the

hole in the broad ligament and underneath and behind the vessels. The hemostat should be held closed

with the tip of the hemostat facing away from you. The tip of the hemostat is then directed above the

vessels as the hemostat is rotated counterclockwise to end up facing you. The hemostat is opened and

used to clamp the ovarian vessels. The vessels are cut or torn between the hemostat and the ovary and the

knot is gently pushed off the tip of the hemostat. The knot should be pulled tight before releasing the

hemostat.

**Miller’s Knot**

The Miller’s knot is a very secure, self-locking knot that can be placed either with an instrument or with a

hand tie. The Miller’s knot can be used on spermatic cords, on ovarian pedicles in dogs and uterine

bodies of dogs and cats. To place a Miller’s knot, pass the suture under the tissue to be ligated, bring the

suture back over the tissue and under the tissue one more time. This creates a small loop of suture above

the tissue to be ligated. Position the needle holder through that small loop, wrap the long strand once

around the needle holder, grasp the short strand of suture with the needle holder, and pull the needle

holder toward you while pulling the long strand of suture away from you. Gentle upward tension while

pulling this knot tight facilitates placement of the ligature. Complete the knot by placing three or four

more square knot throws.

**Scrotal Castrations in Adult Dogs**

Scrotal castrations are rarely ever taught in veterinary school; in fact, for decades veterinary students

have been taught to avoid making incisions in the scrotum of dogs. Scrotal castrations appear, however,

to offer several advantages over the prescrotal approach, including smaller incisions, less surgical time,

and less tendency for scrotal hematomas. The justification for avoiding scrotal castrations in dogs had
been to prevent self-mutilation. As long as no external skin sutures are placed in the scrotum, there
appears, however, to be no greater risk of self-trauma in a scrotal castration than in a prescrotal
castration.

Position the patient in dorsal recumbency. Grasp one testicle and position it in a manner that
elevates and exposes the median raphe. Make an incision through the skin and subcutaneous tissue along
or near the median raphe over the displaced testicle. Continue the incision through the spermatic fascia to
exteriorize the testicle. In the closed castration technique, care is taken not to incise the parietal vaginal
tunic and tunica albuginea. Use gentle traction to exteriorize the testicle and reflect fat and fascia from the
parietal tunic of the spermatic cord using a gauze sponge. Place three hemostats on the spermatic cord
and transect the cord distal to the third hemostat. In smaller dogs (under 18 kg), a single ligature tied
with a Miller’s knot and placed in the crushed area of the most proximal hemostat is sufficient for
hemostasis. In larger dogs (18 kg and above), a transfixation ligature is placed in addition to and just
distal to the Miller’s knot. The second testicle is exteriorized through the same scrotal incision. A second
incision in spermatic fascia is made over the second testicle to allow exteriorization, transection, and
ligation of the second spermatic cord and is accomplished in a manner identical to the first testicle.

The technique for closure is the surgeon’s preference. Incisions can be left open to heal by second
intention, can be partially closed with one buried subcutaneous suture of absorbable suture material, or
can be closed fully with skin glue. All three of these techniques are considered acceptable.

**Age at Which Surgery is Performed**

As a general rule, the larger the animal is (dog or cat), the more obese the animal is, and the older the
animal is, the longer it will take to perform a spay or neuter surgery. Even though most of us were taught
to wait until a dog or cat is sexually mature (six to nine months) before sterilization surgery, there is
growing evidence that there is no reason to wait until the animal is an adult. Pediatric spay/neuter has
been shown to have little or no adverse physiologic effects on the animal, and spay/neuter in the
pediatric patient is much easier and quicker than that in the sexually mature patient.

**Conclusions**

Becoming efficient at spays and neuters is a combination of many factors. One of which, of course, is the
skill and comfort level of the surgeon. Adoption of specific techniques that are used commonly in high-
volume spay/neuter clinics is a key factor in improving efficiency. Being willing to question why you
were taught specific manipulations in veterinary school and recognizing that it is acceptable to abandon
some of them (such as always double ligating pedicles) will improve surgical efficiency greatly.
**Unusual Spay/Neuter Surgeries**
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**INTRODUCTION**
Not all spays and neuters are “routine.” Conditions such as cryptorchidism, hermaphroditism, uterus unicornis, mammary hyperplasia and lactation may present surgical challenges, but approaches to each of these nontypical cases are actually quite simple.

**CRYPTORCHIDISM**
Cryptorchidism is defined as the failure of one or both testicles to descend into the scrotum. The cryptorchid testicle can be located anywhere along the path from the area of fetal development of the gonads (just caudal to the caudal pole of the kidney) to the subcutaneous tissue between the external inguinal ring and the scrotum. Thus a cryptorchid testicle can be located in the abdominal cavity, in the inguinal canal, or in the subcutaneous tissue between the external inguinal ring and the scrotum.

Testicles should be easily palpated in the scrotum of dogs and cats greater than 2–4 months of age. If one or both testicles are not located in the scrotum, careful palpation will reveal which testicle(s) are involved and whether the testicle(s) are located in the subcutaneous tissue. Failure to palpate a testicle in the scrotum or the subcutaneous tissue leads to a presumptive diagnosis of abdominal cryptorchidism. Palpation of the testicle in the subcutaneous tissue leads to a diagnosis of subcutaneous cryptorchidism.

**Subcutaneous Cryptorchidism**
If the cryptorchid testicle is palpated in the subcutaneous tissue, incising directly over the testicle will allow exposure and removal of the testicle.

**Abdominal Cryptorchidism**
Locating an abdominal testicle is generally very easy. The critical factor to remember is that both ductus deferens enter the urethra at the prostate. If you trace the ductus deferens from the prostatic urethra cranially, it is located dorsal to the bladder until it passes the junction of the ureter and the bladder. Cranial to the point where the respective ureter enters the bladder, the ductus deferens turns laterally on its course to the inguinal canal and the testicle. This anatomical feature makes it extremely easy to find an abdominal testicle.

In the dog, the skin incision is made in the caudal abdominal skin just lateral to the prepuce on the side of the cryptorchid testicle. Entry into the abdomen is either on the midline through the linea alba by undermining the prepuce to the midline or by a paramedian incision incising the external rectus fascia and separating rectus abdominus muscle fibers. I first make a very small incision and pass a spay hook from medial to lateral, lateral to the bladder wall. Often that will catch the ductus deferens, allowing exteriorization of the testicle. If that fails, I extend the incision, exposing the urinary bladder. Caudal reflection of the urinary bladder exposes the dorsal surface of the bladder, allowing visualization of both ductus deferens. Gentle retraction of the ductus of the cryptorchid testicle will allow delivery of the testicle into the surgical site, ligation of the testicular vessels, and excision of the testicle.

In the cat the skin incision is made in the caudal abdominal skin on the midline. Entry into the abdomen is on the midline through the linea alba and allows exposure of the urinary bladder. Again, using a spay hook and sweeping laterally from the bladder wall will often catch the ductus deferens. If this fails, caudal reflection of the urinary bladder, exposing the dorsal surface of the bladder, will allow visualization of both ductus deferens. Gentle retraction of the ductus of the cryptorchid testicle will allow delivery of the testicle into the surgical site, ligation of the testicular vessels, and excision of the testicle.

On occasion, cryptorchid testicles are trapped between the muscle layers in the inguinal canal. When this occurs, gentle tension on the ductus deferens will allow visualization of the ductus deferens entering the inguinal canal. Gently teasing the musculature of the internal inguinal ring apart with a blunt instrument is often enough to allow delivery of the testicle back into the abdomen for removal.
Frequently cryptorchid testicles are smaller than normal, and it is possible that the cryptorchid testicle will be in the subcutaneous tissue but not be palpable. Entry into the abdomen, assuming abdominal cryptorchidism, would, therefore, fail to reveal the cryptorchid testicle. Gentle tension on the ductus deferens would confirm that the ductus deferens passes through the inguinal canal. The caudal abdominal skin incision is of value here, as from that incision you can undermine the skin between the incision and the external inguinal ring. Gentle traction on the abdominal ductus will allow you to locate the ductus deferens as it exits the inguinal canal and will lead you to the cryptorchid testicle.

Once the cryptorchid testicle is located, either in the abdomen or the subcutaneous tissue, it can be excised using any standard technique. For very small testicles with small vessels and a small ductus deferens, I will use the cord tie or figure-eight knot in the spermatic cord. For larger testicles with larger spermatic cords, I will clamp the spermatic cord with hemostats, transect distal to the most distal hemostat, and place a ligature using a Miller’s knot in the area of the spermatic cord crushed by the most proximal hemostat. In dogs weighing over 18 kg, I will clamp the spermatic cord with three hemostats, transect distal to the most distal hemostat, place a ligature using a Miller’s knot in the area of the spermatic cord crushed by the most proximal hemostat, and place a transfixation ligature in the area of the spermatic cord crushed by the second hemostat.

**Uterus Unicornis**

Uterus unicornis is congenital absence of one horn of the uterus, but both ovaries are always present. So when performing a spay and discovering that one uterine horn is absent, you must search for the 2nd ovary. It will be in the normal location and, if a broad ligament is present, is rather easy to find. If no broad ligament is present on the involved side, use the biological retractors to help localize the ovary.

**Mammary Hyperplasia/Lactation**

Cats with mammary hyperplasia or lactating queens still nursing kittens are ideal candidates for flank spays. Performing a flank spay will avoid any damage to mammary tissue, preventing abscesses due to leakage of milk into the tissues. A flank spay should be performed with the patient in left-lateral recumbency. An incision is made paralleling the last rib 2/3 the way back from the last rib and cranial to the wing of ilium and just ventral to the transverse spinous processes. Dissect through the subcutaneous tissue, separate fibers of the external abdominal oblique muscle and the internal abdominal oblique muscle entering the abdomen. If the incision is positioned properly, the right uterine horn and right ovary will be clearly visible. If not visible they can be retrieved using a spay hook. The spay is then performed the same as with a ventral midline approach. A three-layer closure is performed suturing internal abdominal oblique, external abdominal oblique and subcuticular tissue.

**Hermaphroditism**

Hermaphroditism is the presence of both ovarian and testicular tissue in the same gonad or the same individual. Most frequently hermaphrodites are presented as a female for ovariohysterectomy. The patient often has female genitalia with an enlarged clitoris. The “ovariohysterectomy” is performed routinely.

**Conclusions**

While conditions such as cryptorchidism, hermaphroditism, uterus unicornis, mammary hyperplasia and lactation may present as challenges to the veterinary surgeon, understanding the conditions, the anatomy involved and the surgical techniques that can be used will make spay/neuter in these nontypical conditions relatively easy.
Feline Retroviral Management for Animal Shelters
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Feline leukemia virus (FeLV) and feline immunodeficiency virus (FIV) are retroviruses that represent two of the most important infectious diseases of cats worldwide. Recent data indicate the seroprevalence of FeLV is 3.4% in Canada and 3.1% in the United States, while the seroprevalence of FIV is 4.3% in Canada and 3.6% in the United States, with marked regional variation in both countries.\(^1,2\) Prevalence of both viruses is much higher in sick cats than in asymptomatic cats. Guidelines for retrovirus testing and management have been published in Canada and the United States.\(^3,4\) The American Association of Feline Practitioners (AAFP) document contains a section on considerations for cat shelters.\(^3\) The Association of Shelter Veterinarians (www.sheltervet.org) has published position statements on retrovirus testing in shelters.

Virus Transmission and Risk Factors
Viremic FeLV-infected cats shed virus in body fluids, including saliva, feces, milk, and urine. FeLV transmission occurs through sustained close contact among cats. Behaviors such as mutual grooming, sharing of food and water bowls and litter boxes, and fighting can contribute to transmission, primarily via saliva. Kittens less than 16 weeks of age are most likely to develop progressive infection after exposure. However, adult cats may be susceptible to FeLV infection after long-term exposure. Vertical transmission of FeLV can occur. Infected pregnant queens may suffer reproductive loss; kittens that survive to term are generally born viremic and fade quickly. Up to 20% of vertically infected kittens may survive to develop progressive infection as adults. Transmission to kittens may also occur via the milk from an infected queen or via saliva when the queen cleans the kittens.

Unlike FeLV, kittens do not appear to be more susceptible to infection with FIV than adults. FIV is present in saliva of infected cats, and infection is most likely to occur in male cats and free-roaming cats, reflecting efficient transmission by bite wounds. Transmission via sustained contact among infected and uninfected cats, as with FeLV, may also occur. In addition, in utero and lactogenic transmission to kittens from queens may occur, especially if the queen is experiencing high levels of viremia.

Both FeLV and FIV are very labile outside the host and remain infectious for only minutes in the environment; in moist secretions, virus may survive until dried. The viruses are readily inactivated by soap, disinfectants, heat, and drying. FeLV and FIV are not zoonotic.

Several studies have found similar risk factors for infection with FeLV and FIV. Seropositivity is more likely in adult cats than in those less than 6 months of age, in cats with current illness than in healthy cats, and in cats with access to outdoors than in indoor cats. Seroprevalence for FIV is highest in intact males, and seroprevalence for FeLV is highest in intact females and males.

Outcome of Infection
Recent research suggests that many cats may remain infected with FeLV for life following exposure, but may revert to a regressive state with a low risk of clinical disease. Following exposure, cats may exhibit mild clinical signs, such as fever and malaise, or may remain asymptomatic. For cats that remain persistently infected, this acute phase is followed by a period of asymptomatic infection that may last months or years. Ultimately, progressive infection occurs with development of one of several FeLV-associated disorders (e.g., lymphoma, anemia) or a secondary disease associated with immune dysfunction (e.g., opportunistic infections, oral inflammatory disease).

After infection with FIV, cats enter an asymptomatic phase that may last for many years. Virus replication continues, but at very low levels. Over time, the level of both CD4+ and CD8+ lymphocytes may gradually decline, ultimately leading to immunodeficiency. The clinical signs and illnesses associated with FIV are varied and nonspecific and are usually not a direct effect of the virus, but due to secondary infections that may be treatable. Other common associated diseases include inflammatory ocular and oral disease, neoplasia, neurologic disease, and possibly renal disease.
**Diagnostic Testing**

Serological diagnosis of FeLV relies on detection of the core antigen p27 in peripheral blood using ELISA, either as a patient-side kit or at a referral laboratory. Patient-side kits may be used with anticoagulated whole blood, serum, or plasma, although the test kit should be checked for the manufacturer’s recommendations on sample type. Tests performed on tears or saliva are less reliable and are not recommended. Most cats will test positive with ELISA within 30 days of exposure. Confirmatory testing for cats with positive test results is strongly recommended, especially for low-risk cats. A second soluble antigen test can be performed, preferably using a test from a different manufacturer. Immunofluorescent antibody (IFA) tests available from referral laboratories detect p27 antigen within infected leukocytes or platelets, and are recommended as confirmatory tests. IFA tests do not detect infection until secondary viremia has occurred with infection of bone marrow (6 to 8 weeks after initial infection). PCR is offered by a number of commercial laboratories for the diagnosis of FeLV. PCR detects viral DNA sequences and can be performed on blood, bone marrow, saliva, and tissues. Blood PCR tests for FeLV are usually positive within 1–2 weeks of FeLV exposure. However, information regarding sensitivity and specificity of specific PCR assays offered by commercial laboratories is frequently unavailable.

Kittens can be tested for FeLV at any age, as passively acquired maternal antibody does not interfere with testing for viral antigen. Newborn kittens infected from an FeLV-positive queen may not test positive for weeks to months after birth. Testing for FeLV infection is not generally compromised by vaccination.

FIV infection and antibodies to the virus persist for life. Hence, the most common method for diagnosis of FIV infection is screening for antibodies (typically against p24 and p15) using an ELISA, either with a patient-side kit or at a referral laboratory. Most cats will produce antibodies to FIV within 60 days of exposure, but the time to seroconversion can be longer. As for FeLV, confirmatory testing for cats with positive FIV test results is strongly recommended, especially for low-risk cats. Western blot and immunofluorescent antibody assays detect antibodies against an increased number of viral antigens, and are suggested as confirmatory tests in seropositive cats with no history of FIV vaccination.

The release of the first vaccine against FIV has complicated the ability to diagnose FIV infections. Vaccinated cats produce antibodies that cannot be distinguished from antibodies due to natural infection using most available tests. Antibodies due to vaccination persist for more than one year, and possibly for more than 4 years. In addition, kittens born to naturally infected queens, or queens vaccinated against FIV, may acquire FIV antibodies in colostrum. In one study, FIV antibodies persisted past 8 weeks of age in more than 50% of kittens born to FIV-vaccinated queens, but were no longer detectable at 12 weeks of age.\(^5\)

Since it is uncommon for kittens to acquire FIV infection, most kittens that initially test positive are not truly infected and will test negative when reevaluated, especially at or over 6 months of age. Kittens over 4–6 months of age with FIV antibodies are more likely to be infected. Due to concerns regarding detection of passively acquired FIV antibodies, it is tempting to delay testing kittens for FIV until after 6 months of age. Since they are a low-risk group, most kittens test negative and can then be reliably considered clear of infection. However, infected kittens could be a source of infection for other cats if they are not identified and isolated. Hence, kittens should be tested for FIV at the first opportunity.

In some cats, it may be difficult to determine if a positive FIV antibody test means the cat is truly infected with FIV, is vaccinated against FIV but not infected, or is vaccinated against FIV and also infected. PCR has been promoted as a method to determine a cat’s true infection status. However, PCR tests offered by some commercial laboratories may be unreliable, with misidentification of both FIV-infected and uninfected cats.\(^6,7\) PCR testing should be reserved for FIV antibody-positive cats that have an unknown vaccination history or that have been vaccinated against FIV but where infection is still suspected. PCR test results must be interpreted with caution. A positive FIV PCR result from a laboratory with stringent quality control should confirm FIV infection and should not be affected by FIV vaccination. However, a negative FIV PCR result does not rule out infection, but may reflect a level of viral nucleic acid below the limit of detection, or a strain of FIV that is not detected by the test.

While it may be tempting to test only a queen and not her kittens for FeLV and FIV in an attempt to conserve resources in shelter or rescue settings, it is inappropriate to test one cat as a representative for
others, as it may lead to erroneous assumptions about infection status. Even young kittens may be exposed to cats other than their mother; for example, stray and feral queens often share mothering of kittens. If a queen or any one of her litter of kittens tests positive, all should be considered potentially infected and isolated, with follow-up testing to resolve status. If a queen or one kitten in a litter tests negative, it cannot be guaranteed that the others are also negative. Shelters sometimes test pooled blood samples from litters of kittens in order to save money; the reliability of this method is unknown and cannot be recommended.

**CONSIDERATIONS FOR ANIMAL SHELTERS**

Testing strategies in shelters will depend on variables such as resources, staffing, housing, nature of the cat population, and program goals. Diagnosis of FeLV and FIV infection in the shelter setting follows the same principles as for pet cats. While screening tests are often performed in shelters, confirmatory testing poses challenges due to increased costs and delays. All cats entering a shelter should be considered potentially infected, regardless of their background. In shelters that rarely euthanize adoptable cats and have available resources, all cats and kittens may be tested upon admission. Cats returned to a shelter because of a failed adoption should also be tested. In shelters with limited resources, priority may be given to testing higher-risk cats or testing cats only at the time of adoption. Priority may also be given to testing cats before an investment in care, such as fostering, spay/neuter, treatment for upper respiratory tract disease, etc. Also, in limited resource situations, kittens under 4–6 months of age may be tested only for FeLV. In all cases where cats have tested negative in the shelter but only a single test has been performed, the test result must be viewed as representing a snapshot in time. Adopting owners should be clearly informed about the need to keep the cat isolated until confirmatory testing has been performed 60 days after the initial test. If one cat in a litter or a group is later found to be infected, the new owners of all in-contact cats should be informed. If testing for FeLV and/or FIV is not performed at the shelter, the new owners should also be informed about the need for isolation and testing and should be encouraged to consult their own veterinarian as soon as possible.

All cats not tested at admission should be housed singly. Before entering group housing, whether in a shelter or a foster home, cats should have negative test results for both FeLV and FIV. Whenever possible, seronegative cats housed long term should be retested in 60 days before introduction to the group. Resident cats in foster homes should be tested before the introduction of foster cats. In shelters or sanctuaries where cats are housed long term, annual retesting of seronegative cats is recommended.

Shelters should have clear policies on management of cats found to be retrovirus positive on a screening test. In many shelters, holding cats for confirmatory testing presents serious risks to the shelter population and the individual cat and strains shelter resources so that all cats testing positive are euthanized. In shelters with some resources, FeLV confirmatory testing of only the most adoptable cats may be chosen and other seropositive cats are euthanized. Cats that test FIV positive may have true infection, or the test result could be due to vaccination. Many shelters routinely euthanize sick cats that test FIV positive, and some euthanize all adult cats testing FIV positive regardless of health due to the difficulty of determining their true infection status. Kittens that test FIV positive present a special challenge, as it may not be feasible to hold and retest even though most of these kittens are not truly infected. Holding kittens for weeks in a shelter may not be in their best interests, both socially and medically. One option is to utilize foster homes, but the kitten must be isolated from any other cats in the home until retested. In shelters with high kitten euthanasia rates, it may be reasonable to euthanize FIV-positive kittens rather than healthy, adoptable kittens. Alternate forms of testing, such as PCR, to determine the FeLV or FIV status of these cats and kittens is seldom economically feasible for shelters.

**LONG-TERM HOUSING OF INFECTED CATS**

A growing number of shelters or sanctuaries are refusing to euthanize healthy retrovirus-infected cats and consider them adoptable. However, this creates significant challenges, because finding homes for infected cats is often difficult and both FeLV- and FIV-infected cats can live for many years. In one study, median survival of FIV-infected cats was 5 years, and the median survival of FeLV-infected cats was 2.4 years (compared with 6 years for uninfected control cats). Shelters are not the best environment for
housing infected cats that may have compromised immune systems due to risk of exposure to infectious
diseases and the effects of stress. Long-term care of infected cats in sanctuaries or shelters often requires
detailed and expensive medical care, both for wellness and in illness. The AAFP has published guidelines
for management of retrovirus-infected cats that should be consulted. Retrovirus-infected cats require
wellness examinations at least every 6 months as well as periodic monitoring of complete blood count,
serum biochemistries, and urinalysis. Intact cats should be spayed or neutered. Clinical illness requires
prompt and accurate diagnosis as well as aggressive therapeutics. Situations of neglect and suffering can
easily develop if facilities do not have the long-term economic resources to care for retrovirus-infected
cats as well as the expertise and appropriate housing.

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Anorexia in Shelter Cats
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One of the most common problems affecting cats in shelters or rescue situations is anorexia. Since anorexia is a nonspecific clinical sign that can be associated with a variety of underlying disorders, the clinician and shelter worker may find it challenging to diagnose and treat affected cats. Effective intervention requires an understanding of common causes and management options.

Anorexia is the loss of appetite for food. Another useful term is hyporexia, which can be defined as a reduction in appetite. Both are important in feline medicine and should be monitored for in the shelter setting. The distinction is useful both for descriptive purposes and because totally anorexic patients often require more aggressive diagnostic and therapeutic intervention, including assisted feeding. In order to understand if a cat or kitten is consuming adequate nutrition, guidelines for daily caloric intake should be familiar to shelter staff. For adult cats, the starting point for daily resting energy requirement (RER) is calculated by the equation \(30 \times \text{body weight in kg} + 70\). Multiplying by an illness factor is no longer recommended, as it has been associated with higher complication rates. The amount fed should then be adjusted according to monitoring of body weight (BW) and body condition score (BCS). For kittens under 2 kg (4.4 lb), it is more accurate to use the equation \[70 \times \text{BW (kg)}^{0.75}\] to calculate RER.

The detrimental effects of anorexia/hyporexia are well known. Metabolic changes include increases in glucose, lactate, cortisol, glucagon, and norepinephrine as well as increased proteolysis leading to loss of muscle while fat may be preserved. Immune function and wound healing are impaired, and morbidity and mortality are increased. Hepatic lipidosis can occur in older obese cats that have undergone a stressful experience accompanied by anorexia. It is associated with acute and rapid weight loss (40–60%), depression, and icterus; muscle mass is lost while abdominal & inguinal fat is often retained. This disease requires aggressive nutritional management.

There are many potential causes for hyporexia/anorexia in shelter cats. An attempt should be made to prevent hyporexia/anorexia when possible and to find a specific cause when it is identified. Some common causes include:

**Diet Change**
Cats are affected by many qualities of food, such as the taste, smell, format (canned, dry, semi-moist), kibble shape, kibble size, mouth feel, etc. Cats are notorious for developing fixed food preferences that are often shaped by early experience. Food acceptance and intake can also be affected by environmental factors such as the feeding location, timing of feeding, type of bowl, presence of other animals or people, etc. Most cats entering shelters are not accompanied by a diet history, but capturing the information whenever possible for relinquished cats may help with continuity of diet. Cats housed in shelters for any period of time should be fed a consistent diet. Shelters that rely on food donations may find it difficult to provide consistency; feeding programs offered by the major pet food companies should be considered as an alternative. Food should always be fresh and stored appropriately. Poor-quality or spoiled diets may cause anorexia, vomiting, and diarrhea.

**Stressors**
Many external stressors cause changes in feline behavior such as anorexia/hyporexia, vomiting, diarrhea, etc. Stressors may also have wide-ranging effects on cat behavior, such as suppression of normal behaviors (e.g., resting, grooming) and increased vigilance and hiding. Undesirable physiologic responses to stress include hyperglycemia, decreased serum potassium, increased serum creatinine phosphokinase, lymphopenia, neutrophilia, erratic response to sedation and anesthesia, immunosuppression, hypertension, and cardiac murmurs. Examples of stressors include change in diet, change in caretakers, change in daily routine, unnatural day/light cycles, lack of interaction with caretakers, cold ambient temperatures, noisy environments, presence of other cats and dogs, unfamiliar caretakers, unfamiliar environment, rearrangement of cage contents, and lack of environmental resources (e.g., places to hide, perch, etc.). Cats in shelter environments are subjected to many of these stressors on a
routine basis. Many shelter cages were designed at a time when shelter stays were shorter, as animals were likely to be euthanized if not adopted quickly. Therefore, many cages are small (< 6 sq ft floor space) and inhibit normal behavior, because they lack places for cats to perch, hide, and scratch. Small cages also make it difficult to place food and water away from the litter box. Food intake is adversely affected when less than 2 ft of triangulated distance between litter box, resting area, and food/water bowls is available. As well, cats may be housed within the sight of dogs, or at the least, within the smell and sound of dogs. While it is not possible to avoid all stressors, care should be taken to minimize them, and resources are available to maximize the shelter and cage environment for cats. In addition, some group-housed cats may not eat well due to competition and may benefit from single housing.

DISEASE
One of the most common diseases affecting cats in shelters is upper respiratory tract disease (URTD), especially that caused by herpesvirus (FHV-1). In one multicenter shelter study, FHV-1 shedding rates increased from 4% at admission to 50% one week later. Both latent FHV-1 infections reactivated by stress and new infections would have accounted for the increase. As well, concurrent respiratory infections can occur as other pathogens (e.g., Mycoplasma, Bordetella, calicivirus, etc.) are often present in shelters. A recent study evaluated the associations among changes in BW and BCS, behavioral stress score, food intake, and development of URTD in cats admitted to an open-intake municipal shelter. Healthy adult cats (n = 60, > 12 months old) were monitored for a maximum of 21 days. The majority (82%) of cats lost weight during at least one week, primarily during their first and second week in the shelter. Within 21 days of shelter entry, URTD developed in more than 50% of the previously healthy cats. As expected, food intake and stress scores were negatively correlated, and many of the cats that lost weight also developed URTD. Detection and prevention of stress and weight loss could improve wellbeing and may prevent URTD in shelter cats.

Shelters should have procedures in place for monitoring of food intake, BW, and BCS, as well as guidelines for intervention. Cats should be weighed and body condition scored at intake; this should be repeated regularly during the shelter stay. Initially, BW and BCS should be determined weekly and then every two weeks or monthly for stable, longer-term residents. More frequent evaluation is indicated for sick cats and those on medications (many drugs cause hyporexia/anorexia or vomiting in cats). Daily assessment should be performed before cage cleaning and should include food and water intake, presence of vomitus, urine, and fecal frequency and quality (e.g., Nestle Purina fecal scoring system). With ad libitum feeding plans, it can be difficult to detect hyporexia/anorexia, so daily weighing of food bowls is recommended, especially for group-housed cats. A good compromise for monitoring food intake is free-feeding of dry diets in consistent measured quantities combined with one meal daily of a canned diet. Cats prefer shallow bowls for food and water, such as dog-size water bowls and paper plates.

For older kittens and adults, nutritional intervention should be implemented if the cat is eating less than 85% of RER, the cat is anorexic for 3 or more days, or the cat has lost 10% BW or more in a short period of time. Young kittens and sick cats or kittens are more vulnerable to the detrimental effects of inadequate nutritional intake and need closer monitoring. Intervention should begin if a kitten has not eaten for a 24-hour period. Assessment of hyporexic/anorexic cats therefore must include at a minimum: complete physical examination, evaluation of available medical history, nutritional assessment, and environmental assessment. Whenever possible, underlying diseases should be identified and treated where feasible. Cage enrichment and stress amelioration should always be considered in the treatment plan. Use of feline facial pheromone in confined cats is associated with increased food intake and increased normal behaviors such as grooming. Basic supportive care may be required for hyporexic/anorexic cats and could include hydration, vitamin B12 supplementation, and treatment of fever, pain, or nausea if present.

Food aversion occurs readily in cats if they learn to associate eating or the sight or smell of food with feeling sick, nauseous, or painful. Once they are feeling better, they may avoid that food or similar foods for prolonged periods of time. It is important to recognize signs of nausea in cats (e.g., gULING, drooling, dropping food from the mouth, turning away from food) and institute treatment early. Avoid leaving food in the cage of a nauseous cat; instead, offer food intermittently and then remove it. Offering
food that is chilled may help overcome aversion by reducing the odor. Whenever possible, avoid administering medications in food unless they are formulated for that purpose. Cats recovering from food aversion may be helped by offering a different diet, feeding in a new but quiet environment, and even being fed by a new caretaker.

Feeding strategies can be grouped into three levels based on level of intervention:

- **Level 1 (simple interventions):** The sense of smell is particularly important for cats to eat normally and may be impaired in cats with URTD or facial trauma. Enhancing smell and palatability can be accomplished by feeding fresh, canned foods that are warmed no higher than body temperature, and adding water or chicken or tuna broth. Canned foods not only have higher water content than dry diets (> 75% compared with < 10%) but are more palatable due to their higher fat and protein content. For cats that do not accept canned diets, dry kibble can be soaked in water. Human baby foods are a popular choice for tempting cats to eat and are acceptable as long as they do not contain onion and are used for less than 2 weeks. In the short term, caloric intake is more important than nutritional balance. Offer small, frequent meals, as early satiety is common in illness. Hand-feeding, petting, and praise may also be helpful. Appetite-stimulating drugs (Table 1) are not very reliable, and many have undesirable side effects. Their effect on appetite is short-lived, and they are best reserved for convalescent cats with hyporexia and cats overcoming food aversion. They are not likely to induce adequate nutritional intake in an anorexic sick cat.

- **Level 2 (syringe feeding):** Syringe feeding has limited usefulness and is best for cats that are not totally anorexic and will tolerate the procedure. It may be difficult to meet daily caloric needs with this method, and many cats will struggle and resist. Orogastric feeding with a mouth speculum and tube is not recommended by this author, as it is too stressful. However, orogastric tube feeding is an appropriate and effective way to feed neonatal kittens.

- **Level 3 (tube feeding):** Tube feeding is indicated when nutritional support will be needed for more than a few days. In a shelter setting, the easiest method is nasogastric (NG) or nasoesophageal (NE) tube feeding. The tube is easy to place, does not require anesthesia, is relatively easy to maintain, and is inexpensive. The drawbacks are that NG/NE tubes are easy to dislodge, are not useful for long-term feeding (up to about 7 days), and only liquid diets (e.g., CliniCare, Rebound; both 1 kcal/mL) can be fed. Contraindications to placement of NG/NE tubes include the inability to swallow or lack of gag reflex. Complications may include rhinitis, esophageal reflux, aspiration, inadvertent tube removal, or obstruction of the tube.

**Table 1. Common antiemetic and appetite-stimulating drugs for cats**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metoclopramide</td>
<td>0.2–0.4 mg/kg SC, PO q 8 h</td>
<td>Also prokinetic</td>
</tr>
<tr>
<td></td>
<td>1–2 mg/kg/day CRI</td>
<td>Centrally acting?</td>
</tr>
<tr>
<td>Maropitant</td>
<td>1 mg/kg IV, SC, PO q 24 h</td>
<td>Inhibits substance P binding to NK-1 receptors</td>
</tr>
<tr>
<td></td>
<td>(up to 5 days)</td>
<td></td>
</tr>
<tr>
<td>Phenothiazines:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prochlorperazine</td>
<td>0.1–0.5 mg/kg SC q 8 h</td>
<td>Centrally acting via multiple mechanisms</td>
</tr>
<tr>
<td>Chlorpromazine</td>
<td></td>
<td>May cause sedation</td>
</tr>
<tr>
<td>Mirtazapine</td>
<td>1.9–3.75 mg/cat PO q 48 h</td>
<td>5-HT3 receptor antagonist</td>
</tr>
<tr>
<td></td>
<td>Often given as ¼ of 15-mg</td>
<td>Appetite stimulant</td>
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<tr>
<td></td>
<td>tablet</td>
<td></td>
</tr>
<tr>
<td>Cyproheptadine</td>
<td>1.0–2.0 mg/cat PO q 12–24 h</td>
<td>Do not give with mirtazapine (can be used as</td>
</tr>
<tr>
<td></td>
<td></td>
<td>antidote for serotonin syndrome</td>
</tr>
</tbody>
</table>
ONLINE RESOURCES
137. Cornell University College of Veterinary Medicine, Maddie’s Shelter Medicine Program: Enrichment of shelter animals: www.sheltermedicine.vet.cornell.edu/Resources/Enrichment.cfm (VIN editor: Original link was modified as of 3-29-2014.)

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Prevention of disease is one of the primary goals of veterinary medicine. While there is no question that the widespread use of vaccines has improved the health of cats, the current prevalence of vaccination in worldwide cat populations is not enough to achieve herd immunity and eliminate infectious diseases. For example, it is estimated that only 25% of cats in Canada and the U.S. have ever been vaccinated.\(^1\)

The first feline vaccination guidelines were published by the American Association of Feline Practitioners (AAFP) in 1996, with updates in 2000, 2006, and 2013. Other international panels have produced feline vaccination guidelines and feline infectious disease fact sheets, and all documents are available free of charge online (see Recommended Reading). Guidelines documents represent a consensus of a panel of experts in immunology, infectious disease, internal medicine, and clinical practice. The documents are a comprehensive review of the literature with recommendations for feline vaccination protocols derived from evidence-based medicine wherever possible.

The term ‘shelter’ encompasses a broad range of facilities that hold animals that are awaiting adoption, rescue, or return to owners. The facilities may have large or small populations that may be stable or transient. The term also encompasses specialized programs, such as those providing foster care for orphans. Each shelter is unique and so individualized infectious disease management programs are required. In most shelters, eradication of infectious disease is not possible, so efforts are aimed at minimizing risk and controlling the spread of infectious diseases. Vaccination programs are an integral part of disease management in these settings.

Shelter environments often put cats at increased risk of exposure to infectious disease due to the random source of populations, presence of endemic disease, high rates of turnover, environmental stressors, and sustained exposure to infectious disease. Leading vaccine guidelines groups (i.e., WSAVA, AAFP, European Advisory Board on Cat Diseases) have addressed the special situations of shelter animals.

All agree on some basic principles:

- Vaccinations should be limited to those diseases that are likely to be transmitted within the shelter itself.
- Vaccinations may be indicated at an earlier age (e.g., 4–6 weeks) and may be administered at shorter intervals (e.g., 2–3 weeks) compared with schedules for pet cats.
- Modified-live virus (MLV) vaccines should be used in most cases for rapid onset of protection in the face of maternal immunity, and improved protection against illness.\(^2,3\)

A full physical examination should always be performed before vaccination to determine the patient’s age, presence of illness, and other factors that might affect the immune response. The majority of kittens and cats in shelters should be vaccinated regardless of physical condition. Cats with high fever, acute disease, severe injury or debilitation should not be vaccinated until they are recovered.

Other commonly encountered situations include:

- Vaccination is not necessarily contraindicated in cats with stable chronic illness and may be done at the discretion of the clinician.
- Cats with feline leukemia virus (FeLV) or feline immunodeficiency virus (FIV) infection may be at increased risk of infection and so may be candidates for vaccination if they are otherwise in good health. Supportive data is limited, but some experts recommend only inactivated vaccines in these patients.
- Vaccination does not need to be delayed in patients with mild illness (such as upper respiratory tract disease or diarrhea) as long as there is no fever or inappetence and the patient is not debilitated.
Anesthesia and elective surgery do not appear to impair the immune response in otherwise healthy cats.\textsuperscript{4}

For pregnant queens, vaccination is a decision based on risk of exposure versus risk of vaccination. Generally, the use of MLV vaccines containing FPV is not recommended due to potential adverse effects on the fetuses. However, risk of exposure to FPV may be very high in some shelters, endangering the kittens and the queen. In those cases, the overall benefits of MLV vaccination of the pregnant queen may outweigh the risks. Vaccinations against FHV and FCV during pregnancy may be beneficial not only for the queen, but also for the kittens by providing higher levels of maternal immunity in the first few weeks of life.\textsuperscript{5}

Table 1 contains a summary of the most commonly administered vaccinations in shelters. Important points to consider:

- MLV injectable or intranasal (IN) vaccines containing feline panleukopenia (FPV) should not be given to kittens less than 4 weeks of age due to the risk of cerebellar hypoplasia or clinical panleukopenia.\textsuperscript{6}

- Inactivated multivalent feline calicivirus (FCV) vaccines may provide broader cross-protection than single-strain vaccines.\textsuperscript{7,8} Use of a multivalent product may be beneficial in cats in long-term group housing where calicivirus infections are a problem.\textsuperscript{9}

- IN vaccines may result in early onset of protection (e.g., 2–4 days) especially for feline herpesvirus (FHV).\textsuperscript{10} However, study results have been mixed regarding reduction of risk for upper respiratory tract infection in shelter settings when IN vaccination is used simultaneously with parenteral vaccination.\textsuperscript{11,12}

- Only products licensed and approved for IN administration should be given by this route. Mild but transient signs of upper respiratory tract infection may develop following the use of IN vaccines that may be difficult to distinguish from natural infection.

- Intranasal vaccines may not provide sufficient protection against FPV in shelter settings, so parenteral MLV vaccines are preferred for this pathogen.

- Rabies vaccinations are necessary where legally mandated or in endemic regions. For shelters where the length of stay for cats may be weeks or months, rabies vaccinations should be administered at intake.

- FeLV vaccination is recommended for cats housed long term or in group housing. However, vaccination is not a substitution for testing and isolation of infected cats.

Trap-neuter-return (TNR) programs also present unique circumstances for vaccination decisions. Most free-roaming cats entering these programs lack protective antibody titers against panleukopenia, herpesvirus, and rabies.\textsuperscript{13,14} Cats can develop protective antibody titers against FPV and FCV regardless of whether inactivated or MLV vaccines are used. However, in one study, only inactivated FHV vaccines provided protective titers after one inoculation.\textsuperscript{14} Protective titers against rabies will develop in nearly all cats after a single dose of inactivated vaccine.\textsuperscript{14} Most experts recommend that cats in TNR programs receive FPV, FHV, FCV, and rabies vaccinations at the time of surgery.

<table>
<thead>
<tr>
<th>Vaccine</th>
<th>First inoculation</th>
<th>Subsequent inoculations: kittens</th>
<th>Subsequent inoculations: cats &gt; 16 weeks old</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Panleukopenia + herpesvirus + calicivirus</td>
<td>Administer at intake; as early as 4–6 weeks of age</td>
<td>Revaccinate every 2–3 weeks until 16–20 weeks of age</td>
<td>Revaccinate once, in 2–3 weeks</td>
<td>All cats should be vaccinated unless the cat is known to have received inoculations</td>
</tr>
<tr>
<td>(injectable or intranasal)</td>
<td>Rabies</td>
<td>As for pet cats</td>
<td>As for pet cats</td>
<td>If local regulations prohibit shelters from issuing rabies vaccination certificates, cats should be vaccinated by a local vet within 1 month of adoption</td>
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<td>--------------------------</td>
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<td>---------------------------------------------------------------------</td>
</tr>
<tr>
<td>Rabies</td>
<td>Administer at intake or release depending on risk &amp; length of stay; all kittens/cats over 12 weeks of age</td>
<td>As for pet cats</td>
<td>As for pet cats</td>
<td>If local regulations prohibit shelters from issuing rabies vaccination certificates, cats should be vaccinated by a local vet within 1 month of adoption</td>
</tr>
<tr>
<td>Feline leukemia virus</td>
<td>Administer at intake for cats being group-housed; as early as 8 weeks of age</td>
<td>Revaccinate 2–3 weeks later</td>
<td>Revaccinate 2–3 weeks later</td>
<td>Risk of FeLV transmission is very low if cats are housed individually</td>
</tr>
</tbody>
</table>

**Recommended Reading**


**References**


Welcome to the 2014 CVMA Convention in St. John’s, Newfoundland. The following papers are compiled to accompany the presentations scheduled in the continuing education sessions at the convention. The proceedings are organized by day and by stream as follows:

**SATURDAY, JULY 12, 2014**

Companion Animal – Nutrition  
Companion Animal – Behaviour  
Companion Animal – Anesthesia and Pain Management  
Companion Animal – Feline Medicine  
Equine – Inflammatory Airway Disease: An Update  
Equine – Medicine and Management of Geriatric Horses  
Ovine – Diseases and Management  
Other – Team Wellness  
Other – Social Media for Dummies
The Truth About Unconventional Diets for Dogs and Cats
Adronie Verbrugghe, DVM, PhD, DECVCN
Ontario Veterinary College, University of Guelph, Guelph, ON, Canada

Pet food selection and feeding practices are more complex than nutritional science and are influenced by the same social and cultural factors that direct the pet owner’s personal eating behaviors. Human-animal bonding, ideology, personal identity and investment in the pet’s health and wellbeing are reasons why people seek alternatives to conventional pet foods. Pet owners, who are feeding non-commercial foods, appear to reflect greater mistrust of commercial pet foods and pet-food processing than commercial feeders.

**PET OWNER’S CONCERNS**
A major concern among pet owners seeking alternative options for feeding their pets are the types and sources of ingredients used in commercial pet-food manufacturing. Well-known examples are meat byproducts and corn. When choosing a commercial pet food, owners focus on the ingredient list. Still, the ingredient list is a major marketing tool and can be manipulated in many ways. Moreover, the quality of the ingredients is not stated on the ingredient list.

Also of concern is the use of artificial additives. Preservatives, colorants and flavors elicit fear about adverse health effects. There have been well-published cases of additives that have been harmful and withdrawn for use. In many instances however, this concern is undeserved. Additives have many positive aspects including organoleptic, technologic and nutritional benefits. Still, the demand for pet foods free of artificial additives or those prepared with ingredients that are perceived by pet owners to be more wholesome and safe has increased. It is important to identify and discuss a pet owner’s specific concerns as it is likely that an acceptable, commercial, complete and balanced diet is available.

**THE MEANING OF NATURAL, ORGANIC, HOLISTIC, HUMAN-GRADE**
Consumer preference for pet foods free of artificial ingredients has led to commercialization and marketing of natural pet foods. Some regulatory oversight of ingredient use and labeling exists as AAFCO defined the term “natural.” This definition excludes the use of any synthetic preservatives, flavors and colorants. Formulating pet food free of artificial colorants and flavors poses little challenge. Preservation, however, is necessary to protect against microbial attack and to prevent oxidation. Naturally occurring antioxidant compounds can be used as an alternative to synthetic preservatives, yet are less effective. It is therefore important that owners choose a food labelled with a “best before” date and that they don’t feed the food past the labeled date. Also, because most added trace nutrients are chemically synthetic, AAFCO guidelines allow the use of trace nutrients in “complete and balanced” pet foods with a disclaimer (e.g., “natural ingredients with added vitamins and minerals.”)

“Organic” is not the same as “natural.” The term characterises the procedure by which the ingredients are grown, harvested and processed. The US and Canada have established standards that human foods labelled “organic” must meet. Technically, this makes the production of pet food from certified organic ingredients possible, yet it is currently not clear how the rules originally developed for human food and livestock feed apply for pet food.

The term “holistic” has been applied for a wide range of pet foods with a variety of ingredients and characteristics. The term is not legally defined or regulated and is therefore meaningless.

Currently, there are also no standards for designating pet food ingredients as “human-grade.” A pet food manufacturer is therefore free to interpret and use this designation as it sees fit. Still, the public has often a different perception of what human-grade ingredients consist of.
COOKING AT HOME
More than 90% of dogs and cats consume complete and balanced commercially prepared pet foods for at least half of their diet. However, the use of non-commercial diets, including homemade and raw food diets, has risen in popularity among veterinarians and pet owners. For 30.6% of dogs and 13.1% of cats, table scraps, leftovers and homemade foods were fed as part of the main diet. 3% of dog and cat owners fed their pets exclusively with homemade foods. The pet owner’s motivations for providing these non-commercial diets include a desire to pamper the pet, control over the ingredients, avoidance of artificial preservatives, preservation of natural enzymes and phytocellulars, therapeutic reasons because a veterinary therapeutic diet is unavailable or unacceptable, diagnostic reasons such as food elimination trials, and simply because feeding table food has become a bad habit.

When properly formulated and prepared, these diets can provide complete and balanced nutrition. Yet, major areas of concern with homemade diets that often lead to malnutrition in pets are the use of recipes not designed for pets, failure to follow the recipe, and deviation from the recipe over time. Each of these can lead to many nutritional imbalances and related health problems, which have been discussed in many case reports. Less than a third of the owners that feed their pet a homemade diet actually use a recipe designed for pets, putting our pets at an enormous risk for nutritional imbalances. Formulating a complete and balanced pet food requires specialized knowledge and owners should be advised not to try this on their own, but to seek help from someone with this expertise. Still, even if the owners use a well-formulated recipe, the overall nutritional adequacy depends on the selected ingredients and how closely the person preparing the food adheres to the recipe. Owners may decide to leave ingredients out of the recipe or to substitute one item by another or even add new ingredients. It is therefore important to advise owners to follow a properly formulated recipe exactly, and not deviate from the recipe, as any alteration in amount or substitution of ingredients may unbalance the diet and may be detrimental for the pet’s health, unless the change is permitted. Also owners should be advised to avoid toxic food items such as onions, garlic, grapes, raisins, chocolate etc. When the owner is provided with a complete and balanced recipe and with clear instructions on food preparation, it still remains important to follow up with the owner one or more times a year to monitor owner compliance and to examine the patient for signs of nutrient deficiency or excess.

VEGETARIAN AND VEGAN DIETS
Pet owners may choose to feed a vegetarian diet to their pet because of several reasons: religious beliefs, ethical concerns, health considerations and because conventional commercial diets are perceived as unwholesome. In one study all persons who were feeding a vegetarian diet to their cats also reported being vegetarian themselves. Because selecting this type of feeding is a conscious choice for the pet owner, it should be relatively easy to enter into a dialog about the appropriateness and nutritional adequacy of this kind of diet. Many owners may assume that they have to cook if they want to feed a vegetarian diet. However, commercial vegetarian foods for dogs and cats do exist and can be well balanced. Home-cooked vegetarian diets can also be complete if these include eggs and milk products. Vegan diets are a bigger challenge and should be carefully checked because plant-based diets may be deficient in several essential nutrients unless synthetic additives are added. At-home preparation of vegetarian or vegan foods for cats should be discouraged, as without adequate synthetic supplementation, cats are at high risk for many deficiencies. Formulating vegetarian and vegan pet food is extremely challenging and AAFCO feeding trials and a high level of quality assurance are needed to ensure confidence in the finished product. Because the nutritional adequacy of some vegetarian and vegan pet foods can be questioned, it is advised to follow up with the pet owner on a regular basis.

THE TRUTH ABOUT RAW FOODS
The use of raw food diets for household pets as an alternative for conventional diets is a fairly recent development. In 2008, 8% of dog owners and 4% of cat owners fed raw meat with or without bones to their pet. Raw food can be fed as homemade food, but also commercial raw products are available, ranging from complete frozen foods to grain and supplement mixes, which are combined with raw food.
Advocates of raw food claim that dogs should be fed raw meat because their wild canine ancestors survived on uncooked food. Still, no scientific data exist to support that dogs should eat uncooked food as did wild canids. Claims that dogs are carnivores are due to confusion, as dogs belong to the order Carnivora taxonomically, but their eating habits are those of an omnivore. Raw food diets are promoted enthusiastically because of the purported benefits. There is, however, no objective scientific evidence with regard to disease prevention and resolving or amelioration of preexisting conditions.

Advocates of raw food also emphasize the importance of ingredients with less emphasis on nutrient balance and claim that nutrients from commercial dry and canned food are less or not available or even absent when compared to feeding raw ingredients. No studies are available that compare the digestibility of the different types of food. Reservations have also been expressed with regard to nutritional adequacy. Many home-prepared raw food as well as commercial raw-food diets are not complete and balanced and, therefore, inappropriate for long-term feeding. To date, no scientific evidence exists that demonstrates raw food diets provide additional or exceptionally unique nutrients that cannot be obtained from cooked food.

Another important concern is the potential for bacterial contamination and risk for foodborne illness. Salmonella was isolated from 80% of raw-meat canine and feline diets prepared by Canadian pet owners. Of 25 commercial raw-meat diets of eight different manufactures in Canada, 64% were positive for Escherichia coli, 20% for Salmonella spp. and 20% for Clostridium perfringens. Advocates of feeding raw meat, bones and eggs claim that pathogenic organisms in raw meat do not affect dogs and cats due to the lower stomach pH and shorter gastrointestinal transit time. In fact, these are very similar among humans, dogs and cats and do not lower the risk to pets. Dogs and cats succumb to foodborne pathogens and exhibit clinical signs similar to those in humans. It is important to point out that meat and eggs supplied for human consumption are contaminated with microorganisms and feeding raw meat increases the exposure of owners and pets to foodborne bacterial diseases. Pet owners may not realize that infected dogs may shed bacteria capable of infecting people, yet remain clinically normal. Several studies have demonstrated a link between faecal excretion of organisms in dogs and the presence of the same organism in their raw-meat diet. Furthermore, transmission of Salmonella infection from pets to people has been documented. Safe handling of food, work surfaces and feeding containers is therefore of extreme importance. Extra caution should be emphasized when elderly persons or young children are living in the household or when persons in the household have immunosuppressive infections, are undergoing chemotherapy or are being treated with anti-inflammatory medications. Veterinarians recommending commercial or homemade foods containing raw meat or eggs have an ethical responsibility to fully inform pet owners of this increased potential risk for foodborne pathogens, not only to the pet, but to the entire household.

Overall, the diet history should be used to understand the pet owner’s attitudes towards commercial pet food, feed ingredients, nutrition and nutritional therapy. Furthermore, it is important that veterinarians obtain the client’s beliefs and understanding about how his or her pet should be fed and obtain the pet owner’s viewpoint regarding the need to change feeding practices. When formulating a plan for dietary modification, veterinarians should take into account the pet owner’s beliefs, cultural background, lifestyle, and abilities and assess any concerns that may arise from the proposed dietary modifications. By anticipating problems, the veterinarian should be able to craft the dietary interventions in a way that is more acceptable for the household or look for compromise when the recommendations and the pet owner’s preferences are in conflict. Also communication with the pet owner about the rationale for the dietary changes could be more effective and the veterinarian should be in a better position to explain why the proposed changes are in the best interest of the pet.
The Nutritional Approach to Senior Cats
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More pet cats are getting older; approximately 40% are at least 7 years old. Age-related diseases begin to increase in prevalence around seven or eight years; this prevalence is coupled with the gradual onset of behavioural, physical and metabolic changes. Some changes are obvious, like whitening of hair, general decline in body and coat condition, and failing senses including sight and hearing. However, other less obvious changes include alterations in the physiology of the digestive tract, immune system, kidneys and other organs. Research on the physiological and medical changes affecting the nutrient requirements of older cats is limited.

Geriatric screening should be considered as a preventive medicine service, conducted to identify diseases in their early stages, or to head off preventable diseases, helping to maintain good health and maximize longevity. An important aspect of this evaluation is a thorough nutritional assessment.

Geriatric Nutritional Evaluation
According to the WSAVA nutritional assessment guidelines, a nutrition evaluation should be completed in every patient, especially older patients, before instituting a dietary change. This nutritional assessment should include an evaluation of the patient, the diet and feeding management. The goal is to identify the presence and significance of risk factors related to malnutrition.

Patient evaluation should include a complete medical history and a thorough physical examination. Body weight and body condition score are important to assess. Increases or decreases in body weight or condition should trigger further evaluation. A longitudinal study in cats indicated that weight loss is evident in aging cats approximately 2 to 3 years before death from various causes, often well before clinical signs are apparent. Moreover, middle-aged cats tend to have reduced energy needs. If caloric intake is not adjusted accordingly, weight gain will result. Yet, body weight and body condition are not the whole picture. It is also important to assess changes in muscle mass, using a muscle condition score. Animals, especially those that are sick, may be losing lean muscle mass despite an abundance of body fat. Pronounced loss of lean body mass is associated with increased mortality and morbidity.

A complete evaluation of the diet and feeding management should include a thorough diet history, including the specific diet, additional treats, table food, or supplements, food used for medication administration etc. It is also important to consider how foods are provided and how they are accepted by the cat. Owners should be asked: How much and how often each food is fed? Is the pet fed measured amounts of food or free choice? Are the cats in a multiple cat household sharing a food bowl or do they have access to other pet’s food? The diet history should determine any recent changes in diet, quantity fed or appetite.

The information from the diet history is not only important in determining the adequacy of the current diet situation; it is also important in planning a dietary recommendation that will achieve good client and patient acceptance and compliance. Once the nutritional characteristics of the total diet are known, it should be compared against the individual patient’s needs and key nutritional factors. If the cat’s weight, BCS and MCS are optimal, no illnesses are present, and the cat is eating an appropriate complete and balanced diet for adult maintenance, there is no reason to change the diet of an older cat.

Effects of Aging on Nutritional Requirements
Cats have been shown to have reduced ability to digest energy, fat and protein with increasing age. However, in most of these studies, not all cats showed these changes so these alterations may not be universal among cats. The mechanisms behind the observed reductions in nutrient digestibility were not identified in these studies and additional research is required.

The maintenance energy requirement (MER), the amount of calories a healthy animal with normal activity needs to keep up with its body condition, appears to decrease with age in most animals. Age-related changes in MER in cats are more controversial. Short-term studies have shown no change in MER within aging cats, while longer studies have shown that MER decreases with age until approximately 11
years and increases thereafter. The primary driver of energy requirements in animals is lean body mass, which includes muscle, skin and organs and accounts for about 90% of basal energy expenditure. Across species, including cats, lean body mass decreases with age. Also the decrease in activity can contribute to the reduction in MER seen in middle-aged cats. If the MER increases and energy intake does not decrease accordingly, the cat will gain weight and become overweight. Many commercial senior-cat foods have lower dietary fat levels and fewer calories. Some also have added dietary fibre to further reduce the caloric density.

Not all older animals are less active or overweight. In fact, a great proportion of cats over 12 years of age are underweight. This may be explained by an increase in MER in this age group, but also a reduction in digestive capabilities, changes in smell, taste and appetite and increased prevalence of dental disease, could contribute to weight loss. These patients may benefit from more energy dense, highly digestible diets.

Dietary protein is another important nutrient for aging pets. Many veterinarians recommend protein-restricted diets for older cats because of the increased prevalence of renal pathology in this age group, especially as the perception that excessive protein intake causes kidney damage still remains in human and veterinary medicine. Nonetheless, high protein intake appears not to contribute to the development of kidney disease in healthy animals. No clear consensus about the role of protein reduction in slowing progression of clinical and subclinical renal disease exists. The rationale for reducing protein in diets for patients with renal disease is to reduce production of nitrogenous waste products of protein catabolism that are thought to be important causes of uremic signs and to limit the acid load imposed on patients with chronic renal disease.

However, when dietary protein intake is insufficient, the body responds by decreasing protein turnover, and mobilizing protein from lean body mass to support essential protein synthesis. In addition to the effect of inadequate protein intake, aging itself has a detrimental effect on protein turnover and lean body mass. This has been the basis for the argument that protein intake in mature cats should be higher than for young adults. As with all life stages, healthy mature cats should receive enough protein and energy to avoid protein-energy malnutrition. Still, a recent study shows that at some point in age it may be impossible to keep up with the cat’s protein needs, even when feeding a high-protein diet.

Also a reduction of dietary phosphorus is commonly recommended in foods designed for mature adult cats. This is based on the fact that nearly 30% of older cats may have kidney disease. Renal insufficiency is rarely diagnosed until significant loss of renal function has occurred. Thus, large proportions of older cats have subclinical renal damage and may benefit from reduced dietary phosphorus as it is commonly accepted that phosphorus restriction slows the progression of renal disease in cats. Still, as long as the senior cat does not show any clinical signs and there are no early renal changes notable on blood work, urinalysis and abdominal ultrasound, excessive amounts of phosphorus should be avoided, yet it is not necessary to restrict phosphorus. Many phosphorus-restricted diets also have lower calcium concentrations, which may affect bone mineral density. Osteoporosis is not commonly diagnosed in old cats. Nevertheless, the bone mass of adult cats remains stable until 7 years of age, then declines. Moreover, it is difficult to keep the dietary protein content high enough when restricting phosphorus as animal protein is the main source of both protein and phosphorus. If a complete diagnostic workup is not available and it is not possible to follow up closely with the patient, it is recommended to aim on the side of caution and prescribe a maintenance diet that is not too high in phosphorus. If a complete diagnostic workup is not available and it is not possible to follow up closely with the patient, it is recommended to aim on the side of caution and prescribe a maintenance diet that is not too high in phosphorus. In case the cat is diagnosed with kidney disease, an appropriate veterinary therapeutic renal diet is recommended to prolong survival time, decrease uremic episodes and delay disease progression.

**THE SENIOR DILEMMA**

Given the limited amount of research on the nutrient requirement of senior cats, the Association of American Feed Control Officials (AAFCO) and National Research Council (NRC) have no definition for senior life stage and have not established specific nutrient requirements for this life stage. The AAFCO nutrient profile remains the same regardless whether the cat is 2 or 12 years old. Likewise, AAFCO feeding trails for senior cat diets must follow the protocol guidelines for adult maintenance. The term “senior” or “mature” is not regulated, meaning that the nutritional adequacy statement for this type of...
diets can be “adult maintenance” but also “all life stages.” Some common characteristics of commercial senior-cat foods include reductions in calories and fat, altered protein levels, reduced sodium and phosphorus levels, added fibre compared with adult maintenance diets. However, because no specific guidelines exist, nutrient profiles may vary widely among these diets and finding the right senior diet for an older cat can be a challenging task.

The majority of aging cats are generally healthy but may have special dietary needs. It is critical to base dietary recommendations on a thorough nutritional assessment of the individual cat. Commercially available senior diets vary widely in nutrient composition and should be examined carefully before they are recommended to each patient. Overall, all senior cats benefit from a highly digestible diet. The diet should also be highly palatable and energy dense in order to stimulate appetite and caloric intake, unless the cat is prone to obesity. In this case a lower-fat, higher-fibre diet is recommended. Don’t restrict protein and phosphorus unless medically necessary. Moreover, aging pets should be monitored regularly to confirm that the desired nutritional benefits are being achieved, and to assess any need for new dietary changes.
Altering ingredients, nutrient profile and feeding method can be a powerful tool in managing gastrointestinal (GI) disorders as standalone therapy or in combination with drug therapy. Drug therapy instituted without dietary therapy often yields less than desirable results. Moreover, foods or ingredients may occasionally function as diagnostic tools in evaluating pets with GI disease. As vomiting and diarrhea have numerous causes, feeding plans vary accordingly and multiple dietary manipulations should be considered in each patient individually.

**Highly Digestible/Low-Residue Diets**
A major consideration when choosing a diet for animals with GI disease is the nutrient digestibility. Gastrointestinal foods are highly digestible, meaning that protein digestibilities are higher than 87% and fat and carbohydrate digestibilities exceed 90%. To increase the digestibility, commercial veterinary therapeutic diets formulated for GI disease contain highly refined protein and carbohydrate sources. Crude fibre levels greater than 5% on dry matter basis (DM) are unusual for these diets, because fibre reduces digestibility. More recently, some manufacturers have added small amounts (< 5% DM) of soluble or mixed fibre as short-chain fatty acids (SCFA) produced by intestinal microbial fermentation of fibre may positively affect the intestinal mucosa. These diets are generally recommended for the management of acute gastroenteritis, small bowel disease or exocrine pancreatic insufficiency. Also patients with colitis or constipation may benefit from these diets, as these diets may reduce exposure of the colonic mucosa to ingesta.

**Fat-Restricted Foods**
A reduced fat intake is often recommended when fat malabsorption and maldigestion are present due to exocrine pancreatic insufficiency, short-bowel syndrome and lymphangiectasia, especially as unabsorbed fat in the bowel lumen causes secretory diarrhea. In dogs, diets containing 12–15% DM fat are generally tolerated. For cats with diarrhea, dietary fat content does not appear to affect outcome, making fat levels up to 23% DM acceptable. However, some patients might need a lower fat content (< 10% DM); yet, due to the lower energy density, larger food volumes need to be fed to meet the patient’s caloric needs, which might not be tolerated.

**Elimination Diets**
When patients are suspected of food intolerance or food hypersensitivity, elimination foods are recommended as diagnostic and therapeutic tool.

As most food allergens are thought to be glycoproteins, dietary protein is the nutrient of most concern in patients suspected of food allergy. The number of different proteins in the food, the protein sources and amount of protein comprise the key nutritional factors for elimination foods. Novel protein sources are usually defined as “animal or vegetable ingredients containing proteins that are not commonly used in pet food and/or not commonly associated with adverse food reactions.” Venison, rabbit, duck, kangaroo, various fish, oats, potato, sweet potato and green peas are examples of such protein sources. Beef, dairy products, wheat, egg and chicken are the five most common food allergens in dogs. In cats, beef, dairy products, fish and lamb are most commonly reported ingredients causing adverse reactions. Elimination diets that contain intact proteins should avoid these most common food allergens and have maximum two protein sources to which the patient has not been exposed before, including both animal and vegetable protein sources. A thorough diet history can provide information about previously consumed protein sources and help define the most appropriate novel protein source.

Another approach to providing novel ingredients is the use of hydrolysed proteins. Hydrolysis of proteins to smaller peptides and amino acids reduces the molecular weight of the original protein, by which the antigenicity and allergenicity of the protein are reduced. This means that antigens are no longer recognised by the immune system of patients already sensitised to the intact protein and therefore...
mast cell degranulation and clinical signs are prevented in patients already hypersensitive to the intact protein. Secondly, sensitisation of a naïve individual might also be prevented. Thereby less concern for sensitisation to the novel protein is needed during the initial treatment phase when intestinal permeability might still be higher. Protein hydrolysates of appropriate molecular weight (< 10 kDa) are less likely to induce an immune-mediated response. Protein-hydrolysate diets have been reported to be effective and well tolerated when used as elimination diets for the diagnosis and treatment of adverse food reactions in dogs and cats. Overall it is recommended to avoid excess protein to reduce the amounts of potential allergens to which the patient is exposed. The importance of low-protein contents demands, however, a little nuance: low-protein content is only beneficial in non-allergic reactions (food intolerance), because in cases of food allergy, the smallest amount of protein already evokes clinical response. Foods for dogs should provide 16 to 22% DM protein, foods for cats should have 30 to 45% DM protein. A higher protein level may be necessary to counteract protein losses or impaired absorption in patients with hypoproteinemia and weight loss associated with severe GI disease.

Fibre-Enhanced Diets
This type of diets is beneficial in managing many large and some small bowel diseases, though fibre levels and sources vary tremendously among available foods.

Soluble fibres, such as pectins and gums, increase the viscosity of intestinal contents, which delays gastric emptying, slows small-bowel transit time and reduces the rate of nutrient absorption. Bacteria in the colon ferment soluble fibre with the production of SCFA, which are a key fuel for colonocytes and are capable of stimulating enterocyte and colonocyte proliferation. Certain soluble fibre types also bind toxins and irritating bile acids, and thereby prevent further damaging of the intestinal mucosal surface.

Insoluble fibres primarily composed of cellulose and structural polysaccharides are resistant to digestion and ferment very slowly. One of the major effects of insoluble fibre on the GI tract is the normalization of the gut motility. Increasing the bulk due to increased bacterial cell mass, non-degraded fibre residue, faecal water or a combination of these factors, provides a physical stimulation to normalize intestinal transit times. This may be helpful in patients with colitis or chronic constipation. Still, if the motility patterns of the patients with obstipation are completely abolished (e.g., severe end-stage megacolon in cats), fibre-enhanced diets and fibre supplements will no longer be effective stimulants of colonic motility and, worse, can contribute to obstipation.

Altering Gut Microbiota
Many studies have emerged recently about the importance of the gut microbiota to GI health in particular and immune function health in general.

Prebiotics, defined as “non-digestible food ingredients that selectively stimulate a limited number of bacteria in the colon to improved host health,” have been used extensively in veterinary therapeutic diets for GI disease. Prebiotics are mainly non-digestible carbohydrates such as mannan oligosaccharides, fructooligosaccharides, lactulose, inulin and resistant starch, are resistant to enzymatic hydrolysis and are substrates for “beneficial” bacteria. Administration of prebiotics causes a shift in the composition of the gut microbiota in favor of a “healthy” flora. All prebiotics tested in vitro increased bifidobacteria and most decreased clostridia. Data from canine and feline feeding studies are limited and provide mixed results.

A second approach to modulate gut microbiota is feeding probiotics. Probiotics are live bacteria that have beneficial effects on the health of the host when administered in adequate amounts. Probiotics should be non-pathogenic and resist stomach acid and bile. Some probiotics adhere to intestinal epithelial tissue and colonize the intestinal tract. Adherence but not colonization may be necessary for some beneficial effects. As with prebiotics, only limited studies have been performed in dogs and cats. One study in dogs with food-responsive diarrhea only showed very mild effects of a probiotic cocktail containing three different Lactobacillus spp., while a second study in dogs with acute idiopathic diarrhea examined Bifidobacterium animalis AHC7 and did show reduced time to resolution and a reduced percentage of dogs that were administered metronidazole compared to the placebo. Furthermore, shelter cats fed Enterococcus faecium SF68 had fewer episodes of diarrhea of ≥ 2 days when compared with
controls. Also, many stability challenges exist for incorporating probiotics into pet food. Evaluation of 19 canine and feline products claiming to contain probiotics showed that none of the products contained all the listed microorganisms, 11 products contained additional microorganisms and five products did not have any relevant growth when tested in vitro.

Feeding synbiotics is a third approach to modifying gut microbiota. This new concept, “a mixture of both probiotics and prebiotics that beneficially affects the host by improving survival and implantation of live microbial dietary supplements in the GI tract,” also requires more research.

**Feeding Management**

A last aspect to consider is **meal size, frequency and consistency**. Generally, small meals are fed several times per day. This reduces gastric distension, decreases gastric secretion and may reduce nausea, vomiting and gastroesophageal reflux. Furthermore, the larger the volume of food ingested, the less that can be effectively assimilated. Liquid diets generally empty faster from the stomach than canned foods, and canned foods empty faster than dry food. If liquid diets are fed too fast or in large volumes, diarrhea may occur. The use of liquid diets is therefore limited to specialized circumstances and specific GI disorders such as esophageal stricture, selected cases of achalasia and gastric outflow disturbances, to reduce regurgitation and vomiting.

Each patient with GI disease should be seen as an individual variant of the norm. Therefore, multiple dietary manipulations should be considered as needed for each patient. Because of the diverse nature of GI diseases, a number of food types may be appropriate. Nutrient profiles should be considered as starting points and adjusted for each patient individually as necessary. Relative terms such as “low” versus “high” are too often used without reference point. This should be avoided and changes of nutrient concentrations should always be relative to the previous food, making a complete diet history a necessary tool.
COMPANION ANIMAL – BEHAVIOUR

The Importance of Recognizing Silent Threats in Dogs and Cats
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SILENT THREATS IN CATS
Laboratory studies carried out by Leyhausen\(^1\) led to the description of offensive behaviour seen during tom cat duels. The first sign of an attack was a threat in which the aggressor drew itself up high on all four limbs. The back was straight and slightly elevated caudally. Piloerection along the middle of the back accentuated this slope. The tail was held stiffly and almost perpendicular to the ground. The distal end could twitch back and forth. The head was stretched forward, the pupils were somewhat dilated and the ears were raised upward and swiveled. The cat stared at its opponent, approached slowly, vocalized and swallowed intermittently. Similar threats (posture, facial expression and staring) can also be observed in housecats. They are often silent, without piloerection and thus are generally missed by owners. The threatened cat can respond defensively (hissing) to this silent threat, leading the owners to blame the wrong cat as the aggressor.

Leyhausen\(^1\) also described defensive behaviour in cats. The cat would lower itself close to the ground, shrink and draw its head in as far as possible. Its ears were pulled down sideways, sometimes completely flat. The pupils were dilated.

Threats can be overt (active aggression) or covert (passive aggression). Passive aggression involves mostly silent threats such as staring, a rigid body, and flicking tail. The threatening cat may be standing, sitting or lying down. Ears are directed forward or swiveled. The recipient cat usually tries to avoid visual contact and may be defensive. The victim may crouch down, ears flattened on the head, try to make a huge detour past the aggressor, hiss and swat. The owners may wrongly accuse the victim because they will not recognize the covert silent threats of the aggressor. The aggressor may also be following (stalking) the victim everywhere.

Questions on presence of passive aggression (silent threats) are essential to ask during the clinical evaluation for intercat aggression. Most owners are unaware of the importance of these threats.

BEHAVIOURAL CONSEQUENCES IN CATS
In a study of 736 cats,\(^2\) 185 (25.1\%) presented for intercat aggression and 170 (23.1\%) with urination outside the litter box. The probability of having both diagnoses should therefore have been 5.8\% \((0.251 \times 0.231)\). Of 185 cats presented for intercat aggression, 2.7\% also urinated outside the litter box. Of 170 cats presented for urination outside the litter box, 2.9 \% had intercat aggression. Intercat aggression and urination outside the litter box occurred together less often than chance would predict. Urination outside the litter box is therefore more likely to occur for reasons other than intercat aggression. However, some cases of urination outside the litter box may be associated with aggression between household cats since these problems occasionally do co-occur.

On the other hand, aggressive interactions with other household or outdoor cats were the two factors most commonly associated with the initiation or the continuation of spraying.\(^3\) Case reports also highlight the coexistence of urine spraying, house soiling and intercat aggression.\(^4,5\)

In inappropriate elimination and spraying cases, the owners may be aware of coexisting aggression but may not always realize that aggression is directly linked to the other problems. Treating the intercat aggression (active and passive aggression) becomes essential in order to resolve the soiling or spraying problems.

Sometimes the only sign of passive aggression is a cat that becomes withdrawn. A descriptive study\(^6\) of the use of space and patterns of interactions of 14 unrelated (7 males and 7 females), neutered indoor cats in one home was published. “Lily was the most restricted cat, spending almost all her time on top of the refrigerator. She came down off the refrigerator after the death of a cat named Julius.” Little
overt aggression was reported in the home, but Lily was most likely a victim of passive covert aggression by Julius, unbeknownst to the owners. Recognizing passive aggression allows us to identify the true aggressor and treat accordingly.

The importance of stress and its role in other medical conditions, such as interstitial cystitis, dermatological conditions, and gastrointestinal disorders, is also being reported. Treatment for feline interstitial cystitis (FIC) now includes recommendations to decrease stress factors in the environment. When looking at multicat households, cats with FIC were more likely to be in conflict with a housemate than cats in the control population.

**Silent Threats in Dogs**

Aggression between household dogs can include displacing the victim, blocking access or silent threats. Dr Karen Overall refers to these as nonspecific signs often missed by the owners. Examples can include, but are not limited to, blocking access to a bed, crate, food, water, or stairs by positioning themselves “in the way.” Threats are done by staring directly at the victim, or approaching the victim at a 90° angle (nose of the aggressor is at a 90° angle to the victim’s shoulder). The victims of silent threats may become more defensive and aggressive to protect themselves, leading owners to think the victim is the “problem” dog.

Questions on presence of passive aggression (silent threats) are essential to ask during the clinical evaluation for interdog aggression. Most owners are unaware of the importance of these threats.

**Behavioural Consequences in Dogs**

The victim dog may not want to go outdoors to eliminate because of silent threats from the aggressor. This behaviour occurs only at times when the aggressor is also outdoors. The victim dog may not want to get off furniture and may growl at the owner, when in fact, the nearby presence of the other household dog staring at him is the trigger for the growling. Other signs would include one dog making detours around another one or actively avoiding the other household dog.

This presentation will focus on video examples of silent or subtle threats in cats and dogs and will talk about treatment.

**References**

Obsessive-Compulsive Disorders: Medical, Behavioural or Both?
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**Definitions**
A stereotypy is defined as a repetitive, constant behaviour and appears to serve no obvious purpose.\(^1\) Obsessions are described as recurrent and persistent thoughts, impulses or images that are experienced as intrusive and inappropriate, thus causing marked anxiety and distress.\(^2\) Compulsions are repetitive behaviours performed to prevent or reduce distress.\(^2\) In psychiatry, obsessions or compulsions are time consuming or significantly interfere with the person’s normal routine, occupational functioning, social activities or relationships. The veterinary literature in addition to the term obsessive-compulsive disorders (OCD) also includes compulsive disorders (CD).\(^3\) Compulsive disorders (CD) are defined as behaviours that are usually brought on by conflict, but that are subsequently shown outside the original context. Compulsive behaviours seem abnormal because they are displayed out of context and are often repetitive, exaggerated or sustained.

**History of the OCD Label in Veterinary Medicine**
In 1991, an article\(^4\) published in the *Journal of the American Animal Hospital Association* was entitled “Canine acral lick dermatitis: response to anti-obsessional drug clomipramine.” The authors felt that acral lick dermatitis (ALD) was a good model of human OCD because the dermatitis had been refractory to previous attempted treatments but had improved with psychotropic medication: 50% control of symptoms for clomipramine and fluoxetine.\(^5\) Previously attempted treatments\(^5\) included steroids, bandages, Elizabethan collar and other (unspecified). Several other articles using canine acral lick as a model for OCD in humans\(^6,7\) were subsequently published. No specific details were given in one study\(^7\) as to medical workup done in the 63 study dogs. Unsuccessful treatments in the other study\(^6\) listed local and/or systemic cortisone, iodine ointment, salts and surgical removal. Antihistamines, corticosteroids, topical application of corticosteroid creams and use of bandages and Elizabethan collars were also listed in a different study.\(^8\) Antibiotics were never included among attempted treatments in the above mentioned studies.

MacDonald and Bradley\(^9\) write that infection is almost always present in acral lick dermatitis and that antibiotics are one of the most important treatments of ALD. These agents should be used in all cases and therapy may require 4–6 months. A more recent study\(^10\) concludes that “Lesions associated with ALD warrant tissue bacterial cultures as the majority of cases yielded positive growth of bacteria differing from superficial culture and often resistant to empirical drugs.” Current text books and dermatology conference notes emphasize that ALD should be viewed as a primary disease that is complicated by perpetuating factors. Atopy and food allergy, as well as secondary deep pyoderma, should be high on the list of differentials. Also according to a case series\(^11\) “six dogs were presented with acral lick dermatitis-like lesions as a result of different underlying causes, namely lymphoma, an orthopedic pin, deep pyoderma, mast cell tumor, leishmaniasis, and (presumptive) sporotrichosis.”

As a result of those studies making an analogy between ALD and OCD in people, the veterinary literature then introduced OCD as a diagnosis in cats and dogs presenting repetitive behaviours. Examples of listed compulsive disorders in dogs include shadow chasing, light chasing, spinning, spinning/tail chasing, acral lick dermatitis (ALD), self-mutilation, fly biting, pica, fence running, flank sucking, checking hind end, and excessive licking of objects. Examples of listed compulsive disorders in cats include wool or fabric eating, pica, excessive grooming, hyperesthesia, and tail chasing.\(^12\)

The case of ALD becoming an OCD highlights two important points: 1) Complete medical investigation of dogs with ALD included in those 5 studies\(^4,4,5\) was not described and most likely not done; 2) Improvement with various psychotropic medications is not sufficient to conclude that ALD is a “behavioural disorder” or an OCD/CD. Perhaps, if these dogs had received appropriate medical treatment (for atopy, food allergy and deep pyoderma), the ALD would have resolved completely.
**WHAT WE CURRENTLY SAY**
To be labeled a true CD, the repetitive behaviour should occur in the absence of any primary dermatologic, neurologic or other medical condition. However, extensive medical workup has not been done yet for many of these repetitive behaviours.

**WHAT WE DO NOT KNOW**
There are few published scientific data on compulsive disorders in dogs and cats. In several studies, various different repetitive behaviours (locomotor, oral) are grouped together within one publication.\(^{13-15}\) The number of dogs or cats presenting a specific repetitive behaviour is generally low and treatment outcome is not given by type of repetitive behaviour. Luescher\(^3\) reported that approximately two thirds of CD cases improved to the client’s satisfaction. No details, however, are given about improvement (complete resolution versus decreased frequency and/or decreased duration of the repetitive behaviours) or which specific repetitive behaviour improved and which one did not.

Unanswered questions include how many and which repetitive behaviours could in fact be nonspecific signs of strictly medical conditions and not OCD/CD. Do we have the technology and knowledge to identify all underlying medical causes? Is it possible that there might not be a good or affordable way to diagnose an underlying medical problem? Could the medical condition in some cases be difficult to treat (i.e., no good treatment available)?

**WHAT WE ARE LEARNING**
Neuropathic pain is typically a consequence of injury or disease that damages the axon or soma of sensory neurons or disrupts the myelin sheath of axons. Neuropathic pain\(^1^6\) associated with intervertebral disc herniation is common and results in persistent or intermittent pain. Spinal cord injury (trauma, ischemia, hemorrhage or extradural compression) can result in somatic or visceral neuropathic pain. Radicular (referred) pain is observed with impingement of nerve roots. Behaviours such as scratching motion without touching the skin (Chiari-like malformation), continually biting or attacking an area on the body, frequently turning (looking) at the same area or yelping for no reason, should alert veterinarians to potential neuropathic pain. Therefore, some of the repetitive behaviours labelled as OCD/CD such as “flank sucking,” “self-mutilation,” “checking” could in fact be secondary to somatic or visceral neuropathic pain. Anecdotally, the author has seen a few cases of dogs with self-mutilation of the prepuce and penis that had back pain and improved significantly with gabapentin.

Neuropathic pain is often worsened by stimuli that evoke a sympathetic response such as the startle response and emotional arousal.\(^1^6\) Feline interstitial cystitis (FIC) is associated with visceral neurogenic (neuropathic) pain. Treatment for FIC now includes recommendations to decrease stress factors in the environment. When looking at multicat households, cats with FIC were more likely to be in conflict with a housemate than cats in the control population.\(^1^7\)

Inflammatory bowel disease (IBD) is frequently diagnosed in dogs and cats. Some anecdotal reports\(^1^6\) of amitriptyline use in cats already adequately treated for their IBD but still uncomfortable according to their owners, indicated improvement in behaviour, thus suggesting a potential neuropathic component to the IBD.

Anecdotal, unpublished cases of IBD have been reported in both dogs and cats presenting with pica. The author believes (studies are needed) that conditions labelled as oral stereotypies or oral compulsive disorders are more likely signs of gastrointestinal disease than behavioural disorders. These repetitive behaviours in dogs include fly biting, excessive licking of surfaces, air licking, star gazing, flank sucking, and pica. IBD may in some cases be causing anal sac disease.\(^1^8\) Therefore, “checking behaviours” reported in Miniature Schnauzers should include gastrointestinal disease in the differential. These dogs may be experiencing pain and are therefore looking repeatedly at their hind end.

Pica in cats has been reported as a sign of feline immunodeficiency virus (FIV),\(^1^9,2^0\) a sign of gastric motility disorder,\(^2^1\) and associated with chronic anemia.\(^2^2\) Cats may eat kitty litter or may lick concrete or ceramics. Compulsive licking at concrete, carpeting or other cats was also reported in 3/16 cats with chronic feline infectious peritonitis (FIP).\(^2^0\)
Perhaps in some of the case reports of stereotypic motor behaviour, the abnormal behaviours described (pacing, panting) were associated with gastrointestinal discomfort rather than anxiety.

Feline symmetric alopecia (FSA) is a clinical reaction pattern in which cats present with symmetric alopecia over the thorax, flanks, ventral abdomen or pelvic regions. Excessive grooming of the ventral abdomen may indicate abdominal pain, particularly of the bladder. Radiographs may reveal arthritic changes in older cats, such as intervertebral arthritis. Resolution of the overgrooming and regrowth of the hair following treatment for pain provide evidence that pain may be causing the overgrooming. True behavioural causes of overgrooming are rare. Of 21 cats with presumptive psychogenic alopecia, 16 showed medical causes of pruritus. Three cats had both a psychogenic alopecia and a medical condition and only two were found to have psychogenic alopecia.

Results of two studies will be presented: 1) Gastrointestinal disorders in dogs with excessive licking of surfaces and 2) Prospective medical evaluation of 7 dogs presented with fly biting.

**Conclusion**

Each type of repetitive behaviour should be investigated with a systematic, rigorous, medical approach in order to serve our patients and clients better! We still have a lot to learn!

**References**

2. Diagnostic criteria from DMS-IV-TR. Obsessive-compulsive disorder. Published by the American Psychiatric Association; 2000;217–218.


**Puppy Behaviour in the Veterinary Clinic**

One hundred two puppies (46 males, 56 females), ranging in age from 8 to 16 weeks and adopted at least 1 week prior to the evaluation, were included in this study. Eighteen were mixed breed and 84 were purebred puppies. All puppies were intact at the time of the examination.

**Assessments**

Interested owners recruited from 5 different clinics in the Québec City area were asked to book an appointment with the principal investigator (veterinarian) at Loretteville Veterinary Hospital (hospital assigned for the evaluation). Owners were informed that the technician would take the dog to the veterinarian immediately on their arrival. This step was done to standardize as much as possible the sequence of events prior to each evaluation. Concerns included stimulation of the puppy by other dogs or people present at the veterinary clinic as well as owner interactions with the puppy. The principal investigator performed each assessment in the absence of the owner and always in the same examination room of Loretteville Veterinary Hospital. The assessment was divided into 3 different parts (3 different contexts):

**Free-Floor Evaluation (FF)**

The puppy was initially set free on the floor for approximately 2 minutes while the veterinarian sat in a corner of the room filming but not interacting with the dog. No special objects were available or presented to the dog during this evaluation. The room contained the examination table, one chair, and a rubber doorstop on the floor. It was impossible to control noise made by people and other animals elsewhere in the clinic, but such occurrences were assumed to be random with respect to the independent variables.

**Physical Examination on the Table (PET)**

Next, the veterinarian examined the dog on a stainless steel table (105 cm by 50 cm). This step was standardized and included eye, mouth, and ear examination, palpation of the lymph nodes, the chest, and the abdomen. A brief examination of the locomotor system was also performed, including manipulation of each paw and toes. Finally, heart rate and body temperature were recorded. Duration of this examination varied depending on the animal’s compliance.

**Manipulations of the Puppy on the Floor (MF)**

Following the physical examination, the dog was again released on the floor. The veterinarian asked the puppy to come and sit. If the puppy did not come voluntarily, it was gently approached and taken by the veterinarian. Manipulations included gentle examination of the puppy’s ears, head, limbs, and toes. Next, the investigator restrained the dog by holding the shoulders for 5 seconds and by holding the hips for another 5-second session. Finally, the dog was put on leash and received a treat for its compliance. Manipulations were standardized, but duration of this segment also varied depending on the animal’s compliance. All procedures were videotaped either directly by the veterinarian (FF) or with the camera placed on the counter (PET) or the floor (MF).

**Results**

Most puppies (free on the floor) behaved in a similar fashion. They were very active and oriented to the environment, silent and not panting. They also interacted little with the veterinarian. However, about 10% of outliers “extreme puppies” did not explore, were panting, vocal, and seeking active interaction with the veterinarian.

For the physical examination on the table there was a wide range of values for the 3 categories: not panting, keeping their ears in a normal position, and passive interaction with the veterinarian during handling, but again many outliers were observed.
Presence of outliers was also noted for lip licking and yawning, both during the physical examination on the table and the manipulations on the floor.

Several behaviours expressed by the outliers are compatible with signs of stress or anxiety. Panting, excessive motor activity, active avoidance, increased vocalisation, decreased exploration, flattened ear position, lip licking and yawning.

**FOLLOW-UP STUDIES BY DR MARTIN GODBOUT (UNPUBLISHED)**

**Persistence Over Time of Behaviours and Signs of Anxiety Observed in Puppies**

Forty-two puppies (various breeds) were filmed during an appointment in a veterinary clinic at two to four months of age and again 12 months later. The study included observation of the puppy or adult, free on the floor (FF), as well as various manipulations on the floor (MF) by the veterinarian. During FF, the behavioural categories recorded were: activity, exploration, facial expression, puppy solicitation of interaction with the veterinarian, vocalisation and other behaviours. During MF, the type of interaction with the veterinarian, facial expression and ear position were recorded.

Most puppy behaviours observed in the veterinary clinic environment tended to persist in adulthood. Signs of anxiety showed the highest correlation between the two data-collection sessions. Lip licking, panting and ears back apparently have a similar underlying motivation in puppies and adults.

**Excessive Mouthing in Puppies as a Predictor of Aggressiveness in Adult Dogs**

Sixty-one puppies aged between 8 and 16 weeks were selected and assigned based on presence or absence of mouthing behaviour in specific contexts to a target group (38 “mouthing” puppies) and a control group (23 “non-mouthing” puppies). Twenty dogs (13 “mouthy puppies” and 7 “non-mouthy puppies”) were assessed at three years of age.

Reasons for loss to follow-up included: loss of contact with 13 owners; nine owners no longer returned phone messages (three messages left prior to giving up); nine puppies re-homed (4 puppies from the target group and 5 from the control group); seven euthanized, one for severe hip dysplasia (control group), five for aggressive behaviour (all in the target group) and one (also a “mouthy puppy”) for an unspecified reason.

**Puppy Behaviour Home Alone**

Thirty-two puppies, 16 males and 16 females, ranging in age from 50 to 118 days (mean 82.1 days) were included. The puppies were adopted by their owners between 50 and 85 days of age. Five dogs were of mixed breed and 27 were purebreds. Owners were asked to complete a brief questionnaire, including information on the puppy’s characteristics and history, as well as on the physical and social environment of the dog. Videotaping sessions were carried out under routine conditions with regard to owner absence: 15 puppies were kept in a cage, 3 were allowed to run freely in the apartment, and 14 were locked up in one room.

The dogs were filmed home alone for 60 minutes. The videotaping was repeated after 1 and 2 months, yielding a total of 3 films (film 1, 2, 3) per puppy.

Analysis of puppy behaviour for the 3 subsequent observations showed that they spent most of their time when home alone resting or sleeping (PA = 40.38 ± 18.31 minutes) as opposed to being vigilant (OE = 4.5 ± 5.06 minutes). Puppies were thus mainly inactive.

Puppies exhibited play (PL = 6.1 ± 9.53 minutes) behaviour while separated from the owners. Vocalisation was present (VO = 2.7 ± 6.25 minutes). Locomotion (LO = 0.51 ± 1.25 minutes), exploration (EX = 0.41 ± 1.10 minutes), oral behaviour (defined as any vigorous behaviour directed toward the environment or cage using the mouth including chewing, biting, shaking, pulling with the mouth, and licking; OB = 0.25 ± 1.53 minutes), and grooming (GR = 0.21 ± 1.21 minutes) were observed for shorter periods. Only 5 puppies eliminated during the 60 minutes of separation. Fifty percent of puppies did not show yawning (YA) and lip licking (LL), and 25% of puppies showed these behaviours more than twice.

Vocalisation, lip licking, and oral behaviour, all compatible with stress-related behaviours, tended to cluster together, whereas play, oriented to the environment and exploration were seen together. Three puppies out of 32 (10%) were more “stressed” than the others. These puppies were aged less than 85 days
and were adopted between 50 and 70 days of age. All three belonged to hunting breeds and spent between 2 to 4 hours alone daily. Two of them were male and 1 was female. These dogs were not followed beyond the scope of the study. However, one of these dogs belonged to an animal health technician working for one of the authors. This dog was subsequently diagnosed with separation anxiety.

The observed behaviours did not change significantly over time. No significant influence of age and age of adoption were found on behaviours shown by puppies during the 3 video recordings. The analysis of the temporal distribution of puppy behaviour did not show any statistical relevance, but passive behaviour (sleeping or resting) decreased slightly over time, whereas oriented to the environment (vigilance) and locomotion increased during the three video recordings. Play and exploration were exhibited similarly during the three observations.

Twenty-one of 32 puppies vocalised during the first film, whereas 17 of 32 vocalised during the third one. The duration of vocalisation shown by puppies tended to be higher during the first video recording. Vocalisation and oral behaviour tended to decrease, although not significantly, over time. One puppy vocalised for almost half the time (27.36 minutes) and was oriented to the environment (vigilant) for a quarter (14.06 minutes) of the entire duration of the first video recording. For this puppy, these behaviours changed slightly over time, showing similar patterns of duration, in the second and third video recordings (VO: 17.48 minutes; OE: 15.33 minutes).

A clinical case of a “different” puppy will be presented during the lecture.

References
Update on Small Animal Cardiopulmonary Resuscitation - Is Anything New?
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Objective: Update on the new small animal guidelines for CPR and a discussion of the 2012 Reassessment Campaign on Veterinary Resuscitation (RECOVER).

Cardiopulmonary arrest (CPA) is a dynamic, time-dependent, complex process occurring secondary to failed cardiac contractility, which results in either ventricular asystole, pulseless electrical activity (PEA), pulseless ventricular tachycardia (VT) or ventricular fibrillation (VF). The aims of cardiopulmonary resuscitation (CPR) are to provide maximal blood flow to the heart and brain until restoration of spontaneous circulation. Until recently, veterinary CPR recommendations were largely based on a combination of clinician preference and the ILCOR guidelines. In 2012, the Reassessment Campaign on Veterinary Resuscitation (RECOVER) was designed in order to provide comprehensive CPR guidelines for veterinary medicine.

Basic Life Support (BLS)
In 2010, ILCOR human CPR guidelines changed the order of intervention for all age groups (except newborns) from the classical Airway (A), Breathing (B) and Chest compression (C) - “ABC” - to Chest compression (C) first, followed by Airway (A) and then Breathing (B) - “CAB.” This change is in part because securing an airway in humans is fairly challenging and the prolonged intubation time causes a delay in initiation of chest compression, which negatively impacts return of spontaneous circulation (ROSC). In veterinary medicine however, there is no evidence supporting benefits of CAB resuscitation in comparison to standard ABC resuscitation. When multiple trained rescuers are available, chest compression, securing an airway and ventilation can be initiated almost simultaneously.

Chest Compression
It is essential to provide the highest possible quality chest compression in order to maximize blood flow to the myocardium and brain. CPR guidelines and the 2012 RECOVER veterinary CPR guidelines recommended a compression rate of at least 100 per minute compressing the chest by one-third to one-half of its width to maximize vital organ blood. Myocardial blood flow is determined by the coronary perfusion pressure (C_{oPP}), which is defined as the difference between aortic diastolic and right atrial diastolic pressures. Coronary perfusion pressure can be quantified by the equation: C_{oPP} = DAP - RAP, where DAP is diastolic aortic pressure and RAP is right atrial pressure (diastolic). Institution of effective cardiac compression restores the pressure gradient between the aorta and right atrium, with return of coronary perfusion and a corresponding marked increase in the likelihood of ROSC. If multiple rescuers are present, intermittent abdominal compression (IAC) and chest compression can be performed. IAC has been shown to improve venous return and hemodynamic values in animal CPR models, with little risk to the patient.

Ventilation
During CPR, an open airway should be rapidly established by orotracheal intubation. After the airway is secured, the patient should be ventilated manually with 100% inspired oxygen. No studies have yet evaluated the optimal tidal volume and inspiratory time for dogs and cats during CPR. CPR guidelines currently recommend a ventilation rate of 10 breaths per minute without interruption to chest compression. A full breath should be given in approximately 1 second and with a 10 ml/kg tidal volume. Current guidelines recommend asynchronous ventilation with continuous chest compression.
**Pharmacological Therapy**

1. **Vasopressors**

   Vasopressors increase systemic vascular resistance, increasing aortic pressure and directing more of the intravascular volume from the peripheral circulation to the central compartment. Chest compression therefore now preferentially perfused the brain and myocardium at the expense of less vital organs.

   Both epinephrine and vasopressin are a reasonable choice for all types of cardiac arrest. At the correct dosage, epinephrine acts on both alpha (vasoconstrictor) and beta receptors (inotropic and chronotropic) causing increased intracellular calcium and vascular constriction. An appropriate epinephrine dose regimen is 0.01 mg/kg IV every 3–5 minutes. While a higher dose (0.1 mg/kg) of epinephrine initially increases ROSC, this does not improve hospital discharge rate (survival) and is no longer recommended for CPR. Vasopressin is a non-catecholamine vasopressor that acts on peripheral vessels (V1 receptors), decreasing hyperpolarization and increasing intracellular calcium. In comparison to epinephrine, vasopressin has a longer half-life and seems to be a more efficacious vasopressor in a hypoxic, acidic environment. An appropriate vasopressin dose regimen is 0.8 IU/kg IV every 5 minutes.

2. **Anticholinergics**

   The 2010 ILCOR guidelines do not recommend routine use of atropine during CPR. A human CPR study revealed that use of atropine with epinephrine produced a worse outcome than epinephrine alone. In animal CPA after a high vagal tone event (e.g., vomiting, diarrhea), use of atropine is reasonable.

3. **Buffer Therapy**

   The 2010 ILCOR guidelines do not recommend routine use of sodium bicarbonate during CPR. Bicarbonate administration may cause paradoxical cerebral acidosis, hyperosmolarity and decreased catecholamine effectiveness. An appropriate dose of sodium bicarbonate is 1–2 mEq/kg IV. Bicarbonate use may be considered in prolonged CPA or for CPA due to severe hyperkalemia or severe metabolic acidosis.

**Defibrillation**

In the event of ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT), the recommendation is to start BLS chest compression and then immediately perform electrical conversion (defibrillation). CPR can be frequently interrupted by rhythm check in both the pre-shock and post-shock period; this interruption has a detrimental hemodynamic effect. It is important for the rescuer to understand that BLS compression should resume immediately after shock defibrillation, with no interruption for rhythm check until after a full CPR cycle has been completed. When choosing a defibrillator, preference should be given to biphasic waveform defibrillators. Biphasic defibrillators have been found to be as or more effective than monophasic defibrillators. In comparison to monophasic defibrillators, biphasic defibrillators require less energy for successful defibrillation and produce less tissue trauma and less cardiac damage. Electrical defibrillation should start at 2–5 J/kg with a 50% increase in energy for each subsequent attempt.

**Post-Resuscitation Care**

**Hemodynamic Optimization**

To avoid further morbidity, adequate organ perfusion should be established during the post-cardiac arrest phase. Respiratory goals are to maintain a PaO₂ of 80–100 mm Hg or a saturation of 94–98%. Hyperoxemia (PaO₂ >100 mm Hg) can cause an increase in free radical and neurological injury. It is important to ensure the patient can spontaneously ventilate with a PaCO₂ of ~32–43 mm Hg. Hypoventilation is common after CPR and long-term positive pressure ventilation should be available. The major cardiovascular goal is to maintain systolic blood pressure at 100–200 mm Hg (mean blood pressure at 80–100 mm Hg). Hypotension can be indicative of hypovolemia or decreased vascular resistance. In these cases, use of fluid therapy, inotropes and vasopressors is indicated.
Therapeutic Hypothermia

There is little information about therapeutic hypothermia (TH) for post-cardiac arrest syndrome in veterinary medicine. Advantages of TH include reductions in cerebral oxygen requirement, brain metabolic demand, excitatory neurotransmitters, inflammatory cytokines and free radicals, together with inhibition of neuronal cell apoptosis. Mild hypothermia (97°F) seems to be a safe and logical target. The ideal target temperature and the speed of rewarming the patient have not yet been established.

Adjunct CPR Devices

Intrathoracic pressure (IttP) can be manipulated to improve circulation during CPR. Recent advances have been made to maximize decreased IttP during CPR. Examples of these include impedance threshold devices (ITD) and active compression-decompression devices (ACD). It is important to remember that use of mechanical CPR devices (i.e., the ITD) has the potential to delay or interrupt CPR for the victim of cardiac arrest. Rescuers should be trained to minimize any interruption to chest compression. In other words, these devices should only be used if they are not a distractor; it is essential to start immediate chest compression while paying attention to complete and effective chest recoil with adequate ventilation.
Perioperative Analgesia for Our Feline Patients - Why, When and How to Treat Pain
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Objective: Acute pain in cats can cause severe cardiovascular, endocrine, immune and metabolic changes. Adequate analgesia can have a positive impact on patient morbidity and survival. This manuscript will review the pathophysiological effects of pain, discuss option for pain monitoring and provide some therapeutic options.

Why Treat Pain
Nociceptive stimulation represents a major factor capable of altering the ability of the organism to build, organize and regulate the homeostatic response. Acute pain exacerbates the cardiovascular response to polytrauma by immediate massive release of catecholamine. Somatic and visceral pain is conducted to the spinal dorsal root through C and delta fibers, and then transmitted to the hypothalamus. The consequent activation of the hypothalamic centers induces the secretion of pituitary-adrenal arc leading to a significant increase in blood glucocorticoid concentration. The ultimate results are increased glucose production by gluconeogenesis, inhibition of protein synthesis and increase of protein degradation.

Pathophysiology of Pain (How to Treat Pain)
Pain is a key component of any traumatic event and arises from the activation of both peripheral and central pathways. Pain is classically defined as an “unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.” Pain can also be broadly classified in nociceptive, inflammatory, and neuropathic pain on the basis of its presumed underlying mechanism.

Nociceptive pain comprises the conversion of a noxious stimulus into electrical activity (transduction) represented by action potentials between the periphery and the dorsal horn of the spinal cord (conduction) followed by synaptic transfer of input up to the cortex (transmission). The closing component of the process is the conscious, aversive sensation of the noxious stimulus (perception). Long-lasting nociceptive pain triggers activation of the N-methyl-D-aspartate (NMDA) receptor and induces a functional change known as “wind-up.” This induces a progressive increase in the output from dorsal horn neurons and represents an acute form of pain amplification.

Practical Approach to Analgesia (When to Treat Pain)
Pain control should be an integral part of the overall care plan. Analgesic treatment should be a multistage approach involving pain assessment, drug administration (systemic and regional), and repeated reevaluation. Multimodal analgesia and continuous patient reassessment constitute the cornerstones of patient care. Analgesia on feline patients can be challenging. Special attention should be taken to consider unique pharmacodynamics consideration in this group of patient. Special care must be taken to obtain accurate lean body weight for drug dosage. Cats tend to have more difficulty metabolizing certain classes of drugs. Pain assessment in cats can also be challenging.

Assessment is the first step of pain management and should begin with the patient’s triage. Analgesia may be achieved by the administration of systemic analgesic agents via various routes or by neural blockade. Nociceptive pain comprises (transduction) represented by action potentials between the periphery and the dorsal horn of the spinal cord (conduction) followed by synaptic transfer of input up to the cortex (transmission).

Nonsteroid anti-inflammatory medication (NSAID) is a great analgesic option for most moderate to mild painful procedures. It is important to ensure that the patient is not receiving another NSAID or steroid from owner; it is important to dose on lean body weight, and ensure animal has good water intake. Opioids are effective, titratable, reversible, analgesics with minor cardiovascular side effects. For all those reasons they constitute first-line analgesics for perioperative pain. Alpha2 agonists can provide adequate analgesia and are very useful in feline patients. Alpha2 can produce cardiovascular side effects.
like decrease in cardiac output and increase in systemic vascular resistance. Local anesthetics and regional block techniques are great efficacious and safe option if no opioids are available.
Chronic vs. Acute Pain: What is the Difference?
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Objective: Acute pain is not a pleasant sensation and, if not treated, can develop into neuropathic or pathological pain. In the past called chronic pain syndrome. This manuscript will review the pathophysiological effects of neuropathic pain, discuss some therapeutic options.

Musculoskeletal injuries in dogs and cats are frequently caused by trauma, degenerative disease and/or iatrogenic (surgery). In physiological terms, musculoskeletal injury is a combination of severe tissue injury, haemorrhage and inflammation. All of those activate pain pathway. Inadequately treated pain has been repeatedly shown to increase the stress response, resulting in higher morbidity. Pain is classically defined as an “unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.” Pain can also be broadly classified in nociceptive, inflammatory, and neuropathic pain on the basis of its presumed underlying mechanism (Figure 1 and Figure 2).

Nociceptive pain comprises the conversion of a noxious stimulus into electrical activity (transduction) represented by action potentials between the periphery and the dorsal horn of the spinal cord (conduction) followed by synaptic transfer of input up to the cortex (transmission). The closing component of the process is the conscious aversive sensation of the noxious stimulus (perception) (Figure 1). Nociceptive pain includes somatic and visceral pain depending on the type of tissues triggering the painful stimuli. Compared to somatic pain, visceral pain is often characterized by delayed onset and poor localization; this is due to the relatively low number of afferent receptors in visceral organs when compared to somatic tissue. Nociceptive pain is considered an adaptive physiologic defense mechanism against further tissue injury.

Long-lasting nociceptive pain triggers activation of the N-methyl-D-aspartate (NMDA) receptor and induces a functional change known as “wind-up.” This induces a progressive increase in the output from dorsal horn neurons and represents an acute form of pain amplification. Until “wind-up” arises, nociceptive pain is physiologically inhibited by the release of endogenous opioids, gamma-aminobutyric acid (GABA), serotonin and norepinephrine. For this reason, opioids are the most effective analgesics for controlling nociceptive pain, preventing formation of wind-up. However, when “wind-up” is established, a cascade of mediators causes hypersensitization, which is commonly refractory to opioids and requires the use of other classes of drugs to be controlled, such as NMDA antagonists.

Cytokines and other inflammatory mediators increase the sensitivity of nociceptors in injured tissues. This represents the basis of inflammatory pain. (Figure 1) This type of pain is also considered adaptive as it prevents movement of the injured region until repair is complete. Severe injury and subsequent extensive inflammation result in the release of large amounts of metabolically active chemicals. Those substances over-activate and over-sensitize peripheral nociceptors leading to the shift from adaptive (protective) to maladaptive (pathologic) inflammatory pain. Maladaptive pain is the expression of abnormal sensory processing and persists long after the tissue has healed.
Neuropathic pain occurs in response to both the peripheral and the central nervous system injury (Figure 2). Neuropathic pain is always considered maladaptive and may arise spontaneously in absence of any peripheral stimulus (allodynia) or may be an exaggerated and prolonged response to a noxious stimulus (hyperalgesia). Neuropathic pain is excruciating, recurrent, and poorly responsive to opioids. Examples of types of trauma mainly causing neuropathic pain include vertebral fracture or luxation and nerve entrapment following pelvic fractures.

Other important elements that influence the effect of pain are psychological factors such as stress, fear and anxiety. There are now several animal studies showing the role of stress in the enhancement of nociceptive responses. Use of anxiety-reducing drugs and techniques for individuals experiencing pain is proved to reduce the perceived severity/intensity of pain. Anxiolysis, then, could represent an effective, adjunctive component of the analgesic approach to veterinary traumatized patients. Analgesic treatment in neuropathic pain represents a multistage approach involving pain assessment, drug administration (systemic and regional), and repeated reevaluation. Analgesia of the neuropathic pain patient can be achieved by administration of local anesthetic, systemic administration of Na-channel blockers (lidocaine, mexiletine), NSAIDs and use of drugs that can block NMDA receptor (like ketamine). It may be achieved by the administration of gabapentin; however, studies demonstrate conflicting results. Acupuncture and other alternative modalities have shown success in some cases. Reason for this is still unclear. It is fair to
assume that treatment of neuropathic pain is challenging and frustrating. Prevention is still our best choice.

Prevention of neuropathic pain can be achieved by good surgical techniques and aggressive treatment of pain when it is presented in its “earlier” stages (nociceptive pain).
Anesthesia Protocol in Challenging Cases: Small Changes, Big Results - Part I
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Objective: Case scenarios and anesthetic options for patients with renal disease (renal failure, urinary obstruction), cardiac disease (hypertrophic cardiomyopathy, degenerative valvular disease), geriatric animals, C-section and patients with polytrauma.

Anesthesia for Patients with Urinary Disease

Lower Urinary Tract Obstruction
Patients with lower urinary tract obstruction are at risk of severe hyperkalemia; this condition should be considered an emergency. Clinical signs of severe hyperkalemia include lethargy, muscle tremors and bradyarrhythmias. The main initial goal of therapy is to lower the serum potassium concentration and restore micturition. After relief of obstruction, hyperkalemia usually resolves within 24 hours, while azotemia and hyperphosphatemia require longer periods of time to resolve.

Anesthetic Regimen

Premedication
When the patient is lethargic, urinary catheterization can usually be performed with only sedation. The main concern is bradyarrhythmias due to hyperkalemia. With this in mind, drugs best avoided in urethral obstruction include xylazine, dexmedetomidine and romifidine. An intravenous combination of buprenorphine and midazolam should produce mild sedation. In cases where the patient is not cooperative or after unsuccessful attempts at urinary catheterization, general anesthesia should be elected to minimize stress.

Induction
One of the main concerns during induction is avoidance of bradycardia, stress and myocardial depression. With this in mind, ketamine with diazepam or tiletamine with zolazepam may be good, short-acting induction agents. Induction agents should be given intravenously until the animal is deep enough to permit endotracheal intubation. In cats, norketamine, an active metabolite of ketamine, is excreted unchanged by the kidneys, but prolonged recovery is unlikely unless urinary outflow cannot be reestablished. If this is the case, repeated doses of ketamine should be avoided.

Maintenance
Inhalant anesthetics are a good option for anesthesia maintenance. Hypoventilation can lead to respiratory acidosis. As with any acidosis, this will cause efflux of intracellular potassium in exchange for entry of extracellular hydrogen into the cell, which exacerbates hyperkalemia. Animals with mild hyperkalemia (6-7 mEq/L) can develop arrhythmias during general anesthesia even if they have not earlier demonstrated electrocardiographic abnormalities. Monitoring should include a capnograph, ECG, noninvasive blood pressure (Doppler), pulse oximeter and blood gas analysis. A good intraoperative maintenance fluid is 0.9% sodium chloride.

Therapy for hyperkalemia is recommended, especially if the patient is showing clinical signs of electrocardiographic abnormalities. Palliative therapy for hyperkalemia includes administration of potassium-free fluid (0.9% saline), calcium gluconate, dextrose and sodium bicarbonate. Coadministration of insulin and sympathomimetic drugs can also help in decreasing plasma levels of potassium. Administration of 0.9% saline will help to correct hypotension and hypovolemia as well as dilute the hyperkalemia. Calcium gluconate administration does not decrease serum potassium; however, this increases the extracellular fluid concentration of calcium and increases the threshold potential, thus restoring normal membrane excitability. Administration of sodium bicarbonate can alter the flux of potassium across the cell membrane. Glucose administration increases endogenous insulin release and
increases intracellular transport of potassium. Coadministration of insulin can prevent hyperglycemia and facilitate the movement of potassium. Other therapy modalities include administration of β-adrenergic agonists such as albuterol, terbutaline and epinephrine, which may be used to activate ATP-dependent potassium channels and therefore increase cellular influx of potassium.

Renal Insufficiency
Chronic renal insufficiency or chronic renal failure (CRF) may be caused by primary renal disease or systemic disease, which also affects the kidney. To assess those concomitant problems, a full serum chemistry profile (chem) and complete cell count (CBC) are required for all patients with renal insufficiency that will undergo general anesthesia.

It is important that the patients be normovolemic before anesthesia. However, the kidneys have difficulty handling both large fluid loads and dehydration. Thus, fluid therapy should be initiated long before anesthetic event. Ensuring fluid balance is corrected in a slow progressive fashion.

Anesthetic Regimen

Premedication
The objective of anesthesia in renal patients is to prevent hypovolemia, avoid decrease in renal blood flow and avoid drugs that can cause further kidney damage. With this in mind, nonsteroidal anti-inflammatory drugs should best be avoided. Dexmedetomidine has been shown to decrease peripheral blood circulation (skin perfusion) but not central circulation (renal perfusion). However, this drug should be avoided if patient is dehydrated or hypovolemic. A safe intravenous combination of opioid and midazolam should produce good sedation and analgesia.

Patients with renal insufficiency should be able to metabolize most sedative and analgesics without a problem. Morphine active metabolite M6G is eliminated through the kidney and will accumulate in patients with severe renal disease.

Induction
One of the main concerns during induction is avoidance of hypotension and bradycardia. With this in mind, ketamine with diazepam or tiletamine with zolazepam may be good, short-acting induction agents. Ketamine in cats have renal elimination, where in dogs liver metabolizes large portion of ketamine. Dogs with CRF have little risk of ketamine accumulation.

Maintenance
Inhalant anesthetics produce vasodilation and hypotension. Other drugs like opioid and lidocaine can be administered during the procedure to decrease concentration of inhalant anesthetic. Some drugs can be administered to increase renal blood flow and maintain glomerular filtration rate. This includes low-dose dopamine infusion, fenoldopam infusion and mannitol. The use of “kidney protective” drugs is probably not as important as good fluid therapy during anesthesia to maintain adequate renal perfusion.

ANESTHESIA FOR THE PATIENT WITH CARDIAC DISEASE

Hypertrophic Cardiomyopathy (HCM)
One of the most common cardiac diseases in cats, hypertrophic cardiomyopathy (HCM), is characterized by stiffness of the left ventricle with poor diastolic function. A recent epidemiological study reported that 13% of healthy cats have HCM, although they do not have clinical signs and are normal on physical examination. It is speculated that a good percentage of anesthesia deaths in routine cases may be secondary to undiagnosed cardiac disease.

There is now widespread acceptance that the subaortic gradient and associated increase in left ventricular (LV) pressure cause an outflow obstruction. Outflow obstruction can occur acutely and unexpectedly, and its presence has been correlated with increased risk for congestive heart failure, stroke death and sudden death. Outflow obstruction is determined by a number of structural abnormalities, including small LV outflow tract area, septal bulge and hyperdynamic LV ejection, which pulls the mitral valve towards the septum. Patients with outflow obstruction have a massive drop in cardiac output and
blood pressure. Outflow obstruction can be provoked by conditions that increase myocardial contractility, such as stress and tachycardia. Hypovolemia, hypotension and dehydration may also trigger dynamic outflow obstruction.

Patient with LV outflow tract obstruction benefit from protocols that reduce heart rate and contractility, decrease sympathetic stimulation, and increase filling pressure and afterload. Drugs that mildly depress myocardial contractility and reduce oxygen demand while maintaining systemic vascular resistance are excellent in avoiding LV outflow obstruction.

Anesthetic Regimen

Premedication
The main concern during premedication is stress and a sudden burst of catecholamines. Administration of anticholinergics such as atropine and glycopyrrolate should be avoided because of the potential for tachycardia and increased myocardial work and oxygen demand.

For patients with no clinical signs of cardiomyopathy a combination of opioids and midazolam is a safe choice, although this provides only mild sedation in cats. For animals with a diagnosis of dynamic LV outflow obstruction, an intravenous combination of opioids and low-dose dexmedetomidine should lead to good sedation. Dexmedetomidine causes a decrease in catecholamine secretion and results in myocardial depression, bradycardia and increased systemic vascular resistance. These are all desired effects in HCM with outflow obstruction.

Induction
One of the main concerns during induction is avoidance of severe vasodilation, tachycardia, arrhythmias, stress and myocardial ischemia. With this in mind, ketamine and diazepam or tiletamine with zolazepam are not good choices of induction agent. Etomidate causes almost no change in the hemodynamic state of the patient, with little change in systemic vascular resistance.

Recovery and postanesthetic management
Vigilance during the recovery period with respect to any increase in sympathetic response remains a priority. Stress, pain, hypothermia and hypovolemia can all lead to catecholamine release, resulting in increased myocardial oxygen demand and promoting dynamic outflow obstruction and malignant arrhythmias. Analgesics that can be titrated and are easily reversible should be provided; depending on the procedure, IV infusion of fentanyl for 24 hours may be an adequate choice.

Anesthesia for Dogs with Degenerative Mitral Valve Disease
One of the most common cardiac diseases in dogs is degenerative valvular disease. About 1/3 of geriatric small-breed dogs have some degree of mitral regurgitation (MR), and about ½ of patients presented for congestive heart failure have MR as underlying disease. Anesthetizing patients in the early stages of MR is fairly easy and patient can compensate to significant changes in contractility and vascular resistance. In the late stages of MR the patient is prone to congestive heart failure. The objective of anesthesia for patients with MR is promoting forward blood flow and reduce regurgitation fraction. Thus, drugs that promote bradycardia, vasoconstriction, pulmonary hypertension and reduction of cardiac output are all contraindicated.

Anesthetic Regimen

Premedication
Administration of anticholinergics such as atropine and glycopyrrolate can be a good option since they will reduce the chance of intraoperative bradyarrhythmias. For patients with no clinical signs of cardiomyopathy, a combination of opioids with phenothiazinic (acepromazine) is a safe choice. For patients with more advanced stage of MR, a combination of opioids and midazolam is a safe choice, although this provides only mild sedation. Opioids that do not reduce heart rate like meperidine are a good option for premedication.
**Induction**
Bradyarrhythmias should be avoided and a mild elevation of heart rate will reduce regurgitation fraction. With this in mind, ketamine and diazepam or tiletamine with zolazepam are good choices of induction agent. Etomidate causes almost no change in the hemodynamic state of the patient, with little change in systemic vascular resistance.

**Recovery and postanesthetic management**
Heart rhythm and blood pressure (if possible) should continue to be monitored for about three hours after general anesthesia because this is the most likely period in which anesthesia-related fatalities will occur; significant bradyarrhythmias should be treated. Analgesics that can be titrated and are easily reversible should be provided; depending on the procedure, IV infusion of fentanyl for 24 hours may be an adequate choice. The patient should be recovered in a quiet area and with oxygen insufflations.
Anesthesia Protocol in Challenging Cases: Small Changes, Big Results - Part II
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ANESTHESIA FOR C-SECTION
Anesthesia for patient with C-section is challenging. As pregnancy develops, severe physiological changes happen that can affect your anesthetic protocol. Patients’ functional residual capacity (FRC) decreases; there is a drop in vascular resistance, decrease in pack red blood cells (PCV), increase in gastric pressure and increase in abdominal pressure. Abdominal compartment syndrome leads to venous congestion and venous engorgement of the sinus vein (especially in the epidural region). This can lead to higher risk of epidural forward spread and accidental venous injection. Patients can present signs of hypocalcemia, hypokalemia or hypoglycemia and alteration in the nervous system due to electrolyte dysfunction.

Anesthetic Regimen
Premedication
Preoperative evaluation of the pregnant patient is typically more complex. All drugs that can cause sedation will lead to some fetus cardiovascular depression. Complete blood work, including hematocrit, plasma protein, and hepatic functional panel should be considered the minimum for preanesthetic laboratory testing.

The main concern during premedication is to avoid oversedation. Preference should be given to drugs that are very short lasting and easily reversible (opioids or benzodiazepine). Opioids are a good choice for premedication (especially the short-lasting ones like fentanyl). They provide adequate sedation in most animals, with minimal cardiovascular and respiratory signs. The use of opioids will reduce the amount of induction and inhalant anesthetic required.

Induction
Propofol is rapidly metabolized and has been correlated with good puppy survival rate. It is preferable to reduce the dose of induction agent (10–30%) and administer it slowly to compensate for pharmacodynamic changes. Supplemental oxygenation should be available in all cases.

Maintenance
Inhalant agents offer a good option for maintenance of anesthesia. Whenever possible, local anesthetic techniques like epidural should be used to provide analgesia and reduce the necessary amount of anesthetic. There is extra risk of epidural administration in animals that are pregnant. Intensive monitoring during anesthesia is essential to ensure rapid diagnosis and timely treatment of cardiorespiratory complications. The veterinarian should be able to monitor patient oxygen saturation, blood pressure, heart rate and end-tidal carbon dioxide. Monitor serum glucose during surgery and supplement dextrose if needed.

ANESTHESIA FOR GERIATRIC PATIENTS
Anesthetic Regimen
Premedication
Preoperative evaluation of the geriatric patient is typically more complex than that of the younger patient. Particular attention should be given to auscultation of the heart and lungs. Complete blood work, including hematocrit, plasma protein, and renal and hepatic functional panel should be considered the minimum for preanesthetic laboratory testing in this age group.

The main concern during premedication is to decrease stress and provide good analgesia. Age by itself minimally changes anesthetic requirements. The increase in anesthesia-related morbidity is usually related to concurrent age-related diseases. Degenerative disease, decreased organ reserve capacity and neoplasia can significantly influence plasma protein concentration and renal function, influencing the free
fraction, potency and duration of anesthetic drugs. As a general rule, geriatric patients require less anesthetic agent for equivalent effect. Depending on the comorbidity present, some drugs, including xylazine, dexmedetomidine and acepromazine, are best avoided in geriatric patients. Opioids are a good choice for premedication. They provide adequate sedation in most animals, with minimal cardiovascular and respiratory signs. The use of opioids will reduce the amount of induction and inhalant anesthetic required.

**Induction**
One of the main concerns during induction is to avoid stress and myocardial depression. The response of the older patient to surgical stress is often unpredictable. Extra care must be taken to ensure good analgesia during the perioperative period. Most short-acting, intravenous induction agents are suitable for geriatric patients. These include propofol, thiopental, dissociative anesthetics (ketamine and Telazol) and etomidate. It is preferable to reduce the dose of induction agent (10–30%) and administer it slowly to compensate for pharmacodynamic changes due to age-related disease. Supplemental oxygenation should be available in all cases that require heavy sedation or general anesthesia.

**Maintenance**
Inhalant agents offer a good option for maintenance of anesthesia. It is important to remember that the minimum alveolar concentration (MAC) for inhaled anesthetics is 30% less in geriatrics compared to young dogs. Whenever possible, local anesthetic techniques should be used to provide analgesia and reduce the amount of anesthetic necessary. Intensive monitoring during anesthesia is essential to ensure rapid diagnosis and timely treatment of cardiorespiratory complications. The veterinarian should be able to monitor patient oxygen saturation, blood pressure, heart rate and end-tidal carbon dioxide.

**Recovery and Postanesthetic Management**
The patient should be recovered in a quiet area. It is advisable to monitor the patient for about three hours after general anesthesia because this is the most likely period in which anesthesia-related fatalities will occur. Appropriate analgesics should be administered. Geriatric patients may have comorbidities that make them prone to chronic (pathologic) pain syndrome. Opioids can lead to excessive sedation, which is not always advantageous during the postoperative period. Nonsteroidal anti-inflammatory drugs (NSAIDs) can be good alternative analgesics. In geriatric populations, NSAID use is not contraindicated as long as there are no signs of renal impairment, hypotension or dehydration.

In conclusion, anesthetic protocols for geriatric patients do not differ significantly from those for younger patients. The difference will be in how and when the drugs are administered, and the detailed preoperative evaluation, intensive intraoperative monitoring and extra effort required ensuring adequate postoperative analgesia.

**Anesthesia and Analgesia in the Polytrauma Patient**
The cardiovascular system should be monitored continuously once the patient is admitted to the hospital. Patients should have a large-bore catheter placed for vascular access. After initial assessment, further diagnostic tools should be used to rule out chest contusion, pneumothorax, traumatic myocarditis, intracavity hemorrhage, hypovolemia and neurologic lesions.

Polytrauma causes severe cardiovascular, endocrine, immune and metabolic changes. In physiological terms, trauma is a combination of severe tissue injury, haemorrhage, and inflammation. All of those activate pain pathway. Pain is a primary component of trauma and plays a major role in the alteration of homeostasis. Adequate analgesia can have a positive impact on patient morbidity and survival. As pain is a major component of polytrauma and has many deleterious systemic effects, it is incumbent upon the clinician to consider aggressive pain management a core tenet of care in trauma. Pain control of the trauma patient should be an integral part of the overall care plan, and can be divided into two main stages: the pre-hospital phase (patient restraint and temporary coaptation) and the hospital phase (assessment and analgesic treatment in the ER). Trauma-associated pain is a complex and multifactorial symptom that requires a thoughtful approach using a variety of treatment modalities to obtain an optimal outcome. The goal is control of both nociception and neuroplasticity.
**Analgesic Regimen**

Analgesia of the trauma patient may be achieved by either administration of systemic, analgesic agents via various routes or neural blockade. The intravenous (IV) route is always advisable for systemic analgesia. Injection directly into the circulatory system allows immediate distribution of the analgesic to the nervous tissue. Drugs can be administered IV either as a bolus or preferably by continuous infusion, which can be titrated to effect to reduce the risk of rebound pain. Intramuscular and subcutaneous administration would lead to slower and unreliable systemic absorption, especially in patients that are hypovolemic and peripherally vasoconstricted. Epidural analgesia is achieved by the introduction of analgesics into the epidural space. Injuries to the abdomen, pelvis, hind limbs and soft tissues of the posterior portion of the body may benefit from epidural analgesia. The most frequently used medications for epidural analgesia are local anesthetics, opioids or combinations of these drugs. Alpha-2 agonists have also been used, either alone or in combination with local anesthetics or opioids. When combined with morphine they induce a longer-lasting analgesia in comparison to morphine alone (13 hours vs. 6 hours). However, practitioners should expect that systemic absorption of alpha-2 agonists from the epidural space may result in some undesirable effects. The most significant advantages of the epidural route in comparison to IV administration are the reduced dose of drug required, longer duration of analgesia and superior analgesic effect. Epidural analgesia is contraindicated in patients with coagulation disorders, spinal injuries or skin infections.

Trauma-associated pain is a complex and multifactorial symptom that requires a thoughtful approach using a variety of treatment modalities to obtain an optimal outcome. Due to the significant homeostatic changes in traumatized patients, there is no set dosage for any given analgesic drug. It is recommended to start at a fraction of the published dose and then titrate based on the patient response. Multimodal (or balanced) analgesia represents an approach to manage pain using a combination of several drugs, with different mechanism of action, minimizing each single agent dose. A possible initial approach to multimodal analgesia in traumatized patients may be to include an anxiolytic, an opioid and a locoregional block. However, use of several drugs in combination introduces a significant variable, which is the effect of one drug on the concentrations of the others at their sites of effect. Most of the time, this is unpredictable. It is therefore important to stress that the minimum number of drugs necessary to achieve optimal control of pain should be chosen. Opioids are effective, titratable, reversible analgesics with minor cardiovascular side effects. For all those reasons, they constitute first-line analgesics for trauma patients. The choice whether or not to include a sedative depends on the patient’s temperament. It is not uncommon for trauma patient to be aggressive on presentation. Often, this is secondary to pain, anxiety and fear. When dealing with aggressive or potentially aggressive patients, sedation may be required in order to ensure the safety of personnel and allow appropriate treatment. Before sedative is administered, it is important to state that most sedatives have profound cardiovascular effects that are deleterious in hypovolemic animals. Thus, it is important to reestablish normovolemia before giving sedatives like alpha2 agonists and acepromazine.

Improved pain management in the trauma patient not only increases comfort and reduces unnecessary suffering, but also reduces morbidity and improves short- and long-term outcomes.

**Anesthetic Management**

Before anesthesia, it is important to adequately stabilize the animal. Patients with hemorrhagic shock may benefit from restrictive fluid-resuscitation and permissive hypotension. Maintaining those animals at mean blood pressure ~ 60 mm Hg for a short period of time would lead to less morbidity than attempting aggressive fluid resuscitation and aiming for supraphysiological blood pressure values. Initial therapy for septic shock consists of adequate fluid resuscitation, inotropic drugs and oxygen supplementation should be administered to improve oxygen delivery in a timely fashion. A quick checklist of the anesthetic area will ensure that all equipment is working properly and that no steps have been overseen due to haste. Allowing anesthesia induction runs smoothly.

**Premedication/Induction**

Animal should have at least 2 large-bore catheters preplaced. Before induction, animal should be preoxygenated for at least 5 minutes and noninvasive monitoring equipment connected. Anesthesia
should be achieved with drugs that have minimum cardiovascular effect, can be reversible and are short acting. Most of the time, in polytrauma patients, anesthesia induction can be achieved with the use of opioid and benzodiazepine followed by a low dose of ketamine.

**Maintenance**
Anesthesia maintenance can be achieved with inhalant anesthetic plus infusion of ketamine, lidocaine and/or opioid to reduce the total amount of inhalant anesthetic used. When appropriate, local anesthetic blocks are an excellent option to reduce inhalant anesthetic use even further.

**Recovery**
Animal should be monitored for at least 24 hours after surgery. Adequate analgesia is paramount to ensure smooth recovery and decrease morbidity. See analgesic protocol.
**Advanced Monitoring for the Critical Care**
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**Objective:** Discuss advantages/disadvantages and alternatives for advanced hemodynamic monitoring and talk about microcirculatory monitors

**ECG**

Electrocardiographic (ECG) monitoring during general anesthesia has become a care standard over the past two decades. ECG monitoring can provide important information regarding cardiac function during the perianesthetic period. As such, the ability to understand and interpret information provided by the ECG signal is important for all anesthesia-related personnel. Surface mount or esophageal leads are designed to detect low-voltage signals generated by the heart during a normal cardiac cycle. These signals, which are millivolt in strength, are conducted from the patient to the monitor base via the “lead system” wires. Once the generated signal enters the monitor, it is conditioned, amplified, and displayed on a CRT or LCD panel. The appearance of the signal can be altered by changing the sensitivity or “gain” on the display. It may also be altered by changing the way in which the lead montage is viewed on the screen. We know this montage as leads I, II, III, and the augmented leads based on which lead sites are active at a particular time. This works well for limb lead placement; esophageal leads behave in a different manner.

It is important to realize that ECG monitoring during anesthesia **cannot** be used as a diagnostic ECG. In most cases, patient position and lead location are incompatible with diagnostic ECG benchmarks. Measurement of cardiac chamber size and primary electrical vector are inaccurate because a standard lead system placement and patient position may not be present. Also ECG does not monitor cardiac contraction and a patient in cardiac arrest can still have an ECG rhythm. As such, information obtained during anesthesia monitoring is limited to electrical rate and rhythm.

**Arterial Blood Pressure**

Blood pressure is the pressure exerted by the circulating blood against the vessels walls. Blood pressure can give an estimation of cardiac contraction (cardiac output) and tissue perfusion. It is important to remember that blood pressure is influenced not only by cardiac output but also by systemic vascular resistance. A patient can have an adequate cardiac output with a low systemic vascular resistance yielding a low blood pressure. Or on the other hand, a normotensive patient does not necessarily have a normal cardiac output.

During each heartbeat, pressure varies between maximum (systolic) and minimum (diastolic). Systolic blood pressure (SAP) can be an indication of the stroke volume and arterial compliance. Diastolic blood pressure (DAP) is more dependent on how the systemic vascular resistance is performing. The mean arterial pressure (MAP) is an average of both values (MAP = (SAP + 2xDAP)/3) and can be an estimation of average driving tissue perfusion pressure.

The noninvasive blood pressure method is user-friendly and causes less patient morbidity than invasive blood pressure. However, noninvasive blood pressure method may yield lower accuracy, especially on small animals (less 10 lb). Noninvasive blood pressure can also give erroneous values if the cuff is not of correct size, if there is patient motion or if machine setting is not appropriate.

Hypotension is a common occurrence during general anesthesia. It can be an indication of pharmacologically induced vasodilation (drop in systemic vascular resistance). It can also be an indication of decreased contractility, decreased stroke volume, sepsis, hemorrhage or hypovolemic shock. If hypotension is persistent, it can lead to decrease in tissue perfusion, precipitate anaerobic metabolism, metabolic acidosis and ischemic organ damage (kidney, liver and brain injury). Therapy includes decreasing the pharmacological agent that is causing the vasodilation (usually inhalant anesthetic), restore circulating blood volume (fluid bolus or transfusion) or administering positive inotropes (dopamine, dobutamine, vasopressin or norepinephrine infusion).
Hypertension can be an indicator of systemic problem and, if not treated, may have long-term adverse effect. Persistent hypertension can be pharmacology induced, due to pain, hypermetabolism or disease. Treatment consists in finding and removing the underlying cause. Drugs like nitroglycerine and nitroprusside can decrease blood pressure by decreasing systemic vascular resistance.

**Pulse Oximeter**

Pulse oximetry is a simple and noninvasive method of monitoring arterial hemoglobin oxygen saturation. It also provides information about the patient’s heart rate. Oximetry is based on the Lambert-Beer law, which relates the absorption of light by a solution (here blood and specifically hemoglobin) to the concentration of a desired (in this case, oxygen) substance. Oxygenated hemoglobin absorbs light in the infrared spectrum (920–960 nm), whereas deoxygenated hemoglobin absorbs light in the red spectrum (640–660 nm). Analysis of the differential absorption spectra of oxygenated and deoxygenated hemoglobin by the microprocessor in the monitor provides a value for oxygen saturation.

Pulse oximeters are especially useful in patients where oxygenation is compromised by the patient’s condition, the nature of the procedure or because of anticipated physiological changes. Because they provide continuous information regarding oxygenation, changes in pulse oximeters are frequently used to guide the need for blood gas analysis.

In general, pulse oximeters tend to be most accurate (to about 2%) within the normal range (SpO₂ 90–100%). Their accuracy may be further influenced by pigment and tissue thickness, patient movement or shivering, ambient lighting, intravenous dyes and poor perfusion. Abnormal hemoglobins, while not common in veterinary medicine, will also result in an erroneous reading. Carboxyhemoglobin will result in a falsely high reading as its absorption spectrum in the 660 nm wavelength is similar to that of oxyhemoglobin. The absorption coefficient for methemoglobin is the same for red and infrared light wavelengths and corresponds to a saturation of 85%. Newer pulse oximeter (Massimo Ltd) can determine concentration of carboxy- and methemoglobin. The accuracy of pulse oximeters has not been verified for non-mammalian species.

It is important to remember that saturation is just part of the oxygen delivery (DO₂) equation and a normal saturation does not mean normal blood perfusion nor normal oxygen delivery.

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\text{Oxygen delivery (DO₂) = oxygen content x cardiac output}
\]

\[
\text{Cardiac output (CO) = heart rate x stroke volume}
\]

\[
\text{Oxygen content (mL/dL) = (1.36 x [Hb] x \% saturation) + (PaO₂ x 0.003)}
\]

\[
\text{Saturation: pulse oximeter (％)}
\]

\[
\text{Percent of ‘available hemoglobin’ saturated with oxygen}
\]

\[
\text{HbO₂/Hb + HbO₂}
\]

Hemoglobin (Hb): measured or approximated (1/3rd PCV)
Partial pressure of oxygen (PaO₂): blood gas analyzer (mm Hg)
95% saturation or better is desirable
90% saturation or less indicates a problem with oxygenation
100% saturation is the maximum reading

**Capnography**

The measurement of the respiratory carbon dioxide tension is called capnometry. The graphic representation of carbon dioxide concentration during the respiratory cycle is called capnography. The end tidal carbon dioxide (EtCO₂) is the maximum CO₂ concentration that is measured at the end of expiration. EtCO₂ can be expressed in percentage or millimeters of mercury. The measurement and graphical analysis of EtCO₂ can be used to: estimate arterial carbon dioxide tension (PaCO₂); confirm correct endotracheal intubation; assess patient ventilatory and circulatory status; alert anesthetist to the occurrence of apnea; and ensure anesthetic circuit integrity. EtCO₂ can be measured continuously and in real time every time that an endotracheal tube is in place. And help in cardiovascular monitoring during CPR.
Capnography is a great tool to evaluate cardiorespiratory and anesthetic machine functions. In this regard, it is very important to understand the anatomy of a normal capnographic wave and its components. Also, the user should be familiar with the most common problems that will affect the capnographic wave and also the EtCO₂ measurement. Some of its most common abnormalities and their causes should also be recognized by the clinician.

The waveform above is the only normal shape for the capnograph. The wave can be divided in 4 phases. Phase I is the mid-part of the inspiration. It is represented by the E to A distance. In this phase, carbon dioxide should be zero, which means that the patient is breathing in fresh gas. Phase II is the start of expiration (BC). Its fast rising line represents the mixing between the dead space gas, which is full of fresh gas from the last inspiration, with the alveolar carbon dioxide. The phase III is the rest of expiration representing exhaled alveolar gas (CD). This phase is also called the plateau portion of expiration. The angle α should be between 90 and 110°. D is the highest point of the wave and represents the EtCO₂. The EtCO₂ is the closest estimation of arterial carbon dioxide concentration (PaCO₂). Phase IV is the start of inspiration (DE). The decline on CO₂ concentration should go down to zero and the angle β should be close to 90°. Below there are some examples of abnormal capnograph waves and their most common causes. (VIN editor: no figure was provided at time of publication)

**Hypercapnia**
Hypercapnia is the same as hypoventilation. The ventilation status of a patient can only be accessed by knowing their PaCO₂. EtCO₂ can predict PaCO₂ and in this case above we note that EtCO₂ is greater than normal (normal = 30 to 43 mm Hg). Also, take a minute to analyze the waveform itself. Be systematic. First, the shape of the wave is normal (normal angles and anatomy of the phases). The inspiratory phase is going all the way to zero, as it should. The problem then is that the EtCO₂ is greater than the normal range. Common causes for that: Deep plane of anesthesia; opioid respiratory depression, obesity, position of patient impeding breathing.

**Hypocapnia**
Hypocapnia: EtCO₂ is lower than normal (normal = 30 to 43 mm Hg). Common causes for that is hyperventilation, decrease in cardiac output, decrease in blood pressure, pulmonary thromboembolism or decrease in pulmonary perfusion (cardiac arrest).

**Rebreathing**
This is different from the hypoventilation. The wave shape is still normal; however, the inspiratory phase I does not reach zero. This means that the patient is inspiring some amount of carbon dioxide when breathing in. This will cause not only hypoventilation, but also an elevation of the baseline (greater than zero). Common causes: exhausted absorber; faulty one-way valves in a circle system; low flow than required in a non-rebreathing system.

**Airway Obstruction**
Waves are abnormal due to an airway obstruction. The α-angle is increased and phase III is a slope. The obstruction will make expiratory times more labored. Also, the rise of carbon dioxide to true alveolar sample will take much longer.

It is important to realize that EtCO₂ cannot replace PaCO₂ measurement, although in healthy dogs and cats the PaCO₂-EtCO₂ difference is generally between 2-5 mm Hg. Cardiorespiratory changes as well as certain conditions may increase this difference. Capnography is a great tool for the clinician during anesthesia; however, judicious interpretation must be made and, therefore, measurement of PaCO₂ may be necessary to make a more accurate assessment of ventilation status.

**Preload Monitor**
(Central venous pressure, pulse pressure variation and lactate).
Overaggressive fluid therapy has been shown to increase abdominal compartment syndrome and cause severe morbidity. There are several preload monitors (or monitor of fluid-therapy status) used to predict when the patient received enough fluids. The most common one used clinically is still the central venous pressure (CVP). Recently, CVP accuracy and sensitivity have been put into question. There has
been poor correlation between CVP and fluid responsiveness in large human studies. Other preload monitors include pulse pressure variation and echocardiogram. Pulse pressure variation (PPV) is the difference between maximum and minimum systolic arterial pressure minus diastolic arterial pressure in one ventilator cycle. PPV number is an indicator of the cardiovascular system compliance and therefore preload. Pulse pressure variation requires the patients to be anesthetized, intubated and under mechanical ventilation in order to give an accurate value. A new class of pulse oximeter (Massimo Ltd) can use the plethysmograph wave analysis for to predict the pulse pressure variation and is a good alternative when direct arterial line is not possible. Echocardiogram is a very useful tool to predict fluid responsiveness and evaluate contractility. However it needs special equipment and can’t be used in all anesthetized patients.

Lactate value has little predictability for fluid responsiveness and preload. However, one can use the temporal drop of lactate after treatment (lactate gap) to evaluate if more aggressive fluid therapy is needed. Lactate gap might have a prognostic value in critical patients as well.

There is not one perfect hemodynamic monitor available for critical care and critical anesthesia. Each one has advantages and disadvantages. Each one complements the other. The best monitor is still an attentive and dedicated anesthetist.
COMPANION ANIMAL – FELINE MEDICINE

Cat-Friendly Preventive Health Care Protocols and Practice Tools: It’s All About the Cats
Margie Scherk¹, DVM, DABVP (Feline Practice); Susan Little², DVM, DABVP (Feline Practice); Elizabeth O’Brien³, DVM, DABVP (Feline Practice)
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SEE ATTACHED DOCUMENT TITLED: CAT HEALTHY PREVENTATIVE HEALTHCARE PROTOCOLS
Not Just Old, But Sore - Recognition, Prevention, and Treatment of Feline Arthritis
Elizabeth O’Brien, DVM, DABVP (Feline)
The Cat Clinic, Hamilton, ON, Canada

There is a public perception that dogs get arthritis and cats just get old. Cat owners/guardians know that cats get many diseases, but often arthritis (DJD) is overlooked. It has been assumed that osteoarthritis (in the cat) is very rare and/or the cat does not suffer with clinical disease. We need to change this public perception by educating our veterinary teams and our clients about arthritis in cats.

In this lecture, case examples will be used to illustrate the recognition, treatment, and prevention of arthritis.

Twenty-two percent of cats in the general population older than one year of age and ninety percent of cats older than twelve years of age demonstrate radiographic evidence of arthritis. Sixty-one percent of cats aged six years of age or older have osteoarthritis in at least one joint, yet only nine percent of cats were diagnosed with osteoarthritis. Major cartilage pathology can be present in the absence of any bony change, meaning that radiographic identification of DJD in the cat can also be problematic. The coxofemoral joint is often involved and usually bilaterally. Cats do get hip dysplasia and it is underestimated in cats as they often have no clinical signs. Appendicular and axial can both be affected. Appendicular more frequently, but axial involvement increases with age. Hips, stifle, tarsus, elbow, carpus, and shoulder may be affected in decreasing frequency.

RECOGNITION
Recognition of arthritis in the cat is a challenge to both the clinician and the owner/guardian. The challenge is: most cats will not exhibit lameness, it is not always evident on radiography, and a meaningful physical orthopaedic examination is a challenge in most cats. Recognition of the disease in the cat is based on recognition of behavioural changes in the cat. The main features of feline DJD are changes in behaviour and lifestyle which develop gradually and which owners tend to interpret as simply being the effects of old age. A variety of changes should alert the owner and clinician including, but not limited to: reduced activity, difficulty or a hesitation when jumping or using the stairs, a decreased appetite, weight loss, irritability, aggression, inappropriate urination and defecation, decreased grooming, alopecia over affected joints, lameness, or a stiff gait.

A questionnaire and history taking using a checklist about the cats behavior is invaluable and should be included on all visits. Topics that need to be included are ability to jump, agility when jumping, change in activity (decreased hunting, increased sleeping, decreased playing both in frequency and length), change in sleeping location (less locations - or the same spot all the time), inappropriate defecation/urination, change in socialization (less social or more needy), decreased activity, lower scratch marks on scratching posts, poor hair condition (result of lack of grooming) are amongst a number of behavioural changes that need to be recognized.

The use of a 6-day trial of analgesia will often show a dramatic improvement in the cat’s abilities and behavior and can be used as a tool for the diagnosis of arthritis.

TREATMENT
Treatment requires analgesia, a slowing of the disease process, and in the majority of the cases, weight loss. A list of a few items below can be used to control or prevent the pain of arthritis in the cat and slow the progression of the disease.

Chondromodulating Agents
Chondroitin sulfate and glucosamine may slow progression, or alter the disease process, involved with osteoarthritis by: stimulating cartilage matrix synthesis, inhibiting catabolic enzymes, and increasing fluidity of synovial fluid.
Omega-3 Fatty Acids
Omega-3 fatty acids have an inhibitory effect on arachidonic acid which is a key player in the inflammatory pathway in arthritic joints. Antiinflammatory = pain reduction!

Green-Lipped Mussel
Native to New Zealand, these shellfish have been consumed by the Maori people for thousands of years. The health benefits of the green-lipped mussel came to the attention of westerners in the 1960s because of the notable absence of inflammatory illness among the Maori people. Further studies have shown that this unique shellfish is a rich natural source of omega-3 fatty acids and naturally contains glucosamine and chondroitin sulphate.

Diet
My first pick off the shelf choice is to select a joint diet where possible. Both Hill’s and Royal Canin have exceptional joint diets. In my opinion. It is key to not only recommend the diet, but to recommend a dose of the diet (amount to feed) based on lean body weight.

    If the cat will not eat the diet, which is unlikely as both of the above diets are highly palatable, then a high quality veterinary diet is advised and supplementation with products that contain glucosamine, chondroitins, omegas, and green-lipped muscle is my next step.

    If the cat is obese, the choice may be to put the cat on a calorie-restricted, weight-loss diet with joint supplementation.

Injectable Chondroprotectants GAGs
Provide the raw materials to build more cartilage and produce more synovial fluid - cartilage is made of glycosaminoglycans.

    In addition, they contain antiinflammatory properties that slow down the actual damage to the cartilage and inhibit degradative enzymes and free radicals and other entities and promote enzyme systems that facilitate joint repair beyond simply making more matrix.

Cartrophen (pentosan polysulphate):
- Dose: 3 mg/kg once weekly for 4–6 weeks and then once monthly, but there are a variety of doses and treatment regimes.

Adequan® (polysulphate glycosaminoglycan) is currently unavailable during the writing of these notes:
- Dose: 5 mg/kg SQ once weekly for 4–6 weeks and then once monthly, but there are a variety of doses and treatment regimes.

Therapeutic Lasers
Have no clinical evidence, but clinicians feel anecdotally that they work well. Lasers do not affect normal cells, but inflamed cells respond to laser. Laser can be used to moderate pain and inflammation. Laser results in an activation of enzymes, modulation in the levels of cytokines and mediators of inflammation, and an overall increase in tissue oxygenation.

Analgesia is an important aspect of treatment of feline DJD.

NSAIDs
In the past, NSAIDs could not distinguish the COX enzymes; they inhibited them both. With the development of COX-preferential and COX-selective NSAIDs, we can inhibit COX-2 and leave COX-1 alone. The introduction of COX-2 preferential NSAIDs has reduced stomach and intestinal side effects by 50% in humans and has made FDA approval of certain NSAIDs possible for pets. They have made a profound difference when dosing at the ideal weight and using the least dose possible the least number of days to enable a cat with DJD to live a quality of life. Gabapentin is been used frequently in addition to NSAIDs on severe cases, or instead of NSAIDs in mild cases, or cats with pronounced renal disease.

    Buprenorphine (0.01–0.03 mg/kg q 8–12 hours (buccal mucosa) can be used as well, but remember to start with a low dose in the geriatric cat and increase the dose in small increments. Dermal codeine at 2.5 mg/5 kg cat BID possesses similar activity to other opiate agonists. Very limited information available for
domestic animals on this product, but again, it has been a useful addition in multimodal pain control. Tramadol given once a day at half the suggested dose is well tolerated by cats and can be used for analgesia. At this lower, once-a-day dose the euphoria that cats traditionally have exhibited is reduced or nonexistent.

**Prevention**
Prevent obesity - short and sweet as it is the major culprit. The excess presence of “white fat” is detrimental for the body and the inflammatory process is heightened when the patient is obese, creating a Pandora’s box syndrome with the joint damage and destruction resulting as an innocent bystander.

**Solution**
Nutritional assessment - *every cat, every time!* In addition to an assessment a nutritional recommendation, dose recommendation, and how to feed recommendation needs to occur - *every cat, every time!* The rest of the lecture will focus on the challenge of the “obesity battle in cats.”
Feline Hyperthyroidism: A Review of an Old Disease in Old Cats
Elizabeth O’Brien, DVM, DABVP (Feline)
The Cat Clinic, Hamilton, ON, Canada

Using case examples this lecture will be a review of hyperthyroidism in cats. An emphasis on early detection, clinical presentation in 2012, and various treatment options and considerations including surgical, medical therapy, I131, and diet will be discussed. Recommendations based on the cat’s age, the client’s budget, the ability to medicate, and concurrent conditions will be considered.

Below are some of the facts about the disease that will be intertwined throughout the case presentations.

EPIDEMIOLOGY OF HYPERTHYROIDISM
Hyperthyroidism is the most common endocrinopathy of cats. It is considered to be a “new” disease that was first described in 1979. Enlargement of the thyroid gland had been noted at necropsy in cats before this date, but they were not associated with clinical signs. The cause of the disease is still unknown.

Canned food appears to be a risk factor and the “pop-top” cans may pose more of a risk because of the release of chemicals such as bisphenol-F and -A from the lacquer lining of the cans during the heating process. Environmental factors such as insecticide use, herbicides, and fertilizers, and the routine use of flame retardants are all things that became present around the onset of this disease.

PATHOGENESIS
The clinical signs of hyperthyroidism are a direct result of adenomatous hyperplasia of normal thyrocytes. These cells replicate in an autonomous fashion in the absence of TSH. Bilateral thyroid involvement is present in more than 70 percent of cases. Thyroid carcinoma is rare occurring in less than 2-4% (depending on the study) of cases. In my clinical experience, I have seen it much less than what is reported in the literature.

Hyperthyroidism most commonly occurs in middle-aged to older cats, but the speaker has diagnosed it in a cat of age one, so always check T4 on any cat that exhibits the clinical signs listed below. The severity and frequency of the clinical signs are often reduced in comparison to what we have seen historically, but the clinical signs remain the same: weight loss, heart murmur, tachycardia, mild hypertensive (moderate-severe hypertension should make the clinician look for other causes), behavioural changes, hair-coat changes, increased appetite, vomiting/diarrhea, PU/PD (watch for UTIs due to low SG).

The complete blood count is usually normal, although polycythemia, eosinophilia, or a stress leukogram may be present. A biochemical panel usually reveals mild to moderate increases in ALT and alkaline phosphatase. Other common findings include azotemia (concurent renal or prerenal), hypokalemia, and hyperglycemia (stress induced). Apathetic hyperthyroidism appears to occur in about 5-10% of cases. These cats rather than being restless are lethargic and weak. Anorexia or decreased appetite, rather than a ravenous appetite, is the cause of the weight loss. Approximately 80% of cats have a palpable thyroid nodule which can be palpated by the classic, Norsworthy or two-handed technique - these techniques will be quickly reviewed during the lecture.

APPROACH TO DIAGNOSIS
The minimum database necessary for evaluation of a cat with hyperthyroidism includes a detailed history and physical examination, serum thyroxine (T4) concentration, complete blood count, serum chemistry panel, urinalysis, thoracic radiographs, and arterial blood pressure. A diagnosis of hyperthyroidism can usually be confirmed by measurement of a single, serum T4 concentration. In some cats with early hyperthyroidism and those with concurrent non-thyroidal disease, the T4 concentration may intermittently fluctuate into the normal range. If the T4 is high normal or borderline, diagnostic options include repeating the measurement of serum T4 at a later date after concurrent diseases have been treated and/or the hyperthyroid state has become more severe, or measurement of a free T4 concentration (by equilibrium dialysis or equivalent assay). Other diagnostic options include a T3 suppression test.
**TREATMENT OF FELINE HYPERTHYROIDISM**

A number of options are available for the treatment of feline hyperthyroidism. The choice of treatment depends on the presence and severity of concurrent, non-thyroidal illness, the age of the cat, the cat’s tolerance for hospitalization, availability of I131, adverse effects of antithyroid medications, owner preference, and concurrent illness.

**Antithyroid Drugs**

The antithyroid drug of choice is methimazole which should be initiated at a starting dose of 2.5 mg q 12 hours and then titrated to effect. Most cats (90%) become euthyroid within 2–3 weeks of starting therapy. The dose required to maintain euthyroidism is quite variable from cat to cat (2.5–20 mg/day). Adverse clinical reactions can include anorexia, vomiting, and lethargy which are usually dose related and can be corrected with cessation of medication for a few days and then started at half of the dose. Excoriation of the head and neck, icterus, and bleeding diatheses can occur idiosyncratically and medication must be permanently stopped. Mild hematological abnormalities develop in 16% of cats and include leucopenia, lymphocytosis, and eosinophilia. More severe hematologic abnormalities develop in 4% of cats and include agranulocytosis and thrombocytopenia. A small percentage of hyperthyroid cats may be resistant to the effects of methimazole.

**Thyroidectomy**

The advantages of surgical thyroidectomy include rapid response to treatment, short hospital stay, convenience in the private practice setting, and opportunity to evaluate surgically excised thyroid tissue histopathologically. Disadvantages include the need for general anesthesia, the risk of inducing iatrogenic hypoparathyroidism, and morbidity associated with the surgical procedure. Ectopic thyroid tissue is found in as many as 12% of hyperthyroid cats which limits surgical correction. In our practice, due to access to I131, good results with medication, and a dietary option to control the disease, I no longer offer surgery as an option.

**Radioactive Iodine**

The thyroid gland concentrates iodine within the colloid of the gland. Radioactive iodine emits β particles which destroy functional thyroid tissue without causing damage to normal tissues such as the parathyroid glands. Normal thyroid tissue is spared because it is atrophic due to lack of TSH, and does not concentrate much iodine. Iodine-131 is the radionuclide of choice for the treatment of hyperthyroidism. Antithyroid drugs or iodine-reduced diet should be discontinued several weeks before treatment. T3 and T4 concentrations decline in 5–10 days post treatment and clinical improvement is usually observed within 2 weeks of treatment, although in some cats the response may be delayed. Hypothyroidism may occur in cats secondary to radioactive iodine therapy; in many cases this is transient and unassociated with clinical signs. Persistent hypothyroidism decreases glomerular filtration rate and has been associated with decreased survival in azotemic cats. Clinical signs of hypothyroidism in cats include anorexia, lethargy, weight gain, poor hair coat, and alopecia.

The advantages of I131 therapy are that all sites of thyroid tissue are destroyed, there is no risk of hypoparathyroidism, no anesthesia is required, and it is possible that the risk of recurrence may be lower. The disadvantage is the requirement for isolation in an approved facility; currently 7 days in Ontario. Radioactive iodine therapy should be avoided in patients with severe, concurrent, non-thyroidal illness especially those that require therapy during isolation, in cats with CRD that worsens with the use of antithyroid drugs, and in those patients that do not tolerate hospitalization well.

**Nutritional Management**

Limiting dietary iodine to less than 0.32 ppm reduces the circulating thyroid hormone concentrations into the normal range in hyperthyroid cats. Dietary iodine restriction has potential as an alternative management strategy for feline hyperthyroidism because thyroid hormone synthesis requires iodine and the thyroid gland is the only gland in the body that uses iodine. Hill’s y/d has proven to be a wonderful option for those cats that cannot be treated with radioactive iodine therapy or those who do not tolerate the medication or medicating well. The most common reason for failure to control the hyperthyroidism is access to iodine containing food such as treats, human food, or other pet foods. Even small amounts of
other iodine-containing foods can result in an increase in the TT4 concentration. An iodine-limited diet should not be combined with oral antithyroid drugs because of the risk of profound hypothyroidism. Nutritional management is not a good option for cats that go outside and can access other sources of dietary iodine, and for those cats that need to be on a controlled diet to manage concurrent non-thyroidal illnesses that require dietary therapy such as inflammatory bowel disease and allergic dermatitis. In cats in early renal insufficiency and concurrent hyperthyroidism, Hill’s y/d may be an acceptable diet because it is supplemented with omega-3 fatty acids, contains controlled amounts of phosphorus sodium, and high quality protein. Hyperthyroid cats in multiset households need to be fed individually and access to food of other pets in the household must be prevented. Alternatively, the iodine limited diet can be fed to all cats in the household providing the euthyroid cats are supplemented daily with a small amount of food with higher iodine content. In cats that do not become euthyroid within 4–8 weeks of starting a limited-iodine diet a detailed history should be investigated for evidence of other sources of iodine. Possible sources of iodine in addition to access to other pet foods include well water, use of medications or supplements, contaminated food bowls, and access to human food.
Bloodwork in the Feline - Exactly What Are Those Results Telling Me About My Patient?
Elizabeth O’Brien, DVM, DABVP (Feline)
The Cat Clinic, Hamilton, ON, Canada

OBJECTIVES
To demonstrate a personal approach to the hemogram and blood chemistry interpretation in cats using actual case studies.

Cases will include:
- A physiologic leukogram
- Anemia - regenerative and nonregenerative
- Kidney disease
- Liver disease
- Concurrent disease
- A stress leukogram

KEY POINTS

Hemogram

Anemia is a disease syndrome rather than a specific disease. Anemia is classified as regenerative or nonregenerative based upon peripheral blood morphology and reticulocyte count.

Regenerative anemia includes blood-loss anemia and hemolytic anemia. They are characterized by decreased PCV, decreased RBC, increased reticulocyte count, and polychromasia, and anisocytosis. Markedly regenerative anemias have elevated MCV values and reduced MCHC values (macrocytic and hypochromic anemias).

Nonregenerative anemia is the most common anemia seen in the cat and is a result of long standing, often hidden, chronic disease. The vague nonregenerative “anemia of chronic disease” may be macrocytic normochromic; normocytic normochromic, or macrocytic hypochromic. In my experience as a feline practitioner, a nonregenerative anemia in a cat may be viewed as a guarded prognosis, but it can fool the practitioner because it may be the result of long-term, “hidden” chronic disease that will become regenerative, when the disease is appropriately managed.

All anemic cats, like all sick cats, should be FeLV/FIV tested.

White blood cell parameters in the CBC are the white blood cell count (WBC) and differential cell count from the peripheral blood film. Although the differential cell count is always evaluated as a percentage, it should only be interpreted in terms of absolute numbers.

Acute to subacute inflammation is suggested by the presence of increased numbers of immature neutrophils (band cells). Most inflammatory processes are also accompanied by a leukocytosis with neutrophilia and lymphopenia, but a leukopenia with neutropenia and left shift (degenerative left shift) may be seen with severe, overwhelming inflammatory disease. Toxic neutrophils should alert the clinician that severe inflammation is present.

A stress leukogram from chronic illness without accompanying inflammation is usually characterized as a mild leukocytosis with a mature neutrophilia, lymphopenia, and eosinopenia. Unlike dogs, monocytosis is an unusual finding in a cat with chronic illness and inflammation. Mild to moderate lymphopenia is an indicator of illness. The severity of the lymphopenia is often an indication of the severity of the illness and personally, I do see this in acute cases as well as cases with a chronic illness.

A physiologic leukogram which is transient and nonpathologic and a result of an epinephrine release due to fear and excitement results in an increase in total WBC with an increase in both neutrophils and lymphocytes resulting in a mature neutrophilia and mature lymphocytosis. No matter how “cat friendly” we aim to be, this is a common occurrence in the cat.

It is important to note the difference between a “stress leukogram” and a “physiologic leukogram.” I often think the terminology should be changed as the wording can be confusing to the clinician.
Eosinophilia can occur in the cat commonly from endoparasitism, ectoparasitism, hypersensitivity reactions such as asthma and eosinophilic gastritis, eosinophilic granuloma complex, hyperthyroidism, and mast cell tumors.

Thrombocytopenia can be caused by increased platelet destruction and DIC, but look for clumped platelets and that is commonly the source of the thrombocytopenia.

**Blood Chemistry**

Elevated total protein levels are most often associated with either dehydration or chronic antigenic stimulation with hypergammaglobulinemia. Although we see this commonly with FIP, we see it with any chronic antigenic stimulation. Commonly in my practice, this is seen with severe dental and gingival disease. Elevated total protein levels in conjunction with elevated packed cell volume (PCV) suggest that the animal probably is dehydrated with relative polycythemia as a result. If normal hydration were restored, PCV would most likely be normal. An elevated total protein in conjunction with a low PCV may well be masking a more severe anemia. Whenever total protein is elevated secondarily to dehydration other chemistry changes are to be anticipated. For example, electrolyte levels should be higher due to simple concentration. Prerenal uremia secondary to hypovolemia and characterized by mild to moderate elevations in blood urea nitrogen (BUN) and Creatinine (Cr) is often present and a normal urine specific gravity would be expected.

**Albumin**

Albumin is the major plasma protein synthesized in the liver. Hypoalbuminemia in the cat is often, in my opinion, a result of intestinal malabsorption and is a red flag for lymphoma. It may also result from protein losing enteropathy or nephropathy, or be the result of decreased protein production by the liver.

**Alanine Aminotransferase (ALT)**

ALT is a cytosolic/leakage enzyme and is considered relatively liver specific as it is in abundance in the hepatocellular cytoplasm. Hepatocellular injury by damage to the cell membranes causes release of the enzyme into the extracellular space and subsequent elevations in serum levels. ALT may leak from the hepatocyte in any condition that alters membrane permeability to a significant degree (leakage does not require cell death). The serum half-life is 24 hours and the magnitude is directly proportional to the number of hepatocytes affected. Acute damage causes an elevation within 12 hours and the highest magnitudes indicate tissue necrosis. ALT elevations are not specific for primary hepatocellular disease. Bile duct obstruction (e.g., pancreatitis) causes ALT elevation within 3 to 4 days, and the elevation persists for at least several days. It is probable that retained bile salts physically damage the cell membranes of surrounding hepatocytes and result in enzyme leakage. The magnitude of ALT elevation does not correlate with reversibility. It is simply a screening test for hepatocellular damage. Nonspecific elevations of serum hepatic enzyme activity can occur secondary to a number of causes. In most cases serum enzyme values do not increase beyond 2 or 3 times normal.

**Alkaline Phosphatase (ALP)**

Serum ALP is considered a reflection of liver or bone activity. Bone growth in young animals may result in ALP values up to 4 times normal adult values. In adults, an increase is a result of increased production secondary to intrahepatic or extrahepatic cholestasis, not cell leakage as with ALT/AST. Any cause of hepatobiliary inflammation may result in secondary hepatobiliary disturbances leading to intrahepatic cholestasis. **Pancreatitis may result in marked increases in SAP.**

**Gamma Glutamyl Transferase (GGT)**

In the feline, GGT is more sensitive in diagnosis of liver disease than is alkaline phosphatase (but is more specific for hepatobiliary disease). GGT generally parallels changes in SAP with one notable exception in cats. Cats with hepatic lipidosis have marked increases in SAP while only mild increases in GGT occur. This fact may be useful for strengthening suspicion that hepatic lipidosis is present. Remember that hepatic lipidosis is no longer considered a primary disease entity and is secondary to some other disease process.
**Bilirubin**
Bilirubin is formed from the breakdown of hemoglobin by the mononuclear phagocyte system in the body. An elevation in bilirubin should be then approached as prehepatic, hepatic, and posthepatic. A normal HCT rules out prehepatic. A normal ALT, AST, ALP indicates posthepatic and in the face of a normal HCT, ALT, AST, ALP, inflammation of the pancreas should be considered in my experience. The total bilirubin level provides the most useful clinical information. Again, classify jaundice as: prehepatic, hepatic, posthepatic. The first step in evaluation of an icteric patient is to rule out hemolytic anemia (may be life threatening). Bilirubinuria, even in concentrated urine, is an abnormal finding in cat. Bilirubin is less sensitive, but more specific than liver enzymes in identifying hepatobiliary disease. Using bilirubin values in conjunction with liver enzymes and serum bile acids concentrations improves the diagnostic specificity of the enzymes.

**Serum Bile Acids**
Abnormal serum bile acid concentration approaches 100 percent specificity for hepatic disease. It is not necessary to run a bile acid assay in a hyperbilirubinemic patient that is not anemic. Hyperbilirubinemia in the absence of hemolysis indicates that a hepatic or post hepatic disorder is present. However, the test may be run in hyperbilirubinemic patients because bile acids do not compete with bilirubin. The degree of bile acid elevation in icteric cats is greatest with hepatic lipidosis. Cats and dogs with portosystemic shunting may have normal or only slightly increased fasting levels, but postprandial concentrations are always abnormal.

**Creatine Kinase (CK)**
Creatine kinase is a cytosolic enzyme found in numerous tissues although its activity is highest in skeletal muscle, cardiac muscle, and brain. Abnormal elevations in CK are the result of direct injury to the musculature and leakage of the enzyme from the cytoplasm of damaged myocytes.

**BUN/Creatinine**
Blood urea nitrogen (BUN) and creatinine are the most commonly assessed indices of glomerular filtration and, thus, renal function in mammals. As these components are freely filtered by the glomerulus, any reduction in the glomerular filtration rate (GFR) results in increases in the concentration of these analytes in plasma and serum.

**Blood Urea Nitrogen (BUN)**
BUN is produced in the liver by breakdown of dietary proteins. Therefore, BUN may increase in the serum due either to decreased renal filtration or due to increased protein ingestion. Gastrointestinal blood loss and catabolic states increase urea concentration. Low protein diets and liver disease can decrease urea concentration. Thus, urea concentration has limitations with respect to measurement of GFR.

**Creatinine**
Creatinine is freely filtered by glomeruli so that its concentration in plasma is equal to that of the glomerular. Creatinine is not affected appreciably by diet in cats. The rate of excretion is relatively constant in the steady state and serum creatinine concentration varies inversely with GFR. Thus, serum creatinine concentration provides an estimate of GFR. Both urea and creatinine concentrations need to be assessed with urine specific gravity (USG).

Conventional wisdom is that urine concentrating ability will become compromised once approximately two thirds of the nephrons have been lost and that azotemia reflects loss of at least three quarters of overall nephron function. These fractions are not literally correct in all circumstances, but they do indicate that a large proportion of renal function must be compromised before renal function becomes clinically evident. Increasing creatinine concentrations even within normal range are suggestive of a declining GFR if USG is not concentrated.

**Staging chronic renal disease (IRIS):**
The International Renal Interest Society (IRIS) developed a four-level system for staging the continuum of progressive renal disease to use as a guide in diagnosis, prognosis, and treatment. Staging is based on the level of kidney function as determined by creatinine in the rehydrated patient.

- **Stage 1**: Creatinine < 1.6 mg/dl (< 140 mmol/L) = non-azotemic renal disease
- **Stage 2**: Creatinine 1.6–2.8 mg/dl (140–250 mmol/L) = mild renal azotemia
- **Stage 3**: Creatinine 2.8–5.0 mg/dl (251–440 mmol/L) = moderate renal azotemia
- **Stage 4**: Creatinine > 5.0 mg/dl (> 440 mmol) = severe renal azotemia

**Proteinuria - UPC ratio:**
- Nonproteinuric = UPC < 0.25
- Borderline proteinuria = UPC 0.25–0.5 - reevaluate after two months
- Proteinuria = UPC > 0.4

**Classification of blood pressure:**
- **NH** = non-hypertensive = < 150 mm Hg no complications
- **BP** = borderline hypertensive = 150–160 mm Hg with no complications
- **Hnc** = hypertension no complications = consistent systolic blood pressure values >160 mm Hg
- **Hc** = hypertension with extrarenal complications = signs + > 150 mm Hg

**Calcium, Phosphorus, and Magnesium**

Calcium, phosphorus, and magnesium are regulated via three main body systems: the gastrointestinal tract, kidneys, and the parathyroid glands. Therefore, any dysfunction in any one of these organ systems may result in severe imbalances of cellular function.

Hormonal control of calcium is maintained by the action of parathyroid hormone (PTH), formed by the parathyroid glands, and by calcitonin, produced by the C cells of the thyroid glands, where the serum concentrations of calcium regulate the activity of these hormones by feedback control. The majority of calcium is located intracellularly (bone) in animals. Only a small amount of calcium is found in circulation with approximately 50% being present in the ionized/active form. Measurement of total calcium, therefore, reflects not only the active ionized form of this mineral, but also the amount bound to protein and complexed to other molecules. Imbalances of calcium should be verified by measurement of the ionized form. Common causes of hypercalcemia are spurious (lipemia, hemolysis), hyperparathyroidism, idiopathic, renal disease (normal iCa with elevated tCa), and tumors such as lymphoma, carcinoma, or multiple myeloma. Hypocalcemia is not a common finding in cats and can be seen with eclampsia and iatrogenic hypoparathyroidism post thyroidectomy.

Like calcium, **phosphorus** is regulated by renal, parathyroid, and gastrointestinal mechanisms. Absorption of phosphorus by the gastrointestinal system is also under the control of PTH and vitamin D3 activity on skeletal, renal, and gastrointestinal systems. The majority of phosphorus is located intracellularly and mainly stored as inorganic hydroxyapatite in bone with lesser amounts in muscle. Hypophosphatemia can be caused by decreased absorption in the intestine due to malabsorptive diseases, vitamin D deficiency, or intestinal losses from vomiting and diarrhea. Severe hypophosphatemia can lead to hemolysis. Hyperphosphatemia is most commonly seen in renal disease and the use of decreased phosphorus containing diets and phosphorus binders early in kidney disease have become an important part of the treatment regime for CRD in cats. In acute situations, hyperphosphatemia is treated with diuresis until the phosphorus binders can be added orally with food. Remember that phosphorus binders need to be given with food as they bind with the phosphorus in the diet and prevent intestinal absorption. Other causes are vitamin D toxicosis, phosphate containing enemas, hypoparathyroidism, and hyperthyroidism.

**Potassium**

Potassium is the major intracellular cation and is largely responsible for intracellular volume. Hypokalemia most commonly results from chronic renal disease, but it also occurs from gastrointestinal losses during vomiting and diarrhea and is influenced by the acid-base balance of the animal. Translocation of potassium may occur from extracellular fluid to intracellular fluid with the administration of insulin, sodium bicarbonate, and catecholamine release. By the same token, a sick cat
that may be acidotic may have a normal serum potassium level, but will be exhibiting clinical signs of hypokalemia with the classic ventroflexion of the neck and muscle weakness.

Maintenance of normal electrolyte balance is tightly regulated by the body and occurs mainly via renal-tubular resorption and secretion of these molecules in response to variations in hydration status, serum osmolality, and acid-base balance of the individual. In the case of sodium, the most osmotically active particle in the body, hydration status is closely linked to its rate of resorption or loss and variations in sodium are often coupled to loss of other electrolytes due to the body’s attempt to maintain electroneutrality. Imbalances of electrolytes are useful to predict, not only the cause of disease, but also response to therapy.

**T4**

Thyroid hormones are released by the thyroid gland in response to thyroid stimulating hormone (TSH) release from the pituitary. Thyrotropin releasing hormone (TRH) secreted by the hypothalamus binds to receptors on the pituitary gland to cause the release of TSH. The thyroid gland releases triiodothyronine (T3) and thyroxine (T4) and a small amount of the parent hormone, thyroglobulin, into the bloodstream. T4 is the major thyroid hormone released by the thyroid gland, while T3 is most active in the cell. T4 is elevated in cats with hyperthyroidism, but T4 can be suppressed by illness resulting in euthyroid sick syndrome. In my opinion, T4 should be evaluated in all cats with illness regardless of age. A low T4 level is usually an indication of severe illness. Remembering that they are the masters of disguise, a low T4 makes me search further for a reason of ill health and treat the cat more intensively than I may have if the T4 was normal.

**Endnote**

a. A normal specific gravity for a cat is a concentrated specific gravity; sometimes as high as 1:060 without any clinical signs of dehydration. They are from the East and are like desert animals in that they tend to live with concentrated urine. Their normal state would appear on blood work to be a dehydrated state for other species such as the dog.
Defining Inflammatory Airway Disease: A Moving Target
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Much confusion still exists about the definition of inflammatory airway disease (IAD) in horses. An important first step in our understanding of this syndrome involves a review of the similarities and differences between IAD and its closely related sibling, recurrent airway obstruction (RAO), also known as ‘heaves.’ This presentation is the first of three sessions, which examines IAD, first from the perspective of its relationship to RAO, second, as it relates to diagnosis based on recent research, and finally, from the standpoint of treatment options, recommendations and perceived outcomes. In all sessions, recent literature is cited where appropriate.

IS IT HEAVES OR IAD? WHY WE SHOULD CARE
What We Know About Heaves...

Terminology
Over the years, numerous terms have been used to describe the noninfectious diseases of the lower respiratory tract of horses. These include heaves, chronic obstructive pulmonary disease (COPD), chronic obstructive bronchitis/bronchiolitis, allergic bronchitis, broken wind, RAO, IAD, and small airway inflammatory disease. This plethora of terminology serves only to reflect our incomplete understanding of the pathogenesis of these disorders. The best example is the term COPD. In the past, equine COPD was used to describe any accumulation of neutrophils and mucus in the airways of horses in the absence of infection. From this definition, both the older horse with severe heaves and the younger horse with reduced performance (both of which have increased airway neutrophils and mucus) would be diagnosed as having COPD. But, the two conditions, as we understand them today, are very different, and although there is no evidence that the former is a consequence of the latter, the suspicion of such, at least in some horses, remains.

A second source of confusion has been the interchangeable use of the term ‘COPD’ to describe the syndromes of noninfectious, lower-airway obstructive disorders in both humans and horses. In human medicine, COPD is usually a disease of aged smokers, and this term would not be applied to a young athlete with cough, increased mucus production and decreased exercise tolerance. Equine and human COPD differ in that in humans, the obstructive disorder is irreversible, whereas in horses, the obstruction of severe heaves is reversible with changes in management, bronchodilator drugs and corticosteroids. As a result of these differences, and at the recommendations of a team of delegates who participated in the Havemeyer Foundation Workshop in 2002, the term COPD has been replaced with recurrent airway obstruction (RAO), or ‘heaves,’ the latter being descriptive in the clinical sense, the former being a more accurate reflection of the reversible nature of the disease as it exists in horses.

General
Heaves is a noninfectious, inflammatory, reversible, obstructive airway disease of middle-aged to older horses with a mean age of greater than seven years and an estimated prevalence of 7–20%. The disease is most prevalent, albeit sporadic in nature, in the Northern Hemisphere where horses are stabled and fed hay, particularly where adequate drying of hay is difficult to achieve. Conversely, a seasonal, naturally occurring obstructive pulmonary disease affecting horses residing on pasture in the southeastern United States (termed summer pasture-associated obstructive pulmonary disease [SPAOD]) is clinically indistinguishable from heaves, the primary difference being that affected horses develop signs after exposure to pasture during the warm and humid months of the year. Remission of signs occurs during colder months.
Heaves is initiated by exposure of the airways to a combination of different airborne allergens and particulate matter. The presence of dust, molds (especially *Faenia rectivirgula*, formerly *Micropolyspora faeni*), pollens and their spores, forage mites, bacteria and endotoxin, and perhaps viruses, in the breathing zone of susceptible horses has been associated with the development of pulmonary hypersensitivity in RAO-affected horses. The identification of allergen-specific immunoglobulin E (IgE) in bronchoalveolar lavage (BAL) fluid and sera of some affected horses supports the involvement of a late phase IgE-mediated hypersensitivity; however, it has also been proposed that RAO may result from a nonspecific, inflammatory response to inhaled, proinflammatory agents. Regardless of the precise etiopathogenesis, affected horses appear to have an exaggerated, inflammatory response to environmental stimuli that might only cause mild inflammation in ‘normal’ horses.

A genetic susceptibility to RAO was first proposed by Schäper in 1939 who discovered that 14 out of the 24 descendents of the stallion “Egmont,” affected by RAO, also developed disease. In another study which looked at 90 German warm-blooded horses and 42 Lipizzaners, Marti et al. (1991) showed that the risk of contracting RAO was 3.2 times higher when one parent had the disease, and 4.6 times higher when both parents were affected. More recently, Gerber et al. (2009) demonstrated that RAO has high heritability and a complex mode of inheritance. In one family, the mode of inheritance was shown to be autosomal dominant, whereas in another family, it was autosomal recessive.

**Clinical Signs**

The earliest clinical signs that an astute owner observes are occasional coughing and exercise intolerance. With progression, affected horses have prolonged recovery, typically a slight nasal discharge, and may have intermittent periods of respiratory distress. During later stages of disease, horses show signs of severe respiratory distress manifested by anxiety, paroxysmal coughing, nostril flaring, anal pumping, tachypnea, and abdominal push (often accompanied by a heave line). Adventitial lung sounds are evident during thoracic auscultation.

**Diagnosis**

Diagnosis of RAO is most commonly made on the basis of signalment, history and clinical signs. In uncomplicated cases of RAO, the complete blood count, serum chemistry panel and fibrinogen findings are normal; these tests may be useful, however, for ruling out pneumonia or an advanced neoplastic process. Chest radiographs are variable, but more commonly nondiagnostic. Mildly affected animals may have normal-appearing lungs; in older, moderately affected horses, it may be difficult to differentiate radiographically, between a mild interstitial or peribronchial reaction, and normal aging changes. Overall, one should look for a bronchial pattern and/or signs of pulmonary overinflation. Radiographs are helpful in ruling out neoplasia or infectious disorders. Thoracic ultrasound may confirm a caudal shift (past the 16th intercostal space) of the caudal lung border caused by alveolar hyperinflation in severely affected horses. Excessive mucopurulent tracheal secretions are evident on endoscopy and fluids collected from either a transtracheal wash (TTW) or BAL reveal a marked increase in the percentage of neutrophils relative to other cell populations. Bacteriologic culture from a TTW sample is necessary to rule out an infectious cause. In severe cases, arterial hypoxemia (pO₂ < 82 mm Hg), in the absence of arterial hypercapnia, may be noted. A bronchodilator challenge test may provide temporary relief if chronic, irreversible changes have not yet occurred in the airways (atropine challenge test: 5–7 mg/450-kg horse, butylscopolamine [Buscopan™] at label dose, IV, or an inhaled bronchodilator). Severely affected horses that repeatedly fail to respond to bronchodilators and steroids may have irreversible airway remodeling (peribronchial fibrosis) or bronchiectasis leading to collapse of the affected airways during expiration. Mucus plugging of airways also may contribute to failure to fully respond to bronchodilation. Pulmonary function testing (PFT) demonstrates a decrease in lung compliance, an increase in lung resistance, an increase in the work of breathing, and airway hyperreactivity in response to nonspecific stimuli such as histamine. Changes in pleural pressures (as measured with an esophageal balloon) of > 6 mm Hg are a consistent finding in horses with advanced disease. The use of allergen skin testing (intradermal skin testing) and serum allergen testing is highly controversial.
Treatment and Prevention
The primary goal of therapy should be aimed at environmental modification. Drugs are needed when management measures are either not possible or are insufficient to control clinical signs. While in early stages, antihistamines may provide some benefit, the mainstay of therapy should include bronchodilators to ease the work of breathing, and corticosteroids to control inflammation (See session three: Inflammatory Airway Disease: Therapeutic Options, for a more complete discussion on approaches to treatment). Careful attention to good management practices in young, stabled horses is very likely to dramatically reduce the incidence of RAO in older horses.

Consequences
In affected horses, the distal airways are the primary sites of microscopic lesions. Airway remodeling is characterized by inflammation of the distal airway wall, epithelial metaplasia with desquamation, peribronchial fibrosis, increased smooth muscle mass, and ‘air trapping,’ which, in the horse, is more common than emphysema. Bronchiectasis (a chronic inflammatory or degenerative condition marked by dilatation and loss of elasticity of the walls of the bronchi or bronchioles) may occasionally develop, and when present, contributes to the poor response to bronchodilation observed in severely affected horses. Other consequences include weight loss and debilitation, secondary infection of compromised airways leading to pneumonia, and in rare circumstances, right heart failure and cor pulmonale.

Prognosis
Due to the reversible nature of RAO, treatment is usually effective if identified and treated early. The prognosis for horses with RAO therefore depends upon the severity and chronicity of clinical signs, the ability to control the environment or eliminate inciting causes, the amount of drugs needed, and the work expected of the horse. Performance horses may be more difficult to manage.

What We Know About IAD...
General
Inflammatory airway disease, although similar in many respects, has a different phenotype compared to horses with RAO. While IAD is also a noninfectious, inflammatory, obstructive airway disorder, it is a disease of predominantly young performance horses; however, any age (and horses of any discipline) can be affected. The disease is common, and highly prevalent in stabled horses with estimates of 20–80% prevalence in racehorses in training, and in this group of horses, prevalence tends to decrease with age. In non-racehorses or in sport horses of other disciplines, IAD is usually diagnosed at an older age. Like RAO, IAD is initiated by inhalation of organic dust, which is composed of molds, bacteria and the toxins they release (endotoxin), minerals, insect parts and various chemicals, including ammonia, although the relative contribution of any of these factors is unknown. Air pollutants, mostly gases (ozone sulfur and nitrous dioxide), but also particulate matter (heavy metals, carbon, smoke) are also believed to play a role. The role of viruses is unknown, but there often is a history of viral infection prior to the onset of IAD. In contrast to RAO, which presents with fairly classical clinical signs of airway obstruction (the ‘heavey’ horse), IAD presents with a wide range of clinical signs. Finally, while there have been a limited number of studies investigating heritability of IAD, a genetic susceptibility has not been shown at the time of this writing.

Clinical Signs
IAD-affected horses generally are normal on physical examination at rest and fail to display signs of obstruction typical of the heavey horse, even when exposed to dusty environments. The most common complaint by owners of athletic horses with IAD is poor performance. Racehorses reportedly perform well until the ¾ mile pole, after which time, they drop back. Sport horses are “sluggish,” or unwilling to move forward in their training. Dressage horses resist collection. Structurally, performance suffers due to chronic exposure of the lower airways to particulate matter in the stable setting. Histologically, there is increased mucus production along with cellular infiltrates into the walls of the small airways as well as into the lumen of the bronchioles, which serves to compromise luminal diameter. Functionally, performance suffers because obstructed airways lead to airflow restriction, causing air to be diverted to
unobstructed airways. As a result, fewer alveoli fill, which results in insufficient oxygen uptake to meet the demands of maximal aerobic activity. Airflow restriction also leads to more forceful breathing, further increasing the need for oxygen. The ultimate result is an imbalance between the need for oxygen and oxygen consumption. It should be noted that IAD (and RAO) is a disease of the terminal bronchioles. The alveoli are not affected.

Other clinical signs, which are generally reported in older sport horses of other (non-racing) disciplines are chronic cough, nasal discharge (although the relationship between nasal discharge and IAD is currently unknown), and, less frequently, behavioral changes. Coughing may occur at any time, may occur frequently throughout the day, at the beginning of exercise, or may be paroxysmal in nature. Coughing and nasal discharge are rarely reported in racehorses, which may suggest a greater degree of inflammation, or a different type of inflammation when they occur in older sport horses. It is important to note that, because all stabled horses likely experience some degree of lower-airway inflammation, clinical signs may be lacking entirely ('silent inflammation') in horses that are not involved in any type of athletic discipline. The concern here is that, given the proper environment and genetic predisposition, some of these horses may go on to become heavy later in life.

**Diagnosis**

A clinical suspicion of IAD should initially be based on the history, presenting complaint, and lack of clinical signs referable to the respiratory system in the case of racehorses with unexplained poor performance, or by the lack of fever or other signs of systemic infection in the case of coughing horses or horses with nasal discharge. Routine hematology and serum biochemistry are normal. As with RAO, endoscopic examination often reveals increased amounts of mucus along the length of the trachea (suggestive of inflammation), although excessive mucus is common in otherwise normal racehorses with the highest prevalence observed in yearlings and two-year-old horses with decreasing frequency with increasing age. Endoscopic examination of the respiratory tract also facilitates examining the nasopharynx and trachea for other abnormalities and permits collection of a tracheobronchial aspirate (using a guarded swab) for culture, which helps to rule out infectious causes of lower-airway inflammation. While evidence of lower-airway inflammation based on BAL was previously considered the gold standard for a diagnosis of IAD, new criteria emphasize the need for PFT used in conjunction with BAL, as it is known that some horses without cytological evidence of inflammation on BAL exhibit pulmonary dysfunction and vice versa. Therefore, a diagnosis of IAD, according to the 2007 ACVIM consensus statement, may be based on either abnormal BAL or abnormal pulmonary function. It should also be noted that tracheal wash cytology is considered insufficient for a diagnosis of IAD due to a discordance of tracheal wash findings with BAL cytology and PFT. Overall, a history of unexplained poor performance or altered performance, or a history of chronic cough with or without a nasal discharge in an otherwise healthy horse in the presence of an abnormal BAL and/or abnormal lung function is supportive of a diagnosis of IAD.

**BAL Findings**

Three cytologic profiles have been described for IAD. The first is characterized by an increased total nucleated cell count with mild neutrophilia, lymphocytosis and monocytosis. The second is characterized by an increased percentage of neutrophils, mast cells or eosinophils. The third is characterized by mixed inflammation with increases in one or more of the inflammatory cell types. Studies have demonstrated associations between BAL mast cell and eosinophilic inflammatory profiles with airway hyperreactivity (see below), and BAL neutrophilic profiles with chronic cough. In comparison, RAO-affected horses typically show moderate to severe neutrophilic inflammation (> 20%), and demonstrate increased respiratory effort when exposed to moldy hay. A normal BAL cytologic profile derived from an initial infusion of 500 ml saline includes alveolar macrophages 50–70%, lymphocytes 30–50%, neutrophils < 5%, mast cells < 2%, and eosinophils < 0.5%. A normal horse is defined as a horse with no clinical signs of IAD, that has normal endoscopy after exercise [i.e., no mucus], normal airway reactivity [reaction at ≥ 6 mg/ml histamine], and one, which is in full work with no history of exercise intolerance. A cytologic finding of an increase in neutrophils > 5%, mast cells > 2%, and/or eosinophils > 1% is consistent with a diagnosis of IAD.
Pulmonary Function Testing (PFT)
Several studies have documented abnormal pulmonary function in horses with IAD, both at rest and during exercise. Gas exchange has been shown to be impaired during exercise in horses with IAD, as indicated previously. The basis for lung function testing in IAD is the need to identify the mechanical property of flow limitation. There are a number of different methods by which flow limitation can be studied, including the conventional measure of pleural pressure using an esophageal balloon, forced maneuvers, oscillometry, and plethysmography. Most of these methods are performed on a referral basis because of associated technical difficulties, the necessity for laboratory support, lack of portability, and cost. Regardless of the method employed, the tests must be sensitive enough to detect the low-grade obstruction associated with IAD (in contrast, horses with RAO generally do not require PFT to confirm flow limitation due to the presence of classical clinical signs of airway obstruction). Generally speaking, horses with IAD have either normal or increased baseline respiratory resistance, normal or slightly decreased dynamic compliance, mild to moderate frequency dependence of total respiratory resistance, and airway hyperreactivity based on histamine challenge testing. Airway hyperreactivity (also referred to as airway hyperresponsiveness) is a prominent feature of IAD (and RAO!), especially in horses in which the BAL fluid reveals increased proportions of eosinophils and mast cells. The property of airway hyperreactivity is based on the fact that inflamed airways become overly sensitive, or ‘twitchy’ in response to various environmental challenges, including allergens, irritants, pollutants, viruses and inert particles, as well as nonspecific agonists, such as histamine or methacholine. Hyperreactivity is a trait of horses with both IAD and RAO, and, in IAD horses, is best measured with controlled exposure to increasing concentrations of histamine. As per the 2007 ACVIM Consensus Statement on IAD, identification of a horse with airway hyperreactivity (response to histamine at concentrations of < 8 mg/ml), irrespective of the results of a BAL, confirms a diagnosis of IAD.

Treatment and Prevention
Treatment goals for IAD are the same as for RAO, with the primary goal of therapy being aimed at environmental modification (minimizing dust while improving ventilation). Bronchodilators remove the stimulus for coughing and bronchospasm, thereby enabling the horse to breathe easier, while corticosteroids control inflammation. Prevention is aimed at providing good stable hygiene. The majority of horses do better outside. A closer look at treatment options will occur in session three.

Consequences
Short-term consequences of IAD include poor performance for the majority of affected horses, and loss of fitness as a result of both poor performance and prolonged downtime during treatment. Lung remodeling may be a long-term consequence in untreated horses or in horses with “silent inflammation.” Finally, given the right environmental circumstances and the right genetic background, some horses may go on to become heavy later in life.

Prognosis
Prognosis is very good to excellent for return to previous level of performance if identified early and treated appropriately. Failure to respond to appropriate therapy suggests that the underlying cause has not been removed from the horse’s environment.

Summary
Heaves vs. IAD, the Similarities...
To summarise, both heaves and IAD are noninfectious, inflammatory, obstructive disorders of the peripheral airways of horses. Both conditions are initiated by dust or other aerosolized particulate matter. Both may manifest with cough, exercise intolerance and excessive respiratory secretions, and both conditions are associated with airway hyperreactivity.

Heaves vs. IAD, the Differences...
Whereas heaves is more common in older horses, IAD is more common in younger horses. There is a known heritable susceptibility to heaves, but not IAD. Clinical signs of obstruction occur in heaves-affected horses, but not IAD-affected horses upon exposure to moldy hay. Clinical signs of heaves are
usually respiratory in nature, whereas clinical signs of IAD are often non-respiratory in nature (poor performance). Moderate to severe neutrophilic inflammation characterizes heaves, whereas low-grade neutrophilic, eosinophilic and/or mast cell inflammation typically characterize IAD. Finally, PFT is not necessary to make a diagnosis of heaves, whereas very sensitive tests are required to detect the low-grade obstruction of IAD.

References
Diagnosis of Inflammatory Airway Disease: A Comparison Between Old and New Modalities
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BACKGROUND TO INFLAMMATORY AIRWAY DISEASE (IAD) DIAGNOSIS
Equine airway researchers in North America have arrived at a provisional consensus on the diagnosis of IAD (Couetil et al. 2007). They recommend that the diagnosis of IAD be made using two criteria, both of which must be satisfied:
1. History of poor performance, exercise intolerance, and/or cough
2. Presence of nonseptic inflammation detected by bronchoalveolar lavage (BAL) cytology and/or evidence of pulmonary dysfunction

Assessing the first criterion is relatively straightforward, especially in horses in training. However, adequately assessing the second criterion can present difficulties in the field. Pulmonary function testing (PFT) is relatively underutilized in the field due to technical difficulties, the necessity for laboratory support, lack of portability, and cost, leading to reliance on BAL in many situations. There are a number of concerns with this reliance on BAL: it is a relatively invasive procedure; results may vary depending on which parts of the lung are sampled (Depecker et al. 2014); and BAL cytology may not correlate with pulmonary dysfunction (Hoffman, Mazan 1999). These latter two issues may lead, potentially, to underdiagnosis of IAD, given our currently accepted criteria.

Currently, we have insufficient data to assess the degree to which IAD is underdiagnosed. If the results of PFT correlate well with BAL cytology or other measures of airway health, the degree of underdiagnosis is likely to be minor. On the other hand, if there is poor correlation between PFT results and other measures of airway health, we may need to find ways to specifically include PFT whenever IAD needs to be ruled out. With the availability of portable flowmetric plethysmography, it is possible for ambulatory veterinarians to test pulmonary function and airway reactivity in the field with favorable reproducibility (Nolan-Watson et al. 2009).

Flowmetric plethysmography is a sensitive and well-tolerated test used to detect airway hyperreactivity. Earlier methods used to assess pulmonary function in subgroups of horses with lower-airway disease (IAD, RAO) have demonstrated that IAD is indeed an obstructive phenomenon, and that a large proportion of IAD horses have airway hyperreactivity (Klein, Deegan 1986; Doucet et al. 1991; Hare, Viel 1998; Hoffman 1998a).

Airway hyperreactivity results from repeated exposure of the airways to a variety of both direct and indirect stimuli, including allergens, chemical irritants, cold air, hypoxia and viral infections. Elevated leukotriene C4 levels (Hoffman et al. 1998b), mast cells (Hoffman et al. 1998a), eosinophils (Hare, Viel 1998) and equine influenza virus infection (Hoffman et al. 1992) have been associated with airway hyperreactivity in young horses. Repeated or prolonged exposure of susceptible individuals to such irritants sets into motion a complex interplay between the resulting inflammatory response and the nervous system, epithelium, and airway smooth muscle cells, culminating, ultimately, in an increase in the ease and degree of airway narrowing upon reexposure to the offending agents. The initial inflammatory response acts to "prime" the airways, and these primed airways, over time, remodel, which contributes to maintenance of hyperreactivity. The tissues remain primed and "hyperreactive" until the stimuli are removed and the cycle is broken.

Evidence exists to support the fact that hyperreactivity and evidence of inflammation are not always observed concurrently (Hoffman, Mazan 1999; Doucet et al. 1991). In this talk, I present the results of a study which lends further support to this observation, and suggests the need to assess both markers of inflammation and pulmonary function to ensure practitioners identify all possible clinical manifestations of IAD.
**PORTABLE FLOWMETRIC PLETHYSMOGRAPHY: HOW IT WORKS**

So, how does portable flowmetric plethysmography work to test pulmonary function? The technique of flowmetric plethysmography combines measurements of flow, measured directly, at the nose, with flow, measured indirectly (through volume displacements), at the chest and abdomen (RIB/ABD). The technique requires an accurate flow collection device (face mask with pneumotachograph) and elastic bands that sense volume displacement of the chest and abdomen. The excursions of the bands are calibrated to the pneumotach. The pneumotach and RIB/ABD sensors are both attached to an amplifier and laptop computer, and the differences in flow at the two sites are recorded as a signal proportional to constriction of the airways. Under normal circumstances, flow at both levels is roughly equal. During airway obstruction, however, flow at the RIB/ABD increases relative to flow at the nose as the breathing effort of the horse increases in an attempt to overcome compression of gas behind the obstructions. The difference between the volume of compressed gas (as measured by the elastic sensors) and the uncompressed gas (as measured at the nose) is a well-established measure of airway obstruction (Pennock *et al.* 1979; Dorsch *et al.* 1981; Hoffman *et al.* 2001).

The primary benefit of flowmetric plethysmography is its ability to detect subclinical, low-grade IAD. In contrast, despite the fact that horses with heaves show dramatic plethysmographic changes, PFT is not needed for diagnosis or treatment in this group of horses. Flowmetric plethysmography is used to evaluate airway reactivity in response to histamine in the case of IAD, or the response to a bronchodilator in the case of heavey horses. Compared to unaffected horses, horses with IAD bronchoconstrict in response to lower concentrations of nebulized histamine. For testing airway reactivity, horses are first lightly sedated with xylazine or detomidine. The bands are placed around the ribcage (11th intercostal space) and abdomen (behind the last rib), the facemask is applied and the flow sensor (pneumotach) attached. Once the system has calibrated and baseline data have been collected, histamine is nebulized at increasing concentrations until the difference in flow (delta flow) between the RIB/ABD and the nose increases during early expiration by 50% for at least 5 consecutive breaths. Each concentration of histamine is nebulized for two minutes. The nebulizer is then turned off and data are recorded for three minutes before moving to the next dose. Finally, a dose-response curve is generated and the concentration of histamine that evokes a 35% increase in delta flow above baseline is computed by interpolation of the data. Unaffected horses generally do not react to histamine at concentrations below 6–8 mg/ml (Hoffman *et al.* 2001; Hoffman *et al.* 2007; Nolen-Walston *et al.* 2009).

**THE PROJECT**

Given the uncertainty around the optimal methods for diagnosis of IAD, and the fact that underdiagnosis may be common if pulmonary function is not tested, we decided to examine the relationships between equine airway reactivity, as measured by flowmetric plethysmography, and other indicators of airway inflammation (Wichtel M, Burton S, Gomez D, Hoffman A), with the ultimate goal of formulating optimal field diagnostic approaches for IAD, and to increase the chance of identifying horses likely to respond favorably to therapy and/or environmental modification.

**Study Objectives**

We used a case series of horses presenting for poor performance and/or cough to examine the practical application and importance of PFT in the diagnosis of IAD. Our research hypothesis was that airway responsiveness, measured using flowmetric plethysmography and histamine bronchoprovocation, is poorly associated with bronchoalveolar lavage cytology in horses presenting for unexplained poor performance and/or cough. Specifically, we examined the relationships between airway hyperreactivity in response to histamine bronchoprovocation (as measured by flowmetric plethysmography) and other indicators of airway inflammation, including: abnormal BAL cytology; tracheal wash cytology; tracheal wash culture; tracheal mucus accumulation; and degree of pharyngitis. Finally, we formulated a recommendation regarding the importance of using a test of pulmonary function when screening for IAD.
Methods

Animals
This case series comprised 63 privately owned horses that were presented to the Atlantic Veterinary College over a seven-year period (2006–2012) with a complaint of poor performance and/or cough where the poor performance was unexplained by history or physical examination. Exclusion criteria included evidence of systemic signs of infection (fever, hematologic abnormalities compatible with infection) and a history of (or evidence of) labored breathing at rest (heaves). Five horses were excluded from the study because further workup revealed causes for poor performance, cough and/or nasal discharge other than IAD (e.g., septic tracheitis/bronchitis) leaving 58 horses satisfying the enrolment criteria.

Procedures
Upon admission, horses were weighed and placed in a stall. A complete history was collected and physical examination performed followed by a rebreathing bag examination.

Endoscopy was performed to the level of the carina. The upper airways were examined and a pharyngitis score was recorded based on the scoring system described by Holcombe (2005). The endoscope was then passed into the trachea. A tracheal mucus score was recorded based also on the scoring system described by Holcombe (2005). A tracheal wash was performed transendoscopically using a sterile guarded tip catheter and the aspirate collected was submitted for cytology and routine aerobic culture.

For histamine challenge testing, horses were pretreated with detomidine (0.01 mg/kg, IV) and intranasal phenylephrine to prevent/minimize nasal edema. Horses were fitted with the flowmetric equipment, the system was calibrated, and baseline measurements were collected. Histamine was aerosolized at concentrations of 2, 4, 8, 16 and 32 mg/ml. The concentration of histamine that elicited a 35% increase in delta flow was recorded.

For the BAL procedure, horses were sedated with xylazine (0.2 mg/kg, IV). Bronchoalveolar lavage was performed using a commercial cuffed BAL tube (Bivona Medical Technologies, Gary, IL) and two aliquots of 250 ml of room-temperature sterile saline. Thirty to 60 milliliters of a 0.66% lidocaine solution was instilled at the point of maximal coughing. The tube was then advanced until it wedged. The cuff was inflated and the first 250 ml of sterile saline was infused. The sample was retrieved using gentle negative pressure generated by a syringe. The procedure was repeated using the second 250 ml aliquot. Collected fluid was pooled, placed into sterile jars, and submitted for cytology. All samples were submitted to the lab within one hour of collection.

Transtracheal wash cytology was considered abnormal if neutrophils exceeded 20% of total cells counted. A diagnosis of IAD was made on the basis of a BAL if the proportion of cell types exceeded 5% for neutrophils, 2% for mast cells, and/or 0.5% for eosinophils. A diagnosis of IAD was made on the basis of PFT if the concentration of histamine required to increase delta flow by 35% was less than 8 mg/ml.

Results
The 58 study horses were between 2 and 14 years of age (mean 4.3 ± 2.5 years), with 3-year-olds being overrepresented (26 horses). Twenty-five were female, 23 were geldings, and 10 were entire males. Eighty-eight percent of horses were Standardbreds and all horses were in training or in some type of work at the time of presentation.

The number of cases presenting for poor performance, cough, or both, were 32, 11 and 15, respectively.

A valid histamine response test was conducted in 43 of 58 horses. Flowmetric plethysmography data were considered invalid because of calibration problems (4), evidence of an obstructive pattern at baseline (4), excessive noise in the data (1), upper-airway obstruction (1), a ‘heave’-like pattern of breathing at baseline (2), motion artifact (3) or other patient concerns (1).

Twenty-six of 43 horses (60%) were determined to be hyperreactive (delta flow was increased by 35% at a histamine concentration of less than 8 mg/ml). There was no significant relationship identified between abnormal histamine response and signalment, presenting complaint, physical findings, or results of TTW or BAL cytology, or culture.
A diagnosis of IAD was made if the results of BAL cytological examination were abnormal, and/or if the histamine challenge response was abnormal. A diagnosis of IAD was made in 33 of 43 horses (77%). Of the 33 horses in which a diagnosis of IAD was made, only 14 (42%) were both hyperreactive and had abnormal BAL cytology. There was no significant relationship identified between a diagnosis of IAD and signalment, presenting complaint, physical findings, results of TTW cytological analysis or culture.

Figure 1. The proportion of horses (n = 43) presenting with a history of cough and/or poor performance that satisfied none, either, or both of the diagnostic criteria for diagnosis of inflammatory airway disease (IAD)

Discussion
The results of this research suggest that IAD will be underdiagnosed in a significant proportion of horses in which IAD is suspected if reliance is placed solely on the results of a BAL. Of the horses in which a diagnosis of IAD was made, less than half of the horses (14 of 33) had both hyperreactivity as well as abnormal BAL. Other measures of inflammation, such as TTW cytology and mucus accumulation, were also non-predictive of hyperreactivity. The lack of a significant relationship between an abnormal histamine response and other measured indices of respiratory health, suggests that airway hyperreactivity may be intrinsic in some horses, whereby its presence is not always corroborated by evidence of inflammation.

This lack of agreement between airway reactivity and cytological evidence of inflammation has been previously reported (Doucet et al. 1991; Hoffman, Mazan 1999); however, the reasons for this discrepancy are not clear. A possible explanation for the inability to demonstrate hyperreactivity in 7 of 33 horses that had an abnormal BAL is that there may have been an insufficient amount of time for hyperreactivity to develop. Another possible explanation is that a genetic predisposition for BHR exists, as has been shown in humans (Postma et al. 2000) and other animals (De Sanctis et al. 2001), and that the horses in this study lacked such a predisposition.

Conversely, the lack of inflammation in 12 of 33 horses in the face of hyperreactivity may be explained by sampling a non-representative portion of the lung (Depecker et al. 2014), by the presence of bronchial wall as opposed to luminal inflammation (Busse 2010), by recently resolved inflammation, or it may be that hyperreactivity is a normal finding in some horses (Hoffman, Mazan, 1999). It is also possible that, through unknown mechanisms, inflammation is the prominent feature in some, whereas airway reactivity is the prominent feature in others, while the two may occur simultaneously in a proportion of horses. Further research into the relationship of lower-airway inflammation and the occurrence of BHR in horses is warranted.
Regardless of this apparent diagnostic conundrum, we acknowledge that horses satisfying either criterion for IAD (nonseptic airway inflammation or hyperreactivity) will generally benefit from therapy and/or environmental modification. Clinicians should use all of the diagnostic tools available to them to identify horses with clinical manifestations of IAD and institute appropriate case management to reduce its effects on performance and wellbeing.

**Recommendations for Diagnosis of IAD**

The results of the above study support the recommendations recently set forth by the ACVIM Consensus Statement on IAD in horses. It suggests that a definitive diagnosis of IAD must be made based on cytological evidence of inflammation and/or abnormal pulmonary function, and one cannot necessarily substitute for the other. The discordance between inflammation and airway hyperreactivity is significant; thus, it would appear that the likelihood of underdiagnosis of IAD would be high if only one of the two tests was applied. This obviously presents challenges for field practitioners.

So, how does one go about making a diagnosis of IAD? As a general rule, the more tests you apply, the more information you obtain, and the more likely you will treat the right horses. Despite the apparent lack of agreement between many of the diagnostic tests employed in the present study, many of these tests do allow the practitioner to rule in or rule out other potential problems that might mimic IAD such as upper-airway mechanical issues and septic inflammation.

First, presenting complaint assists with identifying a population at high risk of IAD: although horses of any age and athletic discipline can develop IAD, it is a common cause of poor performance in young racehorses. In our study, horses presenting with cough were older (5.4 ± 3 years) when compared to horses presenting without cough (3.4 ± 1.5 years; p < 0.01). In younger horses, especially those at the track, poor performance is the most common presenting complaint.

Having established that the horse in question is at risk of IAD, the next step is to perform endoscopy, with the intent of ruling out upper-airway abnormalities, establishing the amount of tracheal mucus, and performing a tracheal wash for cytology and culture. While an increased amount of tracheal mucus is not specific to IAD, the present study showed that the triad of chronic cough, increased mucus and abnormal tracheal wash predicted an abnormal BAL in ~ 90% of horses with all three signs; however, using these criteria alone, a significant number of horses with an abnormal BAL would still be missed (data not shown).

The next step includes histamine challenge testing, if available, to determine if the horse is hyperreactive, followed by a BAL, to determine if there is cytological evidence to support a diagnosis of IAD. The results of all of these tests should then be examined together to determine whether inflammation is present, whether or not it is septic inflammation, and where along the respiratory tree, approximately, the inflammation is present.

Unfortunately, in the real world, BAL and PFT are often either impractical, or the necessary equipment is unavailable, and it is acknowledged that we have a way to go before PFT becomes an expected part of every workup. The current “standard of practice” is that horses at risk of IAD should be worked up with a physical examination, endoscopy and BAL. Using this standard, and in the absence of PFT, we will miss treating approximately one third of cases that would benefit from therapy for IAD.

If we want to minimize the impact of failure to treat horses that would benefit from therapy, and in situations where neither BAL nor PFT can be performed, a rational approach may be to initiate treatment in all horses presenting with cough and/or poor performance, once other non-IAD causes of poor performance and cough have been ruled out, realizing that under such circumstances, at least one quarter of the horses would be treated unnecessarily.

Where possible, a definitive diagnosis is always desirable, and practitioners are strongly encouraged to continue to collect as much information as possible (including TTW and BAL cytology, and a PFT if available) prior to initiating treatment. Despite the relative invasiveness of BALs, it is a procedure that is safe and easy to perform, and it should be included as part of the routine diagnostic workup of lower respiratory tract conditions in the horse. Finally, our understanding of IAD is still in early stages of development. In the future, we expect there will be greater availability and access to portable PFT for use in the field, and we may find that hyperreactivity and inflammation are treated as
distinct entities. In the meantime, there is a need to continue to seek answers to the multitude of questions that remain regarding the precise etiopathogenesis of IAD and the most accurate means of arriving at a diagnosis.

REFERENCES
Inflammatory Airway Disease: Therapeutic Options
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INTRODUCTION
The respiratory tree of the stabled horse is bombarded with massive quantities of particulate matter on a daily basis. The combination of poor ventilation, living in a dusty environment, and the feeding of hay all contribute to lower-airway inflammation. Dust is composed of molds, bacteria and their toxins (endotoxin), minerals, insect parts and a range of chemicals. Stable air also may contain high levels of ammonia, a known respiratory irritant. Air pollutants, such as ozone, sulfur dioxide, and nitrous oxide, as well as particulate matter, such as heavy metals, carbon and smoke, contribute to pollution in urban horses and are likely to also play a role in the initiation and maintenance of inflammatory airway disease (IAD). In young horses, viruses have also been incriminated as initiators of inflammation. And, to add insult to injury, stabled horses, who are also in training, sample thousands of gallons of air in just a few minutes of intense exercise, contributing further to the introduction of dust particles into the airways. While the upper airways are relatively efficient at filtering out inhaled particulate matter, the upper respiratory tract is not capable of preventing very small particles (< 5–10 microns in diameter) and gases from reaching the terminal airways. Arrival of these irritants to the small airways initiates a nonspecific immune response that culminates in infiltration of the walls of the bronchioles with inflammatory cells, increased production of mucus, and/or airway hyperreactivity. Inflammation also activates irritant receptors, triggering reflex bronchoconstriction and coughing, which is somewhat protective in that it helps shield the alveoli from irritants. It is the combination of airway-wall inflammation, increased mucus production and the release of a variety of inflammatory mediators leading to edema and exudation that further decreases the functional diameter of the airway. Persistence of this low-grade ‘simmering’ inflammation may result in airway remodeling and permanent impairment of pulmonary function. Rational therapy must therefore be aimed at early recognition, reducing exposure to dust, controlling inflammation, and dilating the airways. This presentation will examine the current treatment options applicable to both IAD and recurrent airway obstruction (RAO), and the pros and cons of each, will report on past experiences, and, where appropriate, will provide updates based on recent literature.

MANAGING THE ENVIRONMENT
Environmental management is not only the most important means of control during all stages of IAD and RAO, but it is also the most difficult aspect of treatment to implement long term. Most horses improve when moved outside. Horses allergic to pollens, molds or plants may develop clinical signs in as short as one hour after exposure to a barn environment. In contrast, clinical remission and normalization of conventional pulmonary function tests take 4–8 weeks once chronically affected horses are turned out to pasture. For affected horses, which must be housed, the primary goal of management should be to minimize exposure to respirable dusts and molds in the air.

As our knowledge of the role of dust in the initiation and maintenance of lower-airway inflammation has improved, so has our interest in identifying management changes that help to reduce, or at least minimize, exposure of the horse’s respiratory system to inhaled irritants. It is important to emphasize that dust is a feature of life, and cannot be eliminated. Further, one can have a state-of-the-art stable that has high standards of hygiene and excellent ventilation, yet still have the occasional horse that coughs. As with humans, individual horses have different tolerance levels to environmental irritants, and, what significantly impacts one horse in the barn may have no significant impact on the others.
Because it is difficult to effectively control the environment of a single horse in a stable setting, the recommendations below should be applied to all horses in the barn as part of best practices in respiratory hygiene. It is understood that not all of the recommendations are possible to implement under all circumstances.

**Suggested Environmental Modifications**
1. Maintain horses outside 24/7 (unless pasture-associated).
2. Avoid stabling horses in barns that are attached to indoor arenas.
3. Wed down aisle before sweeping.
4. Avoid the use of blowers to clean the aisles.
5. Refrain from throwing bedding at the stall wall to separate wet from dry material.
6. Use an alternative type of bedding material (paper, wood pellets; avoid straw).
7. Wet down or steam hay. Do not feed hay in water, as the hay takes on a bitter flavor. Some horses do better on chopped hay (Dengie). Others require complete removal of hay. These horses are fed a complete pelleted feed instead.
8. Turn horses outside while cleaning stalls and for at least two hours afterwards.
9. Consider storing hay somewhere other than in the same barn where the horses are stabled.
10. Assess/improve ventilation.
11. In the case of ‘summer pasture-associated’ allergies, affected horses should be kept off pasture during the late spring and summer months.

**Controlling Inflammation**
When management measures are either not possible or are insufficient to control clinical signs, medical intervention is required. Effective control of lower-airway inflammation involves the use of corticosteroids, for both RAO and IAD, although, the amount of drug necessary to control the inflammation may vary between the two phenotypes. Systemic corticosteroids, such as dexamethasone at 0.02–0.05 mg/kg, IV or IM, or 0.04–0.08 mg/kg PO, once daily, and prednisolone at 1.0–2.0 mg/kg, PO, once or twice daily are usually very effective, and although both drugs have been shown to improve pulmonary function in the presence of continuous antigen exposure, low-dose daily dexamethasone (0.05 mg/kg, orally) has been shown to be more effective than prednisolone (2 mg/kg, orally, once daily) in the treatment of acute exacerbations of RAO (Leclere et al. 2010). It has also recently been demonstrated that oral prednisone has poor efficacy because it is poorly absorbed and the active metabolite, prednisolone, is rarely produced in the horse. In contrast, prednisolone tablets or liquid have excellent bioavailability and should therefore be used in preference to prednisone for the treatment of IAD and RAO. Other steroid preparations, which have been used in the treatment of lower-airway inflammation, include the more potent steroids, triamcinolone acetonide (0.09 mg/kg IM, once) and isoflupredone (0.03 mg/kg IM, SID for several days); however, both drugs have a higher potential for side effects when compared with dexamethasone and prednisolone. Potential side effects include immunosuppression, adrenal suppression and laminitis, and in the case of isoflupredone (because of its mineralocorticoid effects), hypokalemia; thus, serum electrolytes should be monitored with the use of the latter. Regardless of the drug used, all steroids must be tailored to the individual using the lowest dose possible with the least frequency. Steroid therapy must be tapered gradually and never abruptly discontinued. Once-daily treatments should be administered in the morning. Corticosteroids also are contraindicated in horse predisposed to laminitis or those exhibiting other endocrinopathies.

**Bronchodilation**
Bronchodilators are used to open mucus-plugged airways, thereby easing the work of breathing. Generally speaking, they are used prior to exercise in horses with IAD, and to relieve bronchospasm in heavy horses. In cases of severe heaves, bronchodilator challenge tests are sometimes performed in order to assess the reversibility of obstruction. Bronchodilators fall into three categories: the methylxanthine derivatives (aminophylline), anticholinergic drugs (atropine, glycopyrrolate, ipratropium bromide, N-butylscopolammonium bromide) and β₂-adrenergic agonists (clenbuterol, albuterol,
salmeterol, fenoterol, trimetoquinol). While the xanthine derivatives were some of the first to be studied in horses, a recent publication demonstrated that a combination of theophylline and low-dose dexamethasone was ineffective in the treatment of RAO (Cesarini et al. 2006) Although β₂ agonists are more commonly given by aerosol, oral clenbuterol is frequently used in equine practice both for its bronchodilating effects and its ability to stimulate mucociliary transport. As a general rule, bronchodilators should be used in combination with anti-inflammatory drugs, although recent evidence has shown that IV clenbuterol exerts significant beneficial effects on lung function, total cell counts and BAL neutrophil influx when administered prior to hay dust challenge in otherwise untreated RAO-susceptible horses (Laan et al. 2006). Recently, treatment of IAD-affected horses with clenbuterol alone was shown to be less effective as a bronchodilator after treatment for 14 consecutive days (Read et al. 2012), likely due to downregulation or desensitization of receptors. Finally, N-butylscopolammonium bromide, also known as Buscopan™, has recently been shown to be a potent bronchodilator, due to its anticholinergic properties, reaching maximum effect within 10 minutes of IV administration and dissipating by one hour post-administration (Couetil et al. 2012).

Overall, in horses affected with IAD or RAO, bronchodilators should be used on an as-needed basis, or prior to exercise, rather than on a routine basis.

**The Use of Aerosol Medications for the Treatment of IAD and RAO**

Over the years, there has been an increased interest in aerosol therapy as an alternative to systemic administration of corticosteroids and bronchodilators in the treatment of both RAO and IAD. A distinct advantage of inhaled drugs is that they are delivered directly to the tissues of interest.

**Inhaled Anti-inflammatory Drugs**

**Steroids**

The primary advantages of inhaled over systemically administered steroids are their high inherent potency (fluticasone and beclomethasone are more potent than triamcinolone, dexamethasone, and prednisolone!), low spill-over into the systemic circulation, fewer side effects, shorter withdrawal times, and good safety profiles with both short- and long-term use. The primary disadvantages include the high setup and treatment costs, trial-and-error dosing, varying degrees of intolerance to the delivery devices, drug wastage (only approximately 10–20% of total drug delivered reaches the lungs), and finally, the relative paucity of evidence-based clinical trials.

The most commonly used inhaled corticosteroid for both RAO and IAD is fluticasone propionate. Alternatively, beclomethasone (QVAR) can be used; however, because of its lower potency, it must be administered at twice the dose. Fluticasone (FloVent™ HFA 250 μg/puff) administered at a rate of 8–12 puffs/day is generally effective for IAD-affected horses, whereas twice-daily treatment is generally required for heavy horses. Recently it was shown that high-dose fluticasone (6 mg q 12 h) was as effective as dexamethasone (0.1 mg/kg q 24 h) for prevention of acute exacerbations of RAO; however, high-dose fluticasone was not as effective for treatment of RAO exacerbations (Robinson et al. 2009). This, coupled with the higher cost of the newer metered-dose inhalers (MDI) that are HFA propelled, suggests that initial control of inflammation in severely affected horses may best be achieved with dexamethasone or prednisolone, after which time, a switch to inhalants may be more appropriate. As previously mentioned, fluticasone has been shown to be safe when used long term. Treatment of RAO-affected horses with inhaled fluticasone at therapeutic dosages for 11 months had no significant effect on innate and adaptive immune parameters, suggesting that prolonged administration would not compromise the systemic immune response to pathogens or vaccination in adult horses (Dauvillier et al. 2011).

Overall, because it has been demonstrated that aerosolized steroids are more effective at preventing, rather than stopping heaves, it is recommended that inhaled steroids be used for long-term control of IAD or heaves, once these conditions are clearly in remission. Because of the higher cost of these medications, accurate diagnosis is warranted.
**Mast Cell Inhibitors**

The use of mast cell stabilizers, such as sodium nedocromil (Tilade™) and disodium cromoglycate (Intal™), have been less well studied, and what literature is available, the results are conflicting. Tilade™ is available as a 2 mg/puff concentration, while Intal™ is available as a 1 mg/puff concentration. Both drugs are administered at the same rate of 8–12 puffs twice daily. Long-term use of these drugs appears to be required before a therapeutic effect is observed, thus, compliance by owners may be observed (personal communication with Dr Melissa Mazan, Tufts University).

**Inhaled Bronchodilators**

As with inhaled steroids, the ability to rapidly deliver bronchodilators directly to the tissues of interest makes the inhaled products very attractive. Compared to the steroid preparations, the bronchodilators tend to be less expensive, and pretreatment of horses with aerosolized bronchodilators facilitates deposition of inhaled steroids. The majority of products that have been studied are the β2 agonists, including albuterol, salmeterol, fenoterol and trimetoquinol. Of the β2 agonists, albuterol (Salbutamol™, 100 μg/puff), administered at a dose of approximately 1 μg/kg (5 puffs) is short-acting. A dose of 360 μg was associated with an onset of action of 5 minutes, and a duration of action of 30 minutes to three hours (Derksen et al. 1999). Ipratropium bromide (Atrovent™, 18 μg/puff) is an anticholinergic drug with slightly longer duration of action (6–8 hours). This drug is administered by inhalation, either by MDI (0.36–0.72 μg/kg q 8–12 h) or nebulizer (2–3 μg/kg). A combination of albuterol and ipratropium (Combivent; 90/18 μg albuterol/ipratropium, respectively/puff) is also available when a slightly longer duration of action is needed. Current dosage recommendations for Combivent are 5–8 puffs q 12 h, depending upon the severity of obstruction. Finally, salmeterol (Serevent™, 50 μg/puff; 5–10 puffs once or twice daily) also provides long-lasting relief. At a dose of 210 μg, maximal bronchodilation occurs within 30–60 minutes and duration of action is approximately 6 hours (Henriksen, Rush, 2001). The use of this drug should be reserved for RAO-affected horses that require 24/7 control.

**Delivery Devices**

Aerosolized medications can be delivered via metered-dose inhalers (“puffers”) or by nebulization, using either an ultrasonic or jet nebulizer. Studies over the years have shown that delivery of aerosol by MDI is superior to aerosol delivery by nebulization, which likely reflects the smaller particle sizes that MDIs are capable of generating. Delivery devices for MDIs have come in many shapes and sizes over the years. The Equine Aeromask™ (Trudell), one of the earliest devices, was later followed by the Equine Haler™ (Equine Health Care Systems), and, most recently, by the AeroHippus™ (Trudell). The basic premise for all of these devices is that the medications are released under pressure, in the form of an aerosol cloud, into a holding chamber (known as a ‘spacer’), which ‘poises’ the drug for inspiration at the next available breath. A one-way valve, which is attached to the holding chamber, permits inhalation without exhalation of the breath into the chamber. High sensitivity of this valve is required to permit rapid emptying of the holding chamber, even under conditions of low flows typical for tidal breathing in horses. Of the currently available products, valve sensitivity is greatest for the AeroHippus™. The amount of deadspace is also an important feature in terms of efficiency of delivery, with larger amounts of deadspace being less desirable. The amount of deadspace is greatest for the Aeromask™, and least for the AeroHippus™. In these respects, the AeroHippus™ appears to be perfectly designed. Data collected by Dr Andy Hoffman at Tuft’s University support the notion that the Equine AeroHippus™ is the most efficient, cost-effective device available on the market. When delivery of fluticasone diproprionate was studied in vivo, lung deposition was found to be greatest for the AeroHippus™ (18%), 9.5% for the Equine Haler™ and 6–7% for the Aeromask™. Not only is the AeroHippus™ now the delivery device of choice, but it is also the least expensive ($80.00–$100.00 compared to $250.00–$300.00 for the Aeromask). An additional advantage of the AeroHippus™ is the ability to load the ‘puff’ off the horse and then place it onto the horse’s nose (within 5–10 sec) with little decay of efficiency. This feature is useful for horses which do not tolerate the sound associated with activating the MDI (Hoffman, 2010).
SAMPLE PROTOCOLS FOR TREATMENT OF IAD (AND/OR RAO)
(Reprinted with permission of Dr Andy Hoffman, Head, Lung Function Testing Laboratory, Hospital for Large Animals, Tuft’s University)

Bronchodilators
While it is difficult to make specific recommendations for the use of bronchodilators, horses with severe exercise intolerance, severe coughing during exercise, or poor recovery following exercise (“blowing”) may benefit from their use. In general, bronchodilators are used only during the first two weeks of treatment, and then on an as-needed basis thereafter. Inhaled bronchodilators should be administered 10–15 minutes prior to inhaled steroids. If needed, they may be administered 30 minutes before exercise.

The following bronchodilators are listed in order of Dr Hoffman’s preference:
- **Albuterol**: 5 puffs = 500 μg (Salbutamol™, 100 μg/puff) (1 dose = 1 puff)
- **Albuterol/ipratropium combination**: 5 puffs = 450 μg albuterol + 90 μg ipratropium (Combivent™, 90 μg albuterol/18 μg ipratropium per puff)
- **Ipratropium bromide**: 5 puffs = 90 μg (Atrovent™, 18 μg/puff)

Antiinflammatory Treatments
- For mild IAD without increased neutrophils; mild exercise intolerance, occasional cough, near-normal baseline lung function, airway hyperreactivity, and increased mast cells (≥ 2%) in BALF
  - Nedocromil sodium: 8–12 puffs = 16–24 mg twice daily (Tilade™, 2 mg/puff)
  - Disodium chromoglycate: 8–12 puffs = 8–12 mg twice daily (Intal™, 1 mg/puff)

  Note: The use of mast-cell inhibitors requires long-term use before positive effects are noted; thus, compliance by owners is often poor.
- For mild IAD with increased proportions of neutrophils (≥ 5%; ≤ 15%)
  - Weeks 1 and 2: Fluticasone, 8–12 puffs (use 8 puffs for 800-lb horse & 12 puffs for 1500-lb horse) once daily in the morning (Flovent™, 250 μg/puff)
  - Weeks 3 and thereafter: Fluticasone, 8–12 puffs every other day in the morning, or continue daily if signs recur

  Note: Use a short-acting bronchodilator 10–15 minutes prior to steroid administration.
- For moderate to severe IAD; moderate to severe exercise intolerance, persistent cough, abnormal baseline lung function, positive response to a bronchodilator, and increased neutrophils (≥ 15%) in BALF
  - Week 1: Prednisolone, 0.8 mg/kg, orally, twice daily
  - Week 2: Prednisolone, 0.6 mg/kg, orally, twice daily
  - Week 3: Prednisolone, 0.4 mg/kg, orally, twice daily
  - Fluticasone, 8–12 puffs once daily in the morning
  - Week 4: Prednisolone, 0.4 mg/kg, orally, once daily in the morning
  - Fluticasone, 8–12 puffs, once daily in the morning
  - After week 4: Fluticasone, 8–12 puffs, once daily, every other day, in the morning

  Note: Use bronchodilators to control cough and bronchospasm on an as-needed basis only.

Treatment Costs
- Flovent HFA 250 μg/puff; 120 puffs/MDI: $89.60/MDI
- Salbutamol HFA 100 μg/puff; 200 puffs/MDI: $8.50/MDI
- Prednisolone
  - Suspension, 20 mg/ml (Trutina™; compounded; for oral use); 500 ml: $40.00
  - Tablets, 5 mg each: $0.05/tab
  - Injectable, 50 mg/ml; 30-ml vial: $22.00/vial
- **Dexamethasone**
  - Powder, 10 mg/packet: $1.25/packet
  - Tablets, 4 mg each: $0.37/tab
  - Injectable:
    - 2 mg/ml, 100 ml vial: $10.00/vial
    - 5 mg/ml, 50 ml vial: $10.00/vial

**ENDNOTE**
a. www.haygain.com

**REFERENCES**


There is at least the perception that the care of geriatric horses (often defined as horses older than 15 or 20 years) plays an increasing role in veterinary practice. Longevity of horses may be increasing, which may in part be attributable to improved general management, preventive measures and veterinary care. Horse owners may also increasingly regard their animals as pets and companions, and may be more willing to invest in the care and maintenance of geriatric horses. Of course, many “geriatric” horses (if the term is defined solely based on chronological age) maintain their athletic and work potential and some are still actively competing in equine events.

A horse may be assessed as being “geriatric” based on its chronological age or because it exhibits physical characteristics of old age, or has medical or management concerns related to aging. Definitions based on chronological age should take into account that ponies tend to have longer life spans than horses. Physical characteristics of aging as they pertain to horses include graying of the face and muzzle, development of a swayback, drooping lips, appearance of marked grooves above the eye and increased tooth and sometimes hoof wear. Based on one study, many horse owners perceive distinct signs of aging in their horses at about 22 years of age. As mentioned earlier, many scientific studies include horses older than 15 or 20 years as geriatric and often compare these to one or more younger populations. The 1998 National Animal Health Monitoring System (NAHMS) study in the United States revealed that 92.5% of the equine population was comprised of horses less than 20 years of age, with 7.5% being older than 20 years old. These numbers didn’t change much by 2005 and 7.6% of horses at that time were reported as being greater than 20 years of age, with 0.7% being older than 30 years. I am not aware of similarly detailed information pertaining to Canada; however, the 2010 Canadian Horse Industry Profile Study “Horses in Canada 2010” reported that of an estimated 963,500 horses in Canada at the time, 22.8% (219,884) were classified as “young horses not yet in use,” while “companionship or full retirement” was reported as the primary use for 3.5% or 33,723 horses. Interestingly, 56% of Canadian horse owners also indicated that they intended to own their horses for the horses’ lifetime.

Much information is available about processes of aging as they pertain to the health of humans and, to an extent, animals. Genetic make-up determines longevity; however, this is influenced considerably by environmental factors and the occurrence of disease. As in other species, therefore, veterinarians may be presented with very healthy and active old horses or, conversely, those that require special management or suffer from diseases at a much younger chronological age. The old adage “Age itself is not a disease” certainly holds true for horses, and while veterinarians must recognize the potential impact of age on disease processes and overall health, assessment and client counseling must always be tailored to individual situations. Specific considerations with regard to feeding and dental care for geriatric horses are covered in a separate lecture. Veterinarians must also recognize that horse owners may display a variety of attitudes towards aging and the “usefulness” of an aged horse, and may be faced with communication challenges on both ends of the spectrum.

Comparatively little information is available about the impact of age on the occurrence of specific diseases in horses. Some conditions that are frequently discussed in the context of the geriatric horse include arthritis, laminitis, certain causes of colic, recurrent airway obstruction or heaves, dental disease and sinusitis, weight loss, degenerative valvular disease, uveitis and neoplasia. The incidence of pituitary pars intermedia dysfunction (PPID or Equine Cushing’s disease) is strongly age-related and this condition as well as thyroid disorders are discussed in more depth in a separate lecture. Reproductive considerations for aging horses are not covered here aside from mentioning that some conditions, including postpartum uterine artery rupture, occur more frequently in aged mares.
The knowledge that certain diseases occur with increasing frequency in aged horses should be incorporated in the diagnostic approach and treatment. Importantly, certain conditions such as PPID, even if not clinically obvious, may affect the response to treatment of other problems (such as infections), and a comprehensive assessment of the aged patient is encouraged to provide the best possible treatment outcomes and owner satisfaction. Routine “wellness checks” in geriatric horses should place greater emphasis on the assessment for certain age-related conditions, as it is likely that early recognition and appropriate treatment prolong the healthy lifespan of horses. While more frequent dental examination is often recommended for geriatric horses, I am unaware of a general recommendation to include specific diagnostic tests (such as blood work) in the routine wellness examination of aging horses, and options likely have to be discussed on the basis of the individual situation.

There is some evidence that the immune response to vaccination is reduced in aged horses, but this may only apply to those with poor nutritional status, concurrent diseases or severe parasitism. I am not aware of any general recommendations to adjust vaccination schedules for aged horses although the American Association of Equine Practitioners’ (AAEP) vaccination guidelines mention geriatric horses in the context of West Nile virus vaccination. Geriatric horses should continue to receive core vaccinations, and risk assessment for risk-based vaccinations should take into account the potential exposure of in-contact horses on the farm, even if the geriatric horse itself no longer travels. Increased susceptibility of geriatric horses, especially those suffering from PPID, to parasitism is frequently suggested and an excellent parasite-control program may serve to maximize digestive efficiency and overall health in these patients.

The age of a patient often becomes an issue for discussion when surgical intervention is needed, for example in a horse requiring colic surgery. Many horse owners (and veterinarians) assume that anesthetic risk and the risk of complications are increased considerably in aged horses, and may elect not to pursue treatment. Unfortunately, information specific to horses is lacking, and one has to rely on extrapolation from other species and reports of treatment outcomes. Without questioning the usefulness of the latter type of information, one has to be cognizant of the impact of those cases that were euthanized without even attempting treatment. One study did not identify a consistent effect of age on outcome of colic surgery in horses. Most experienced anesthesiologists appear to be of the opinion that age in and of itself only slightly increases anesthetic risk in horses but that comprehensive preanesthetic examination to uncover medical problems that may affect anesthetic risk, cautious use of anesthetic drugs and careful monitoring of patient parameters during anesthesia are of great importance. Cardiovascular disease, respiratory disease, orthopedic disease and endocrine or metabolic disease, as well as medications used to control these conditions, may all impact on the horse’s ability to withstand general anesthesia and recover successfully from a procedure. In our practice, it is not uncommon, for example, to uncover previously unknown cardiac murmurs or arrhythmias during the preanesthetic examination. If an elective procedure is planned, further investigation and diagnostic testing should be recommended to horse owners but this may not be an option in emergency situations. Comprehensive baseline blood work to assess liver and renal function may be more important for geriatric horses, whereas a minimum panel is often deemed sufficient for younger horses. There is also limited information about the pharmacokinetics of sedatives and anesthetic drugs in horses, but while no specific drug regimen is recommended for aged horses, lower doses may be needed. It may be a safe approach to assume increased sensitivity to anesthetic drugs in aged horses and to tailor dosages as needed based on monitoring. Supportive care to maintain body temperature, tissue perfusion and oxygenation may be of increased importance in geriatric horses, and should extend into the recovery period. Administration of analgesics to geriatric horses suffering from chronic pain (such as arthritic pain) may facilitate smooth recovery from anesthesia, even if pain control is not of major importance for the primary condition the horse is undergoing general anesthesia for. Some older horses appear to behave “smarter” during the recovery period; however, this observation can probably not be generalized for all horses and careful monitoring of the individual is indicated in all age groups. Facilitated recovery may be necessary on older horses that have trouble getting to their feet, especially if they have undergone prolonged anesthesia or are weakened from their primary condition.
According to the 2005 NAHMS study, old age accounted for the highest number of deaths in horses older than 6 months. Horse owners may elect to euthanize aged horses based on their decreased usefulness, presence of disease or a perception that the animal no longer has a good quality of life. In our practice, we are asked to “electively” euthanize several aged horses at the beginning of each winter, and owners should be counseled on the importance of addressing nutritional issues well ahead of the winter time and the necessity of providing extra shelter or indoor housing for horses in marginal body condition. Quality of life assessment tools may be helpful for horse owners having difficulty with the decision to euthanize their horse, and the AAEP euthanasia guidelines can also be helpful when counseling horse owners with regard to euthanasia. For example, the guidelines state the euthanasia may be indicated if a horse suffers continuous or unmanageable pain from a condition that is chronic and incurable, or if it requires continuous pain medication or stall confinement for the rest of its life.

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Specific Management Issues of the Geriatric Horse
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Maintenance of nutritional balance and adequate body condition, maintenance of dental health or correction of dental abnormalities, and management of chronic pain are issues commonly faced by owners of geriatric horses and, therefore, their veterinarians. This lecture attempts to outline current knowledge and recommendations as the basis for discussion. It should be stated from the beginning that research specific to geriatric horses is overall lacking and that some recommendations are based on experience and common understanding rather than scientific fact. With regard to nutrition, this lecture will focus on weight loss rather than obesity, both of which can be problems in geriatric horses.

The new Code of Practice for the Care and Handling of Equines (2013) requires that “geriatric horses [...] receive a diet that is adequate for maintaining health and vigour.” It further requires that corrective action be taken for all horses with a body condition score of 3 or lower, and that veterinary advice be obtained for geriatric equines that are emaciated. Emaciation in the context of the Code of Practice is defined as a body condition score of 2 or less on a 9 point scale for horses, or a body condition score of 1 on a 5-point scale for donkeys and mules. For the purposes of this lecture, the body condition scoring system according to Henneke will be used. Body condition scoring in addition to weighing or weight estimation (e.g., by using a weight tape) is a useful tool for both horse owners and veterinarians, and should be included as part of the medical record for all horses. Horse owners may need to be trained in the correct use of body condition scoring. Some physical changes associated with aging (e.g., loss of muscle mass over the top line and sway-backed appearance) may affect the visual aspects of body condition scoring and need to be taken into account.

According to the National Research Council’s (NRC) latest “Nutrient Requirements of Horses,” the extent to which maintenance energy requirements and protein requirements change in aged horses is poorly understood, while investigation of micronutrient requirements has suggested decreased apparent digestibility of phosphorus. Maintenance energy requirements decrease by 15–20% in aged humans and dogs, which is likely attributable to decreased physical activity and a decrease in fat-free mass. In some individuals, the decrease in maintenance energy requirements due to age may be offset by an increased requirement arising from medical conditions. Even if maintenance requirements do not change in aged horses, digestibility of feed may decrease due to changes in mastication, parasitism and, possibly, inherent changes in large intestinal function. It has been suggested that enhancing provision of calories from non-carbohydrate sources in aged horses may be beneficial, especially in those horses suffering from pituitary pars intermedia dysfunction.

In many cases, the ability to maintain adequate body condition and weight in an aged horse is impacted significantly by the type of feed available to the horse. While roughage-only diets are generally preferable in horses and have many advantages with regard to digestive health and behavior, aged horses with poor dentition may no longer be able to sustain themselves on a hay diet. Investigation of the type of roughage offered, its quality and nutrient value, as well as the horse’s eating behavior and dental health, is of crucial importance when addressing weight loss in an aged horse, and will often reveal that a horse has access to an adequate feed supply but is simply not able to eat enough of it. In my experience, many horse owners have difficulty understanding (or accepting) that their aged horse needs a special diet for maintenance, and I try to address this issue in some detail before embarking on extensive diagnostic testing for a horse presented for weight loss. Some owners will need to commit to the provision of a special diet (and the associated expense) for the rest of their horse’s life. Competition for feed (e.g., due to changes in a herd’s hierarchy as some of its members age), and chronic pain hindering movement over distances to obtain feed and water or making eating off the ground uncomfortable, also require consideration. Careful grouping of aged horses with compatible companions with similar needs can help maintain both nutritional adequacy and meet social needs.
My personal preference is to recommend supplementation with a complete feed designed for “senior” horses while maintaining access to hay or pasture for behavioral reasons. Complete senior feeds provide a balanced ration that is specifically formulated to be palatable, easy to chew and easily digestible. It is important to counsel owners on the meaning of a “complete” feed (i.e., one that is formulated to contain sufficient roughage to meet nutrient requirements if fed exclusively). Most senior complete feeds will be formulated to have an increased protein content (usually 14%) and fat content (usually 4.5–8%). The amount fed will very much depend on the individual horse and the remaining ability to graze or eat hay; some horses do quite well if supplemented with 4–5 lb per day while others need to have all their nutritional needs met by the complete feed. Given adequate pasture conditions, horses may also require less supplemental feed in the summer than winter, where additional cold stress becomes important. While feeding instructions provided by the supplier can be a useful guideline, monitoring of body condition is important to tailor the ration to the individual horse. Remember that the vitamin/mineral needs may only be met by a complete feed if it is fed as an exclusive source of nutrition. I typically caution owners about adding additional fat supplements if the horse is fed a substantial amount of a high-fat, complete pellet; however, other authors state that the addition of fat to a typical senior diet is safe.7,8

Alternatives to complete senior diets include provision of higher-quality forages such as alfalfa hay, easier to chew roughage types (e.g., silage, haylage, chopped hay, soaked alfalfa cubes) or supplements in addition to a roughage diet. In our practice, many owners feed beet pulp to their horses and this seems to be a good supplemental source of energy as long as it is prepared adequately and palatable to the horse and the horse maintains an adequate forage intake. Addition of fat in the form of stabilized rice bran or vegetable oils (approximately 1–2 cups/day) may also help senior horses maintain weight. Vitamin E supplementation (100 IU/100 ml of added oil) is often recommended when additional fat is provided,7 to reduce the potential for oxidant damage. The calcium-phosphorus balance may need to be considered and adjusted depending on the types of feed available, especially in horses with renal disease. I usually caution against supplementation with large amounts of carbohydrate-rich, sweet feeds or grains, especially as many aged horses will have PPID and some degree of insulin resistance.

Water intake must be maintained in aged horses and may require special consideration. Horses with dental disease may have dental pain that can be mitigated by providing warmed water. A reduction in thirst sensation is recognized in aged human beings but has not been described in horses. On the other hand, polydipsia and polyuria are described in some horses with PPID. Water intake and hydration status should be monitored carefully in aged horses, especially those with reduced feed intake. Provision of mashes or soaked feeds (beet pulp, alfalfa cubes) may help to increase water intake; however, in my experience, some horses refuse soaked feeds in the long run and it is not a viable option for horses housed outside in the winter. Addition of salt to increase water intake must be done with caution and requires close monitoring. Maintenance of water and electrolyte intake may be of particular importance in older horses with continued athletic activity as age-related reduced aerobic capacity may result in increased sweating.9

Dental care for the older horse can present unique challenges but also offers an opportunity to significantly improve a horse’s overall health status. Maintaining the ability to eat sufficient amounts of food is crucial in order to maintain body condition, and chronic pain arising from dental diseases can be assumed to impact a horse’s overall comfort and quality of life. In my opinion, a dental examination is indicated in all geriatric horses presented for routine examination or medical concerns, and especially in those presented for weight loss, choke, or colic. There is an increased recognition of the importance of regular dental care in equine medicine as a whole; however, it is still not unusual to be presented with an aged horse that has never received a dental examination until problems become obvious. Unfortunately, dental disease in these situations is often advanced and requires more and longer treatment to achieve resolution. In some cases, one may be left with “palliative” care and management changes that address nutritional needs and pain control. Given the subjective impression that the population of aged horses is increasing and that geriatric horses represent an increasing proportion of veterinary patients, more research will hopefully be done on the specific issues affecting geriatric horses and the best management methods to maintain dental health for as long as possible.
Major dental problems encountered by geriatric horses are abnormal dental growth and malalignment due to irregular tooth wear, tooth loss, periodontal disease and tooth root infection, sometimes with accompanying sinusitis. Tooth fractures, neoplasia, and excessive wear due to behavioral abnormalities such as cribbing may also be encountered. Feeding strategies and feed quality can affect dental wear, which needs to be considered for those horses put on special diets (such as complete feeds) that reduce the normal grinding motion. Additional disease processes such as PPID may also increase the risk of tooth infection and sinusitis, or impact the ability to recover from these problems.

The principles of dental examination are the same in aged as in younger horses; however, several additional considerations apply. Thorough physical examination should precede sedation and the owner should be informed of any conditions (e.g., cardiovascular, respiratory, neurologic or orthopedic) that may impact the safety of sedation in an individual horse. Many veterinarians will approach sedation more cautiously in older than younger horses and some avoid certain drugs due to a perception that they are tolerated less well. Conversely, many veterinarians also comment on the apparent difficulty to achieve adequate sedation in aged horses. Pain should always be considered as a potential reason for a horse’s resistance to dental examination, and local anesthesia or, with elective procedures, preemptive administration of pain medication should be considered. It may be more difficult to maintain a speculum comfortably in a horse with very worn incisors, and padded speculum bars instead of bite plates have been recommended for that purpose.10 Veterinarians should also consider using radiographs (if available) for full assessment of a geriatric mouth prior to corrective dentistry. Based on my personal experience, dental radiography is not suited to replace complete and thorough oral examination, but can provide useful additional information in horses with complex problems.

Care has to be taken when approaching dental correction in aged horses. The emphasis should be on maintaining chewing ability and contact of the occlusal surfaces, and on removing or at least reducing any pain the horse may be experiencing. Due to the hypsodont nature of the equine teeth, the length of the reserve crown is finite and overly aggressive dental correction may result in a situation where the horse is no longer able to chew feed properly, without chance of improvement over time. Wave mouths should be corrected gradually and often require several sessions in approximately 3-month intervals.10 Complete correction of wave mouths is often not possible and maintaining a “functional” and pain-free mouth should be the primary goal. Dental examination in 6-month (rather than the usual 12-month) intervals is frequently recommended for aged horses with dental abnormalities, and may be necessary to stay ahead of a recurrence of wave mouth formation or other malocclusions. Smooth mouth or “cupped out” teeth represent an end-stage situation for aged horses’ teeth and should be addressed very carefully, limiting any correction to the removal of sharp edges that may cause pain. Dietary adjustments are frequently necessary for these horses and appropriate counseling of owners is needed to maintain the welfare of the horse.

Periodontal disease and development of diastemata are common problems in aged horses and are related to changes in the size and shape of the apical portion of the teeth. Periodontal disease should be taken seriously as it causes significant pain and also increases the likelihood of tooth root infection and tooth loss.10 Teeth should be carefully checked to detect any loose teeth that may need to be removed; sites of missing teeth should be investigated carefully to detect involvement of associated structures. Luckily, intraoral removal of teeth, especially loose teeth, is often easier in aged than in younger horses. Any removed teeth and the corresponding sockets should be evaluated carefully to ensure complete removal of the entire tooth; radiographs may be indicated to detect any tooth fragments that may have been left behind.

Chronic pain, most notably from arthritis or laminitis, or due to severe dental disease, can become a management challenge in some geriatric horses. Aside from the obvious discomfort of chronic pain, horses in pain may not lie down to rest and, hence, become sleep-deprived, or may be unable to eat sufficient amounts of feed as outlined earlier. The options for pain control are the same as for younger horses and the challenge may lie more in the counseling of owners regarding the welfare implications of chronic pain. The American Association of Equine Practitioners (AAEP) euthanasia guidelines11 state that euthanasia may be indicated if a horse suffers continuous or unmanageable pain from a condition that is chronic and incurable, or if it requires continuous pain medication or stall confinement for the rest of its
life. To date, there is limited information about the effectiveness and safety of nutraceuticals and alternative pain management methods in horses, but these may be considered in individual horses as an adjunct to conventional therapies. In non-athletic horses that can be maintained as “pasture pets,” options for more permanent pain control such as surgical joint fusion may also be considered to reduce long-term administration of medications.

Aged horses are often considered to be more stoic than younger horses, and this may need to be taken into account when evaluating geriatric horses for painful conditions such as colic. Evaluation of other indices of pain (heart rate, respiratory rate, level of depression) should be given more emphasis in these patients and the absence of overt signs of colic alone should probably not be interpreted as evidence that surgical intervention is not needed.

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Endocrine Diseases of Geriatric Horses
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Pars pituitary intermedia dysfunction (PPID), also known as Equine Cushing’s Disease, is a common condition of geriatric horses and ponies. PPID is thought to arise from oxidative damage resulting in dopaminergic neurodegeneration in the hypothalamus;1 the subsequent reduced inhibitory input to the pituitary then results in hyperplasia and increased production and release of pituitary pro-opiomelanocortin (POMC)-derived hormones2. Important POMC-derived metabolites include adrenocorticotropin (ACTH), the melanocyte-stimulating hormones (MSHs), β-endorphin and corticotropin-like intermediate lobe peptide (CLIP). Clinical signs of PPID are likely related to increased concentrations of these POMC-derived peptides; however, the exact mechanisms are incompletely understood.

Clinical signs of PPID include hirsutism (a long, shaggy hair coat that sheds poorly), lethargy, muscle wasting and weight loss, abnormal fat distribution (such as the development of pronounced supraorbital fat pads or a cresty neck), polydipsia and polyuria. PPID is strongly associated with the development of laminitis, which may in part be explained by insulin resistance.3 Infertility can be seen in animals used for breeding. Recurrent infections may be the result of increased adrenal stimulation and overproduction of cortisol; however, hypercortisolemia is an inconsistent finding in horses with PPID. Clinical pathologic findings are variable but may include mild anemia, stress leukogram (neutrophilia, lymphopenia, eosinopenia), insulin resistance (normo- or hyperglycemia in the face of hyperinsulinemia), hyperlipemia, increased liver enzymes and glucosuria. Persistent hyperglycemia indicates diabetes mellitus.

While the diagnosis of PPID in advanced cases is often made based on clinical signs alone, several diagnostic testing strategies have been devised for horses in earlier stages of the disease or where confirmation of the disease process by laboratory testing is desired. Diagnostic tests include both “baseline tests” and challenge tests and the sensitivity, specificity and predictive values of the different proposed tests are an area of intense investigation. Seasonal, regional, breed- and age-specific influences on diagnostic test results also continue to be uncovered4 and results of individual studies always need to be interpreted in the context of the population tested and the specific circumstances in which they were performed. Proper sample collection, storage and shipment are also crucial in order to obtain reliable results, and laboratory-specific reference ranges should be used for interpretation.

Resting plasma concentrations of ACTH are frequently used for diagnosis of PPID but are influenced by seasonal variation, age and environmental factors. Season-dependent reference ranges should be considered to improve diagnostic performance.5 Resting insulin concentrations may be increased in those horses suffering from insulin resistance, and are often measured concurrently with ACTH. Resting cortisol concentrations have not proven to be diagnostically useful.

Several challenge tests have been proposed for the diagnosis of PPID. The overnight dexamethasone suppression test6 has shown good diagnostic performance but is often avoided because of concerns over administering dexamethasone to horses at risk for (or already suffering from) laminitis. For the overnight dexamethasone suppression test, dexamethasone (0.04 mg/kg IM) is administered between 4 and 6 pm following collection of a pre-test serum sample. A second serum sample is collected 19h later, at approximately noon the next day. Serum cortisol concentration is measured to assess the horse’s ability to “suppress” cortisol production; a post-dexamethasone concentration of less than 1 μg/dl (approximately 27.6 nmol/L) is expected in healthy horses. The dexamethasone suppression test is influenced by season with false-positive tests occurring more frequently in the fall months. Testing in the fall should therefore be avoided, or the test (if positive for PPID) should be repeated a few months later for confirmation.
Several variations of and alternatives to the dexamethasone suppression test have been proposed and include the TRH-stimulation test,\textsuperscript{7} combined dexamethasone suppression-TRH stimulation test\textsuperscript{8} and oral domperidone challenge test\textsuperscript{9}.

The most commonly used treatment of PPID is the administration of pergolide mesylate, a dopamine D\textsubscript{2} receptor agonist. A commercially available pergolide product licensed for horses (Prascend\textsuperscript{®}, Boehringer Ingelheim Vetmedica, Inc.) was recently approved in the US and Canada and will enable veterinarians and horse owners to avoid compounded products. Dosage recommendations for pergolide vary somewhat but 2-4 µg/kg/day, in one or divided into 2 doses, are commonly recommended. Inappetence is one of the main side effects of pergolide treatment and can often be successfully addressed by splitting the total daily dose in 2 or reducing the dose temporarily.

The serotonin antagonist cyproheptadine (0.25 mg/kg PO twice daily) was shown to be useful in some patients with PPID and is sometimes recommended as an additional treatment to pergolide. Trilostane, which inhibits corticosteroid synthesis in the adrenal cortex, may also be useful in some patients (1 mg/kg once daily). Supportive care, especially to manage laminitis, infections and hyperthermia (due to hirsutism) is an important aspect of management for horses with PPID and can significantly improve the quality of life of affected patients. Owners of severely affected horses or those in the later stages of the disease should be counseled about reasonable expectations for treatment success; for example, existing laminitis and structural changes to the foot will not be reversed by treatment but worsening of the condition may be prevented. Horse owners also need to realize that treatment will be necessary for the rest of the horse’s life and that intermittent reassessment and dosage adjustment may be necessary. Excellent management of horses with PPID with regard to feeding management, deworming, dental and hoof care should be encouraged to maximize the horse’s quality of life. Feeding strategies need to be evaluated in the context of insulin resistance in some patients.

Clinically relevant thyroid disease in adult horses is generally considered uncommon; however, enlargement of one or both thyroid glands is sometimes the presenting complaint in aged horses. The evidence for naturally occurring hypothyroidism in adult horses is scarce, although decreased thyroid hormone concentrations have been reported in the context of infertility or subfertility, poor performance, anhidrosis, PPID and laminitis.\textsuperscript{10} Interpretation of thyroid hormone concentrations must take into account that these concentrations can be affected by many physiologic conditions and some medications. Clinical signs of hypothyroidism in experimentally thyroidectomized horses include low-grade anemia, decreased body temperature, bradycardia and bradypnea, lethargy, rear limb edema, coarse hair coat, inappetence, decreased libido and infertility.\textsuperscript{10} Diagnostic testing should reveal low thyroid hormone concentrations along with increased thyroid-releasing hormone (TRH) and thyroid-stimulating hormone (TSH) concentrations. Many horses, previously considered to be hypothyroid based on their phenotype, are now usually classified as having equine metabolic syndrome (EMS) and although thyroid hormone supplementation is sometimes transiently employed in the management of these horses, they should not be considered truly hypothyroid.

Thyroid tumors, which occur most commonly in aged horses,\textsuperscript{11} may represent thyroid adenomas or adenocarcinomas. Careful physical examination can usually differentiate thyroid enlargement from other similar-appearing conditions such as submandibular lymph node enlargement, guttural pouch distension or enlargement of the parotid gland. The thyroid glands in horses are located in the dorsolateral aspect of the 3rd to 6th tracheal ring\textsuperscript{10}, where they can be palpated in most normal horses. Enlarged thyroid glands are often quite prominently visible in the throat latch region. True hyperthyroidism has been rarely reported in horses,\textsuperscript{10} but, if present, will present with classical clinical signs including excitability, tremors, tachycardia, sweating and weight loss, sometimes despite polyphagia.\textsuperscript{12}

Most enlarged thyroid glands in aged horses constitute benign, non-functional thyroid adenomas that may occur unilaterally or bilaterally. Aside from concerns over the origin and nature of the “swelling,” horse owners may seek resolution of these enlargements for cosmetic reasons. Hyperthyroidism associated with a thyroid adenoma has been reported;\textsuperscript{13} adenocarcinomas may result in hypothyroidism, euthyroidism or hyperthyroidism.\textsuperscript{10} Surgical removal may be indicated in some cases; however, horse owners should be counseled regarding the invasiveness and potential complications of thyroidectomy in those horses that have non-functional benign tumors.
REFERENCES
This presentation reviews our current understanding of the common gastrointestinal nematode parasites of sheep and goats, and their control - including diagnosis, treatment, and issues regarding anthelmintic resistance.

**Important Gastrointestinal Nematode Parasites (GIN)**

This section contains a description of the most commonly found and important gastrointestinal nematode (GIN) parasites of sheep and goats found in Canada. The most common and the most pathogenic nematode parasites of sheep and goats are: *Teladorsagia circumcincta*, *Trichostrongylus* spp., and *Haemonchus contortus*. *Nematodirus battus*, which is also highly pathogenic, is less commonly implicated.

**Haemonchus contortus**

Also called the barber-pole worm. The worms are easily visible to the naked eye and the female oviduct is visible as a white stripe around the red, blood-filled intestine, giving a barber-pole appearance. It is found in the abomasum.

This parasite also infects llamas, deer, and occasionally cattle. It does not survive well over winter on pasture in temperate continental climates. However, it is a very prolific egg producer - each female worm can produce several thousand eggs every day - enabling it to rapidly increase the pasture contamination to severe levels by midsummer. Pasture contamination comes from two sources: infected lambs and kids are the worst, but early contamination comes from ewes and does where the parasite has overwintered in the ewe and starts to produce large numbers of eggs in the spring at lambing. The eggs can develop to the infective L3 stage in as little as 5 days, but require fairly warm temperatures to do so. Egg development may be delayed up to 2 months if the weather is cool. The L3 larvae can survive for months on pasture under moist conditions.

In central Canada, severe disease outbreaks usually occur mid-July to August in youngstock as well as adults on pasture, but the exact time depends on the air temperature and moisture. Outbreaks occur earlier if the summer is hot and wet, and later in cool summers. Finally, many of the ingested L3 larvae become “arrested” in the abomasum starting in early fall and do not complete development until the following spring.

The adult worm has a lancet mouth piece allowing it to pierce the mucosal surface of the abomasum and feed on the seeping blood. One worm can result in the loss of 0.05 ml of blood per day. A load of 1,000 worms will cause a loss of 50 ml daily. A 25-kg lamb or kid (55 lb) only has 2000 ml of blood in total. However, clinical disease can occur with a load as few as 500 worms. In the course of a few weeks, infected youngstock will become severely anaemic from this blood loss. The animal may drop dead on pasture with very severe infections (e.g., 3,000 worms). Animals with lower levels of infection will be chronically anaemic, hypoproteinemian (exhibiting bottle jaw), and have a poor appetite, and weight loss. The conjunctival mucous membranes around the eye appear pale pink to white. The haematocrit is often less than 12% indicating severe anaemia (normal 25% to 35%).

In central Canada, type II Haemonchosis occurs in the spring, before the animals go to pasture. Ewes or does may develop severe disease in late April and early May, usually when periparturient. This is because the overwintered, immature (L4) parasites in their abomasum emerge in the spring. These animals may not yet have eggs in the faeces.

On postmortem, the carcass is very pale due to the anaemia. The abomasum contains numerous visible worms. The contents are dark brown from the seeping vessels and excreted digested blood. In severe, acute infections the abomasal mucosa may appear haemorrhagic. The marrow of the long bones is often very red from the response to the anaemia.
**Teladorsagia circumcincta**
Brown stomach worm. This is a small parasite found in the abomasum, approximately 1 cm long and barely visible to the naked eye. It is also known as Ostertagia. Most severe infections occur in the late summer or fall, but occasionally severe disease is associated with the re-emergence of the arrested L4 larvae in the spring (type II teladorsagiosis). The latter is seen less commonly than with Haemonchus. The L3 are well adapted to survive over winter on pasture in this climate and do so very successfully! The arrested stage carried in ewes and does will develop in the spring and contribute significantly to pasture contamination with the periparturient rise in egg output.

Disease is mostly seen in youngstock during the first season on pasture. Infection is associated with intermittent diarrhoea, weight loss or reduced gains, decreased appetite, and occasionally bottle jaw. Plasma pepsinogen levels may be elevated due to abomasal damage, and the pH of the abomasum is elevated because of this damage. This interferes with digestion and contributes to ill-thrift or weight loss. A load of 5,000 worms is considered to cause significant clinical disease. It is possible for severe disease to occur prior to eggs appearing in the faeces; lambs or kids put to heavily contaminated pastures may experience severe disease due to the sudden massive infection; if sheep or goats are put in the barn in the fall carrying massive numbers of arrested L4, type II disease may occur in the spring.

On postmortem, the parasites can be seen to have invaded the mucosa of the abomasum and cause swelling and redness of the abomasal folds. Scarring will occur with loss of gastric function - sometimes permanently in severe infections. The appearance of the lining of the abomasum is likened to Moroccan leather or a bird’s eye pattern (swirls around a knot, which is the scar left by the worm).

**Trichostrongylus axei**
Stomach hairworm. The worms live in the abomasum and are < 0.5 cm in length and very difficult to see with the naked eye. It also infects cattle and deer. Like *T. circumcincta*, disease is usually seen in the late summer or fall after a build up of L3 on pasture. The L3 are well adapted to survive over winter on pasture in the central Canadian climate. The arrested stage in sheep/goats will develop in the spring and contribute significantly to pasture contamination with the periparturient spring rise in egg output.

Diarrhoea, hypoproteinemia (bottle jaw), decreased appetite, and weight loss are again all clinical features of severe infections with this parasite. Elevated plasma pepsinogen and abomasal pH are also important features. Again, a load of 5,000 worms is considered to be associated with clinical disease. On postmortem, the parasites are seen to have invaded the glandular mucosa, causing damage to the secretory cells. Plaques may be visible on the abomasal surface with chronic infections.

**Trichostrongylus colubriformis** and **T. vitrinus**
Also known as the black scour or bankrupt worms, they are small (0.5 to 0.75 cm in length), light brown, and hairlike worms found in the small intestine (duodenum and upper jejunum). These parasites infect cattle as well. The larvae burrow into the mucosa of the small intestine to develop and then burst out about 10 days after infection. This may cause severe damage to the intestinal wall with loss of blood and protein. Most disease occurs in the late summer and fall from the buildup of infestation on pasture. L3 can overwinter on pasture very well and can also overwinter in the animals as arrested L4 parasites. While trichostrongyles can cause significant disease alone, the worst disease outbreaks are usually seen with coinfections with *Teladorsagia*.

The parasite causes an enteritis (dark diarrhoea) and hypoproteinemia (bottle jaw) with poor appetite and weight loss. Milder infections are associated with soft stools and poor growth rates. Affected lambs and kids may have manure (dag) sticking to the back end and tail - evidence of diarrhoea. On postmortem, the small intestine will have patches of erosion.

**Nematodirus spp.**
*Nematodirus* is also called the thread-necked worm. They are slender worms approximately 1 to 1.25 cm in length and live in the upper part of the small intestine. The front part of the worm is more slender than the rest of the parasite. With a heavy infestation, they appear like a cotton ball. The worms produce very large eggs in which the larvae develop to the L3 stage prior to hatching. The eggs of *N. battus* are brown in colour whereas *N. filicollis* and *N. spathiger* are colourless, so they can be differentiated under the microscope. These parasites infect sheep, goats, and occasionally calves.
*N. battus* is the most disease-causing species of this genus and is less common. In contrast, *N. filicollis* and *N. spathiger* are common, but cause only mild or no disease. Their life cycle is very different from other Trichostrongyloidea. With *N. battus*, the eggs will only hatch after a prolonged period of cool weather followed by more mild weather in which the temperature stays above 10°C. Usually, eggs laid in the summer do not hatch until the following spring or possibly even for 2 years, so that the biggest risk period for infection and disease is the late spring (May and June). Lambs and kids on pasture in the spring are most at risk of disease.

*N. battus* will cause severe, watery, yellow-green diarrhoea in lambs and kids, often accompanied by dehydration and thirst - and in severe infections, death. Clinical signs may appear before eggs are produced (prepatent period of 14 to 16 days), so faecal egg counts may be of limited value in the face of clinical disease. Mild infections of *N. filicollis* and *N. spathiger* may have no to mild signs of disease - most infections are seen with other gastrointestinal nematode parasites (GIN). On postmortem, large numbers of threadlike cotton balls of worms will be found in the small intestine. Some species tunnel into the mucosa. Severe infections are accompanied by signs of enteritis and marked villous atrophy of the intestinal lining.

**Moniezia expansa**
This is the tapeworm of sheep and goats. The adult is found in the small intestine. It is white and comprised of segments (egg packets) 1- to 1.5-cm wide, and a scolex (head) which is anchored to the intestinal wall. It can be quite long with many segments. The eggs are triangular shaped and easily identified on faecal examination. Each egg contains one embryonic tapeworm.

The life cycle requires an intermediate host - a free-living forage mite which ingests them on pasture. The eggs then hatch and the larvae migrate to the body cavity of the mite where they develop into a cysticercoid (a tapeworm head in a solid structure). When the mites are ingested by sheep, they develop into adults. Ingestion to egg production in sheep takes about 6 weeks. Interestingly, the adult tapeworms do not live long - approximately 3 months. Infection is usually worse in summer months, but the cysticercoids can overwinter in the mites.

This tapeworm is generally believed not to cause significant disease in sheep and goats. However, a severe infection can be associated with diarrhea and unthriftiness, and occasionally the volume of parasites in the gut is associated with intestinal blockage and may be a risk factor for *Clostridium perfringens* type D infection (pulpy kidney, also called enterotoxaemia). On postmortem, the parasite is easily seen, but unless very numerous, is not associated with any pathology. However, because lambs often pass large segments, producers see large, white worms in the faeces and assume that the lamb is suffering from their presence.

**The Typical Life Cycle of a Gastrointestinal Nematode Parasite**
The typical life cycle of the gastrointestinal nematodes *Teladorsagia circumcincta*, *Haemonchus contortus*, and *Trichostrongylus* spp. are all similar (Figure 1). For these parasites, there is no intermediate host. The prepatent period is the period from ingestion of the L3 stage to when eggs are detected in faeces, usually 16 to 21 days.
Epidemiology of Gastrointestinal Nematode Parasites

Normal Patterns of Infection in Adults and Youngstock

Figure 2 summarizes the typical level of gastrointestinal nematode parasite burden in lambs and adults, as well as the number of infective L3 on pasture under conditions in central Canada. This graph is adapted from data obtained on farms in Ontario and Quebec in 2006–2008. In the spring, adult sheep/goats serve to contaminate the pasture for lambs/kids, which then are the major source of pasture contamination. A hot, dry summer will drop pasture contamination, while numbers will rise with the autumn rains.
Youngstock in Their First Grazing Season
These animals tend to have no natural immunity to gastrointestinal nematode (GIN) parasites. The L3 stage on pasture serves to infect naive lambs and kids. The level of L3 on pasture and the level of immunity in the youngstock will determine the level of disease seen. Over the grazing season, the loads in the lambs and kids tend to increase and they become the major contributors to egg contamination on pasture. Towards the end of the grazing season, a proportion of the new GIN infection will not progress to adults, but will rather become hypobiotic or arrested.

Adult Sheep and Goats
Adult sheep tend to have a level of immunity to the GIN parasites if they have been previously grazed, but will still be infected and will contribute to pasture contamination. Adult goats develop immunity less well and may have a more important role than adult sheep in contaminating pasture. The phenomenon of periparturient egg rise (see below) allows for increased egg production by adult female parasites, and thus serves to be one of the most important sources of pasture contamination to new born lambs and kids.

Hypobiosis or Arrested Larval Development
After the L3 larvae infect the host and moult to the L4 stage, they may either develop into adults or stay at the L4 stage. At this stage, little disease is seen in the host and no eggs are passed. In this way, many immature parasites may collect in the host without clinical signs. The trigger for hypobiosis is thought to be unfavourable environmental conditions for egg hatching and development of the free-living larval stages (e.g., autumn in temperate climates or dry periods). In temperate continental climates, arrested development is an important mechanism that allows for survival of *H. contortus*. It is believed that here most L3 larvae ingested in the fall - and sometimes late summer in the case of *Haemonchus* - arrest rather than develop to adults.
Immunity and Parasite Burden

Acquired Immunity to Parasites
Lambs and less well kids will develop immunity to parasites over time. The actual length of time varies with the type of GIN, but generally occurs over the first grazing season and by 4 to 6 months of age. However, this varies between species, or breeds of animals, and between animals in a flock as well as level of challenge. With immunity, the adult parasites are expelled (also called “self-cure”), but the animal will continue to be infected with low numbers. However, without continued exposure to parasites, the animal’s immunity will wane and after 6 to 8 months. Additionally, challenge with high numbers of GIN on pasture can overwhelm the animal’s immunity and cause disease. Immunity is also greatly affected by nutrition, particularly dietary protein as rumen nondegradable protein. Examples of this type of protein are fish meal, roasted soybeans, and corn gluten. It is very important to remember that adult goats do not develop immunity as well as sheep.

Periparturient Egg Rise (PPER)
Also called the “periparturient relaxation of immunity” (PPRI) this term refers to the increase in eggs passed in the faeces of ewes and does from a few weeks before giving birth through the nursing period (around 8 weeks). This typically takes place in the spring months and occurs because of a down regulation (lowering) of immunity in the late pregnant female which allows for the following: 1) arrested larvae to mature to egg-producing adults; 2) ingestion of overwintered L3 on pasture to more likely result in infection; and 3) an increased rate of egg production from existing adult worms. This results in a dramatic increase in pasture contamination in the spring at lambing/kidding. The down regulation of immunity is thought to occur because of nutritional stresses associated with late gestation. PPER tends to be lower in single-bearing females compared to those with multiples, lower in mature females than first-timers, and lower when females are supplemented with bypass protein sources.

PPER in out-of-season lambing: Recent research carried out in Ontario flocks compared PPER in ewes lambing in the winter or fall compared to those lambing in the spring. The data suggest that ewes lambing in the winter also experience a PPER, while those lambing in the fall tend not to - this shows that season appears to play a role as well as nutrition in PPER. Canadian data also suggest that PPER may be extended in dairy ewes. Perhaps this is due to a higher level of nutritional stress.

Genetic Resistance to Infection with GIN Parasites
Some animals develop immunity against parasites more rapidly, and are more able to resist establishment of infection after developing this immunity. Within any population of sheep or goats, a portion of that ability is genetic. Programs have been developed to identify sheep carrying genes for resistance, either through ram selection (e.g., rams raised together and selecting those with lower faecal egg counts or higher levels of antibodies to parasites), or attempting to identify genetic markers in the DNA. As adults, these more immune animals will shed fewer eggs. This reduction of contamination is where the benefit lies to genetic selection.

Resilience to Infection with GIN Parasites
This is the animals’ ability to grow and thrive in the face of parasitic infection. These animals are infected, and pass eggs which contaminate pasture, but appear to be healthier, possibly because they have less inflammation and so don’t feel as sick. But, they will contaminate the pasture and may suffer the direct disease effects of the parasite (e.g., blood loss). So, for this reason, resistance is preferred to resilience.

Normal Patterns of Infectivity on the Pasture
Effect of Environment on Development and Survival of the Free-Living Stages
Temperature: The optimal temperature for hatching, larval development, and L3 survival varies by parasite:

- *Teladorsagia circumcincta* prefers 16°–30°C
- *Trichostrongylus colubriformis* 22°–33°C
- *Haemonchus contortus* requires the hottest temperatures at 25°–37°C
At temperatures < 10°C, larval development and moulting do not occur. At temperatures < 5°C, the metabolic rate of L3 is very low - allowing prolonged survival (e.g., overwintering on pasture). However, if the weather is hot (e.g., > 28°C), the L3 may die more rapidly because their metabolic rate increases and they outlive their stored nutrients (L3 cannot feed) before infecting a host.

**Humidity:** At faecal pellet, or ground level, the humidity should be > 80% to allow for development. L3, but not L1 or L2 can survive desiccation (drying) because of protection of the cuticle covering, even at freezing temperatures. Some species of L3 (e.g., *Teladorsagia*, but not *Haemonchus*) can enter a state of anhydrobiosis which allows them to survive severe cold and desiccation (drying), making them well suited for surviving the freeze-thaw cycles of our winters.

**Survival of L3 Over Winter on Pasture**
It can be assumed that a pasture grazed the previous summer is likely contaminated with L3 that have survived over winter. If pasture contamination was high the previous fall, then the level of L3 from *Teladorsagia* and *Trichostrongylus* will still be high in the spring. Snow cover enhances the survival. Several freeze-thaw cycles or prolonged, cold temperatures without snow cover may lower this survival rate. *Haemonchus* does not survive well in the central Canadian climate. Overwintered L3 of all GIN are considered to survive no longer than the end of June, but this depends on the temperature and humidity. A cool, wet spring may enhance survival, whereas a hot spring will shorten survival.

**Development and Survival of L3 on Pasture During the Grazing Season**
Moderate temperatures and high humidity hasten development to L3; however, hot temperatures will shorten survival. Conversely cool temperatures will prolong survival of L3. The pasture itself will influence development and survival. Old pastures with a mat of dead grasses above the soil, will hold humidity longer as well as reduce temperature extremes, and therefore enhance development and survival. Heavy cropping (e.g., through pasture rotation) will reduce this mat and open the soil to sunlight and desiccation - both limiting survival. Heavy morning dews or moisture that may be present after a rainfall, will allow migration of L3 a few centimetres up the grass blades, enhancing infectivity of the pasture. Hot, sunny days will drive the L3 down to the soil level, thus reducing infectivity.

**Generations of parasites on pasture:** One generation is the time from the eggs passed in the faeces, through hatching and development of the free-living larvae, infection of the sheep or goat, and then passage of eggs again in the faeces. The average time from egg deposition to L3 is 2 to 3 weeks and time from infection to egg production is 3 weeks, so one generation is 5 to 6 weeks.

- **Teladorsagia** and **Trichostrongylus**:
  - It is unlikely that more than 2, or at the most 3, generations of these parasites occur under temperate, continental summer conditions (from passing of eggs to infection and passing of eggs). Therefore, it is likely that if severe parasitism from these parasites occurs on a farm, there was likely heavy contamination from the previous grazing season as well as high stocking densities in the current grazing season - along with optimal summer conditions for L3 development and survival (i.e., warm and moist conditions).

- **Haemonchus**:
  - Although *Haemonchus* does not appear to survive on pasture over the winter, it survives very well in adult sheep and goats in the hypobiotic state. It can increase pasture contamination rapidly because of its ability to produce thousands of eggs per day and hatch to L3 in less than 5 days. Once the adult female worm matures in the spring, it is capable of producing 10,000 eggs per day. Under warm, humid conditions L3 will develop in as little as 5 to 7 days, allowing for multiple generations and therefore massive pasture contamination. This means that within one grazing season, the infectivity of the pasture may become very high.

**Survival and Transmission of L3 Indoors**
Very little is known about survival and transmission of GIN indoors. There is sufficient anecdotal evidence to suggest that transmission can occur, but usually at low levels. However, lambs and kids in a
dry lot, particularly if fed on the ground, may pick up GIN infection. So, to lower the risk of GIN in this situation, it is important to minimize manure contamination of feed.

**DIAGNOSING GASTROINTESTINAL PARASITISM**

**Fecal Egg Counts (FEC)**

FEC is a measure of the adult parasite population in the sheep or goat, but not a measure of total infection (i.e., L4, L5, and adults). There is much animal-to-animal variation in FEC, so it is important to sample a random proportion of the group.

**Who to Sample**

Sheep or goats grazing pasture that are representative of the group should be sampled. Do not sample animals that have been held off feed, or that are off feed due to illness. Submit 10 samples representative of lambs/kids and separately 10 samples representative of ewes/does. Counts will be very different between these groups - even on the same pasture.

**Getting the Samples**

Group the animals into a corner of the pasture (with clean ground), hold them for 15 minutes, and then release. Pick up 10 individual faecal samples that are fresh and place in individual plastic (e.g., sandwich) bags. Immediately place in a cooler with ice packs. Faecal samples can be collected from the rectum. This latter method allows identification of the samples. Animals should be randomly selected. A video showing this method can be viewed at www.youtube.com/watch?v=X2C9sVstce0&feature=relmfu.

**Transportation and Storage of the Samples**

The samples must be kept cool (< 5°C) (not frozen) until they reach the laboratory to prevent hatching. Refrigerated samples should be analysed within 7 days of collection.

**Analysis of the Samples**

It is important that the samples be evaluated using a quantitative technique. The modified McMaster technique is one such method that will allow the number of eggs per gram (EPG) of faeces to be reported. Qualitative counts (e.g., 1+, 2+, 3+) are not useful for differentiating between a moderate infection (e.g., 150 EPG) or a severe infection (e.g., 1,500 EPG) as both will be interpreted as 3+.

**Pooled vs. Individual Samples**

There is considerable animal-to-animal variation in egg output, with 30% of animals responsible for ~70% of the egg output. Pooling of samples, so that only one test is done per group of animals is a valid way of analysing parasite load. However, samples should be pooled at the laboratory (not at the farm) to make sure that equal amounts of pellets are contributed by each animal (minimum 4 grams of faeces each).

**Significant Eggs per Gram Levels**

The following cut-points are often used for individual or pooled samples:

- **Low** = < 500 EPG
- **Moderate** = 500 to 1,000 EPG
- **Severe** = > 1,000 EPG

However, there are several factors that need to be appreciated when deciding what cut-point to use or what action to take:

- **Species of GIN:**
  - *Haemonchus* is a very prolific egg producer and is associated with rapid changes in pasture infectivity. If this species is predominant, the FEC can change very quickly, as can the level of disease in the lambs or kids. Furthermore, even within the 3-week prepatent period of the parasite, youngstock can become very anaemic - before egg levels change significantly. Generally it isn't known which type of parasite is contributing to the FEC, but you can use history of previous infections to help. Larval culture and
identification of GIN species can be done in specialized laboratories. A technique utilizing peanut agglutinin with a fluorescent dye is used in some laboratories to stain and identify the proportion of eggs which are of the *Haemonchus* type.

- **Heavy infection from previous season:**
  - Sheep/goats that grazed the previous summer may have a significant hypobiotic load of *Teladorsagia* and or *Haemonchus*. In the spring, the massive reemergence of these larvae can be associated with significant disease, but may have a negative FEC as the larvae have not yet reached the adult, egg-producing stage.

- **Grazing heavily infested pastures:**
  - Naive animals that graze very heavily infested pastures may experience disease due to *Teladorsagia* and *Trichostrongylus* before the prepatent period is complete. These animals will have watery diarrhoea and bottle jaw with some deaths - along with a very low FEC.

- **Individual variability in FEC:**
  - Approximately 30% of lambs/kids are responsible for 70% of the total egg production. This means that there is tremendous animal-to-animal variation in egg output - also called “over-dispersion” of the values. Mean values may underestimate the severity of infection in some animals. Animals with diarrhoea may actually have decreased FEC because the eggs are diluted, so absence of eggs in an animal with diarrhoea does not mean that animal is not parasitized.

**Clinical Changes in the Animal**

**Diarrhoea/Dag Scores**

Faecal consistency may reflect parasite load, but some animals with parasitic infections (e.g., acute haemonchosis) do not exhibit diarrhoea. Diet type also greatly influences faecal consistency, so interpreting this must be done given the pasture being grazed. Dag is defined as faecal contamination of the wool, or hair coat, around the tail and hind quarters. Soft or diarrhoeic stools will cling to the wool/hair. A dag score will give an approximation of prevalence of diarrhoea in the group.

**Figure 3**

Low Weight Gains/Weight Loss (Body Condition Score)

Low weight gains are primarily due to the decreased appetite from the parasite infection. Additional factors are the energy losses associated with the animal fighting the infection and the losses of protein and blood that the parasites consume. However, there are other causes of poor weight gains (e.g., poor pasture, coccidiosis, pneumonia) so that FEC should be done.
Body condition score (BCS) is difficult to use as so many other factors influence it. Additionally, by the time an animal is thin (≤ 2.0); it is experiencing severe clinical illness. However, an animal in poor BCS may be more susceptible to parasites if debilitated from another disease (e.g., Johne’s disease).

**Anaemia (Haemonchus)**

A major clinical feature of haemonchosis is anaemia. In central Canada, in late July to mid-August, in warm and wet summers, haemonchosis can be the most important type of parasitism on some farms. Lambs and kids can be monitored during this period for evidence of anaemia. This can be done by monitoring the haematocrit. But, it is more commonly done by assessing the colour of the conjunctival (around the eye) mucous membrane.

The FAMACHA® system makes use of this (Figure 4). It was developed in South Africa in regions where the primary type of GIN is *Haemonchus*, and is used successfully in the southeastern USA where the epidemiology of parasites is similar. It allows the producer to monitor individual animals and to only treat those that appear anaemic. Its drawback is that if other parasites are important, then it will fail to detect those infections. For this reason, the FAMACHA® system should only be used under the guidance of a veterinarian and only as an adjunct to routine FEC.

**Figure 4**

[Image of the FAMACHA® system]

**Hypoproteinemia (Bottle Jaw)**

Almost all the GIN parasites feed on protein. In severe infections, the protein levels can drop very low producing the clinical sign of bottle jaw. Additionally, edema in the gastrointestinal lining causes poor absorption of nutrients and diarrhoea. By the time this is clinically apparent, parasitism is very advanced and the animal is in immediate danger of dying.

**Postmortem Examination and Worm Counts**

If animals are dying and internal parasites are suspected of being the cause, it is very important to confirm this diagnosis with a total (adult) worm count from the gastrointestinal tract. A field necropsy
can attempt to identify abomasal and intestinal nematodes. *Haemonchus* are easy to see. *Teladorsagia* and *Trichostrongylus* are small and should be identified and counted in the laboratory using a microscope. The abomasal contents are removed and volume measured, and a sample of a known volume is removed and the worms counted. For example, if there are 10 worms counted in 1/100th of the volume of the fluid in the abomasum, then the abomasum contained 1000 worms.

A system recommended for interpreting burdens, in the manual for the *Sustainable Control of Parasites in Sheep (SCOPS)*, 3rd edition, from the UK, is as follows:

- 2 points = parasitism is likely affecting productivity
- 3 points = parasitism is likely causing clinical signs and even death
- *Teladorsagia* sp.: 3000 worms = 1 point
- *Trichostrongylus* spp.: 4000 worms = 1 point
- *H. contortus*: 500 worms = 1 point
- *Nematodirus* spp.: 4000 worms = 1 point
- Immature worms: 4000 worms = 1 point

**ANTHELMINTIC DRUGS FOR SHEEP AND GOATS**

Anthelmintics are divided into broad spectrum - i.e., those able to kill a wide variety of parasites, and narrow spectrum - those only able to kill one or two types of parasites.

**Broad-Spectrum Drugs**

**Benzimidazoles (BZ)**

White drenches. These chemicals are effective against all GIN and adult tapeworms. The drugs are deposited in the rumen and are slowly released into the gastrointestinal tract. They act on the intestinal cells of the nematode and the skin cells of the tapeworms, inhibiting uptake of glucose and causing starvation. They kill not only the adult forms, but also the immature stages. They are also ovicidal - with activity against eggs being passed by the nematodes and tapeworms.

Currently, fenbendazole (Safeguard 10% suspension, Merck Animal Health) and albendazole (Valbazen, Zoetis Animal Health) are most commonly used. Albendazole also has activity against adult flukes, but should not be used during breeding or the first trimester of pregnancy because of toxicity to the foetus in early gestation. Generally, however, the BZ class of drugs are very safe with low levels of toxicity.

**Imidazothiazoles (LV) and Tetrahydropyrimidines**

This group contains levamisole, pyrantel, and morantel. They are also known as yellow drenches. Levamisole is not ovicidal and the difference between animal toxicity and efficacy is very narrow making overdose and poisoning a potential issue. Levamisole works by paralysing the parasite so that it is removed rapidly from the gut. It works well against a broad range of adult worms, but less so against the immature stages (e.g., L4). However, it is particularly effective against lungworm. Signs of toxicity in animals include salivation, slow heart rate, and muscle tremors with occasional death. Morantel can be used to treat GIN, but is not effective against the immature forms. Pyrantel is rarely used in livestock.

**Macrocyclic Lactones (ML)**

This group contains the avermectins (ivermectin, doramectin, eprinomectin, abamectin) and the milbemycins (moxidectin). These compounds are derived from specific species of the *Streptomyces* genus and all act similarly. MLs have activity against most nematodes including the L4 stage, but not tapeworms or flukes. They are not ovicidal. They also have activity against some arthropod ectoparasites, specifically sucking lice and nose bots (*Oestrus ovis*), as well as some activity against keds (*Melophagus ovinus*) and mange (*Chorioptes, Sarcoptes, and Psoroptes*). Because of this spectrum of activity, drugs in this class are sometimes called endectocides.

When administered, the drugs are stored in fat tissue and then slowly released into the body. These pharmacokinetic properties result in long meat and milk withdrawal times. Moxidectin; however, is the only drug in this class which is considered to have significant prolonged activity - approximately 35 days
when administered as an injection, and 21 days when administered as a drench, against *Teladorsagia* and *Haemonchus*. The mode of activity is believed to be largely against neurotransmitter receptors specific to invertebrates. But, they are considered quite safe for mammals.

**Amino-Acetonitrile Derivatives (AAD)**

The first product from this new class of drugs (monepantel) was released in 2009 in New Zealand and the UK (Zolvix, Novartis Animal Health), but not yet here. This is the first new class of anthelmintics developed in 25 years and appears to have excellent activity against resistant strains of GIN as well as immature forms of nematodes, and in particular *Haemonchus*. The drug is also of low toxicity as it targets a unique, nematode-specific class of acetylcholine receptor subunits.

**Spiroindoles**

Also a new drug, derquantel (of the spiroindole class of anthelmintics) is marketed as a combination drug with abamectin (Startec, Zoetis Animal Health). It is marketed as a combination as there is evidence that the best way to use drugs for which there is no anthelmintic resistance (AR) in the parasite population, is to use in combination as AR develops more slowly.

**Narrow-Spectrum Drugs**

These drugs only act against a few types of parasites.

**Closantel**

This drug (Flukiver®, Elanco Animal Health) is effective only against internal parasites that consume blood. In sheep and goats, this is *Haemonchus* (including larval stages) and the liver fluke *Fasciola hepatica* and at higher dosages, *Fascioloides magna*. It also has persistent activity by binding to the host’s plasma proteins. We are currently engaged in a research project using this drug in ewes at lambing time.

**Praziquantel**

This drug acts against the adult and immature stages of tapeworms and is of most use for control of tapeworms in guardian and working dogs.

**Combination Anthelmintics**

Currently in Canada, no combination products are licensed for small ruminants. In other countries, they are used frequently (e.g., a 3-way combination with ivermectin, fenbendazole, and levamisole) and may be useful in areas with anthelmintic resistance. They are most effective if at least one of the drugs is novel (i.e., there is no anthelmintic resistance present in the flock).

**Route of Administration**

**Drench vs. Injection**

Drenches are deposited in the rumen so that proper absorption can occur. Injection of anthelmintics has been shown to result in a longer action - which may be favourable in some instances, but may select for resistant nematodes because of prolonged subtherapeutic drug levels (i.e., a long tail).

**Use of Pour-On Anthelmintics**

Pour-on products are not as well absorbed in sheep and goats as in cattle. Because of the risk of subtherapeutic dosing by this route, their use is **not** recommended.

**Using an Anthelmintic by a Route Other than Indicated on the Label**

Use of pour-on products as an oral medication is not recommended as the absorption, efficacy, duration of action, and withdrawal times are not predictable and may increase the risk of anthelmintic resistance.

**Appropriate Dose of an Anthelmintic**

In Table 1 is a listing of those anthelmintics that are used in Canada, all of which are considered broad spectrum. Only ivermectin is licensed for use in sheep and none are licensed for use in goats. The dosages provided are based on licensed recommendations from other countries where the drug is approved, or from the literature.
Table 1. Suggested dosages of anthelmintics for treatment of GIN infection

<table>
<thead>
<tr>
<th></th>
<th>Benzimidazoles</th>
<th>Ivermectin</th>
<th>Moxidectin (drench)</th>
<th>Levamisole</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sheep</td>
<td>5 mg/kg BW</td>
<td>0.2 mg/kg BW</td>
<td>0.2 mg/kg BW</td>
<td>7.5 mg/kg BW</td>
</tr>
<tr>
<td>Goat</td>
<td>10 mg/kg BW</td>
<td>0.3 mg/kg BW</td>
<td>0.4 mg/kg BW</td>
<td>12 mg/kg BW</td>
</tr>
</tbody>
</table>

BW = body weight

**Efficacy Against...**

Table 2. Activity of anthelmintics against the different parasite classes

<table>
<thead>
<tr>
<th></th>
<th>Benzimidazoles</th>
<th>Avermectins</th>
<th>Moxidectin</th>
<th>Levamisole</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypobiotic larvae</td>
<td>+</td>
<td>++</td>
<td>++</td>
<td>+/-</td>
</tr>
<tr>
<td>Persistent activity</td>
<td>-</td>
<td>++</td>
<td>++</td>
<td>-</td>
</tr>
<tr>
<td>Tapeworms</td>
<td>+</td>
<td>+/-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>External parasites</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Liver flukes</td>
<td>+/- *</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

+ = good activity; ++ = much activity; - = no activity; +/- = slight activity
* = albendazole has activity against adult flukes, but only at double-dose (10 mg/kg BW sheep)

Benzimidazole = fenbendazole and albendazole
Avermectin = ivermectin, doramectin, eprinomectin

**REASONS FOR TREATMENT (DRENCH) FAILURE**

When a treatment fails to clear up a problem with internal parasites, many might be tempted to believe that this failure is due to anthelmintic resistance. This is only one reason for treatment failure (often called drench failure) which can occur for several different reasons:

**Failure to Administer an Anthelmintic Properly**

The following is a list of reasons why drench failure may occur. The animal’s weight may be underestimated. This can be remedied by the use a calibrated livestock scale to prevent underdosing (underestimating weight). If the animals are variable in weight, dose for the heaviest in the group. The drug may not contain sufficient active ingredient if it is not purchased from a reputable source. Drugs obtained through the internet (for example) may not contain what the label says it does as they may be manufactured in countries that do not have strict legislation on quality control. The wrong dose is used, a risk when using drugs not approved for sheep or goats. Read the label for products approved for sheep. If not labelled for sheep, or if using in goats, obtain the correct dose by veterinary prescription. Goats often require a higher dosage than sheep and cattle, sometimes twice as much. This is because goats will metabolize anthelmintics faster than sheep and cattle. Withdrawal issues for meat and milk must be considered if increasing the dose. Doubling the dose will not always double the efficacy of the drug. Some drugs can be toxic if the dose is doubled, particularly if the animal is ill. The drench gun is not delivering the correct amount and needs to be routinely calibrated. The drench is not administered correctly. It should be done by depositing the entire dose over the tongue, at the back of the throat. This will ensure that the drench is swallowed into the rumen and is more slowly released. If administered in the front of the mouth, loss may occur by spitting or having the drug swallowed directly into the abomasum where it will pass through the digestive tract quickly reducing its effectiveness. Do not lift the head too high as that will prevent proper swallowing. If the anthelmintic is injected, the automatic syringe should be calibrated appropriately and that the entire dose is injected subcutaneously (not intra-wool). The anthelmintic is given by the incorrect route of administration. Do not use a cattle pour-on product either as a pour-on or as a drench. Do not use an injectable product orally. Do not use pour-on products as a pour-on as they are not absorbed adequately to be effective.

The effectiveness of a BZ anthelmintic may be improved by holding the animals off feed for 12 to 24 hours before treatment. This will increase the length of time that the concentration of the anthelmintic is effective. This should not be done if the ewes/does are in late gestation because of the risk of pregnancy
toxaemia. It may also be more effective if, rather than doubling the dose, a second dose is given 12 h after the first.

**Use of the Wrong Anthelmintic**
If an anthelmintic is used to treat a parasite for which it has no efficacy. A common example is the use of ivermectin to treat tapeworms.

**Reinfection After Treatment - Apparent Treatment Failure**
If the pasture that the animals are turned out to graze after treatment is infested with high levels of L3, then there can be apparent treatment failure. Most anthelmintics have no persistency which means that very soon after treatment the lambs/kids are infected from the L3 on pasture. If the challenge is high, then clinically they may appear as if they have not responded to the treatment. Depending on when the faecal samples are reexamined (e.g., 2 weeks later), the FEC may be very low, indicating that the parasites within the animals were killed but that immature adults are numerous enough to cause disease. This is prevented by reducing the challenge after treatment through pasture management.

**Anthelmintic Resistance (AR)**
AR is becoming very common - particularly in *Haemonchus* and *Teladorsagia*, and to all classes of anthelmintics. By the time AR is clinically apparent (i.e., failure of treatment to improve the health of the animals being dewormed), it is well advanced in the flock. The following will explain how AR develops and strategies to avoid its development.

**Definition of AR**
Resistance, or AR, is the heritable ability of the parasite to survive a normally effective dose of an anthelmintic. Usually a parasite is considered resistant if it survives a normal dose of a single anthelmintic. Parasites often survive if the treatment is administered incorrectly - this is not AR, but drench failure (see above). Because resistance is a genetic trait, the parasite may be homozygous resistant (i.e., having two copies of the genes for resistance) or heterozygous resistant (i.e., having only one copy of the gene for resistance).

The homozygous resistant parasite is much more resistant to an anthelmintic than are heterozygous parasites. Heterozygous resistant parasites are still susceptible to the correct dosing of an anthelmintic, but will survive if the animal is underdosed. However, a homozygous resistant parasite may not be affected at all, although repeated dosing at a high level, or dosing with two anthelmintics simultaneously, may be effective for a while. The homozygous resistant parasite is rare in an unselected population of parasites. But, once the selection has occurred, parasites do not lose their resistance.

**How Does AR Develop?**
**Refugia explained:** This term is applied to the free-living stages of GIN on pasture (i.e., L1, L2, and L3 stages of larvae as well as the parasitic GIN in the sheep/goats that are not exposed to an anthelmintic treatment). Traditionally, a higher proportion of the total parasite load on a farm is on the pasture (80%) as eggs and free-living larvae, compared to the parasite load in the animals (20%) which is comprised of L4, L5, and adult parasites. The refugia are the farm’s source of susceptible parasites. Elimination or severe reduction of refugia will hasten the development of this resistance.

**Pressure of anthelmintic use:** Repeat dosing with an anthelmintic will often kill 95% or more of all GIN in an animal. But, it is the surviving, genetically resistant population that will continue to lay eggs and contaminate the pasture. Repeat, frequent dosing, particularly if underdosing occurs, will hasten the development of a resistant refugia. While sheep/goats will clinically respond to a drench that is less than 95% effective, eventually the susceptible parasites are in the minority and the drench ceases to be clinically effective.

**Side resistance:** Resistance is generally shared by all drugs in an anthelmintic class. For example, if the parasite is resistant to fenbendazole, then it is also resistant to albendazole. This is less true with the avermectins and moxidectin, where moxidectin may still be effective in the face of avermectin resistance, but often resistance will develop to moxidectin within a year after it is first used in a flock with ivermectin resistance.
**Parasite fitness**: AR can develop more quickly if the population of parasites is already resistant to one or more classes of anthelmintics. It may be that those parasites have the ability to more quickly metabolize drugs than those that are 100% susceptible. This may play a role in the development of multiclass resistance.

**Consequences of Having Low Levels of Refugia on a Farm in the Face of an Aggressive Deworming Program**

Having susceptible refugia on pasture allows the flock the opportunity to become infected again with susceptible parasites - thus lowering the risk of AR becoming a farm problem. However, it is also important to make sure that pastures are not heavily contaminated, so our parasite control practices include lowering the level of parasites in refugia. However, if an aggressive deworming program is also instituted in the face of low refugia - the development of AR is accelerated.

Two examples of particularly risky methods of parasite control are:

- **“Dose and move” pasture rotation**:
  - The dose and move strategy was designed to prevent animals from carrying parasites into a safe pasture (i.e., one that has no or very few larvae on it). If animals are treated and returned to a contaminated pasture, they will become reinfected with a population of susceptible parasites that were already on the pasture. But, if they are treated and moved to a safe pasture, the only parasites to shed eggs into the new pasture are resistant strains. It might take several grazing seasons to build up a resistant population, but when it happens, it is often too late to reverse the high level of anthelmintic resistance on the farm. For this reason, it is strongly recommended not to practice a “dose and move” deworming strategy. The preferred approach is to delay moving to a safe pasture posttreatment. To prevent the development of AR, the sheep/goats are left on the pasture for a few days to pick up susceptible larvae - enough so that they become mildly reinfected with parasites. This dilutes the resistant eggs being shed on pasture while still keeping low levels in the animals.

- **Dose at lambing/kidding prior to spring turnout**:
  - While *Teladorsagia* and *Trichostrongylus* L3 overwinter well on pasture in our climate, *Haemonchus* does not. This means that spring pastures have very low levels of refugia of this parasite. However, if we deworm all adults prior to turnout (e.g., if we treat all ewes or does at lambing/kidding or at housing in the autumn) we will eliminate all the *Haemonchus* on a farm - with the exception of *Haemonchus* in the animals that have resistance to the anthelmintic used. These resistant *Haemonchus* then infect lambs and kids. It may take a very short time for AR resistant *Haemonchus* to become predominant on a farm, even if the adults are rarely dewormed and may occur more quickly if they are dewormed while still in the barn. In our AR study of 2010 and 2011, almost all the parasites we found on AR farms, were *Haemonchus* - suggesting that this is what may be happening here in Ontario sheep flocks.

**Introduction of Resistant Parasites**

Purchase of sheep or goats that contain large numbers of resistant parasites, may introduce AR to a farm - which when combined with improper parasite control measures, will hasten the development of AR on a farm. Goats are a particular risk as AR tends to develop more quickly with this species. Quarantine of new introductions and proper deworming of new introductions are an important strategy to prevent introduction of AR. For details, see star 4 of the 5 Star Worm Plan in Control of Gastrointestinal Parasitism - Part 2.

**Improper Treatment**

By under dosing, heterozygous resistant parasites are more likely to survive, which will hasten the development of AR on a property.
Detecting the Presence of AR in a Sheep Flock
If AR is suspected in a flock, it is important to review the treatment protocols to make sure that the drug is being administered properly. To confirm AR on a property the following methods can be used:

Drench Response Test
This can be performed with only 1 faecal sample collection time, but only suggests, rather than proves, that AR is present. The group is treated and faecal samples are collected from 10 randomly selected lambs or kids after a period of time (7 days for LV, 10–14 days for BZ, 14–16 days for ML). Failure to achieve low counts may indicate AR, or treatment failure from other causes.

Faecal Egg Count Reduction Test (FECRT)
The FECRT is often used as the gold standard for determining if AR is present on a farm. It is scientifically sound when done correctly and will give an accurate picture of how effective anthelmintics are on a given farm. However, as you can see, the process is labour-intensive and expensive - but gives the best information.

If you decide to perform a FECRT, the following protocol is followed:

- A minimum of 30 lambs/kids or young adults (first grazing season) with elevated faecal egg counts are required.
- Confirm that the level of parasitism is high enough to see an effect. A random pooled sample of a minimum 300 EPG is preferred.
- Ten to 15 lambs/kids are randomly assigned to control and treatment groups. It is necessary to use these many animals per group because of the normal variation in egg output between animals.
- If three anthelmintics are being evaluated (e.g., ivermectin representing the macrocyclic lactones [ML], fenbendazole representing the benzimidazoles [BZ] and levamisole [LV]), then four groups (60 animals) are needed (e.g., control group [no treatment], ML group, BZ group, and LV group).
- Individual faecal samples are obtained per rectum on day 0 (treatment day). This is not necessary if a control group is used.
- The lambs/kids are weighed using a scale and treated appropriately by drenching.
- The control animals are not treated, but are sampled.
- All of the animals are returned to the same pasture to graze.
- All the animals are sampled again later (7 days for LV, 14 days for BZ and ML).
- The posttreatment faecal egg counts are compared to the control.
- Failure to reduce by 95% or greater compared to the control indicates resistance.
- Confidence intervals (CI) are also calculated and AR is present if the lower CI is < 90%.

Larval Development Assays (In Vitro Test)
Larval development assays can be used to detect AR in the laboratory, but they require a specialized laboratory to work properly. Eggs are hatched and developed to the L3 stage while exposed to an anthelmintic. The level of successful hatching, development, or feeding is then measured.
Control of Gastrointestinal Parasitism - Part 2
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SUSTAINABLE INTEGRATED PARASITE MANAGEMENT (SIPM)

Goal
Below is a program which employs the principles of a sustainable integrated parasite management program, titled the “5 Star Worm Plan.” This program, or other similar programs, can be used as the basis of a flock health management approach which is correct for your clients’ farm.

The goal of an integrated parasite management program is to control the parasites on the farm to a level which has minimal effect on animal health and productivity - without allowing for the development of anthelmintic resistance.

STAR 1: MANAGE THE LEVEL OF PASTURE CONTAMINATION
The goal is to have all pastures as “safe” pastures, and there are many methods to achieve this.

Manage the Biggest Sources of Pasture Contamination
The two biggest sources of pasture contamination with parasite eggs are lambs and kids by mid- to late-grazing season (e.g., late July/August), followed by adult females in late gestation and lactation (the periparturient egg rise) - usually in the spring. To manage the contamination by lambs and kids, monitor frequently and treat when needed - particularly from mid-grazing season (e.g., early to mid-July). To manage the contamination from the PPER, if possible, do not graze late gestation or lactating ewes/does, or if not practical to avoid grazing this group, treat selectively (see Section 3).
**Use Our Understanding of the Behaviour of the Free-Living Stages**
By understanding where the infective L3 are in the pasture, we can modify grazing management to try to reduce exposure of the sheep/goats to them.

**Break Up the Faecal Pellet**
Remember that L1 and L2 stages live within the faecal pellet. If the pellet is exposed to moisture, or is broken up using a mechanical means (e.g., harrowing), then it exposes the L1 & L2 to the environment where freezing temperatures or very hot, dry conditions can kill them more rapidly. Soil livestock (e.g., earthworms and dung beetles) assist in this breakdown. L3 are not affected by this.

**Modify Grazing Based on Temperature and Humidity**
Waiting until the dew is off the grass to graze short pastures is often not practical as a summer shower during the day could bring the L3 back up the grass. Exposing the ground to sunlight may have some benefits as it gives the L3 less place to hide and exposes them to heat and drying. This can be done by either dethatching using a harrow, or routinely planting new pastures and ploughing in old pastures.

**Modify Grazing Based on Sward Height**
The L3 are restricted on how high they can climb (usually not higher than 5 cm), so long grass is safer than short grass. Overgrazing pastures will increase the infection rate by forcing the sheep to graze close to the soil.

**Eliminate Areas of the Pasture that Favour L3 Survival**
Areas of the pasture that are wetter, such as marshy areas or around water troughs, may have greener grass and attract sheep and goats to graze, but also favour survival of L3. Eliminate these areas for grazing either by fencing off or gravelling (e.g., around water troughs).

**Use Pasture Plants that Don’t Favour L3 Climbing**
L3 appear only to be able to effectively climb grasses and so clovers and other legumes may be safer to graze.

**Rotate Pastures with Other Livestock Species**
While cattle share some parasites with sheep and goats, rotation with this species has been shown to generally lower pasture infectivity to sheep and goats. Horses will also work. Co-grazing (grazing at the same time) with cattle is less effective, but may help.

**Avoid Grazing Sheep and Goats Together**
Because adult goats do not develop immunity to parasites they are a more important source of pasture contamination. Additionally, they metabolize anthelmintics more rapidly than sheep, require higher doses than sheep, and because of that are at risk of developing AR more rapidly than sheep.

**Rest Pastures That Are Heavily Contaminated**
If a pasture was particularly heavily infected at the end of the previous grazing season, select it for ploughing, reseeding, haying, and/or grazing with another species.

**Be Aware of Risks of Spreading Manure Onto Pasture or Hay Fields**
Although it is unlikely that infective larvae will survive in well-composted manure, fresh manure can be a source of parasites. It is possible that hay crops can be contaminated in this way and perhaps may be a source of infection when fed. Thus, it is safer to spread manure onto fields prior to ploughing for crops.

**Use Low-Risk Pastures for the Most Susceptible Animals**
Graze weaned lambs/kids on newly seeded pasture or hay fields. Annual pastures (e.g., turnips) that are ploughed in at the end of the season will be beneficial.

**Dose and Move vs. Dose and Stay**
If the entire flock is to be treated, there are techniques which allow susceptible refugia to be maintained - ideally, dose and stay for a few days prior to moving is preferred.
Reduce the Contamination of a Pasture by Using Pasture Rotation

Firstly, understand that L3 can survive for weeks or months on pasture if the environment is moist and temperate and that L3 can survive over winter. This makes it very difficult to accurately predict when a pasture is finally “safe” in a pasture rotation system. Most pasture rotation systems require that the flock return repeatedly to the same pasture in a grazing season. Unless the frequency is < 2 weeks, eggs deposited when grazed previously will likely be hatched and the L3 larvae will be waiting to infect. L3, under the right weather conditions (e.g., temperate and moist), may survive for months.

The following are some suggestions that will help with using pasture rotation to control parasites:

- **Evasive grazing:**
  - This technique of grazing pastures when the risk of parasites is lowest requires knowledge regarding the speed of larval development given local conditions. It should be remembered that during the summer, pastures can remain very infective for up to 3 months making evasive grazing not practical if used as the sole method for parasite control.

- **Strip-rotational grazing:**
  - This is a form of evasive grazing and is relatively safe if the following hold true:
    - Animals are moved from the strip before eggs hatch and the larvae develop to the L3 stage (variable depending on weather; longer in cool weather, but shorter in warm weather) and
    - Animals do not return to the strip until the L3 have died (variable depending on weather and moisture, but may be up to several months if temperate and humid).
  - While short-term grazing will limit pasture contamination, returning to the strip several times in a season will result in the buildup of L3 - just as if the pasture was set stocked (i.e., animals put to the same pasture for the entire grazing season). While it may be prudent to strip graze in order to make optimal use of the pasture, monitor FEC closely.

If Heavily Contaminated Pastures Must be Grazed

Often, the producer has only heavily contaminated pastures available for grazing. The following strategies may help to lower risk in the face of grazing heavily contaminated pastures:

- **Lower Stocking Densities**
  By lowering stocking densities, there will be less pasture contamination with faeces. Recommendations vary, but keeping set stocking densities < 6 to 8 sheep/goats per acre is often mentioned. With rapid pasture rotation, these densities can be increased. But, FEC monitoring must also be done.

- **Don’t Graze Nursing Lambs/Kids**
  It is more difficult to manage the parasite exposure of lambs/kids when grazing with their dams. If only heavily contaminated pastures are available, try to avoid grazing nursing lambs/kids. If possible, practice early weaning so their exposure can be better managed. If not, increase the frequency of FEC monitoring. Weaned lambs/kids should be pastured on the pasture with the lowest level of contamination.

- **Rotate Weaned Youngstock Ahead of Adults**
  After weaning, lambs or kids should have first access to safe pastures. This way there is less risk from exposure to PPER contaminated pastures. Adults are better able to tolerate heavily infested pastures.

- **Use Adults to Graze Heavily Contaminated Pastures**
  If pastures are heavily contaminated and safe pastures are in short supply, non-lactating ewes or does not in late pregnancy can be grazed more safely than youngstock on these pastures and may help to lower the infectivity by grazing off L3. This should be done carefully and not without monitoring FEC.
Record Pasture Use and Treatments

STAR 2: USE ANTHelmINTICS APPROPRIATELY

To Avoid Treatment Failure and Development of AR, Treat Appropriately

- Weigh and dose for the heaviest in the group.
- Use drugs approved within Canada/USA.
- Dose correctly using the information provided on the label.
- If not labelled for sheep/goats, use a correct dose as recommended by the scientific literature.
- Goats are generally treated at 2 X (BZ and LV) or 1.5 X (ML) the sheep dose.
- To increase effectiveness when AR is suspected, do not double the dose, but rather give the recommended dose twice, 12 h apart (BZ and ML).
- Calibrate the drench gun frequently.
- Drench correctly by depositing the entire dose over the back of the tongue.
- Oral drenches are preferred to injectable products.
- Use correct route of administration.
- Do not use pour-on products for cattle.
- Holding off feed for 12 to 24 h before treatment with a BZ can increase the length of time that the concentration of the anthelmintic is effective.

Rotate Anthelmintic Classes Slowly
Consensus suggests not to rotate anthelmintic drug classes more frequently than annually. Rapid rotation is thought to lead to multiple-class AR.

Combining Anthelmintic Classes
In many parts of the world, there are commercial deworming products that contain more than one anthelmintic. These were developed for sheep farms with AR and will temporarily improve efficacy of those drugs. If more than one drug is to be administered at one time, they should be delivered in separate drench guns/syringes.

STAR 3: MONITOR AND TREAT ANIMALS SELECTIVELY

Targeted Treatments
This means to treat sheep or goats only when they need it. This is done by monitoring FEC (usually pooled samples) and clinical evidence of disease and then treating the group. Record all FEC results. Increasing the interval between anthelmintic treatments reduces the development of AR. Times to monitor (and possibly treat):

Ewes/Does Prior to Lambing/Kidding
This is to eliminate or reduce the PPER which is considered one of the most important sources of pasture contamination for lambs and kids. For spring or winter lambing ewes and does - this is an obvious time to monitor. Fall lambing ewes (maybe also does) appear not to have a PPER possibly because of seasonal influences.

Ewes/does with a significant PPER will contaminate the spring pasture with eggs starting a few weeks prior to lambing/kidding and continuing through to about 8 weeks into lactation - or until weaning. They will also be more susceptible to infection from any overwintered L3 on pasture.

Animals at highest risk are yearlings lambing for the first time, adults nursing multiples, adults nutritionally stressed, or other disease. Does may also be more prone to disease as immunity in this species is poor for parasites. However, treatment when treatment is not needed will hasten development of AR on a farm - particularly for Haemonchus. Under some management conditions (e.g., if ewes lamb and nurse lambs indoors), it may not be necessary to deworm at this time because they are not contaminating pasture - as long as the females appear healthy.

For these reasons, it is important to make the decision on whether or not to deworm prior to lambing/kidding in light of the farm’s specific parasite issues and to monitor faecal egg counts in nursing adults.
Lambs/Kids at Mid-Summer
The exact date to start to take faecal samples will vary depending on the warmth and humidity of the summer and how early summer arrives in our northern climate. Generally early to mid-July is the earliest that we routinely see clinical evidence of disease. Mostly it is slightly later - late July to August, which appears to be the highest risk period in our climate for haemonchosis.

Monitor the FEC in the youngstock (and adults if grazing together) in early to mid-July, and treating only when high counts are found (or in the case of haemonchosis, evidence of anaemia [FAMACHA® score] can also be used). If the FEC is negative, but animals are showing severe clinical signs of parasitism, determine if another disease is present (e.g., coccidiosis).

If the spring pasture is heavily contaminated from the previous grazing season with overwintered L3 (usually Teladorsagia, but not Haemonchus), parasitism can occur earlier. Lambs/kids on these pastures may encounter such a severe infection with these overwintered L3 that they become clinically ill (diarrhoea, off-feed, depressed) from the immature parasites before eggs are present in their faeces. Any youngstock dying should be necropsied to determine if this is the cause of death.

Repeat Monitoring in the Grazing Season
Monitor frequently at the times of highest risk (i.e., mid-summer to early fall). If/when the lambs/kids are monitored, the FEC is below the cut-point to treat, resample in mid-summer at least every 3 to 4 weeks and perhaps more frequently, particularly if Haemonchus has been a problem in the past.

Monitor After Treatment
Faecal egg counts should be done every 4 (BZ and LV) to 6 (ML) weeks after treatment. If the animals appear parasitized after treatment, it is strongly recommended to resample at 14 days to determine if treatment failure occurred (see above).

Monitor According to Farm History
By knowing the farm history, the time of monitoring can be adjusted. For example, if the previous summer, lambs had elevated FEC in the first week of July, then monitoring should be started in mid-June.

Monitor in the Autumn?
By October there is no point in using FEC to determine infection. Although the animals may be parasitized, most of the development is now to the arrested stage (L4 and L5) which do not produce eggs. Performing FEC at this time will not properly estimate the level of infection present in the animal.

Treatment of Breeding Animals in the Autumn or Pre-Breeding?
Treatment in the autumn may reduce the arrested L4 that overwinter in the animal, and are thus available for a PPER the next spring in housed ewes and does. But, we need to be assured that the treatment is both necessary and actually works at this time of year. The recommendation to treat pre-breeding should only be done if monitoring or poor condition suggests that the adults are parasitized. Usually adults will not show signs of parasitism unless periparturient or debilitated with another disease or poor nutrition. Treatment when not necessary will contribute to the development of AR.

Targeted Selective Treatments
This means treating only those individual animals that need it when they need it and is based on the knowledge that in any given population, only a proportion actually requires deworming. The challenge is to correctly identify the animals that need treating and those that don’t. The development of AR can be slowed or prevented if about 1/3 (30%) of animals are not treated. This approach leaves a susceptible parasite population in refugia - both on the pasture and in the untreated animals and is critical to the success of any sustainable, integrated parasite control program. The producer has only a few options to be able to do this effectively and economically.

Using Faecal Egg Counts
Unless the flock size is very small, it is not economical to perform individual FEC on all animals in order to detect the big shedders (i.e., those 30% of animals that shed 70% of the eggs). There is no method of determining parasite egg load in faeces other than using a laboratory-based test.
Using the FAMACHA® System
The FAMACHA® system can be used very effectively to select individual animals for treatment of haemonchosis - but is not effective at detecting infection of other GIN species. It could be used on farms that know when *Haemonchus* becomes a problem (e.g., starting late July, early August), but should be combined with FEC to rule out other causes of parasitism. Sheep or goats that score 4 or 5 would be drenched (3s too if a large part of the flock is anaemic) and then everybody monitored every 2 to 3 weeks during the high risk period. FAMACHA® cards must be used in good light (i.e., daylight ideally) and replaced annually as the colour may fade with time. It is important to investigate treatment failure. Use the provided record to track FAMACHA® treatment results.

Using Evidence of Diarrhoea
Dag scores indicating diarrhoea may be helpful when the producer can eliminate other reasons for scouring (e.g., coccidiosis or lush pasture) and may work best when combined with monitoring weight gains. However, some research suggests that by the time the lambs or kids have diarrhoea, significant clinical disease is occurring (i.e., waiting until they have diarrhoea is too late).

Using Weight Gains
Routine weighing of lambs or kids (e.g., every 2–3 weeks) can identify those animals that are not gaining as fast as their cohorts, one reason for which may be GIN parasitism. One method of using this information is to only deworm the lighter animals and leave the heavier ones untreated.

Body condition score was not found to be helpful of predicting FEC in a recent Canadian study. It may be that it is not sensitive enough to pick up early parasitism. By the time the animals are thin, parasites have taken a severe toll.

Using Milk Production
Dairy goats in their first lactation may benefit from deworming in terms of improved milk production. While there is evidence that deworming will improve milk production in dairy ewes and dairy does, you must keep in mind that no anthelmintic is approved for use in lactating small ruminants. If deworming is done, it should be an evidence-based decision (e.g., elevated FEC). The producer and veterinarian are responsible for ensuring that chemical residues are not present in milk sold for human consumption.

Using Number of Lambs/Kids Nursing
There is evidence that ewes or does nursing multiples shed more eggs than ewes or does nursing singles. This is likely due to differences in nutritional stresses between the two groups. Deworming only females with multiples - either before parturition based on pregnancy scanning, or after based on number nursing - is one way to target those animals that likely have the highest PPER.

Using the "5-Point Check" Criteria for Treatment
This system identifies sheep and goats that may require deworming and was developed in South Africa. It includes infection from a variety of parasites - not just GIN - and embraces the concept of targeted selective treatment or “leave the best and treat the rest.”

- The nose is checked for discharge that indicates nasal bots (*Oestrus ovis*).
- The eyes are checked for anaemia, indicating blood-sucking worms.
- The jaw is checked for submandibular oedema that also accompanies anaemia and protein-losing infections caused by parasites such as *Haemonchus* and liver fluke (see below).
- The back is checked for body condition score indicating possible infection by internal parasites like *Teladorsagia* and *Trichostrongylus* species.
- The tail is checked for signs of diarrhoea, indicating mainly worms that also cause loss in body condition score.

This approach requires FEC monitoring. This system requires that the plan reduces exposure to parasites on pasture (under Star 1 of the 5 Star Worm Plan).
Alternative Methods of Control to Reduce Reliance on Chemicals

To reduce the use of chemical anthelmintics, some of the following methods have been used to augment targeted selective treatment. Regardless of what methods are employed, make sure they are science-based and can work on your client’s farm.

Genetic Selection
The breeding of resistant or resilient sheep or goats can be done by selecting a breed (e.g., some hair breeds such as Barbados Blackbelly, but not Katahdin) or selecting individuals within a breed - usually rams that have lower FEC or other measures when compared to other rams in the group. Gene marker tests have been marketed in some countries to help identify sheep that will have lower FEC. Hopefully, within the next decade better tests for those genes will become identified. A saliva test (CARLA®) developed in New Zealand measures antibodies to GIN and can help to select sheep that develop immunity more rapidly or to cull animals that do not.

Remember that immunity is acquired and resistant animals still need to be infected with parasites to develop this immunity. Heritability for this trait is moderate ($h^2$ is 0.25 to 0.3), so a producer could use FEC in ram lambs or buck kids (comparison within a group) as a criteria for selecting a replacement male. But, because heritability is only moderate, genetic progress within a flock may take up to 5 years to see an impact on flock levels of parasitism. To properly select parasite-resistant males, it is important to have a large enough group to accurately find the resistant animals without sacrificing important genetic traits of production.

Also realize that goats in general do not develop immunity as well as sheep. Much less research has been done with goats on selecting for genetic resistance and so the following strategy covered in the next paragraph may work better for this species.

Rather than selecting resistant animals, it may be easier to identify and cull parasitized adults that are slow to develop immunity to parasites. This can be done by culling those with a high FEC, repeatedly have scores 4 or 5 on the FAMACHA© chart or require repeated deworming treatments. These adults should be removed from the breeding flock and ideally, lambs/kids from these animals should be sent to market rather than retained as replacements. Additionally, lambs or kids that need repeated treatments should not be retained as replacements as they are also more likely to give birth to offspring with less ability to develop immunity to parasites.

Pasture Plants Containing Condensed Tannins
Grazing pastures seeded with plants that contain high levels of condensed tannins (CT), have variably been shown to reduce shedding of eggs in the faeces. In North America, most research has been published on the legume *Sericea lespedeza* (SL), a warm-climate plant. The mechanism may be 2-fold. While there may be a direct effect by CT on the ability of the adult parasite to produce eggs and for those eggs to develop to infective larvae in the faeces, at least some of the effect is from the elevated levels of bypass protein available to the animal. Animals fed SL also have an improved immune response over animals on a control diet. Low levels of CT in the diet have been shown to increase reproductive performance and wool growth independent of parasite load. However, CT can be toxic to the animal if too high and high levels in the diet decrease feed consumption and have a negative effect on performance.

Nematophagous Fungi
A fungus, *Duddingtonia flagrans*, grows in faeces - sending out hyphae that will trap and kill the free-living forms of GIN in the faecal pellets. While these fungi occur naturally, in order to get them into the faeces in sufficient quantity to be effective, the spores must be fed to the sheep daily for a minimum of 60 days. At this point, daily dosing is not practical for grazing sheep and a bolus is being developed that will deliver spores over a longer term.

Copper Oxide
This product was first developed to supplement sheep and goats in areas of the world with copper deficiency. It appears only effective in temporarily reducing infections due to *Haemonchus contortus*. It
does not appear to improve weight gains (over controls). It does elevate liver copper levels in sheep. It is critical that copper sulphate (bluestone) should never be fed to sheep or goats.

**Vaccination**
A vaccine against *Haemonchus* is nearing commercial production stages and will require 3 vaccinations for each grazing season, usually started post-weaning. A particular antigen known as H-gal-GP appears to confer protective immunity against *H. contortus* when lambs are vaccinated. Research is also underway for a vaccine against *Teladorsagia*.

**Alternative Dewormers**
There have been many alternative or natural deworming products recommended over the years. Some are toxic to sheep and goats as well as the parasites (e.g., nicotine). Some do not work in controlled, peer-reviewed studies (garlic, papaya seeds). Diatomaceous earth has been used as an anthelmintic, but there is no scientific evidence that it is efficacious. It may be useful for control of external parasites, but more research needs to be done to show sufficient efficacy and safety. It is dangerous for humans to inhale. There are other herbal plants that have been hypothesized to be effective parasiticides, (e.g., neem oil), but at this time there is insufficient supportive scientific evidence for this claim, and safety for both animals and humans has not been demonstrated.


**STAR 4: QUARANTINE AND TREAT NEW INTRODUCTIONS**
Purchased sheep or goats may introduce parasites, and possibly AR. While performing a FEC may determine if infection is present, it may be more prudent to effectively treat the animal(s) while in isolation and then expose the animal to the farm parasites prior to mixing. Below are suggestions as to how this may be done.

**Treat All New Introductions While in Isolation**
Purchased sheep and goats should not be turned out onto pastures grazed by the flock until the possibility of AR parasites has been minimized.

**Unknown History of AR in the Farm of Origin**
Hold off feed for 12 to 24 h if possible. Treat with a full dose of ivermectin (following the rules that you need to weigh the animal and give an effective dose). Goats need a higher dose (1.5 X the sheep dose). Treat by drench, not injection. After the animal has swallowed the anthelmintic, follow up this treatment with a full dose of a BZ anthelmintic (don’t mix together) - either fenbendazole or albendazole (not in ewes or does that may be in their first 30 days of pregnancy). Goats require a higher dose (double the sheep dose). The BZ treatment can be repeated in 12 hours.

**If Resistance to ML & BZ Dewormers is Known to Be Present in the Farm of Origin**
Use either levamisole or moxidectin as in previous paragraph.

**Hold Treated Animals Off Pasture**
The sheep/goat should be held off pasture and ideally in a dry lot for at least 48 hours to allow passage of any parasite eggs. Manure from this holding time needs to be properly composted so that the resistant eggs and larvae are killed.

**Turn Animals Onto a Contaminated Pasture**
If the new introductions are still infected with GIN, they will be very resistant. This means it is important to dilute any eggs they may still be passing with “farm” parasites, which is accomplished by turning them onto contaminated pasture, ideally one which has a high level of refugia.
If No Contaminated Pasture is Available
Keep the treated animal(s) in isolation. During the grazing season, have a FEC performed on them 14 days after treatment. If still positive, consider the use of other available treatments. If the animals are purchased during the winter months, FEC may not be useful as the parasites are hypobiotic. You must do FEC in the spring prior to turnout to assess if they are still infected with resistant GIN.

**STAR 5: INVESTIGATE TREATMENT FAILURE**

**Is It Parasites That Are Making the Animals Sick?**
If animals appear not to respond to treatment, or are showing signs of parasitism despite deworming recently, investigate the reason for this. While the signs of parasitism can be very dramatic, other diseases may be the cause of poor growth or diarrhoea, or even sudden death. Poor growth can be nutritional in origin (e.g., poor pasture, selenium deficiency). Diarrhoea can be due to coccidiosis. Pulpy kidney (*Clostridium perfringens* type D) can be the cause of sudden death on lush, green pasture.

Use FEC and/or postmortem with an abomasal worm count to diagnose if a significant parasitic infection exists. Just seeing a few worms in the stomach is not “proof positive” that they killed the animals, they need to be measured and counted as described previously.

**Testing for Presence of Anthelmintic Resistance**
If the FEC is still high, perform a drench response test as described previously. Make sure a sufficient dose of the anthelmintic is delivered. If the treatment fails to reduce the FEC, you should pursue a faecal egg count reduction test. If AR is confirmed, review this document, and with your flock veterinarian develop a plan for managing parasites.

**Reestablishing a Susceptible Parasite Refugia**
If AR has been identified on a property, is it possible to reestablish susceptible refugia? The jury is still out on this one. The refugia is reduced through either leaving the pasture fallow for a long period of time, grazing with another species such as cattle or horses (not goats or sheep), or ploughing and reseeding. Lambs or kids that have been purposely infected with susceptible GIN are then introduced to seed the pasture with susceptible L3 is often not practical.
Control of Coccidiosis in the Barn and on the Pasture
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Coccidia are microscopic, protozoal parasites of the intestine. There are 11 different species of coccidia in sheep and almost an equal number in goats, but not all are considered to be pathogenic. There are subtle differences in the morphology and size of their oocysts which help to distinguish pathogenic from nonpathogenic. These parasites are host specific and do not cross infect.

Pathogenic species of *Eimeria* in sheep and goats

<table>
<thead>
<tr>
<th>Species</th>
<th>Coccidia name</th>
<th>Prepatent period</th>
<th>Pathogenicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sheep</td>
<td><em>Eimeria crandallis</em></td>
<td>15 to 20 days</td>
<td>++</td>
</tr>
<tr>
<td>Sheep</td>
<td><em>Eimeria ovinoidalis</em></td>
<td>12 to 15 days</td>
<td>+++</td>
</tr>
<tr>
<td>Goat</td>
<td><em>Eimeria arloingi</em></td>
<td>20 days</td>
<td>++</td>
</tr>
<tr>
<td>Goat</td>
<td><em>Eimeria christenseni</em></td>
<td>14 to 23 days</td>
<td>++</td>
</tr>
<tr>
<td>Goat</td>
<td><em>Eimeria ninakohlyakimova</em></td>
<td>10 to 13 days</td>
<td>+++</td>
</tr>
<tr>
<td>Goat</td>
<td><em>Eimeria caprina</em></td>
<td>17 to 20 days</td>
<td>++</td>
</tr>
</tbody>
</table>

What makes a species more pathogenic is if it infects both the small and large intestine. The small intestine has an amazing ability to recover from damage, but not so the large intestine. The life cycle of the coccidia is quite complicated as can be seen in the figure below. One coccidial oocyst can result in up to 50 million intestinal cells being destroyed. The act of schizogony (asexual reproduction) and gametogony (sexual reproduction) causes the cells of the intestine to rupture and release the next stage of the life cycle. It is important to understand the basics of this life cycle in order to understand how to best control the infection.
COCCIDIOSIS - THE DISEASE
Most often affected are youngstock - either nursing or weaned, with diarrhoea and poor growth. Adult sheep do not get disease and only very rarely adult goats. Coccidiosis as a herd/flock problem is often seen along with another disease, most often pneumonia and sore mouth (orf). This may be because the kids or lambs are “rundown” from the other. We also more often find coccidiosis as a herd/flock problem when there are other stresses in the herd/flock, for example crowded conditions or weather stresses (e.g., cold, heat, high humidity). Coccidiosis is either acute and severe, or chronic.

Acute, Severe Coccidiosis
Kids or lambs with this form of coccidiosis are notably ill and many may die without prompt and appropriate treatment. Signs may appear fairly suddenly and a kid/lamb only mildly ill the day before, may be very sick the next day. Diarrhoea is an important feature and may be watery and brown or dysenteric. The kids or lambs may be dehydrated and anaemic. They will invariably be depressed, but fever is not always present. Some animals may strain from the inflammation of the lower large intestine and pass only watery blood. Occasionally they may prolapse their rectum. In such an outbreak, it is common to have deaths.

Chronic Coccidiosis
Kids/lambs with chronic coccidiosis may have had acute, severe coccidiosis earlier or may not ever have been noticed ill. The affected group will not appear thrifty and will be growing slowly. A common finding is that they are thin, pot-bellied and small - although their heads may continue to grow giving them a runty appearance. The hind end may be dirty due to the soft stools and intermittent diarrhoea. Kids and lambs with chronic coccidiosis may never fully recover from the effects of the disease.
Age at Which Coccidiosis is Seen
Coccidiosis is a disease of young kids and lambs. The most common age to be affected is 4 weeks to 5 months. Nursing lambs/kids appear more at risk of acute, severe coccidiosis. Occasionally, kids or lambs as young as 2 weeks may be affected and rarely 1 week - although diarrhea in this age group is more often due to agents of neonatal diarrhea (rotavirus, coronavirus, and Cryptosporidium). Older animals can also be affected if not previously exposed as lambs/kids. However, if older animals appear to be suffering from chronic coccidiosis, it may be the lasting effects from an infection from when they were younger. Disease in adults is rare - again, because immunity develops within a month or two after exposure to the coccidia. It is important to remember that shedding of the coccidia eggs (oocysts) is not evidence of disease, but only evidence of infection. Adult goats and sheep often shed a small number of oocysts. It is also important to remember that severe disease can occur before infection is far enough advanced for the sheep/goat to be shedding oocysts, so absence of oocysts in the manure does not mean that the animal is not suffering from coccidiosis.

How the Damage is Done
A single oocyst, once ingested, releases 8 sporozoites which swim through the contents of the intestine and then each penetrate a cell on the lining of the intestine. Inside the cell, the sporozoite changes to a schizont. This schizont divides many times within the cell to produce up to 1,000 structures called merozoites. The cell then ruptures releasing these merozoites which then again swim through the intestinal contents. Each merozoite penetrates another intestinal cell and goes through another cycle of developing into a schizont, dividing numerous times and then again releasing thousands of merozoites. This part of the cycle of the coccidia is called “schizogony” or asexual reproduction. Eventually, the merozoites that are released change into either microgametes (male merozoites) or macrogametes (female merozoites) (“gametogony”). These again infect intestinal cells and male cells divide many times. The male cells fertilize the female cells (sexual reproduction) and in each infected intestinal cell, an oocyst or egg is formed. These oocysts burst out of the cell and are passed in the feces.

At each stage intestinal cells are invaded and then destroyed causing repeated damage to the intestine. The intestinal damage can release blood and cause inflammation of the lining of the gut. If enough damage is done, it becomes extremely ill and may die.

Diagnosing Coccidiosis
The best way to diagnose coccidiosis is based on the clinical signs shown in the group of animals and evidence of infection based on postmortem if any animals have died. Taking a fecal sample and having a quantitative count of the number of oocysts (fecal oocyst count or FOC) in the stool can also be helpful, but there are many ways it can be misinterpreted so caution must be used. A low FOC does not rule out coccidiosis. As mentioned before, acute disease may be present before the prepatent period is reached. A high FOC (even in the tens of thousands) can occur when the kid or lamb is infected with a low pathogenic species. While it is possible to differentiate the species of coccidia based on the microscopic appearance of the oocysts, it is difficult and must be done by a trained parasitology technician using special techniques. Additionally, a moderately high FOC will often be present even long after the animal develops immunity, and may rise if the animal’s immune system is stressed - all without it suffering from the disease.

Factors Affecting if Coccidiosis is a Problem in the Herd or Flock
Why does one herd/flock have a problem with coccidiosis and another does not? Why are some years, or some times of the year worse than others? We need to consider that presence of the disease agent alone is often not sufficient for a coccidiosis problem to occur, but that different factors all play a role. It is easier to consider the factors in three categories:

The Parasite
What species of coccidia are present and how pathogenic are they? How many oocysts are present in the environment and where are the oocysts? Are they contaminating places that allow for easier transmission to the kids/lambs?
The Animal
How immune is the kid or lamb? Younger animals are more susceptible. Are the kids/lambs ill with another disease that could weaken their immune systems (e.g., sore mouth or pneumonia)? Have they been stressed by changes in the diet or a poor diet, by crowded conditions, or bad weather? Have groups been mixed (e.g., younger moved in with older). Has there been fighting and competition at the feeders? Have they recently been weaned?

The Environment
The oocysts must sporulate before they are infective. Time to sporulation depends on moisture, oxygen (e.g., exposed to the air rather than buried in the bedding pack), and temperature. Exposure to sunlight will assist killing of the oocysts so pasture tends to be safer than indoor housing. Animals raised on dirty bedding are more at risk than those on slatted floors or clean bedding. Type of feeders and waters may have an effect if they are designed to prevent contamination with manure. High stocking densities, buildup over the kidding/lambing season - all increase the load of oocysts in the environment and thus increase risk of disease developing.

CONTROLLING COCCIDIOSIS
The information in the rest of these notes is not a recipe for what you should do, but rather the building blocks that you can use to develop a herd health program.

Reducing the Load of Oocysts in the Environment
Reduce Stresses to the Kids/Lambs
Do this by optimizing housing. Keep stocking densities down. Make sure there is adequate space, not just for the dams, but also the offspring. Two meters\(^2\) (2.4 sq yards) per animal is not unreasonable and 3 meters\(^2\) (3.6 sq yards) is preferred. Make sure that ventilation is adequate to prevent buildup of humidity and ammonia levels in the barn. Avoid drafts and daily temperature fluctuations. If outdoors, make sure they have shelter from inclement weather (e.g., a run-in shed). Nutritional stresses may be from artificial rearing with poor quality or poorly managed milk replacer, nutritional deficiencies from a poor diet (e.g., poorly digestible forages, inadequate vitamin E and selenium, other mineral deficiencies). Other diseases such as sore mouth and pneumonia may be worse under stressful conditions. In pastured kids and lambs, concurrent infection with gastrointestinal nematodes may increase the risk of disease from coccidia. Infections can be managed through the judicious and proper use of prophylactic anti-coccidial medications. This will be covered in depth later. Kids or lambs that are ill should be promptly treated to prevent more contamination of the environment.

Reduce Risks From the Environment
The biggest source of contamination of the environment is from kids or lambs with uncontrolled infection so it is important to control infection as mentioned above. One oocyst eaten by a kid or lamb will result in 10,000 new oocysts produced and there can be thousands to millions of oocysts excreted per gram of feces (30 grams = 1 ounce). These oocysts can survive in the barn for many months. So we can see outbreaks of coccidiosis late in the kidding/lambing season because of the buildup through the winter. For example, kidding out doelings in a pen that previously had nursing kids and dams, can be a risk if bedding was not cleaned out. Forage and grain feeders need to be designed so that manure contamination is minimized. This means not only preventing animals from defecating in the feeder, but also preventing dirty feet from going in the feeder. Bedding should also be kept fresh and dry.

Oocysts are very hard to kill. They are resistant to desiccation and many disinfectants. Sunlight will help to kill oocysts on pasture, but those in the barn are protected. Oocysts will sporulate in as little as 2–5 days at temperatures as low as 12°C, so potential for environmental buildup is massive. To disinfect: remove all organic debris. If possible, steam clean the entire pen and equipment. The extreme heat will help to kill the oocysts as well as physically remove them (they are sticky!). Ammonium based disinfectants may be most effective, but surfaces need to be initially cleaned. One product is available in Canada for premise cleaning. OO-Cide (Vétoquinol Canada Inc.) (ammonium chloride and sodium hydroxide) and should be applied with no animals present after the area is cleaned.
Reducing Risks From the Dam

Although kids and lambs are the biggest source of contamination, some advocate preventing buildup in environment from adult animals. All adults will shed a few oocysts, but in the period a few weeks before parturition and through lactation, the number of oocysts excreted may rise (periparturient oocysts rise). Yearlings have higher rises than adults. To control this, some give medicated feed to the adults during this period. This practice alone will likely not prevent coccidiosis in the offspring.

Use of Anti-Coccidial Drugs

Issues

The use of preventive anti-coccidial medications is a common method to control coccidiosis in sheep and goats. However, there are some issues that should be outlined.

- Drugs are an expense and to decide to use them, there should be a benefit to the animal higher than their cost.
- Some products are not licensed for that species necessitating the use of veterinary feed scripts.
- For those producers trying to raise animals in an organic or “natural” way, drugs should not be routinely used.
- Using these drugs requires more exacting feeding management to make sure the kid/lamb gets the correct amount.
- It also requires getting the medication into the very young animal in sufficient quantities to prevent disease.
- Even the best drug cannot protect stressed animals or those in a heavy contaminated environment.
- The goal of using these medications is to control the level of infection so as to prevent the disease, but to still allow enough infection so that the young animal develops immunity. To do that, usually the drug needs to be available from birth to 3–4 months of age, usually delivered in palatable creep feed.
- In Canada, it is legal to script medications not approved for that species into the feed. In the USA, scripts can be written if AMDUCA principles are adhered to and the medication is therapeutically necessary.

Lasalocid

Lasalocid as a feed additive: Lasalocid (Bovatec or Avatec, Alpharma Animal Health), an ionophore antibiotic, is licensed for use in lambs, but not kids. It kills the “free-living” stages of the coccidia (sporozoites and merozoites) as they move from cell to cell in the intestine. Because it kills the coccidia, it may help control disease after the animal is infected. In lambs it is approved as a feed additive to be fed free choice, at a concentration of 36 ppm = 36 mg of drug per kg of feed (36 g/tonne). To be effective a kid/lamb needs to eat 1 mg lasalocid/kg body weight per day. Underdosing is a big problem in animals that are nursing as they may not be eating enough creep to get that dose level. To determine if they are getting enough, weigh feed consumed daily, weigh the animals and calculate what they are eating. For example, a 10-kg (22-lb) kid must eat 0.28 kg of creep daily (at 36 ppm of lasalocid) to receive a therapeutic dose.

Lasalocid in the mineral: Lasalocid is sometimes mixed in the mineral although nursing animals definitely won’t eat enough, so this may be only useful for weaned kids or lambs on pasture or to control the periparturient rise in oocysts that occurs during late pregnancy and lactation in does and ewes. Again, it is necessary to be sure of intakes to prevent under or overdosing. The estimated intake of a free-choice loose salt/mineral premix is 15 g/head/day (or ½ ounce) for an adult goat or sheep. For example, for a 75-lb young adult: 34 kg BW (75 lb) so need 34 mg/day: 2265 mg lasalocid/kg = 2.265 mg lasalocid/g = (2.265 X 15) = 34 mg lasalocid per 15 g premix. But, you must manage the salt premix carefully to make sure you know how much is consumed. Too high of a dose may be toxic to sheep and goats, but fortunately lasalocid is the least toxic of the ionophores. The dose that kills 50% of animals (LD50) is 50 to 100 mg/kg BW for cattle although it is unclear if that is the same for goats and sheep.
Monensin
Monensin (Rumensin, Elanco Animal Health), another ionophore antibiotic, is licensed for goats in the USA (20 g/ton of complete feed which is the same as 22 g/tonne in Canada). It is not licensed for sheep in either country. It works the same as lasalocid. Generally, suggested feeding rates to achieve a therapeutic dose of 1 mg/kg BW/day are 11 g/tonne (ppm) free choice feed and 22 ppm limit fed. Monensin is more toxic than lasalocid. The LD 50 for sheep is 11.9 mg/kg BW/day (i.e., only slightly more than 10 times the therapeutic dose will kill 50% of the lambs!) and for goats is 26.4 mg/kg BW/day. “Decimal place” errors commonly kill lambs when the monensin is included at 110 ppm rather than 11 ppm. Feed refusal and stiffness, as well as death may occur if the levels are too high in the feed. Lambs and kids must be introduced to a ration slowly over 2–3 weeks to acclimatize the rumen organisms.

The USA label for goats in confinement is:

- **Amount**: Monensin 20 grams per ton of feed.
- **Indications**: For the prevention of coccidiosis caused by *Eimeria crandallis*, *E. christensenii*, and *E. ninakohlyakimovae*.
- **Limitations**: Feed continuously. Feed only to goats being fed in confinement. Do not feed to lactating goats. Type C feeds may be manufactured from monensin liquid Type B feeds. The liquid Type B feeds have a pH of 4.3 to 7.1 and their labels must bear appropriate mixing directions. Inadequate mixing of liquid Type B feeds has resulted in increased monensin concentration which could be fatal to goats. Do not allow horses or other equine species access to formulations containing monensin. Ingestion of monensin by these species has been fatal. Monensin medicated goat feed is safe for use in goats only. Consumption by unapproved species may result in toxic reactions. Must be thoroughly mixed in feeds before use, high concentrations of monensin resulting from mixing errors could be fatal to goats. Do not feed undiluted. Do not exceed the levels of monensin recommended in the feeding directions as reduced average daily gains may result.

Monensin is commonly used in lactating dairy cattle in Canada to reduce the risk of metabolic disease in early lactation. It is not approved for lactating dairy goats or dairy sheep and there is little evidence that it has the same benefits in those species. Regardless, to use in adults you need a veterinary script. Using a label claim to prevent coccidiosis in adult goats or sheep while milking for human consumption is difficult to justify as this disease is very rare in adults.

Decoquinate
Decoquinate (Deccox 6%, Alpharma Animal Health) is not an antibiotic nor will it improve feed efficiency. The therapeutic dose is 0.5 mg/kg BW daily for sheep and goats although there is strong evidence that 1 mg/kg BW is much more effective in those species. It is licensed for use in goats and sheep in the USA, but not Canada. Although this drug is not toxic it is much more expensive than lasalocid or monensin. Decoquinate works very early in the life cycle, killing only the sporozoites as they first infect the kid or lamb, and so is not effective in animals that already have disease. Because it is so effective early in the cycle, for the kid or lamb to develop immunity, it should be treated for a minimum of 3 cycles (e.g., 70 days) and perhaps longer.

The UK label for lambs is 1.67 kg of 6% premix per tonne of creep feed. This gives 100 grams decoquinate/tonne feed (100 ppm) and should deliver 1 mg/kg BW if fed appropriately. Feed continuously for a minimum of 28 days.

The USA label for young goats and sheep is quite different:

- **Goats (young)/Sheep (young)**:
  - **Amount**: 12.9 to 90.8 grams per ton of feed.
  - **Indications (goat)**: For the prevention of coccidiosis caused by *Eimeria christensenii* and *E. ninakohlyakimovae*.
  - **Indications (sheep)**: For the prevention of coccidiosis caused by *Eimeria ovinoidalis*, *E. parva*, *E. bakuensis*, and *E. crandallis*. 


Limitations: Feed Type C feed or milk replacer at a rate to provide 22.7 milligrams per 100 pounds of body weight per day (0.5 milligrams per kilogram). Do not feed to goats producing milk for food. Feed for at least 28 days during periods of exposure to coccidiosis or when it is likely to be a hazard. Bentonite should not be used in decoquinate feeds.

Amount: 90.9–535.7 grams per ton of feed.

Indications: See above.

Limitations: Administer as a top dress supplement or mix into daily ration at a rate to provide 22.7 milligrams per 100 pounds of body weight per day (0.5 milligrams per kilogram). Do not feed to goats/sheep producing milk for food. Feed for at least 28 days during periods of exposure to coccidiosis or when it is likely to be a hazard. Bentonite should not be used in decoquinate feeds.

The intent is to deliver 0.5 mg/kg body weight. To compare metric to USA measures requires a bit of calculation: (0.5 lb/ton of feed = [0.227 kg of Deccox/909 kg of feed]) = 0.25 kg of Deccox/tonne of feed or 15 grams decoquinate/tonne of feed - very different from the UK label! A 30-lb lamb or kid should be fed this at a rate of 1 lb/day.

Decoquinate can also be scripted to add to salt/mineral premix, but again, they need to know how much is being consumed in order to make sure enough is added to be effective. However, because decoquinate is nontoxic, it is less dangerous to error on the high side to make sure they receive enough.

As with the ionophores, it is difficult to get nursing kids and lambs to eat enough decoquinate in the feed to be effective, particularly when they are very young and very susceptible to coccidiosis. This is because the milk intake as a portion of their body weight is high so feed intake tends to be low. Winter kidding/lambing does in confinement increases this risk as does having heavily milking does and ewes (which is a good thing - but decreases the desire for the lambs/kids to eat solid feed containing the coccidiostat). You can increase the level of decoquinate in the creep ration to compensate for this if necessary; reducing it once the lambs/kids are weaned. Again this needs to be calculated based on the measured daily intake of creep and the average weight of the kids in the pen.

Decoquinate is available in another form as an additive to milk or milk replacer for kids being raised artificially (Deccox M 0.8%). It contains 8 g decoquinate/kg premix and it is mixed so that the kids/lambs receive 0.5 to 1 mg/kg BW/day. The amount added will depend on whether the milk is limited or free-choice fed. For example, a 10-kg kid needs 10 mg/day. One gram of premix contains 8 mg decoquinate, so each kid will need to consume 0.6 to 1.2 g/day. One level tsp contains 3.5 g (28 mg decoquinate), so each kid will need ~ 1/3 of a tsp per day at that body size. If consuming 10% of BW, then 1 tsp per 3 litres of milk to 3 kids. However, it is important to remember: the milk must be agitated for 5 minutes before feeding as well as during feeding to prevent settling out and under dosing, so only use to feed individuals (e.g., don’t use with a kid bar).

Amprolium

Amprolium (Ampro 9.6%, Huvepharma AD; Corid, Merial) is not approved for sheep or goats in Canada nor in the USA. It is more effective as a treatment than a control. Its mode of action is that it acts on second generation schizonts and so kills the coccidia later in the life cycle, after the coccidia have already done some damage. However, it also interferes with thiamine uptake by the intestine and so overdosing or chronic use can cause thiamine (vitamin B1) deficiency (i.e., polioencephalomalacia). Resistance of coccidia to amprolium has been reported in goats.

There are many suggested doses for kids and lambs, but the following has been reported as working well in kids as a treatment without causing problems: 50 mg/kg per day for 5 days; for lambs the dose is reported at 20 to 50 mg/kg BW/day for 3 to 5 days (higher doses at shorter intervals). Although the amprolium can be added to the water you are less sure of intakes, particularly in nursing animals, so it is highly recommended to drench individuals. Control dosages (not recommended) are 5–25 mg/kg BW/day in feed or water for 21 days.
**Sulfonamides**
These are old drugs and are to be used for treatment only. There are several types with efficacy against coccidia: sulfamethazine, sulfaquinoxaline, and sulfadimethoxine are usually given as a drench or in feed or water. Toxicity is a real risk from overdose or long-term treatment and signs are depression and kidney failure. As with amprolium resistance has been reported.

**Toltrazuril**
Toltrazuril (Baycox 5% Bayer Animal Health) is licensed to control coccidiosis in lambs in Canada. It is not a feed additive, but a drench to be given at a very specific time of the animal’s life. The recommendation is 20 mg/kg BW **once** prior to the first expected onset of disease. It is very important to understand that this drug must have a very long withdrawal for meat and must **never** be used in lactating dairy goats or dairy sheep. The meat withdrawal for lambs is 48 days. What is very different about toltrazuril is that it kills coccidia in the **intracellular** stages. All the rest of the coccidiostat drugs only kill the “free-living” stages as they swim to the next cell to infect, but toltrazuril kills the coccidia in the cell. This means that they effectively wipe out any infection in the animal when they are treated. Additionally, there is persistency of the drug reducing the need for retreatment. Because of how it works, toltrazuril must be used very differently from other anti-coccidial drugs. It is more suited for pasture-based systems when creep feeding is not used.

But, there are several factors that must be considered before deciding that this is the method to control coccidiosis:
- You must drench individual lambs/kids - and all must be treated. Even leaving one untreated lamb or kid can reinfect the remaining animals.
- The animals must be at the correct age. Treatment is done one week prior to the first time coccidiosis is seen in the youngstock and usually before there are any oocysts in the faeces. On most farms that is 4 to 5 weeks of age. This often means treating each animal when it reaches 3 to 4 weeks of age. This means you can’t treat the group at one time, but rather when the individual animal reaches that age.
- To do this, the producer must have a very good identification system, excellent records, and handling facilities - along with enough reliable help, so that it is easy for them to find, catch, and treat the lambs/kids when they reach the appropriate age.
- Because of the persistency of the drug, it can’t be used for kids or lambs slaughtered at light weights. Our recommendation is to use at least a 70-day meat withdrawal for kids and lambs.
- You must continue to monitor the animals for signs of coccidiosis. In situations where there is still a large environmental load of oocysts, it sometimes is necessary to retreat in 3 to 4 weeks - although once they show signs of coccidiosis, toltrazuril won’t work.

**Diclazuril**
Diclazuril is not currently available in Canada. Like toltrazuril, it works against the intracellular forms of coccidia. In the European Union it is available as a sheep drench (Vecoxan, Elanco Animal Health). The label indicates a single administration of 1 mg diclazuril per kg/BW most commonly at about 6–8 weeks of age, or two administrations beginning at 3 to 4 weeks of age and the second about 3 weeks later. Like toltrazuril, you need to give early in disease to prevent damage - it is not a treatment. Unlike toltrazuril, it is not persistent and meat withdrawals are shorter and so can be given at ages closer to slaughter. Again, as with Baycox - the client needs excellent handling facilities, records, and animal identification to make sure are treated correctly.

**Summary**
Coccidiosis is a common cause of disease in kids and lambs and a very important internal parasite. Although environmental control must be part of the herd health approach to this disease, judicious use of anti-coccidial drugs may be necessary to ensure adequate control. Any coccidiosis control program should be designed by the flock veterinarian as each farm and its challenges are unique.
Update on Q Fever
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Infection in animals and humans from the bacterium *Coxiella burnetii* is responsible for significant disease risk. In sheep and goats it is an important cause of abortion, stillbirth, and neonatal weakness and mortality although the organism may be present and shed in birth fluids, milk, and feces without signs of disease. Infection in these species as well as cattle is widespread. Humans who work with infected ruminants are at risk of developing mild to severe disease, called Q fever. Chronic Q fever is particularly dangerous with a high case fatality rate. Treatment with antimicrobials to control abortion or reduce bacterial shedding is unrewarding in sheep and goats. Vaccination has been shown to reduce abortions and degree of shedding, but currently no vaccine is licensed in USA or Canada. Measures to lower risk of infection in humans include lowering the level of contamination of the environment with the bacteria, understanding the signs in people so that treatment can be given promptly, and using protective wear to reduce exposure. This is an important zoonosis and education of clients and service providers is a critical component of reducing the risk of Q fever.

**Introduction**

Q fever is the name of the disease in humans caused by infection with the bacterium *Coxiella burnetii*. It can also cause disease in sheep, goats, and cattle - the disease is called Q fever or coxiellosis. Infection in animals and humans appears to be widespread and in some populations very common. Disease is less common, but when it occurs, the burden of illness is significant. A recent outbreak of Q fever in the Netherlands (2007 to 2010) was responsible for significant illness in over 4,000 people and was epidemiologically linked to nearby dairy goat and dairy sheep farms. In Canada and the USA, the disease in humans is reportable, but it is very likely that cases are underreported.

**The Agent**

*Coxiella burnetii* is a gram-negative intracellular bacterium which can infect a wide range of hosts including ruminants (cattle, sheep, and goats), swine, guinea pigs, cats, dogs, wildlife, rodents, and humans as well as birds and ticks. It has two forms, the large cell variant - which exists intracellular and grows logarithmically in an infected host, and the small cell variant (SCV) - which is spore-like, is shed in birth fluids, semen, milk, and feces, and exists outside the host. The SCV is very resistant to heat, freezing, and desiccation and can survive many months in dust, soil, manure, etc. It is this form that infects animals.

**Distribution of Infection**

Surveys of sheep, goats, and cattle have found that herd-level infection rates are significantly high. In Ontario, based on bulk-tank milk PCR - 30% of dairy goat herds and 60% of dairy cattle herds were positive, based on one sample. Prevalence was higher when 2 tests in parallel were interpreted (either test positive). More recent work in Ontario sheep flocks and goat herds found that, on a herd basis 42% of meat sheep, 64% of dairy sheep, 44% of meat goat, and 79% of dairy goat farms had at least 1 sero-reactor of 35 animals sampled, indicating infection. In a recent Ontario study of the causes of abortion in sheep and goats, *C. burnetii* was found to be the cause in about 20% of goat cases and about 10% of sheep cases submitted. However, the bacteria was found to be present on the placenta, but not the cause of abortion in 76% of goat submissions and 80% of sheep submissions strongly suggesting that *C. burnetii* infection is very common in sheep and goat farms in that province.

**Clinical Picture - Animals**

Goats appear to be most clinically affected, followed by sheep, and then cattle. Abortion rates can vary from 5% to 35%. Severe, suppurative placentitis along with abortion, stillbirth, and weak lambs/kids is commonly seen. Abortion in subsequent years is less common due to flock/herd immunity although
stillborn and weak lambs/kids may persist. Cattle may also abort, but it appears not to be as common as in sheep and goats. There is evidence that *C. burnetii* infection in cattle is associated with an increase in somatic cell counts (i.e., subclinical mastitis), but its role in infertility has not been determined. What is important to understand about this agent, is that infection is much more common than disease. However, because with infection the organism is readily shed into the environment, it is important to consider when determining appropriate control measures that include protecting the people that work with infected animals.

**TRANSMISSION AND PATHOGENESIS**

Inhalation of contaminated aerosols or dust, or mucous membrane contact with aborted materials, vaginal fluids, and membranes from normal birthing can serve as a source of infection. A cloud of organisms is present around aborting animals or even during normal parturition. Ticks may also shed the organism and contaminate the wool. Recent research in Ontario found that people are more likely to be infected when working with dairy goats. This may be because of prolonged exposure to the escutcheon area of the doe during milking, and contaminated vaginal secretions, milk, urine, and faeces. The number of organisms shed is much higher when abnormal birth events occur, but can still be significant when birthing is normal. The organism is shed for weeks after an abortion, or normal parturition, and for months in the feces and/or milk. Sheep intermittently shed in the milk, but goats and cattle are persistent shedders - sometimes for several months, particularly cattle. Pasteurization will kill the bacteria in the milk. The organism can be present in the contaminated bedding and manure for months and be a source of infection when manure is spread on dry, windy days. The bacteria have been detected up to 3 miles (5 km) downwind of an infected farm.

**DIAGNOSIS IN ANIMALS**

It is important to be able to differentiate *C. burnetii* is the cause of disease or if present only as infection. When determining if it is the cause of abortion, demonstration by immunohistochemistry and/or PCR is highly reliable method of diagnosis when combined with histopathology. There should be evidence of inflammation and the pathologist should confirm that if intracellular bacteria are seen on histopathology, that they are confirmed as *C. burnetii* - as they may be confused with intracellular *Chlamydia abortus*. When *C. burnetii* is present, but not the cause of abortion, the numbers of organisms - as determined by quantitative real-time PCR, is much lower than if the cause of abortion (median values for abortion in the range of $10^9$ and when not the cause of abortion, only present at $10^3$). Culture of the bacteria is rarely done as it requires a level 3 containment facility because of its zoonotic nature.

Serology can be done to support a suspected clinical diagnosis as animals which have aborted due to *C. burnetii* are often - but not always - seropositive as determined by IFA or ELISA. However, an animal may be seropositive and have a normal kidding/lambing. Additionally, serological status is a poor predictor of shedding. Sheep and goats may shed vaginally, in the milk, or in the feces and be seronegative. They may also be seropositive and not shed. For this reason, serology is only useful for establishing the infection status of the herd or flock and not for determining which animals in the flock are infected and shedding. It should never be used in a test and cull program.

**TREATMENT OF ANIMALS**

The current level of knowledge suggests that long-acting oxytetracycline, when injected twice at 20 mg/kg BW during mid to late gestation, is not effective in reducing the level of abortion due to *C. burnetii*. However, the studies have been small and more work should be done in this area. What is certain is that any administration of antimicrobials appears not to influence the level of shedding of the organism. For these reasons, there is no justification to recommend the administration of antimicrobials in the feed or water either in the short or long term to control *C. burnetii* in an infected herd or flock.

**Control of Infection in Animals**

Because of the environmental contamination and the longevity of the organism in the environment, control should focus on lowering the level of challenge from the environment by lowering sources of
contamination. As use of antimicrobials will not affect shedding in goats or sheep, nor prevent abortion - when most of the shedding occurs, then it is important to investigate the use of vaccines in those species. There is recent evidence that use of tetracycline at dry-off will reduce the risk of shedding at calving. In Canada, veterinarians have access to a phase I inactivated vaccine (Coxevax, CEVA Animal Health) when a modified biologic import permit is obtained through the Canadian Food Inspection Agency. It is provisionally licensed in Europe and there is strong evidence that it prevents abortion and reduces shedding in goats, sheep, and cattle. There is sufficient evidence that at this time vaccination is recommended as a control measure for preventing abortion and reducing risk to humans. Cats and rodents may also be a source for continuing recontamination of the environment. The organism can remain viable in a dried state in the environment for months and be a source of reinfection. So at this time, continued vaccination is recommended until further research indicates if the organism can be eradicated from a premise.

Q Fever in Humans
The disease in humans makes this organism very important to control in livestock. Although most people who become infected do not become clinically ill (60%), approximately 40% develop flu-like illness with about half of those becoming ill enough to seek medical attention. Approximately 5% of people with Q fever are hospitalized. A similar percentage may go on to develop chronic Q fever. The time from exposure to clinical signs is 2 to 3 weeks, but many patients do not suspect Q fever, believing instead that they have the ‘flu’ and may delay seeking medical attention for another week or two. Signs of acute illness include undulating fevers, headaches, malaise, nausea, rashes, and shortness of breath indicating an atypical pneumonia, and sometimes hepatitis, and more rarely meningitis. Acute Q fever is very responsive to the appropriate antimicrobial therapy (usually doxycycline) if provided in a timely manner. Pregnant women, who develop Q fever, are at risk of severe fetal disease and should be treated as a medical emergency. Chronic Q fever is also very dangerous to the health of the human and more difficult to effectively treat and has a high case fatality rate. It is associated with chronic fatigue syndrome and endocarditis in people with preexisting heart valve problems. Diagnosis is based on serological response of the patient (from a serum sample) and the level of phase I and phase II IgG and IgM antibodies are measured and interpreted. In a very early case, PCR performed on blood may be positive before antibodies are detected. It is important to note that many physicians are unfamiliar with the disease and the vagueness of the signs may delay appropriate therapy.

Control of Q Fever in Humans
Because of the degree of shedding, the highest risk for human infection is likely from working with parturient small ruminants - particularly if abortion is occurring. People may become infected from breathing in contaminated aerosols, handling infected placentas and lambs/kids, from being present in the barn during parturition, and from windborne organisms from infected premises or dried organisms in the dust of barns. Biosecurity precautions are very important to reduce risk to humans. Milk from dairy ruminants on infected farms is commonly infected with C. burnetii and so the consumption of raw milk is discouraged because of this. Pasteurization temperatures will kill the organism.

It is prudent for people who are considered high risk for infection (e.g., people with weakened immune systems - the elderly, those on immunosuppressive drugs), have preexisting heart valve conditions, are pregnant, or are very young - to lower their risk. This can be done by not attending lambings or kiddings and staying out of the area of the barn where ewes/does are giving birth.

However, Q fever can occur in healthy, young- to middle-aged adults so all people should consider that they are at risk. When in the barn, all people caring for livestock should wear protective clothing that is dedicated for animal use. This includes coveralls or overalls, boots, coats, mittens, hats, or any other piece of clothing worn in the livestock areas of the barn. That clothing should remain in the barn and not be brought to the house except in a plastic bag for purposes of laundering. Laundering of the clothing should be done carefully with hot water and soap and no other clothing items be washed at the same time. All lambings/kiddings/calvings should be done wearing shoulder-length plastic sleeves and afterwards, hands and arms washed thoroughly with a disinfectant soap (e.g., chlorhexidine).
potentially exposing themselves to aerosolized organisms (at birthing events or when moving manure or contaminated bedding), it is advisable to wear a fitted N95 respirator which will filter out the bacteria. A renovator’s mask is not sufficient.

Manure should be composted for at least 3 months and only moved or spread on still days to avoid creating contaminated dust. If moved off property, the manure should be covered while transported - again to avoid exposing people to the wind-borne bacteria. Aborted fetuses, placentas, or stillborn kids and lambs should be properly disposed (e.g., covered composting or burning if allowed). They should not be disposed in the manure pile.

Owners and employees on infected farms, as well as service providers that must handle the animals (e.g., veterinarians, shearsers, livestock truckers, abattoir workers), should consider themselves at high risk of becoming infected with C. burnetii. In addition to taking proper precautions, they should visit a physician if they have signs of fever, malaise, headaches, or lower respiratory disease. Because many physicians are unfamiliar with the disease, it is advisable to provide fact sheets on the signs of Q fever, its diagnosis and treatment to the physician at the office visit. These factsheet can be obtained from a number of online sources including the Centre for Disease Control (USA), Public Health Agency of Canada (Health Canada), and the Ontario Ministry of Health and Long-Term Care. A human vaccine is not readily available in Canada or the USA although a licensed vaccine is marketed and widely used in Australia.

**MANAGEMENT OF A Q-FEVER OUTBREAK**

On occasion, both animal and human disease due to C. burnetii infection is significant with more than one human case occurring in a short period of time, or evidence of spread of infection beyond one farm. The Q fever Working Group of the National Association of State Public Health Veterinarians (NASPHV) has developed a document which can be used by veterinarians to assist in managing disease in animals as well as risk to humans. The document contains details beyond the scope of this article. The document can be downloaded at www.nasphv.org/Documents/Q_Fever_2013.pdf.
Neonatal Survival of Lambs
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INTRODUCTION
Lamb losses are often quite high in sheep production, with many studies reporting that over 15% of lambs born are either stillborn or die prior to weaning. Improving neonatal-lamb survival does require understanding of the diseases that kill lambs or makes them ill, and the factors that contribute to the chance of those diseases occurring. This paper will outline some of the reasons that lambs die, and some specific health management practices that will reduce mortality.

What Are Normal Losses in Neonatal Lambs?
There have been many studies examining lamb mortality rates and risk factors. Overall stillbirth and pre-weaning mortality rates in lambs are often in the double digits. The same factors affecting survival tend to come up again and again in these studies: birthweight, dystocia, ewe mothering factors, harshness of the environment. While these figures may be “average,” we should not confuse them with being normal. Goals should be set with what is possible, rather than what usually occurs. A reasonable goal for producers is a stillbirth rate of 3% (lambs born dead/total lambs born dead and alive) and a pre-weaning lamb mortality rate of 5% (lambs dying pre-weaning/lambs born alive). Most lambs are either stillborn or die before 1 week of age, but on specific farms significant losses can occur in older lambs. Ewes tend to be fertile and over the years, most breeds have improved prolificacy - but if we as producers and veterinarians can’t prevent neonatal lamb losses, then all the improvement in lambing percentage and economic gain is lost.

Welfare Implications of Neonatal Lamb Mortality
Producers and veterinarians should also consider the welfare of the lamb. Dystocia - the cause may be due to a number of things (e.g., a large single lamb or a small first-time ewe, or a slow birth process because of poor ewe health or incorrect presentation and position of the lamb, or inattention by the producer at lambing time) can result in immediate death due to trauma or due to asphyxiation while in the uterus or birth canal. Or, if the lamb survives the initial difficult birth, it may have suffered damage to the body and/or brain so that it lacks the ability or strength to rise to drink (death from starvation) or may not be able to properly regulate its body temperature (chilling). This trauma or subsequent chilling or starvation affects its normal behavioural responses to get to its feet and successfully find the teat and nurse adequate amounts of colostrum. If it does manage to drink, it may not be a sufficient volume to protect it from infectious diseases or from starvation if the environment is cool, drafty, or wet. All these struggles: the pain of a prolonged and difficult delivery, hunger, and cold and perhaps the discomfort of illness (e.g., non-weightbearing because of joint ill or the malaise associated with fever), are stresses to that lamb that has just entered the world. As producers and veterinarians, while we may tend to see losses mostly in economic terms, we should not forget that animals also feel pain and discomfort and it is our duty to keep these at a minimum.
Risk factors for early lamb mortality (i.e., less than 7 days of age) have been well studied. Mellor and associates have summarized these risk factors by category. These factors can also be categorized by the time of life when the lamb is susceptible (e.g., *in utero*, during the birth process, < 48 h of age, and so on). Regardless of how the information is categorized, the single greatest risk factor for early lamb mortality is birthweight. Low-birthweight lambs - particularly if born into large litters - are at very high risk of death due to starvation and exposure. Very heavy lambs - in particular singles - are at risk because of dystocia. These risks are higher if born to a first-time (primiparous) ewe. Other factors are litter size independent of birthweight and gender (males have higher risk than females). Lamb behaviour and maternal behaviour influence colostrum intake - both in time from birth and amount ingested. Colostrum status can affect disease development later in life. These host factors are influenced by other factors (agent, host, and environment) in both the prenatal, parturient, and post-parturient period. The following text outlines basic flock health management procedures that will reduce lamb illness and mortality.
HEALTH MANAGEMENT PROCEDURES FOR LAMB SURVIVAL

The following pages address the important factors affecting lamb survival in: prenatal (gestating ewe), parturient (lambing), early postnatal, and later postnatal. Interventions must be attuned to the particular farm, the skill and labour available at that farm, and the goals of the producer.

Gestating Ewe Health Management

Control Common Abortion Diseases

If the annual abortion rate in the flock exceeds 5% of pregnant ewes, or if several ewes abort in a short period of time, it is important to have those abortions investigated by the flock veterinarian and the local diagnostic laboratory. Control measures must be targeted to the disease that is causing the problem so do not institute these measures without a diagnosis. While there are numerous causes of abortion in sheep, the more commonly diagnosed causes are: *Chlamydia abortus*, *Coxiella burnetii* (Q fever), *Toxoplasma gondii* (toxoplasmosis), *Campylobacter jejuni*, *Campylobacter fetus*, and iodine deficiency abortion.

Some of the more common health management strategies are:

- **Use of a vaccine** to control abortion due to *Chlamydia abortus*, *Campylobacter fetus*, or *Campylobacter jejuni*. The vaccines are boostered annually usually before breeding. Both are killed and very safe to use, but should only be used after a proper investigation determines that they are present in the flock or the flock is at high risk of bringing those diseases into the flock.

- **Use of antibiotics** in late gestation to control some of the infectious causes of abortion; best by injection and not the feed or water. Most commonly used to control *C. abortus* when given during the second trimester. Not effective if given late in pregnancy or during an abortion storm. *Campylobacter* bacteria are usually resistant to tetracycline. It is of questionable use if given to control *C. burnetii*. The risks of using long-term antibiotics are residues in the milk after lambing and the development of antimicrobial resistance.

- **Control of toxoplasmosis** by reducing kittens in the barn and fields, by preventing soiling of sheep feed, pasture, and bedding with cat feces, and by feeding an ionophore such as monensin (15
mg/head/day) or decoquinate (2 mg/kg BW/day) throughout gestation. This must be done by veterinary script (Canada).

- Assure that the gestating ewe diet contains **adequate iodine** and that it is fed so that the ewe consumes adequate amounts (e.g., added to ewe supplement or incorporated into a salt mineral premix).

**To Reduce the Risk of Hypothermia/Hypoglycemia Through Preventing Low Birthweight and Fat Stores of the Newborn Lambs**

The last trimester of pregnancy is critical for the lamb when it does most of its growing. Being shortchanged during this period will cause the lambs to be born small (< 3 kg birthweight) and with insufficient fat stores to give it energy to get up and nurse. This is special fat, called **brown fat** and - in a lamb born to a well-fed ewe in a warm environment, can supply its energy needs for over 12 hours. If fat stores are inadequate, the lamb may starve out in as little as 5 hours and in less time if the environmental conditions are cold.

**Pregnancy scanning:** There is an obvious benefit to knowing if a ewe is pregnant and fetal numbers. This will help to formulate rations that are appropriate for the number of fetuses being carried. Don’t underfeed the ewe carrying multiples as those lambs will be small and thin and much more at risk of hypothermia and hypoglycaemia.

**Increasing energy and protein to the late gestation ewe:** Late gestation nutrition is critical not only for lamb growth, but also udder development and manufacture of colostrum. As well, ewes that enter the last month of pregnancy in poor body condition are also prone to developing pregnancy toxaemia and may have other metabolic diseases which interfere with lamb health. A study which compared ability of twins born to ewes with differing body weights (55 kg & less = light; 60 kg & greater = normal), when exposed to cool (15°C) or warm (30°C) temperatures, found that twins born to light ewes and exposed to cool temperatures, developed hypothermia and died in 30 minutes whereas their twin in warm temperature survived. Light ewes, irrespective of body condition score, have lighter lambs with less fat stores and smaller placentas. Season of breeding may play a role in lamb birthweights independent of nutrition. A study performed in New Zealand found that total fetal weight (including placenta) and number of placentomes was lower in ewes bred in summer (December) versus fall (March) (Jenkinson et al. 1996). More work needs to be done to know if this changes lamb survival and growth in this country with our breeds.

**Reduce the Risk of Birth Trauma Due to “Maternal-Fetal Disproportion”**

High-birthweight lambs (> 5 kg) are at a higher risk of trauma during birth, and thus also of hypoglycaemia and hypothermia. Lambs are most often of high birthweight because ewes carrying singles are overfed during late pregnancy - either because no scanning was done and the flock is fed as if all pregnant with twins, or because the ewes are thin in mid-gestation and to increase the body condition score, they are overfed in late gestation.

Undernourished ewe lambs that are undersized will have difficulty managing the birth of a normalbirthweight lamb, let alone a “big single.” If ewe lambs are to be bred to lamb at 12 months, they must be properly fed before breeding and achieve a weight which is 70% of their breed’s mature weight. For example a breed with a mature weight of 150 lb should not be bred before the ewe lamb is 115 lb.

Overnourished ewe lambs (e.g., females pulled out of a feedlot or fed to be show fit) have 2 problems. They may be carrying too much fat in their pelvis - making them more prone to dystocia; and in the udder - reducing the quantity of milk they will produce during lactation. They also are more infertile and more prone to early pregnancy loss than ewe lambs kept at a body condition score of 3 to 3.5. Replacement ewe lambs should not be overfed once they achieve puberty - usually ~ 55% of their mature body weight.

**Optimizing Quality and Quantity of Colostrum Produced by the Ewe**

The lamb must get its neonatal immunity from the ewe’s colostrum. If the quantity is sufficient without quality, the lamb may still not be protected.
Some strategies are:

- **Vaccination of the ewe against enterotoxaemia (pulpy kidney) and tetanus.** This must be done on an annual basis with a multi-way vaccine for clostridial diseases. The vaccine when first given, must be given twice - usually about 4 to 6 weeks apart as recommended on the label, and then boostered annually. This booster is best given 4 to 2 weeks prior to the first expected lambing date, or ~120 days after the ram is introduced to the flock. Vaccinating later means that the colostrum is already in the udder and the vaccine will not improve the level of protective antibodies for the lamb. The vaccine should contain antigens of *Clostridium perfringens* type D (enterotoxaemia, also called pulpy kidney) and *Clostridium tetani* (tetanus). The vaccines contain many more antigens, but those are the most important for lambs.

- **Shearing** of the ewe can be done at the same time at vaccination as long as the ewe has protection from wind and cold for a few weeks. Shearing has many benefits: it increases the amount the ewe eats so that the foetal lambs will be a better size when born; the udder and rear of the ewe are cleaner so that the lamb doesn’t get a mouthful of manure-coated wool tag when looking for the udder; it reduces the humidity in the barn which is a risk factor for lamb pneumonia; and it keeps the udder cleaner for milking later on. Some dairy breeds have no wool on the tail but shearing is still beneficial for them as well. If the ewes are to be kept in a cold environment, they will require ~1 cm of wool coat to prevent hypothermia. If it is not possible, the wool should be removed from the escutcheon and udder - a procedure called crutching.

- **Control of mastitis** during the dry period (when the ewe is not lactating) will not only improve colostrum production, but also the subsequent lactation of the ewe. Stocking density and ventilation is important during lactation and these same rules should be applied to the late gestation ewe. Stocking density of 2 m$^2$ per ewe (just over 20 square feet); an airspace of 7 m$^3$ per ewe; with a ventilation rate of 47 m$^3$/h has been shown to improve udder health and milk production of dairy ewes. Treatment of the ewe at weaning can be done with intramammary infusion products (dry-cow mastitis treatments) or with injectable tilmicosin (Micotil, Elanco). Consult your veterinarian regarding the advisability of using these extralabel products in your flock.

- **Nutrition of the ewe** - as above - is critical to the lamb’s growth *in utero.*

- **Vitamin E and selenium.** White muscle disease, caused by a deficiency of selenium and vitamin E, can be prevented in the neonatal lamb by proper supplementation of the ewe during pregnancy. Selenium will cross the placenta right into the lamb during pregnancy whereas vitamin E needs to be ingested from the colostrum. With proper supplementation of the ewe, there is no need to inject lambs. Work with your nutritionist to make sure the correct amounts are in the ewe’s ration. Se supplementation to the ewe: 0.2 to 0.3 mg/day to the pregnant ewe; 0.7 to 1.0 mg/day to the early lactating ewe. For example, a mineral containing 60 ppm (mg/kg) of selenium, fed free choice will be usually consumed at ~15 gm/day of mineral. This means that the ewe will generally ingest 0.9 mg of selenium/day. Vitamin E should be supplemented so that the ewe ingests 250 to 500 IU/day. Stored forages usually provide none, but pasture will be adequate.

**Management of Dystocias (Difficult Births)**

Encourage your client to know when the ewes are due to start lambing. This can be done by:

- Restricting the time the ram is with the ewes (e.g., 35 days = 2 opportunities to breed for each ewe and = a lambing period of ~40 days).
- Using a ram marking harness and recording breedings (marks). Change the colour of the crayon every 2 weeks so can visually identify “remarks.”
- Having the ewes scanned and estimate foetal age.
- House the late pregnancy ewes’ close together so appetite and behaviour can be closely monitored. This will allow for detection of abortions, vaginal prolapse, and metabolic diseases such as pregnancy toxaemia or hypocalcaemia (milk fever in cows).
- Observe the ewes at least once/day for udder development starting ~1 week before first expected lambing date.
Keep lambing supplies on hand including gloves, sterile lubricant, lamb puller, etc. Be clean and use a disinfectant soap, gloves, and lots and lots of lubricant. After day 142 after the rams were introduced, observe ewes as frequently as can be managed (i.e., at least every 4 to 6 hours). This includes the middle of the night. Although it is advantageous to have ewes that “do it on their own,” you have already invested considerably in keeping the pregnant ewe bred, housed, and fed - the lamb is your profit! If she needs assistance, cull her after the lamb is weaned or at the end of her lactation. This is a reasonable compromise to “easy-care lambing” in that animal welfare is not compromised, but you should be able to apply selection pressure to the ewes.

Educate your client to intervene in a lambing if:
- Only part of the lamb appears (e.g., only the head, just the tail, just one leg).
- After the water breaks (amniotic sac), there is no progress for 30 minutes.
- The ewe has been lambing for more than 90 minutes.
- After washing up the vulva put on a glove and lubricate well. Have an assistant hold the ewe steady while she is standing. Insert your hand and identify the cervix (feels harder than the vagina). Make sure that the cervix is dilated enough to fit your hand through into the uterus. Be gentle! It’s very easy to cause damage to the lamb or ewe (e.g., rip the cervix or uterus).
- Malpresentations can be very daunting for the new producer. Here are a few tips to help you, keeping in mind that it is easy to be too rough.
  - If you can feel the lamb, identify the legs and head and which way the lamb is positioned.
  - A front leg will have the first 2 joints bend the same way (the fetlock and the carpus or knee). The third joint will bend the opposite way (the elbow).
  - A hind leg will have the first two joints bend opposite ways (the fetlock and the hock). The third joint (the stifle) will bend the opposite of the hock.
  - The head can be identified by feeling the dome of the skull and the jaw.
  - A normal presentation is the lamb is upside right and forwards (the head can be felt in the pelvis and the dome of the head is up and jaw down) and both front feet are presented (feet bend down).
  - Lambs can also be backwards (no head to feel, but can feel the tail and fetlocks bend upwards).
  - Or can be upside down (check which way the legs bend).
  - Or can have the head bent back (may feel the front legs and neck, but no head).
  - Or can have only one leg or no legs presented.
  - Twins can be tangled up so it is difficult to tell which leg belongs to which head, etc.
  - If they cannot feel the lamb (e.g., closed cervix) or gentle manipulation will not correct or produce a lamb, or if things are just too confusing call your veterinarian.

A lamb delivered from a prolonged lambing or an assisted lambing is at very high risk of mortality from hypothermia/hypoglycaemia and should receive special attention. Resuscitation of the newly delivered lamb that is not breathing can be done by clearing the airway and reviving it. Swinging or hanging upside-down should be kept to a minimum as this put stress on the diaphragm and makes it difficult for the lamb to draw air into its lungs. Rub the lamb vigorously with a clean towel. Cold water in the ear stimulates it to shake its head and breath in sharply (try it on yourself!).

**LAMB HEALTH MANAGEMENT**

**Improving Passive Transfer of Antibodies From the Colostrum to the Lamb**

When Mother Nature is working well, the lamb will be on its feet in a few minutes, attracted to the smell of the waxy gland in the inguinal region next to the udder, and to the curve of the ewe’s abdomen. These are cues the lamb uses to locate the teat. The ewe assists this process by licking and nudging the hind end
of the lamb towards the teat. This stimulates a sucking response. Ideally, the lamb should be ingesting colostrum within an hour of being born. But, we need to be prepared to intervene if things don’t go well.

This may involve:

- Putting the ewe and her lambs in a claiming pen to assist with bonding.
- Make sure the ewe’s udder is clean and dry. Crutching or shearing will help this.
- Checking the ewe’s udder to detect mastitis and to strip the plugs from her teats.

With a weak lamb, or a nervous ewe that won’t allow suckling, strip the colostrum and hand-feed the lamb. Stripping can be done by tipping the ewe up, cleaning the udder and teats and milking into a clean container. She should be able to provide a litre or more. Oxytocin can be used to help let milk down. Consult your vet for recommendations on its use. Handfeeding can be done by bottle, or if the lamb is weak - by stomach tube. How to stomach tube a lamb can be found in many publications.

Colostrum Management

If the average weight of a newborn lamb is 4 kg (slightly less than 10 lb), and the lamb needs to ingest 20% of its body weight in the first 24 hrs of life - this means ~ 200 mL/kg BW or a total of 800 mL in the first 24 hr. It is impossible for a lamb to consume this in 1 feeding - but the first feeding should be ~ 50 mL/kg or 200 mL for a 4-kg lamb. If the lamb is still too weak to nurse effectively, repeat this feeding every 6 hrs for the first day.

Make sure that in the first 24 hr, this is the first milking colostrum only. Colostrum from a ewe that lambed yesterday contains insufficient antibodies. The longer you wait to milk out the newly lambed ewe, the more dilute and less effective the colostrum will be. So waiting even a few hours can make a big difference to the quality of the colostrum.

Timing is critical! The lamb can only absorb antibodies for the first 24 h of life - but, although this sounds like a lot of time - sooner is much, much better than later.

Issues of concern are:

- If the lamb first ingests bacteria or viruses from the environment or dirty udder, then it doesn’t matter how much colostrum it ingests later - it won’t be protected from their effects.
- The ability of a lamb to absorb colostrum decreases over time so that waiting even a few hours will impair the lamb’s ability to absorb antibodies.
- Hand feeding a weak lamb may make the difference between success and failure preferably by stomach tube. Be clean about how you hand feed the lamb.

Selecting Ewes for Storing Colostrum

Older healthy ewes usually provide the best colostrum in terms of concentration of antibodies. But, careful of the following:

- Higher producing ewes may have lower levels of antibodies due to dilution.
- The health status of the “donor” ewe is important (see heat treating below).
- Selected ewes should have up-to-date vaccination programs.

Using Cow Colostrum

- It must be first milking colostrum only as well.
- Use only from a healthy, older cow without mastitis.
- Many diseases that can infect cows can also infect sheep and can be transferred in the colostrum (e.g., Johne’s disease, bovine leucosis virus) so pick your “donor” cow carefully. Cows should be vaccinated against clostridial diseases if you choose to use cow colostrum routinely.
- Cow colostrum may not last as long or be as effective in fighting disease as sheep colostrum, but is much, much better than nothing.
- Occasionally cow colostrum contains antibodies that attack the lambs’ bone marrow and the lambs become very anaemic within a few weeks of birth. When freezing cow colostrum, label the cow ID and don’t use if an issue - or pool colostrum from several cows to dilute any potential issue.
Using Goat Colostrum?
It should be avoided as goats carry so many diseases that can be shed in the milk (e.g., infectious causes of abortion, CAE, Johne’s) that can infect sheep.

Heat-Treating Colostrum
The general recommendations are to heat the colostrum to 56°C and hold the colostrum at that temperature for 60 minutes. Overheating it will cook the colostrum and destroy the antibodies. Under-heating will allow survival of disease agents. If stirring, the utensil must be cleaned between stirrings so that the colostrum is not recontaminated. Water baths - with accurate thermostats - are best for holding the temperature (must be heated prior to putting in the bath). Slow cookers are not regulated enough. Stove top can work, but requires constant attention.

Colostrum Replacement Products
There must be a concentrated source of antibodies in the product. It should use serum or colostrum as a source and have listed that it contains 100 g/litre of IgG (antibodies). Any substitute without antibodies may keep the lamb alive for a day or 2, but they generally die of septicemia or other disease within a week or two.

Storing and Freezing Colostrum
No matter how clean when milking the ewe, bacteria will contaminate the colostrum. If the colostrum is not used promptly - those bacteria will grow and degrade the quality of the colostrum quickly. If refrigerated for a few days, the colostrum should be heat treated as described above. Make sure all colostrum is clearly labelled with ewe ID, date collected, other issues about that ewe (e.g., disease status), age of ewe (older is better). Freezing is best done immediately after heat treating. Freeze in an ice cube tray and then transfer to a labelled freezer bag. Or freeze in Ziploc® freezer bags - 2 bags is preferred, one inside the other. Use a warm water bath to thaw. It is best used within 6 months, but can be used up to a year. If older, you will have to increase the volume to overcome the loss of antibodies.

Preventing Mismatching
Mismatching is a broad term and includes all the reasons for failure to mother the lamb appropriately. This can include: difficult delivery, failure to clean off the lamb, failure to allow the lamb to nurse, lambing in an unsuitable environment, poor milk and colostrum supply, and likely many other factors. What influences the ability of the dam to properly mother their lambs is also broad and can include nutritional status, size of pelvis or stature of the ewe, behavioural traits - some of which may be determined by genetics and some by management. Breed selection will solve some genetic factors (e.g., maternal genetics versus meat trait animals for milk supply), but lamb survival can also be determined by inheritance.

Protecting the Lamb in the First 24 Hours
Colostrum intake and ewe-lamb bonding is most critical to ensuring lamb survival. If the lambs are to be removed at birth for artificial rearing, then it might be simpler to do this as soon as possible. Make sure the lamb is handled colostrum as described above. Identify the lamb by ear tag or paint branding and record. Weigh the lamb and record. Record any particulars with regards to difficulty of lambing, health of ewe, status of littermates.

Preventing Navel Ill & Joint Ill
Dip the navel in a 2.5% tincture of iodine solution (alcohol-based rather than water-based) at birth. Don’t use teat dips or udder-wash products. Make sure the whole navel up to belly is included. A non-return teat dipper can be used (label for lamb navels only) or a disposable paper cup (Dixie® cup). Use a fresh dip on each lamb.

If joint ill is still a problem in the lambs and navels are being dipped properly, there may be bacteria in the soil such as erysipelas. Have the lamb joints cultured and discuss environmental cleanup or the use of prophylactic antibiotics.
**Preventing White Muscle Disease**
If the ewe has not been properly supplemented with selenium and vitamin E during pregnancy, the lamb can be injected at birth with a suitable product. Do not inject in the muscles of the hind leg at this may damage the nerve. Inject under the skin of the neck with a sterile needle. Read the label directions and only use if indicates it is appropriate for newborn lambs. Use the label dose only (e.g., ¼ cc of 3 mg Se/mL product)! There have been several cases of overdosing newborn lambs at birth with selenium and vitamin E. This will result in death.

After 24 h when the lamb has consumed adequate colostrum and only if it is strong and healthy:
- Tail dock breeds with long, woolly tails.
- Castrate if males are to be raised for meat and kept over 5 months of age into the fall.

**Protecting the Lamb in the First Week of Life**

**Artificially Reared Lambs**
Instruct the owner to hand feed 3 to 4 times per day for the first few days if weak. Each feeding should be at 50 mL/kg BW. Keep in a warm, draft-free location in the barn that can be frequently disinfected if diarrhoea should develop. Although a head lamp is great to help dry off lambs after birth, do not use afterwards as this will encourage piling and may lead to pneumonia.

If strong at 3 days, move to a lamb bar, but continue to pay close attention to the lambs’ feeding behaviour. The care the shepherd takes now to make sure the lamb gets up and suckles from the lamb bar will pay back in improved lamb survival and growth.

Only use high quality milk replacer products developed specifically for lambs (e.g., 22% milk protein and 28% fat). Cow milk and calf milk replacer doesn’t have enough energy for lambs.

**Preventing Hypothermia/Hypoglycemia**
This disease can be prevented if the lambs are drinking enough (see above) and are in an environmentally suitable area (draft free and dry). Stocking density, airspace, and ventilation are critical as outlined previously above. If with dams, keep lambs < 7 days close so that can be observed frequently (4 times per day) for abomasal fill and attitude. If the lamb is empty, wrinkled, and tucked-up, then prompt treatment using the Moredun method will save many lives.

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**TREATING HYPOTHERMIA (CHILLING) AND HYPOGLYCEMIA (STARVATION) IN VERY YOUNG LAMBS**
The following was developed by: Dr. J. Jansen (OMAF), P. Menzies (University of Guelph), and J. Martin (OMAF, retired). This is a summary of the factsheet “Hypothermia in Newborn Lambs.” Two other factsheets are available concerning lamb survival: “Assisting the Ewe at Lambing” and “Care of the Newborn Lamb.”

Talk to your veterinarian before lambing season begins. Discuss and review any techniques used to revive chilled lambs.

**Reasons for Illness in Lambs**
There are many reasons for very young lambs to be sick. This section addresses only chilling and starvation. For other disease conditions, consult your flock veterinarian.
Reasons for lamb death

- Abortion
- Death during Lambing
- Chilling - Exposure
- Chilling - Starvation
- Infectious Disease
- Congenital Disease
- Misc

Supplies to Revive Chilled Lambs

Have these items on hand before lambing begins:
- Digital rectal thermometer which measures subnormal body temperatures
- Frozen colostrum in small batches (150–250 mL or 5–8 ounces)
- Lamb stomach tube (soft rubber) and feeding syringe (60 mL) or squeeze bottle (250 mL)
- Warming box with heater and thermostat
- Dry, well-bedded aftercare unit with heat lamp
- Draft-free pens that are dry and well-bedded
- Bottle of sterile 50% dextrose (500 mL bottle)
- Kettle for boiling water
- Sterile 60-mL syringe and 20-gauge (pink), 1-inch hypodermic needles

Recognizing and Treating Hypothermia

- To best recognize hypothermia, take the lamb’s rectal temperature and observe its behaviour.
- The rectal temperature should be checked of any dull, weak lamb that seems unable or unwilling to suckle. The sooner action is taken, the better the lamb’s chances of survival.

The basis of treatment of the hypothermic lamb is to **warm it up and provide a source of energy** to start heat production again.

**Mild Hypothermia - Any Age (Temperature 37–39°C)**

- The lamb is weak, depressed, empty appearing, but can stand.
- Move lamb into shelter; dry off if wet.
- Feed colostrum by stomach tube. Feed 50 mL/kg of body weight (within the first hour of birth is best). Additionally feed 200 mL/kg body weight spread over 3 more feedings within the first 24 hours.
- Lamb can stay with ewe provided she is in a sheltered area.
- **Ensure** lamb is nursing.
- **Recovered** once rectal temperature returns to normal; lamb and ewe can return to flock.
- Small lambs < 1.5 kg (3 lb) at birth, may not have sufficient fat reserves to initiate heat production, even with colostrum.

**Action:** In addition to colostrum, feed these small lambs an **extra 50 mL/kg** of a 20% dextrose solution by stomach tube 1 hour after the colostrum feeding.
Moderate to Severe Hypothermia (Temperature ≤ 37°C)

How old is the lamb?
- Lambs over 5 hours old should be considered hypoglycaemic (starved) as well as hypothermic. Do not warm before administering colostrum or glucose.

Can the lamb suckle and swallow?
- Lambs with a suckle reflex can be tube fed. Lambs without a suckle reflex will need to be revived using intraperitoneal dextrose and then warmed prior to being tube fed.

≤ 37°C; < 5 h old; & suckle reflex (able to swallow)
Lamb is weak, empty, depressed, and may be unable to stand.

Actions:
- Remove lamb from ewe and dry off if wet.
- Place in warming box until rectal temperature is > 37°C.
- Administer warm colostrum by stomach tube - 50 mL/kg BW.
- Additionally feed 200 mL/kg body weight spread over 3 more feedings within the first 24 hours.
- Move to hospital pen with heat lamp and feed until strong and maintaining normal temperature (39°C).
- Once strong, return to dam, but make sure is nursing (identify using livestock paint or marker).

≤ 37°C; > 5 h old; & suckle reflex (able to swallow)
Lamb is tucked up, empty appearing, and depressed. Assume that lamb has no fat stores and is hypoglycaemic (starved). You must provide an energy source before warming.

Actions:
- Remove lamb from ewe and dry off if wet.
- Administer warm colostrum by stomach tube - 50 mL/kg BW prior to warming!
- If you warm the lamb first, it will convulse and die.
- Place in warming box until rectal temperature is > 37°C
- Again administer warm colostrum by stomach tube - 50 mL/kg BW. Additionally feed 200 mL/kg body weight spread over 3 more feedings within the first 24 hours.
Move to hospital pen with heat lamp (e.g., box in warm environment) and feed until strong and maintaining normal temperature (39°C).

Once strong, return to dam, but make sure is nursing (identify using livestock paint or marker).

≤ 37°C; > 5 h old; & no suckle reflex (not able to swallow)

Lamb is often unable to stand. Do not attempt to stomach tube, as this will result in the milk/colostrum being deposited in the lungs, which will kill the lamb.

**Actions:**
- Reverse the hypoglycaemia first before warming or lamb will convulse and die!
- The lamb must first be injected with a sterile solution of warm 20% dextrose at a dose rate of 10 ml/kg body weight into the abdominal cavity (intraperitoneal). See below for method.
- Place in warming box until rectal temperature is > 37°C.
- Once revived and with a suckle reflex, administer warm colostrum by stomach tube - 50 mL/kg BW.
- Additionally feed 200 mL/kg body weight spread over 3 more feedings within the first 24 hours.
- Move to hospital pen with heat lamp (e.g., box in warm environment) and feed until strong and maintaining normal temperature (39°C).
- Once strong, return to dam, but make sure is nursing (identify using livestock paint or marker).

**Techniques Used to Revive Hypothermic and Hypoglycemic Lambs**

Using a stomach tube to administer warm colostrum:

- Sit with the lamb restrained on your lap. Measure the tube.

  - The tube is passed into the side of the mouth in the space between the front and side teeth.
  - Using gentle pressure, the tube is slid into the esophagus and down to the stomach.
  - The tube will move easily; **any** resistance or **coughing** indicates that the tube has entered the windpipe and it should be removed immediately.
  - The accidental passing of colostrum into the lungs will result in aspiration pneumonia and the death of the lamb.
  - The esophagus is behind/beside the windpipe on the lamb’s left. By placing your fingers on each side of the lamb’s throat, you should be able to feel two tubes while sliding the stomach tube in; you will feel the windpipe and the tube passing down the esophagus.
- Slowly administer the warm colostrum either using a 60-mL feeding syringe or a 250-mL squeeze bottle.
- Colostrum should be administered over 5 minutes.
- Crimp the end of the tube over prior to removing to prevent aspiration.

Sourcing and warming colostrum to feed to hypothermic lambs:
Colostrum from a lamb’s dam is best, other options listed in order of preference:
- Individual healthy ewe colostrum from the same flock.
- Pooled ewe colostrum from the same flock.
- Pooled ewe colostrum from another flock (same disease status or better).
- Pooled cow colostrum (use 30% more; feed every 5 hours in the first 24-hour period).
- Any combination of the above.
- Commercial colostrum replacement product.

Johne’s disease can be spread from infected cows and ewes through their colostrum. Use cows from a Johne’s tested herd only. Occasionally lambs may develop severe anaemia from cow colostrum. Always identify source of colostrum so problem colostrum can be discarded.

Thaw frozen colostrum in a water bath at 35°C. Never microwave colostrum; it will destroy the proteins destroying the antibodies in the colostrum.
Administering dextrose solution using an intraperitoneal (IP) injection:

- With a sterile, 60-mL syringe, draw up 20 mL of sterile 50% dextrose using a sterile needle.
- Boil clean water and draw up 30 mL of this water into the same syringe.
- This will provide 50 mL of warm (38–40°C) 20% dextrose solution.
- The dose is 10 mL per kg bodyweight; 50 mL is sufficient for a 5-kg lamb.
- The lamb is suspended vertically by the forelimbs.
- The injection site is 2.5 cm (1 inch) below and to the side of the navel.
- Use a 20-gauge (pink), 1-inch needle.
- The needle is inserted at 45° to the body wall (the needle is pointed in the direction of the lamb’s pelvis). Ask your veterinarian to show you how to do it.
- The internal organs will be pushed away by the needle and not damaged.
- Both the conscious and comatose lamb can be injected in this manner.

Warming a hypothermic lamb - temperature ≤ 37°C:

**Slowly** warm the lamb to restore body temperature (until it rises to 37°C). There are several acceptable methods to warm a lamb, but some are more effective at increasing temperature.

- A warming box which allows circulation of warm air around the lamb (see diagram below).
- A water bath warms most quickly, but requires holding the lamb to prevent drowning, and immediate drying (towels and hair dryer) to prevent chilling again. This requires the most labour.
- Heating pad and radiant heat. Both will warm the lamb, but there is a risk of burning if used improperly.
- Heat lamp alone is not recommended as it only warms one side.

- Do not warm before administering an energy source (IP dextrose or warm colostrum).
- Check rectal temperature every 30 minutes to avoid overheating. A warm air heater is the preferred method.
A warming box can be constructed from plywood and wire mesh. Heat source may be a fan heater with thermostat (preferred) or hair dryers (must monitor temperature). A piece of Plexiglas in the lid allows for lamb’s condition to be monitored.

Warming a hypothermic lamb - temperature 37°C to 39°C:
- A heat lamp can be used to warm the lamb along with warm colostrum.
- Keep separate from the ewe until strong.
- Suitable containers are disposable cardboard boxes, washable tubs, or small pens made with square straw bales.
- Make sure that can disinfect area if a disease outbreak occurs (e.g., scours).
- Return to the ewe once lamb is strong enough to nurse unaided.
- Identify the lamb with livestock marker and keep in a small area so can observe easily. Watch for signs of rejection.
- Lamb may need to be reared artificially if fails to thrive on the ewe.

Preventing Other Diseases of Nursing Lambs

Abomasal Bloat
- Feed cold and free-choice to reduce gorging too much milk at once. Feeding free-choice will increase intakes as well as growth rates and decrease losses from bloat.
- Adding 1 cc of 37% formalin to each litre of milk will reduce the growth the bacteria in the milk, but may also reduce intakes.
- Work has been done on feeding acidified milk to kids (adding formic acid to obtain a pH of ~ 4.5) similarly to calves. This is to decrease bacterial growth in milk that if fed free-choice in a lamb bar.

Diarrhea
Diarrhea disease agents are shed by the ewe and buildup in the environment over the lambing season. When a lamb becomes ill, it sheds even more of the disease agent and contaminates the lambing pen and wherever else it is housed. Control of diarrhea is a function of optimizing passive transfer of antibodies, keeping the environment clean, and preventing exposure of newborn lambs to the disease agent from the older lambs.

If an outbreak of diarrhea occurs:
- Move pregnant ewes into a new lambing area.
- Clean and disinfect the old lambing area before using again.
- Do not add newborn lambs to the pens containing sick lambs, but start a new area that is clean.
- If possible, have different people tend to the affected pens of lambs from the newborn lambs. If not, then have different coveralls between groups and wash hands with a disinfectant soap (e.g., chlorhexidine).
Any equipment used between groups of affected and healthy lambs should be disinfected first. Diarrhea can be treated with oral electrolytes used in treating calf diarrhea. Consult your veterinarian on how to do this.

**Pneumonia**
The risk factors for pneumonia in lambs are not difficult to understand. The organisms responsible for making the lamb sick are already present in most animals, but the environmental conditions and general lamb health can determine if the animal gets sick or not.

What is difficult is observing lambs closely enough to determine if they are ill. If you jump in the pen, then even a very sick lamb may jump up and run away. Watch from outside the pen and observe abnormal behaviour and stance that may indicate it is sick (e.g., tucked up, separated from the group, not up at lamb bar or feeder when other lambs are, head down).

What you often won’t see in the acute stage of the disease is heavy breathing, nasal discharge, and coughing - those come later after the lamb has been sick for 3 to 5 days (if it is still alive).

Catch the lamb and take it’s temperature. A normal temperature for a lamb is 39 to 39.5°C (about 103°F). If the temperature if 40°C or above (104°F) then the lamb has a fever. Set up a treatment protocol with your veterinarian on how to treat lambs with pneumonia.

Treating lambs with antibiotics in the feed or water is often done, but has significant risks. Lambs that are ill don’t eat or drink enough to get a therapeutic dose so are under treated. Using that antibiotic often will result in resistance and it won’t work when you really need it.

**Coccidiosis**
There are few - if any - flocks that do not have some level of coccidiosis in the lambs. Like neonatal diarrhoea, the disease agent builds up in the environment so that new lambs, housed with older animals, are more likely to develop severe disease. See Control of Coccidiosis in the Barn and on the Pasture presentation for more information.

**Gastrointestinal Parasites in Pre-Weaned Lambs**
Only lambs at pasture, or a dry lot with grassy sections that allow grazing, are at risk of being infected with gastrointestinal nematode parasites. Lambs raised indoors, or during the winter months, are not at risk. See Control of Gastrointestinal Parisitism – Part 2 for more information.

**Urinary Calculi**
Most stones in young lambs are due to too much phosphorus and not enough calcium in the diet and are not common when still receiving milk or milk replacer. Signs may appear like constipation (i.e., straining as well as depression). Treatment is often unrewarding.

Calcium phosphorus ratio of the diet should be ~ 2:1 and lambs must have fresh, palatable water all the time. Addition of sodium chloride (1%) to the diet will help increase water consumption. Vitamin A should be added to the diet if no fresh forages are available. Ammonium chloride at 0.5% to 2% will drop the urine pH to 6.5 which will help to dissolve stones. Avoid diets high in magnesium.

**Pulpy Kidney/Enterotoxaemia**
The spores of the bacteria *Clostridium perfringens* type D are very common in the environment so lambs are always at risk of this disease - which most commonly presents as sudden death and rarely as a lamb with neurological signs. If the ewes are properly vaccinated as outlined above, then the lamb is protected through the antibodies in the colostrum, until about 3 months of age. At that time all lambs that will be retained past 4 months should be vaccinated with a multi-way clostridial vaccine - exactly according to the label recommendations. Lambs that will enter the breeding flock will need to receive a booster once/year to remain protected.

**INVESTIGATION OF LAMB DEATH**
Prompt necropsy of all dead lambs is important when trying to prevent lamb losses. By weighing and making observations of the external and internal appearance of a dead lamb, it can be decided the most
likely cause of death. See Appendix 1 for a simple form that can be used to record findings of your lamb necropsies.

**Easy-Care Lambing**

In New Zealand, many flocks are not closely monitored at lambing. This is because of the large numbers of animals and large geographic areas grazed by ewes. There is debate that by grouping ewes and interfering with birth, we are interfering with nature and not necessarily improving lamb survival. Easy-care lambing dictates that ewes should be able to adapt and survive in adverse climatic conditions, to successfully lamb and rear at least one lamb without assistance, to have a high survival rate, lower lamb mortality, and require less shepherding. Lower lamb mortality would be attributed to a lower incidence of dystocia, reduced lamb starvation, and better maternal behaviour.

One method of accomplishing these goals is through strict ewe selection. The producer must keep good records of ewe performance and cull all of those animals that don’t meet these criteria. For example, cull all ewes that require assistance at birth, have udder problems, develop a vaginal prolapse, or don’t raise or require help to raise lambs. Ewes with pregnancy toxaemia, if treated and survive, are not kept. Ewe mortality at lambing should decline to less than 2%, but may be high in the early years. Lambs from those ewes that survive to be culled, should not be retained as replacements. Lambs that require treatment, or supplemented with milk replacer should not be retained as replacements. It can also be done by breed selection (i.e., use maternal breeds that have a record of effectively rearing lambs in the required environment).

Additionally, ewes need to be familiar with their environment so they can better exploit it. This can be done by set stocking ewes to a property or pasture and not moving them. Called hefting, lambs born on this property learn the best places to graze, or to rest, or to give birth from their mothers. They in turn, use that information when giving birth and raising lambs. Moving them to new pastures, or bringing in sheep that aren’t used to the environment, is associated with poorer survival of lambs.

**Ewe Longevity**

A mature ewe is generally a better mother than a ewe-lamb. But, only to a certain point. If the ewe has a history of lamb rejection, pregnancy toxaemia, vaginal prolapse, mastitis, or difficult lambing, she is not a
good bet to be better the next time. But, what characteristics of ewes are important in maintaining their productivity as mothers in the flock (i.e., length of production life [LPL])? Most ewes are culled after their first lambing. Lamb mortality is highest in primiparous ewes and doesn’t generally reach that high again until ewes have lambed 8 times. Age at first lambing affects culling, with ewes lambing at < 13 months of age at lower risk of leaving versus those giving birth between 13 and 15 months of age. So, selection for maternal characteristics of ease of lambing, early maturing, and suitability for the production system should be done to assure ewe longevity.

**SUMMARY**
The difference between profit and loss on most sheep operations is the number of lambs weaned per ewe in the flock. Keeping lambs alive and growing well is one of the most profitable things you can do for your sheep client.

**APPENDIX 1 INVESTIGATION OF NEONATAL LAMB DEATH**

<table>
<thead>
<tr>
<th>Farm name:</th>
<th>Date:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Lambing start date:</th>
<th>Expected end date:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Ewe and litter information**

<table>
<thead>
<tr>
<th>Ewe ID:</th>
<th>Age of ewe (yrs):</th>
<th>Body condition score (circle):</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1/2/3/4/5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Litter size: 1/2/3/4+</th>
<th># stillborn:</th>
<th># currently alive:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Type of birth (circle): unobserved/unassisted/easy assist/hard assist/C-section</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
</tbody>
</table>

**Lamb information**

<table>
<thead>
<tr>
<th>Date of birth:</th>
<th>Date of death:</th>
<th>Birthweight:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Born dead? yes/no/unknown</th>
<th>Term birth? yes/no/unknown</th>
<th>Nursed? yes/no/unknown</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Other history:
Include any treatment history, history of abortions, etc. that is relevant

**SUMMARY OF FINDINGS**
Using the information obtained from the necropsy, fill in your tentative or final diagnosis below:

<table>
<thead>
<tr>
<th>Summary of important findings:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Death occurred</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before birth</td>
</tr>
<tr>
<td>----------------</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

Death appears to be due to:

Consider: Abortion, trauma or hypoxia due to difficult birth, mismothering, starvation, hypothermia, trauma/misadventure, diarrhoea, pneumonia, septicemia, tail docking infection, white muscle disease, predator attack, iodine deficiency, coccidiosis. Consult veterinarian if diagnosis is open.

**Recommendations for control:**
<table>
<thead>
<tr>
<th>Observation</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight of lamb:</td>
<td></td>
</tr>
</tbody>
</table>
| < 3 kg: poor ewe nutrition or in utero disease  
| > 5 kg: may have birth trauma |

**Lay lamb with its left side down and observe:**

**External appearance:**

- O Yellow (meconium)  
  *Meconium indicates in utero stress prior to birth*
- O Birth fluids/placenta on wool coat  
  *Indicates that the ewe didn’t clean the lamb off*
- O Swelling of head or legs or tongue  
  *Suggests a prolonged/difficult birth*
- O Short or absent wool coat  
  *Suggests the lamb is premature or iodine deficiency*
- O Yellow fluid under tail  
  *Likely diarrhoea. Meconium is thick and pasty*
- O “Watery” abdomen (anasarca)  
  *Suggests an abortion disease or congenital deformity*
- O Infected tail docking site  
  *Lamb may have died from septicemia or tetanus*

**Feet:**

- O Foot pads intact or worn  
  *If foot pads present, suggests lamb never walked*

**Umbilicus is:**

- O Wet, square end, no blood inside  
  *Lamb died before or during birth process*
- O Wet, tapered end, no blood clot  
  *Lamb alive at birth, but died very soon after*
- O Moist, tapered end, blood clot inside  
  *Lamb alive for at least 2 h, but died before 1 day old*
- O Dry, but present  
  *Lamb alive for longer than 1 day of age*
- O Swollen, may express pus  
  *Navel infection present (navel ill)*

Using a sharp scalpel blade, cut the skin along the bottom from the pelvis to the chin. Skin the lamb so that the right front leg can be reflected back. Cut through the hip joint of the right hip to reflect back the right hind leg. Carefully cut the abdominal muscles and through the ribs near the sternum and pull back the ribs and muscles to reveal the inside of the thorax and abdomen. Assess:

**Fat stores** (white-pink, lumpy material):

- O Present on both heart & kidney  
  *Lamb did not starve to death*
- O Present in kidney, but not heart  
  *Lamb was in negative energy balance, but didn’t starve*
- O Absent on both (fat appears jelly-like)  
  *Lamb starved*

**Lungs:**

- O Are dark and do not float  
  *The lamb did not breathe after birth*
- O Are pink and float  
  *The lamb did breath after birth*
- O Are solid and dark in the bottom front sections  
  *Pneumonia may be present*
- O Have abscesses  
  *May have chronic pneumonia or septicemia*

**Abomasum:**

- O Is empty  
  *Lamb did not drink*
- O Contains milk or colostrum  
  *Lamb either drank or was tubed (see history)*
- O Contains foreign material (e.g., straw)  
  *Lamb attempted to nurse, but did not find the teat*

**Intestines:**

- O Are empty  
  *Lamb died before colostrum left abomasum*
- O Contain milk or colostrum  
  *Lamb alive long enough for colostrum to enter intestines*
- O Contain fluid material (light or dark)  
  *Consider diarrhoea. If bloody may be coccidiosis or salmonellosis*

**Internal organs:**

- O Blood in the abdominal cavity and carcass is pale  
  *Organs or umbilicus ruptured, usually during birth, and lamb bled out*
- O One or more ribs are fractured  
  *Ribs fractured during birth if haemorrhage is present*

**Legs:**

- O One or more joints are swollen  
  *Lamb may have joint ill (open joint will have pus inside)*
- O Tissue underneath skin of legs is red & edema  
  *Lamb may have been very hypothermic prior to death*
<table>
<thead>
<tr>
<th>Other:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>O Thyroid glands in neck are enlarged</td>
<td>*Lamb may be iodine deficient (goitre)</td>
</tr>
<tr>
<td>O Muscles of heart, hips, or shoulder are white</td>
<td>*Lamb may have white muscle disease (vitamin E/selenium deficiency)</td>
</tr>
<tr>
<td>O Brain has blood clots or haemorrhages</td>
<td>*Brain trauma may have occurred, likely during birth</td>
</tr>
<tr>
<td>O One or more limbs are fractured</td>
<td>*If haemorrhage present, fracture occurred before death</td>
</tr>
</tbody>
</table>
OTHER – VETERINARIANS AND ANIMAL HEALTH TECHNICIANS (AHT)
WELLNESS

The Ups: What Veterinarians and Technicians Love About Their Jobs
Jean E. Wallace, PhD
Department of Sociology, University of Calgary, Calgary, AB, Canada

ABSTRACT
Growing awareness about increasing numbers of professionals suffering from stress, addictions and suicidal ideation has resulted in an emphasis on “what’s wrong” with the professions and/or their members. In this session, we explore “what’s right” with veterinarians’ and technicians’ work experiences and what enables them and their work community to thrive. Both interview and survey data were used to identify the best parts of their work and what gives them the greatest satisfaction. These factors are examined in relation to positive wellness outcomes to see which are most beneficial to veterinarians’ and technicians’ wellbeing.

INTRODUCTION
Much of the research on worker experiences looks at how to prevent work-related problems such as job stress, burnout or quit behavior. An alternate approach is to examine what contributes to positive work experiences that enhance worker potential and happiness. By examining what veterinarians and technicians love about their jobs, we can gain a better understanding of the subjective work experiences that may enhance their wellbeing, their hope and optimism for the future, and their day-to-day happiness. This newsletter explores the positive aspects of veterinarians’ and technicians’ jobs that study participants identified as the best part of their jobs that give them the greatest job satisfaction.

Job satisfaction is defined as the degree to which workers enjoy their job. Job satisfaction has been linked to many positive outcomes for individuals, such as a greater sense of wellbeing and contentment. It has also been linked to positive outcomes for the workplace, such as greater productivity and commitment to remain in their job or profession.

THE STUDY
Table 1 shows the extent to which veterinarians and technicians are satisfied with their work. Most feel they are enthusiastic about their work most days (78%), they find real enjoyment in their work (79%) and they definitely like their work (87%).

As one veterinarian wrote: “I really enjoy doing my job. I love the work that I perform as a veterinarian. I love working with the patients and the clients. I love the physical nature of the job and traveling from call to call.”

And a technician expressed her job satisfaction as follows: “Working with the patients and their owners to improve their pet’s quality of life. Building rapport with the owners and working with a great team of individuals.”
Table 1. How satisfied are you with your work? (N = 829)

Comparisons were made across a number of factors to see if any groups are significantly more satisfied than others. There were no statistically significant differences in the satisfaction scores for veterinarians vs. technicians, by supervisory status or job position, or by age group. There are too few men to make meaningful gender comparisons for technicians, but the gender comparisons for veterinarians’ satisfaction scores showed no significant difference. While there was no significant differences in job satisfaction for veterinarians who work in clinic settings compared to those in other settings, technicians working in clinics reported significantly higher job satisfaction scores than those working in other settings.

WHAT THEY LOVE ABOUT THEIR JOBS

Both interview and survey data were used to identify the best parts of veterinarians and technicians’ work by asking them to describe what gives them the greatest job satisfaction.

Four popular sources of enjoyment were identified that include:
12. Making a difference by helping animals
13. Making a difference by helping clients
14. Having challenging interesting work
15. Having supportive coworker relationships

Participants’ descriptions of these sources of job satisfaction are presented below. In addition, data from the survey provide frequency distributions of how often they experience these different aspects of their jobs.

Making a Difference by Helping Animals

Veterinarians and technicians alike identified making a difference in the lives of animals in terms of working with and helping animals as one of the most satisfying parts of their job. They described the enjoyment of caring for animals and making a difference in their quality of life. When asked to describe what they enjoy the most or gives them the greatest satisfaction in their job, some participants responded as follows:

“Helping animals live longer healthy lives.”
“Contributing to healthy special relationships between people and their pets.”
“Promoting/emphasizing and protecting the human-animal bond.”
“When I am able to successfully help an animal improve their mobility and quality of life and their owners have more time to spend with them or are able to continue their activity of choice.”
“Knowing I’m doing something good in the world. Helping those who can’t help themselves.”

Table 2 presents the frequency distributions that show how often veterinarians and technicians feel that their work allows them to make a significant contribution to the health and wellbeing of animals, to
do good things for animals, and to be involved with the human-animal bond in a positive way. On average, about half of the respondents report they experience these different ways of making a difference and helping animals “most of the time” in their work and one third reported they experience them “often.”

Table 2. How often do you do make a difference helping animals in your work? (N = 829)

<table>
<thead>
<tr>
<th>% of Responses</th>
<th>Significant contribution to animal health and well being</th>
<th>Do good things for animals</th>
<th>Involved in human-animal bond in a positive way</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never/Not very often</td>
<td>37%</td>
<td>34%</td>
<td>37%</td>
</tr>
<tr>
<td>Sometimes</td>
<td>48%</td>
<td>48%</td>
<td></td>
</tr>
<tr>
<td>Often</td>
<td>56%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Most of the time</td>
<td>48%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Making a Difference by Helping Clients
Making a difference by helping clients was also identified as a source of satisfaction for many veterinarians and technicians. For example, they described how it is satisfying to get to know clients, to interact with them, and to educate and help them care for their animals and pets:

“The good days for me mostly come from the clients. Client reactions and interactions. I feel good “inside” and I feel appreciated.”

“Giving an answer to a client about a pet’s ailment, good or bad, and seeing them understand.”

“Getting to know clients and their horses and becoming a part of their team, as well as the many friendships and relationships I have formed as a result of my work.”

“I love seeing the look on an owner’s face when their sick animal is better and they get to take them home. I like the feeling I get when I am able to make a difference in people’s and their pets’ lives.”

“The good clients that are truly appreciative for what I do here and never want me to leave. Being able to touch lots of lives in so many ways through what I do.

“The ability to feel valued for the work that you do and the ability to help someone’s best friend when they feel so helpless.”

Table 3 shows how often veterinarians and technicians feel they make a difference in their work by helping their clients. About three quarters (72%) feel satisfaction from helping their clients “often” or “most of the time” and about two thirds (68%) report that their clients regularly thank them for caring for their animals. One third (39%) regularly feel invigorated after working with clients.
Table 3. How often do you make a difference by helping clients in your work? (N = 829)

Challenging Interesting Work
Challenging, interesting work was another popular theme regarding the most satisfying aspects of the job. Participants described how their work was never the same from one day to the next, how they enjoyed the variety of tasks, problems and/or animals they worked with, and how they are constantly learning new things and using different skills.

“Having a career that challenges me every day. Diagnosing and treating a medical issue that gives an animal better quality of life.”

“Work is always different... I’m always learning something new.”

“I get to use both my brain and my technical skills every day, I feel my work is stimulating.”

“Variety in my day-to-day work with new challenges to face and learn from. Constantly learning new skills and challenging myself.”

“Wide range in health care abilities I perform: nurse, pharmacy tech, medical imaging tech, dental hygienist, anesthesiologist, lab tech, etc. I enjoy the change and variety.”

Table 4 shows how often veterinarians and technicians experience challenging, interesting varied work. More than half feel that most of the time their work is meaningful (57%) or that it lets them use their skills and abilities (55%). Almost half feel that most of the time their work requires that they learn new things (45%), is varied and interesting (44%) or involves doing a lot of different things (46%). Far fewer report that they are regularly required to be creative in their work.

Table 4. How often do you have challenging work? (N = 829)
Supportive Coworkers

Lastly, participants described how important their relationships with coworkers are in making their job enjoyable. They explained how having good people to work with who supported one another was key to enjoying work. Some of their descriptions are as follows:

“I enjoy when I have accomplished or completed tasks and when I see coworkers doing well and enjoying themselves. I work with great people.”

“Surround myself with people who care about animals as much as I do.”

“Working and collaborating with like-minded, dedicated colleagues.”

“Working with other compassionate people with a passion for animals.”

“Having the “work” family that I do.”

“Freedom to share your opinion about the medical care of a patient.”

Table 5 shows how often veterinarians and technicians feel supported by the people they work with. About two-thirds of participants indicate that their coworkers regularly listen to each other’s opinions and ideas (69%) or thank one another for their work (63%). About half (54%) report that their coworkers give each other positive feedback often or most of the time. Slightly less than half (43%) report that their coworkers regularly do or say things so that others are proud of their work.

Table 5. How often do your coworkers support one another? (N = 829)

<table>
<thead>
<tr>
<th>% of Responses</th>
<th>0</th>
<th>20</th>
<th>40</th>
<th>60</th>
<th>80</th>
<th>100</th>
</tr>
</thead>
<tbody>
<tr>
<td>Listen to others' opinions &amp; ideas</td>
<td>24%</td>
<td>39%</td>
<td>37%</td>
<td>42%</td>
<td>27%</td>
<td>12%</td>
</tr>
<tr>
<td>Thank one another for their work</td>
<td>27%</td>
<td>39%</td>
<td>37%</td>
<td>42%</td>
<td>24%</td>
<td>12%</td>
</tr>
<tr>
<td>Give each other positive feedback</td>
<td>24%</td>
<td>39%</td>
<td>37%</td>
<td>42%</td>
<td>27%</td>
<td>12%</td>
</tr>
<tr>
<td>Do things so others take pride in their work</td>
<td>24%</td>
<td>39%</td>
<td>37%</td>
<td>42%</td>
<td>27%</td>
<td>12%</td>
</tr>
</tbody>
</table>

HOW DO THESE FACTORS RELATE TO JOB SATISFACTION?

The items listed in the tables above were summed to compute mean scores for each of the factors identified as relevant to veterinarians and technicians’ job satisfaction. These factors were then simultaneously entered into a multivariate regression analysis. This allows us to determine the relative contribution of each factor to job satisfaction, net of each other and net of the control variables also included in the analysis. Figure 1 shows the relative contribution of each factor to veterinarians’ and technicians’ job satisfaction.

Figure 1 shows that challenging interesting work is the most important determinant of job satisfaction for veterinarians and technicians. That is, the more challenging and interesting their work, the most satisfied they are with their job. This is closely followed by making a difference by helping clients, which also contributes to greater job satisfaction. Helping animals is third in importance of these four factors, where the more time spent helping animals is related to greater job satisfaction. Lastly, a good work environment, in terms of supportive coworkers, is also important in increasing job satisfaction.
Figure 1. Relative importance of key factors to job satisfaction

Note: The coefficients represent standardized regression coefficients (β) while controlling for occupation (veterinarian vs. technician), workplace (works in clinic vs. other), city size, marital status, parental status and age. ***p < 0.001; **p < 0.01; *p < 0.01

Figure 2. Relative importance of job satisfaction on key outcomes

Note: The coefficients represent standardized regression coefficients (β) while controlling for occupation (veterinarian vs. technician), workplace (works in clinic vs. other), city size, marital status, parental status and age. ***p < 0.001; **p < 0.01; *p < 0.01

**How Does Job Satisfaction Relate to Key Outcomes?**

A similar approach was used to determine the relative importance of job satisfaction to five key outcomes. The outcomes include three that are particularly relevant to individual veterinarians' and technicians' wellbeing including: burnout, wellbeing and life balance. The other two are relevant to the workplace including: whether the individual intends to stay in the profession and work performance.

Figure 2 shows the relative importance of job satisfaction to the five key outcomes. Of the five outcomes, job satisfaction has the strongest relationship with intent to stay in the veterinary or technician professions. That is, those who are more satisfied with their jobs are significantly more likely to intend to remain working in their current occupation.

Job satisfaction is also strongly related to the three individual outcomes - burnout, wellbeing and life balance. Veterinarians and technicians who are more satisfied with their jobs experience significantly less burnout and a significantly greater sense of wellbeing and life balance. While not as strongly related as the other factors, it appears that work performance is also enhanced by job satisfaction. Those who enjoy their work more also report that the quality and performance of their work is higher than other workers in their line of work.
**SUMMARY POINTS**

- **Most veterinarians and technicians are highly satisfied with their work** as indicated by the degree to which they are enthusiastic about their work and find enjoyment in doing it. There are almost no differences across a variety of individual and work arrangement characteristics.

- **Making a difference by helping animals and clients, challenging interesting work, and supportive coworker relationships were identified as key sources of job satisfaction** in the open ended questions. All four factors were statistically significantly related to job satisfaction.

- **Challenging interesting work and making a difference by helping clients** were more strongly related to job satisfaction than helping animals or having supportive coworkers.

- **Job satisfaction appears very important in retaining veterinarians and technicians** in their respective professions, as well as enhancing a sense of wellbeing and life balance and reducing burnout. Job satisfaction is also important in enhancing work performance.

**THE LAST WORD**

“I have always wanted to be a Veterinarian and feel blessed to have fulfilled that dream and aspiration. There can be difficult days as in any job, but the most important factor I can feel happy about - if I could do over - I would not change a thing - I would still be a Veterinarian! I feel blessed and privileged to be part of this profession.”

“Being an AHT is the most rewarding career - it is diverse and there is always something to learn. At times it can be stressful but at the end of the day you still have accomplished good things be it saving a life or making a client feel good or teaching a new staff member something new!”
The Downs: What Stresses Veterinarians and Technicians Out
Jean E. Wallace, PhD
Department of Sociology, University of Calgary, Calgary, AB, Canada

ABSTRACT
All jobs have stressful moments and this paper explores the unique factors that are particularly stressful to veterinarians and animal technicians. While some stressors may seem obvious, others may be more subtle but still harmful to wellness. Both interview and survey data were used to identify stressful parts of veterinarians’ and technicians’ work. Several popular wellness outcomes, such as burnout, compassion fatigue and suicidal ideation, are examined in relation to these stressors to see which are particularly harmful and in what ways.

INTRODUCTION
Veterinarian wellness is receiving more attention in both the practice and academic arenas where it is being documented that more and more veterinarians are suffering from compassion fatigue, burnout, and suicidal behaviors. Several studies have been carried out in Australia, Europe and the United States that identify factors related to these indicators of unwellness among veterinarians. Very few studies have examined these issues among Canadian veterinarians. While veterinarian wellness is receiving more attention in both the practice and academic arenas, scant if any research has examined the work experiences of animal health technicians.

THE STUDY

INDICATORS OF WELLNESS
The paper focuses on three different wellness outcomes: burnout, compassion fatigue and suicidal ideation.

Burnout refers to feeling emotionally overextended and drained as a result of one’s work. It was measured by five items that reflect how often veterinarians and technicians feel emotionally drained, used up, tired or burned out from their work.

Compassion fatigue refers to caregivers’ reduced ability or interest in being empathic or being able to bear the suffering of their clients. It was measured by seven items indicating how often veterinarians and technicians share the emotional pain or traumatic stress of their patients and/or clients, and feel affected, preoccupied or depressed because of it.

Table 1 shows that technicians and veterinarians do not differ in how often they experience feelings of burnout, but veterinarians report feeling symptoms of compassion fatigue slightly more often than technicians.
Table 1. Technicians’ (N = 407) & veterinarians’ (N = 437) burnout & compassion fatigue scores

Suicidal ideation refers to suicidal thoughts that are the immediate precursors to planning and carrying out suicide attempts. Suicidal ideation was measured by three items from Bartram’s study of UK veterinarians asking whether they felt the following over the last 12 months: Life was not worth living? Wished you were dead? or thought about taking your life, even if you would not really do it? Table 2 shows the percent who responded “yes” for Alberta veterinarians and technicians and UK veterinarians.

Table 2. Comparison of suicidal ideation among AB veterinarians and AB technicians and UK veterinarians

The results show that similar proportions of Alberta veterinarians and technicians have had some suicidal thoughts in the past year: 21% of veterinarians and 18% of technicians have thought life was not worth living, wished they were dead or thought about taking their own life. This is comparable to the results of the 2012 CVMA National Survey on veterinarian wellness that reported 19% of their respondents had seriously thought about suicide. The results for Alberta veterinarians and technicians are significantly lower than the 29% of the UK veterinarians who had such thoughts over the past year. It is important to note, however, that 21% of Alberta veterinarians represent 294 of the 1401 practicing at the time of the study and 18% of Alberta technicians represent 222 of the 1234 practicing at that time, which is perhaps a timely call for action.
Sources of General Work Stress

A series of work characteristics have been identified as stressful in other research and in the interviews of this study. These factors were measured in the online survey. Table 3 shows the percent who regularly encounter these situations as indicated by their responses that they experience them “often” or “most of the time.”

Table 3. Technicians’ (N = 407) & veterinarians’ (N = 437) sources of work stress

- About 20% of both technicians and veterinarians regularly experience unrealistic client expectations (see Table 3). As two veterinarians noted:
  “Clients with unrealistic expectations are difficult to deal with. Clients who “researched it online” and “know” what their pet has.”
  “People have an unrealistic perception about the economics of veterinary medicine and try to hold their veterinarians responsible when we fail to live up to their (often misguided) expectations.”
  A technician working in a rural mixed practice described stressful parts of her job as: “The increasing demands of the public for instant service while they challenge you with Dr. Google…”
- Conflict between client interests and what is best for the animal is a regular source of stress, particularly for veterinarians. For example:
  “Clients that want us to help the pet for free, and blame us for costs of health care.”
- Trying to meet the financial expectations of the employing organization is stressful, especially for veterinarians. Two veterinarians describe financial issues as the most stressful part of their work:
  “Dealing with financial/business management issues that I have no training in. Dealing with rising costs, rent and an aging clinic with a half-million dollar business loan and tens of thousands of dollars in student loans as well.”
  “Low revenue day (especially when you feel like you have been working your ass off all day).”

Health Risks

Table 4 shows some of the health risks that AHTs and veterinarians encounter in their work.
Table 4. Technicians’ (N = 407) & veterinarians’ (N = 437) health risks during work week

- Few technicians and veterinarians are regularly at risk of serious injury, but one-quarter are regularly at risk of minor injury.
  “A few years ago due to a workplace injury I had to not only leave the job I loved, but potentially change careers completely.”
  “Numerous staff have been injured by bulls upon semen testing, restraint and proper care of companion animals has been below an acceptable level... and in the end, those were among deciding reasons why I left in pursuit of learning more about animals in a happier, healthier environment.”
- Almost half of both groups regularly report improper posture, but more technicians report improper lifting.

CLINIC-SPECIFIC WORK STRESS
In the survey, technicians and veterinarians reported on how often they experience specific work stressors relevant to working with clinical cases. Table 5 shows work stressors related to animal care, finances and clients.

Table 5. Technicians’ (N = 279) & veterinarians’ (N = 393) clinic work stressors
Veterinarians more regularly have concerns about the financial situation of clinic and inadequate resources than technicians. Two veterinarians wrote:

“When I was an associate, I only had the stress of patient outcomes (and the aforementioned mistakes). As an owner, I have HR and financial stress as well. The HR and financial stresses come in waves but when they are bad, they are really bad.”

“Finances are usually part of most stress - it drives client responses to situations, it complicates practicing the way we want too, it is a problem for new graduates, it affects our options in staffing, products, equipment, continuing education, working environment etc.”

Relatively few technicians and veterinarians report that they regularly encounter animal suffering, but about 70% of both groups regularly deal with euthanasia of animals. For example:

“I drove home crying tonight as I was upset about a case of mine that was euthanized but I still love my job.”

“I had to stop working as a veterinarian because euthanasia bothered me so much. It would upset me for days/weeks. Still does. I do much better as a manager and am much happier.”

Veterinarians more regularly deal with client grief than AHTs. As one veterinarian wrote:

“Euthanizing animals has become very stressful, some CE on how to deal with grief would be beneficial.”

About half of technicians and veterinarians regularly find clients’ financial situation is a barrier to best care for their animals (Table 5). Both raised this concern in the e-interviews as being particularly stressful:

“Frustrating clients that can’t treat due to lack of money or just not caring about their pet.”

“I find it stressful when a client cannot afford something that I feel would benefit their animal(s). I feel “guilty” if I know there is something I can do to help them but they cannot afford it and I cannot afford to do it pro bono. I know that I undervalue some of the things we do so I stay away from the billing side of things.”

**HOW DO THESE FACTORS RELATE TO BURNOUT?**

The items in the tables above were simultaneously entered into a multivariate regression analysis. This determines the relative contribution of each factor to burnout, net of each other and the control variables included in the analysis. Figure 1 shows the relative contribution of each factor to veterinarians’ and technicians’ burnout. The health risk of improper lifting or posture appears to be an important contributor to burnout, followed by unrealistic client expectations and trying to meet the financial expectations of the employing organization. The more often animal healthcare workers experience conflict between client and animal interests also contributes to burnout. Lastly, risk of injury at work is also significantly related to burnout.

**Figure 1. Relative importance of general work stressors and health risks to burnout**

Note: The coefficients represent standardized regression coefficients (β) while controlling for occupation (veterinarian vs. technician), workload, work support, pay satisfaction, work setting (works in clinic vs. other), city size, marital status, parental status and age. ***p < 0.001; **p < 0.01; *p < 0.05
Figure 2 is limited to veterinarians and technicians working in clinical settings. Dealing with client grief and concern about the financial situation of the clinic are both important contributors to burnout. Inadequate resources in terms of lack of equipment or ability to perform certain tests are also significant. While animal suffering and euthanasia significantly contribute to burnout, having clients with financial barriers to best care for their animal is not significantly related.

**Figure 2. Relative importance of clinic-specific work stressors to burnout**

Note: The coefficients represent standardized regression coefficients (β) while controlling for occupation (veterinarian vs. technician), workload, work support, pay satisfaction, city size, marital status, parental status and age. ***p < 0.001; **p < 0.01; *p < 0.05

Figure 3 shows that feelings of burnout are significantly related to both compassion fatigue and suicidal ideation. That is, those who are more emotionally exhausted with their jobs more report experiencing compassion fatigue and suicidal ideation.

**Figure 3. Relative importance of burnout on compassion fatigue and suicidal ideation**

Note: The coefficients represent standardized regression coefficients (β) while controlling for occupation (veterinarian vs. technician), workload, work support, pay satisfaction, work setting (works in clinic vs. other), city size, marital status, parental status and age. ***p < 0.001; **p < 0.01; *p < 0.05

**How Does Burnout Relate to Compassion Fatigue and Suicidal Ideation?**

A similar analytic approach was used to determine the relative importance of burnout to compassion fatigue and suicidal ideation. Figure 3 shows that feelings of burnout are significantly related to both compassion fatigue and suicidal ideation. That is, those who are more emotionally exhausted with their jobs more report experiencing compassion fatigue and suicidal ideation.
SUMMARY

- It is important to understand the factors that lead to burnout given the strong relationships between burnout and compassion fatigue and suicidal ideation.
- Physical strain, client expectations and interests, and financial demands are all relevant to veterinarians’ and technicians’ feelings of burnout and fairly frequently experienced by many animal healthcare workers.
- Clinical workers are more likely to burnout from dealing with client grief and financial stresses, but inadequate resources and animal suffering and euthanasia are also important factors.
- Based on the findings of this paper, some of the factors leading to burnout may be effectively reduced in the workplace and these are identified below.

TO REDUCE BURNOUT...

- **Reduce the physical stressors by:**
  - Promoting **proper lifting and posture** habits and equipment
  - Exploring ways to **reduce risk of injury** in the workplace
- **Reduce the client/patient stressors by:**
  - Promoting **effective communication with clients** about realistic expectations, animal welfare and animal care costs
  - Accessing **grief counseling** for clients and animal healthcare workers
- **Reduce the financial stressors by:**
  - Promoting **effective communication** about **realistic financial expectations** between employees and employers
  - Accessing **financial planning resources** for clinics and office managers
Maintaining Momentum: How Veterinarians and Technicians Cope
Jean E. Wallace, PhD
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Abstract
Veterinarians and animal technicians encounter numerous stressors in their work. In dealing with work-related stress, they may use a variety of coping strategies. Unfortunately, not all coping strategies may be effective in reducing feelings of stress and some may actually be more harmful than beneficial. Veterinarians’ and technicians’ own descriptions of the coping strategies that they use are presented in addition to statistical data that show how often they typically use these strategies. How these coping strategies are related to feelings of emotional exhaustion are examined to illustrate which may be beneficial and which may be harmful in dealing with work-related stress.

The Study
When asked to describe how they cope with work-related stress, veterinarians and technicians described a number of different strategies in the e-interviews that reflect different coping responses.
- At work, cope by calming down, trying to relax, or turning to colleagues for help. Calming down may involve taking a break or time out or talking to a coworker. Here are examples of ways veterinarians and technicians attempt to calm down, take a break or relax while at work:
  - “I try to take a moment to myself and regroup and “pull it together” if you will. Whether I run across the street to [gas station] to get a drink, listen to music, or look at something else that is unrelated to my stressor for a few minutes.”
  - “I try to make the best of it. Will sometimes take a break and go outside for some fresh air. I always go home at lunch time which makes the day seem less long. I will talk to a co-worker about my issue and vent a bit.”
  - “Taking a break, having a coffee, reading the paper - can also be helpful to cope.”
  - Talking to co-workers and seeking their opinions or help is another strategy used to cope with stress.
    - “I will discuss it with the office staff. I will take a deep breath. I will think of what is important in life and put things in their proper perspective.”
    - “The rest of the staff is supportive, we have the same concerns. We vent to each other and try to support each other.”
    - “I think it’s so important to talk about what happened, don’t lay blame, give reassurance, identify the mistake and get it out there.”

Table 1 presents the results from the survey data that shows the frequency with which respondents use four different strategies in dealing with work-related stress that are similar to those described in the e-interviews. All are fairly popular coping strategies in that almost half of respondents report using them.
“sometimes” and about one third report using them “often”. One-fifth of veterinarians and technicians also report that their colleagues help them to figure out how to solve a work-related problem “most of the time.”

Table 1. How often do you do the following in dealing with work-related stress? (N = 805)

- After work, two popular coping strategies are physical exercise and spending time with family.
  - “Workout, spend time with my family.”
  - “I play agility with my dogs or take them for a walk.”
  - “I spend time with my husband and my dogs. I go for a walk with my dogs. I drink wine or eat sweets although I have adopted a better diet and that makes a huge difference in my mental state and energy level.”
  - “Watch TV and have a glass of rum!!! Go golfing. Talk to my wife. Phone my grandchildren and talk to them!! Take my dog for a long walk.”
  - “Exercising is good.”

Table 2 shows the survey results where slightly more than half of the respondents usually eat dinner with their family most days of the week (i.e., five or more days a week). Table 2 also shows that 25% exercise almost daily and almost half (40%) several times a week in moderate physical activities for at least 30 minutes.
Internalization of stress is also a popular response described in the e-interviews. This may involve ignoring the stress and trying to work through it, or keeping it to one’s self and not talking about it with others.

- “Work harder... shrug and move on.”
- “I may or may not tell my family. I usually keep it in and may talk about it later.”
- “Just push through - no sense in getting stuck on things and deal with it with whatever means you have available.”
- “I internalize it so that the staff and clients don’t see that there are issues and to keep everyone happy and keep things moving along... Sometimes internalize so my family does not have to hear about. Sometimes I let loose and vent about it to my wife.”

Table 3 shows different internalization strategies and that keeping it to themselves is the most popular of the four in this table, where 25% keep it to themselves “often” or “most of the time” and almost half (43%) “sometimes.” Almost half (42%) “sometimes” carry on as if nothing has happened with 18% doing this “often” or “most of the time.”

Table 3. How often do you do the following in dealing with work-related stress? (N = 805)

<table>
<thead>
<tr>
<th>How often do you do the following in dealing with work-related stress?</th>
<th>Never/Not very often</th>
<th>Sometimes</th>
<th>Often</th>
<th>Most of the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>I keep it to myself.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I do nothing and try to carry on as usual.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I feel uneasy talking to others about my work stress.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I have a drink at the end of the day.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 3 shows that one third of veterinarians and AHTs (32%) “sometimes” feel uneasy about talking about their work-related stress with others and 22% feel this way “often” or “most of the time.” The majority (65%) report that they “never” have a drink at the end of the day in coping with work stress, but one quarter (25%) report that they do this “sometimes” and the remainder (12%) report doing this “often” or “most of the time.”

**How Do These Coping Strategies Relate to Burnout?**

In the survey, burnout was measured by a five-item scale that assesses how often veterinarians and AHTs experience the following: I feel emotionally drained from my work; I feel used up at the end of the workday; I feel tired when I get up and have to face another day on the job; I feel that working all day is really a strain for me; and I feel burned out from my work. Responses were summed and averaged to get an overall burnout score that ranges from 1 (low burnout) to 5 (high burnout).

To determine the relationship between each of the coping strategies and burnout, zero-order correlations were computed. The correlation (r) indicates the direction and magnitude of the relationship between each coping strategy and burnout. All of the correlations presented below are statistically significant. A negative correlation means that the more frequently that coping strategy is used, the less often they feel burned out. This means the coping strategy is effective in that it reduces feeling of
burnout. A positive correlation means that the more frequently veterinarians and AHTs use that coping strategy, the more often they experience symptoms of burnout. This means it is a harmful coping strategy.

Coping Strategies That May Help
Below are the six coping strategies that appear effective in reducing feelings of burnout. These strategies may be used at work by taking a time out or short break, having a cup of coffee, or talking to colleagues.

- Think calmly about what to do ($r = -0.30$)
- Try to refresh self by relaxing activities ($r = -0.19$)
- Do something that calms me down ($r = -0.11$)
- Colleagues help solve work problem ($r = -0.32$)

After leaving work, the more main meal times that veterinarians and AHTs spend with their family and the more frequently they exercise, the less often they feel burned out from their work.

- Eat main meal with entire family ($r = -0.31$)
- Exercise several times a week ($r = -0.25$)

Figure 1 shows how spending meal time with family significantly reduces burnout.

Figure 1. Burnout and number of meals with family ($N = 256$)*

![Figure 1](image)

Figure 2 shows how moderate physical activity is most beneficial when veterinarians and technicians exercise at least several times a week.

Figure 2 shows how moderate physical activity is most beneficial when veterinarians and technicians exercise at least several times a week.
Coping Strategies That May Hurt
The four internalization strategies used to cope with work stress may be harmful because they are correlated with increased feelings of burnout. That is, keeping it to one’s self, doing nothing, and not talking to others all appear to contribute to feeling more burned out from one’s work.

- Keep it to myself (r = 0.25)
- Do nothing and carry on as usual (r = 0.25)
- Feel uneasy talking to others (r = 0.33)

In addition, it appears that having a drink at the end of the day to cope with work-related stress is also harmful to veterinarians’ and technicians’ wellbeing.

- Have a drink at the end of the day (r = 0.21)

Figure 3 shows that the more often veterinarians and AHTs have a drink to cope with work-related stress, the more burned out they feel from their work.
ADVICE FROM VETERINARIANS AND TECHNICIANS
In the e-interviews, participants were asked what advice, if any, they would offer to other veterinarians or technicians. Some of their suggestions refer to coping with work stress and also appear effective.

“Teams that do not work well together jeopardize the health and wellbeing of the animal and lead to burnout among the staff.”

“Have hobbies outside of work. Make time for your family and friends.”

“I would recommend making sure you have good balance between amount of work and play.”

“Take time for yourself and be healthy.”

Based on the findings reported in this newsletter, several effective coping strategies have been identified that may help in reducing feelings of burnout for veterinarians and technicians and several that may be harmful were identified as well. These are summarized below.

HOW TO EFFECTIVELY COPE WITH WORK-RELATED STRESS
- **Calm yourself** during your work day - take a time out, walk, or coffee break
- **Do relaxing things** - quiet time at work, other activities after work
- Avoid keeping it to yourself - **talk to others**
- Talk to colleagues and **ask for help** - they may be able to help you
- **Don’t ignore it** - the stress isn’t likely to just go away
- Spend time with family - share activities with **family and friends** outside of work
- Try to **avoid alcohol** as a coping strategy
- **Exercise** several times a week - it helps!
Social Media and Veterinary Medicine: Is It for Me?
Jean Gauvin¹, DVM; Eric-Norman Carmel², DVM, DACVR
¹Lachine Veterinary Clinic, QC, Canada; ²Centre Veterinaire Laval, QC, Canada
Social Media: Is There Life After Facebook and Twitter?
Jean Gauvin¹, DVM; Eric-Norman Carmel², DVM, DACVR
¹Lachine Veterinary Clinic, QC, Canada; ²Centre Veterinaire Laval, QC, Canada

SEE ATTACHED FILE TITLED: IS THERE LIFE AFTER FACEBOOK AND TWITTER?
The Good, the Bad, and the Ugly: Transform Your Website into a Client-Attracting Machine!
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