2016 Annual Convention Proceedings

NIAGARA FALLS

2016 CVMA CONVENTION
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Welcome to the 2016 CVMA Convention in Niagara Falls. 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.
EMERGENCY PROCEDURES

CPR FOR TECHNICIANS

David Liss, RVT, VTS (ECC, SAIM)
Los Angeles, CA

These guidelines are taken from two different sources:
• RECOVER initiative: Reassessment Campaign on Veterinary Resuscitation
  • www.acvecc-recover.org
• American Heart Association 2010 Guidelines

There has been some interesting advancements in the field of human and veterinary CPR research. The initial change is the mnemonic change from CPR to CPCR-Cardiopulmonary Cerebral Resuscitation- highlighting the importance of supporting and maintaining brain function in the goal of return of spontaneous circulation (ROSC).

These proceedings will be presented in outline/paradigm format for easy reference.

Initial assessment/preparation
The ideal goal of cardiopulmonary arrest (CPA) is to prevent it. However, that can’t always happen. Patients that are in-hospital or under anesthesia should be labeled as “at-risk” and special attention paid to their monitoring. Crash supplies should always be readily available and include:

<table>
<thead>
<tr>
<th>Blood pressure equipment: Doppler, Cuffs, Sphygmomanometer, Ultrasound gel</th>
<th>Clean clippers</th>
<th>IV Fluids</th>
<th>Catheters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Syringes</td>
<td>Needles</td>
<td>Scrub</td>
<td>Tape</td>
</tr>
<tr>
<td>ECG machine</td>
<td>Oxygen source</td>
<td>Back boards</td>
<td>Bandage supplies</td>
</tr>
<tr>
<td>Good quality lighting</td>
<td>Defibrillator</td>
<td>Emergency cardiac drugs: Lidocaine, Atropine, Epinephrine, Calcium, Magnesium, Vasopressin, Amiodarone</td>
<td>Ambu bag</td>
</tr>
</tbody>
</table>

At-risk patients include those with single or multiple cardiovascular, respiratory, neurological diseases, and also those with multiple physiologic parameter disturbances:
• Hypotension
• Tachycardia/Bradycardia
• Pale MM’s/Anemia
• Tachypnea/Hypoxemia
• Shock patients
• Patients with trauma/hemorrhage
This list is by no means exhaustive. By implementing aggressive resuscitation strategies and monitoring, the critical care technician can help potentially identify problems before they start, and ward off CPA.

**Basic Life Support**
Another very important change is the approach to the patient in CPA- instead of ABC the current recommendations are to start with CBA.

1. Initially ensure the patient is in CPA by confirming no palpable pulses, apnea, and lack of heart sounds
2. Next, begin compressions at least 100 compressions/minute. Compression technique will be discussed shortly.
3. After compressions are initiated, the next step is to provide positive pressure ventilation (PPV). Typically in a veterinary hospital this is done by intubation and then providing PPV. Intubation should be done in lateral recumbency to minimize disruption of compressions. Technicians should start to practice intubating in lateral recumbency as it’s difficult. Breathing rates are 8-10 breaths/minute.
4. After compressions are initiated, intubation is successful and PPV is being provided, attempts at IV catheterization can be performed. Simultaneously an ECG should be hooked up.
5. Only after 2 full minutes of compressions should the ECG be evaluated. The rhythm can be analyzed and advanced cardiac life support (ACLS) administered as needed.
6. This cycle of 2 minutes compressions, rhythm strip analysis, and treatment continues until DVM or owner decision to discontinue CPCR.

**Compressions:**
Patients should ideally be in right lateral recumbency. For small dogs, <15 kg, one-hand can be used, and the thumb placed directly over the apex of the heart. The thorax is compressed 1/3 of it’s width at least 100 times per minute. Adequate recoil of the chest is important to allow for myocardial perfusion. Two-hands can be used as well, directly compressing the heart (cardiac pump theory). For larger patients, a stool will be of benefit to stand directly over the patient. Two hands should be placed on the widest part of the thorax- no longer over the cardiac apex. The thorax should be compressed 1/3 of it’s width, and adequate recoil provided to maximize cardiac perfusion. Rates should be around 100 compressions per minute. “Another one bites the dust” by Queen, or “Stayin’ Alive,” by the BeeGees provides a beat one can follow. Compressions should be performed for 2 minutes or less as needed to prevent operator fatigue. The airway and compressor should alternate to prevent fatigue and patient detriment.

**Advanced Cardiac Life Support (ACLS) - Drug administration & Fluid Therapy**
Copious and unreasoned fluid therapy is no longer recommended in CPCR. Increasing blood pressure can lower perfusion to the myocardium. Thus, in the face of a hypovolemic arrest (hemorrhage) aggressive fluid resuscitation strategies can be used. However, most arrests are euvolemic and other than a small “test-dose” of fluids (20ml/kg), keep-open fluids, or excessive boluses are discouraged.
EMERGENCY PROCEDURES

IV administration of drugs is preferred, but intratracheal and intraosseous routes can be used. Intraperitoneal or intracardiac administration are also no longer recommended. Venous access can be performed in the following order to maximize proximity to the heart and drug delivery: jugular, cephalic, saphenous. Saphenous administration is least ideal. Following administration of a drug bolus, 3-6 mL of a flush (saline) can be used to guide a drug to the heart to enhance its uptake and delivery. Intratracheal drug administration is less predictable and drug doses should be doubled when using this route. The ambu bag, or other PPV source is rapidly disconnected, a red rubber catheter is advanced down the ET tube, the drug is administered, 3-6 mL of saline is followed, and the catheter is removed. PPV is immediately reinstituted. Atropine, Epinephrine, Vasopressin, and Lidocaine can be given via the IT route. IO drug administration can also be performed and flushes used to move the drug from the intramedullary space to the intravascular space.

The following table lists various cardiac drugs, their effects, and dose/route for administration in CPCR:

<table>
<thead>
<tr>
<th>Drug</th>
<th>Concentration</th>
<th>Effect</th>
<th>Indications</th>
<th>Dose</th>
<th>Routes administered</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epinephrine (low-dose)</td>
<td>1:1000 solution (1mg/mL)</td>
<td>a/B receptor stimulation-vasoconstriction</td>
<td>Asystole-initially V-fib after Defib</td>
<td>0.1 mL/10 kg</td>
<td>IV, IT, IO</td>
</tr>
<tr>
<td>Epinephrine (high-dose)</td>
<td>1:1000 solution (1mg/mL)</td>
<td>a/B receptor stimulation-vasoconstriction</td>
<td>Asystole-after low dose V-fib after Defib</td>
<td>1mL/10 kg</td>
<td>IV, IT, IO</td>
</tr>
<tr>
<td>Atropine</td>
<td>0.54 mg/mL</td>
<td>Anti-vagolytic-Removes vagal involvement in CPA</td>
<td>Asystole/PEA</td>
<td>1mL/10 kg</td>
<td>IV, IT, IO</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>20mg/mL (2%)</td>
<td>Na channel blocker- anti-arrhythmic</td>
<td>Refractory V-fib, Ventricular Tachycardia</td>
<td>1mL/10 kg 0.1mL/10 kg (Cat)</td>
<td>IV, IO, IT</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>20U/mL</td>
<td>Non-adrenergic vasoconstrictor</td>
<td>Asystole/PEA</td>
<td>0.8U/kg</td>
<td>IV, IO, IT</td>
</tr>
<tr>
<td>Calcium Gluconate</td>
<td>100mg/mL</td>
<td>Electrolyte therapy</td>
<td>HYPO-calcemic arrests HYPER-kalemic arrests (must be documented)</td>
<td>0.5-1mL/kg</td>
<td>IV</td>
</tr>
</tbody>
</table>
**Advanced Cardiac Life Support (ACLS)- ECG Analysis, Drug administration, Defibrillation**

Evaluation of the ECG is an important part of the CPR effort. ECG analysis should be performed after 2 minutes of compressions have been performed. The following chart illustrates treatment of various arrhythmias:

<table>
<thead>
<tr>
<th>Asystole/Pulseless Electrical Activity</th>
<th>Ventricular Fibrillation</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPCR for 2 minutes</td>
<td>CPCR for 2 minutes</td>
</tr>
<tr>
<td>Administer Epinephrine low-dose</td>
<td>ECG reveals V-fib</td>
</tr>
<tr>
<td>Consider Atropine</td>
<td>Continue CPCR until defibrillator charged</td>
</tr>
<tr>
<td>Resume CPCR for 2 minute cycle</td>
<td>Initial defibrillation at 3-5 J/kg</td>
</tr>
<tr>
<td>Re-administer epinephrine (high-dose), atropine</td>
<td>Resume CPCR for 2 minutes</td>
</tr>
<tr>
<td>Consider one-dose Vasopressin</td>
<td>Re-evaluate ECG</td>
</tr>
<tr>
<td>Search for cause: H's and T's (below)</td>
<td>If still V-fib, readminister shock at 2x original dose</td>
</tr>
<tr>
<td></td>
<td>Continue pattern until 3 shocks have been administered</td>
</tr>
<tr>
<td></td>
<td>If still V-fib after 3 shocks, consider Amiodarone or Lidocaine</td>
</tr>
</tbody>
</table>

**Advanced Cardiac Life Support (ACLS)- Monitoring of CPR**

Our goal of CPR is not only to perform it, but to attempt to ascertain if it is effective. There are various methods to use to assess monitoring of CPR, but End-tidal CO2 monitoring has proven to be highly useful in assessing effectiveness of CPR effort. The theory behind its use is that in CPA no blood flow occurs at the capillary-alveolar junction resulting in no gas exchange. Unlike a PaCO2, which would indicate buildup of carbon dioxide in the blood, no CO2 is released and thus there is no End-Tidal CO2. In effective CPR, some movement of blood will occur across the junction, and CO2 will be exchanged for O2. CO2 will move into the alveoli and eventually be exhaled as PPV is being instituted. This will create an end-tidal CO2. Thus an end-tidal CO2 of around 10mmHg represents some measure of blood flow and perfusion.
Advanced Cardiac Life Support (ACLS)- Assessing cause of CPA
The best treatment for CPA would be to treat the inciting cause. Often it cannot be found, but once CPA efforts are underway, it may be beneficial to examine the following causes and attempt to ascertain the underlying cause of the CPA event. Once the cause has been determined, appropriate treatment can be instituted.

<table>
<thead>
<tr>
<th>H's</th>
<th>T's</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxia</td>
<td>Tension</td>
</tr>
<tr>
<td>Tamponade</td>
<td></td>
</tr>
<tr>
<td>Hypothermia</td>
<td>Toxicity</td>
</tr>
<tr>
<td>pneumothorax</td>
<td>Trauma</td>
</tr>
<tr>
<td>Hypokalemia</td>
<td>Thrombosis</td>
</tr>
<tr>
<td>Hyperkalemia</td>
<td></td>
</tr>
<tr>
<td>H+ (Acidosis)</td>
<td></td>
</tr>
<tr>
<td>Hypovolemia</td>
<td></td>
</tr>
</tbody>
</table>

Advanced Cardiac Life Support (ACLS)- CPCR after-care
CPCR after-care is complex and highly involved. Often these patients are completely dependent on life-support. These proceedings will address major concerns and discuss post-arrest interventions that should be employed.

Blood pressure monitoring:
- If patient’s MAP is <80 mmHg:
  - Fluid status should be assessed
  - If the patient is hypovolemic- IV fluids can be administered and titrated to specific end-points (BP improvement, lactate improvement, CVP improvement)
  - If patient is euvoletic- vasopressor therapy should be instituted:
    - Norepinephrine at 0.1-0.5ug/kg/min
    - Dobtamine at 5-20ug/kg/min

Respiratory system
Supplemental oxygen should be provided and the patient may benefit from sedation, continued intubation, and provision of PPV. A ventilator may be necessary.

Body temperature
Benefits of hypothermia are an on-going area of research in human medicine, yet those techniques employed are probably not feasible in small animals. Patients may potentially benefit from no active rewarming, but this remains to be seen.

Nursing care
These patients are recumbent and require constant monitoring as incidence of re-arrest after ROSC is high. Monitoring, bedside care, urinary output monitoring, perfusion status (CVP) and ECG monitoring should be instituted on these patients and hospitals should be prepared to deal with the after-effects of an extremely critical patient if ROSC were to occur.

References available upon request.
EMERGENCY PROCEDURES

ADVANCED CATHETERIZATION AND SKILLS WORKSHOP

David Liss, RVT, VTS (ECC)
Platt College
Los Angeles, CA, USA

Procedures to be covered:
• Central lines
• Intracath style (TTN)
• Guidewire style
• Arterial catheters
• Measurement of direct arterial blood pressure
• NG Tube placement
• CVP monitoring
• Abdominocentesis

Central Lines
Types:
• Through the needle:
• Guidewire (Seldinger technique)
• Peel-away introducer

What does “central” mean vs. PICC?
• Central line means a catheter placed in a “central” or LARGE vein
• Jugular, Cranial/caudal vena cava
• PICC: Peripherally-inserted central catheter
• Central line placed in a saphenous vein and “threaded” to the CVC

Why do we use them?
• Serial Blood Sampling
• Longevity
• Polyethylene can last 3 + weeks!
• Incompatible medications
• Multiple ports allow multiple fluids for one line

What does “multi-lumen” mean?
• Catheters of this type have multiple exit points
• In addition, these catheters have separate chambers!

Placement- Seldinger technique
Step 1: Gather Equipment
• Appropriate catheter
• Sterile instruments
• Needle driver
• Pickups
• Suture
• Sterile syringes
• Bottle of Saline
• Bottle of Heparin
• Gauze
• Introducer catheter
  • 5.5 Fr caths = 20ga
  • 7Fr Caths = 18ga
• #11 scalpel blade
• Bandage material
• Preparing New Hep Saline flush
  • To keep things as sterile as possible:
    • Grab new small bottle of 0.9% Saline
    • Add 1cc/L of 1000U/mL Heparin
    • Open syringes with sterile technique for placer
    • Have placer draw up saline with sterile syringes

Step 2: Open all materials
Step 3: Drape the patient
Step 4: Locate your vein
Step 5: Make skin nick with #11 scalpel blade
Step 6: Place introducer cath
Step 7: Feed guidewire
Step 8: Remove peripheral cath
Step 9: Feed dilator over wire
Step 10: Feed dilator ALL the way
Step 11: Remove dilator, they WILL bleed
Step 12: Feed catheter over wire
Step 13: Feed catheter to the skin
Step 14: Remove the guidewire
Step 15: Check/Flush all ports
Step 16: Cleanup
Step 17: Suture
Step 18: Catheter check rad
  • Catheter should be JUST cranial to right atrium
Step 19: Bandage!

After-care
• Wear non-sterile gloves when working with central lines
• Keep Closed ports and sample through them with needle
• Swab ports with alcohol prior to sampling
• Keep TPN/PPN ports connected
• Change bandage q 24 hours
• Look at site and clean AROUND site with Chlorhexidine solution

Arterial Blood Gas Sampling
Typically can get all information from a venous, EXCEPT:
• Oxygen parameters
  • PaO2
• Arterial blood gases are invasive
• It hurts!! Patient must be obtunded or mild sedation required
• Operator skill high—No use “fishing”
• Ideally require special syringes
  • Can use TB syringe with NEW needle
Fill syringe with heparin then flush ALL out so small amount remains in hub

Procedure
Step 1: Gather equipment
- Blood gas syringe
- Clippers
- Scrub
- Bottle of heparin (if not using pre-hep syringe)
- Rubber stopper
Step 2: Locate artery
Step 3: Shave and scrub
Step 4: Make your approach
Step 5: Watch for flash
Step 6: Allow syringe to fill
Step 7: Cap IMMEDIATELY!!
Step 8: Run tests quickly

Arterial catheterization
Why place an art line?
- Direct BP monitoring
- Gold standard
- Excellent on critical patients
- Real-time BP measurements
- Continuous blood gas sampling
- Respiratory patients
- Ventilator patients

Placement
Step 1: Gather equipment
- Peripheral catheter
- Tape
- Scrub
- Clippers
- Hep saline flush
- 18 gauge needle
- Luer-lock male adapter +/- T-port
Step 2: Locate artery
Step 3: Shave and scrub
Step 4: Make skin nick
Step 5: SLOWLY advance catheter
Step 6: Wait for flash
Step 7: QUICKLY advance cath
Step 8: Remove stylet, FLUSH!
Step 9: Secure and Tape

Direct Blood Pressure Monitoring
How does direct BP work?
EMERGENCY PROCEDURES

- Artery is cannulated
- Pressure is created and vibrated across rigid tubing
- Rigid tubing minimizes loss of pressure
- Pressure is sensed via a transducer
- Transducer converts pressure into electricity
- Sent to electronic monitor
- Pulse wave and pressure readings displayed
- Little error involved

Procedure
Step 1: Gather materials
- 0.9% Saline bag
- IV line
- Extension
- Transducer
- Monitor with IBP capability
- Stiff tubing
- Bottle of heparin
- Pressure bag
Step 2: Prepare Hep Saline
  - 1cc/L of 1000U/mL Heparin (Be clean!)
Step 3: Prime IV line
Step 4: Attach IV line to transducer
Step 5: Prime transducer/rigid tubing
Step 6: Pump up pressure bag
Step 7: Attach transducer to pole
Should be level with mid-thorax
Step 8: Plug transducer into cable
Step 9: Zero machine
Step 10: Open stopcock to arterial line
Troubleshooting
- Absence of pulse wave
  - Check “pigtail” and flush
  - Check all connections (including cable)
- Improper readings
  - Check all connections
  - Check transducer level with heart

Nasogastric Intubation
Why do we need NG tubes?
- To provide enteral nutrition
  - Fairly well tolerated
- To measure/empty gastric residuals
  - Contribute to fluid loss and nausea

Placement
Step 1: Gather materials
- Appropriate size tube
EMERGENCY PROCEDURES

- Local anesthetic (Proparacaine)
- Wet-Proof tape
- Marker
- Suture
- Needle drivers

Step 2: Measure for placement
Step 3: Apply local anesthetic
Step 4: Lubricate NG tube
Step 5: Placement 1- Anatomy
Step 5: Placement 2- Procedure
Step 6: Check for gut juice
Step 7: NG Tube check rad
  - Should be in lumen of stomach
  - Tubes can go into duodenum
  - Tubes can be just cranial (nasoesophageal)
  - Tubes can be “poking” stomach
Step 8: Suture
Step 9: E-collar!

Abdominocentesis

Why do we perform abdominocentesis?

Diagnostic:
- To identify type of abdominal effusion
  - Exudate (blood, septic abdomen)
  - Transudate (liver failure, heart failure)
  - Modified transudate (perforation, septic abdomen)

Step 1: Gather materials
- 4- 1” or 1.5” 22 ga needles
- 4- 1cc syringes
- Plain red top tubes
- EDTA tubes
- Gloves
- Scrub/Alcohol
- Clippers
Step 2: Shave and prep
Step 3: Place needles around umbilicus
Step 4: Obtain samples
Step 5: Remove needles
Step 6: Testing
- Blood
  - PCV/TS
    - If matches with peripheral = acute bleed
    - If appears “dehydrated” (High PCV and TS) = chronic bleed
- Fluid
  - Serosanguineous
    - PCV/TS
- Cloudy
  - Blood Glucose
• 20 points lower than peripheral means sepsis!
• Lactate
  • 2 points higher than peripheral HIGHLY suspicious for sepsis
• Possible urine
• Creatinine
  • 2x peripheral = uroabdomen
• K+
  • 1.4x peripheral = uroabdomen

CVP Monitoring
What does CVP stand for?
• Central Venous Pressure
• Arterial blood pressure is often used to tell us about vascular volume (preload)
• Yet it has its limitations
• CVP measures the actual “volume” not PRESSURE in a “great” vein
• Save for some complicating factors it tell us about vascular volume

Why do we run CVP’s?
• To monitor trends on patients and their fluid status
• HIGH means too much volume
• LOW means not enough

Limitations of CVP
• CVP estimates the left ventricle END-diastolic volume (preload)
• If there is intrathoracic pressure changes or disease
• If there is myocardial disease
• CVP can be inaccurate
• Pneumothorax, ventilators, myocardial disease, Pleural effusion, pericardial effusion can affect CVP measurement

Procedure
Step 1: Gather materials
• Must have jugular catheter placed in cranial vena cava
• CVP manometer
• Fluid stand
• 0.9% saline IV bag
• IV line
• Extension set
Step 2: Assemble/prime IV line
Step 3: Prime extension set
Step 4: Fill H2O column to 30cmH2O
Step 5: Attach to jug cath
Step 6: Check jug cath patency
Step 7: Turn off to fluid line
Step 8: Obtain CVP
• Allow fluid to lower until it reaches a plateau
• Read at the middle of the meniscus
• Should vascillate with respiration
Step 9: Disconnect
Welcome to the 2016 CVMA Convention in Niagara Falls. 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.

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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 in the patient’s best interest. The pain associated with orthodontic adjustment, and the numerous anesthetics required, often makes 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!
Supernummary, 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 premolar into the furcation of the mesiobuccal and mesiopalatal roots of the fourth premolar 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. Non vital 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. 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 gingivla 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.

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 mm² 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 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 non-vital 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 non-vital (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 (please see the article on composite bonding later in the issue for further information).

**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 non-vital. Non-vital teeth lose their natural defence ability and are often infected via the bloodstream, which is known as anachorisis. 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 (<1mm) 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.

**Tooth resorption**

While this is typically thought of as a cat condition, we are seeing more and more of this in our canine patients. As of yet, we do not know why they occur. What we do know is that they result from the activation of cells called odontoclasts. These cells are responsible for the normal remodeling of tooth structure. In this disease process, however, they will continue to resorb tooth structure until in some instances the entire tooth is lost.

These lesions start below on the roots where the cells start destroying the underlying root structure. Over time, the resorption will extend into the crown of the tooth. The enamel is undermined which will cause it to break off, exposing the underlying tooth.
structure which is called dentin. These lesions are excruciatingly painful, especially when they are advanced. If you probe one of the lesions even under general anaesthetic, the cat will react. However, most cats will not show evidence of oral pain, even when the tooth is fractured with an exposed root canal.

Because they start below the gum and progress into the crown, they are first seen at and just below the gumline. The teeth most commonly affected are the premolars, followed by the molars and finally the canines. The majority of the lesions are on the buccal surface (on the outside of the tooth, against the cheek), however you can also see them on the inside.

Diagnosis is done by a combination of visual, tactile and radiographic means. The lesions will usually start out as little erosions along the gumline with associated inflammation to the gums in the area. They can progress to large holes in the teeth, and eventually can destroy most of the tooth. In severe cases, the entire crown of the tooth can be lost, with only the roots remaining. These lesions will usually be rough when an explorer is rubbed along the suspected lesion.

Dental radiographs are absolutely critical for treating patients with tooth resorption. First, it is used to diagnose lesions under the gumline. However, they are also important to determine the proper method of therapy (See below).

Currently, the only accepted method of treatment for clinical (meaning the lesion has progressed into the mouth) tooth resorption is extraction. In general, complete extraction of the tooth and all root structure is recommended. However, extraction of these teeth is often quite challenging. This is because it is very common for the roots to be significantly resorbed and replaced by bone, making extraction by a surgical approach necessary. On occasion, the roots will be completely replaced by bone. This finding has led to the development of a technique called crown amputation, where the crown of the tooth (area within the mouth) is removed, the area smoothed, and the area sutured over, allowing the body to continue resorbing the roots. This is a much less invasive procedure than a surgical extraction.

However, crown amputation can only be performed with advanced resorptive lesions where no root structure remains. Dental radiographs are required to determine this. Always insist on dental radiographs when taking your cat to the veterinarian for a dental procedure.

Restoring (filling) tooth resorptive lesions is not recommended. This is because there is no way to stop or slow down the resorptive process and it continues under the filling. In a short period of time (usually around 6 months), the tooth loss will progress to the point of the filling falling out and needing extraction regardless.

**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, where as 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 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 peripheral odontogenic fibromas (previously called epulids). These are benign overgrowths of the periodontal ligament (harmatomas). These can grow very large, but are not aggressive. Acanthomatous Ameiloblastomas (epulids) 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.

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.

Fibrosarcomas and SCC in dogs are very aggressive locally, but tend to spread only very late in the disease course. The best therapeutic option at this point is early, aggressive surgery (2-3 cm surgical margins). In dogs, radiation therapy has shown promise as effective for palliation (1 year survival)

Key Points:
• All fractured teeth with direct pulp exposure require root canal therapy or extraction
• All pathologic processes require dental radiographs
• Uncomplicated crown fractures are painful and can become infected.
• Early, aggressive surgery is the best treatment for oral cancer.

Further Reading:
• Niemiec BA: Dental, Oral, and maxillofacial pathology, a color handbook.(Manson)
• Niemiec BA: Dental Applications in Emergency Medicine (Practical Veterinary Publishing)
• Niemiec BA: Veterinary Endodontics (Practical Veterinary Publishing)
• Niemiec BA: Veterinary Orthodontics (Practical Veterinary Publishing)
DENTAL EXTRCTIONS MADE EASIER

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Extractions are surgery, and therefore need to be treated with appropriate respect. Patience and gentle technique is the best way to achieve a successful outcome. All extractions can be broken down into simple, single rooted extractions. Therefore proper elevation and extraction techniques learned and performed on incisor teeth will make all extractions easier. Proper and well maintained equipment is critical for successful extractions. This author prefers luxating elevators to standard or winged elevators. Small extraction forceps and needle holders will also benefit the surgeon.

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 post-extraction 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 post-operative, and it is therefore important to administer the drugs BEFORE the painful procedure. (See notes in previous lecture for pain management)

SINGLE ROOT EXTRACTIONS

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.

**EXTRACTION OF MULTI ROOTED TEETH:**
Section all multi-rooted teeth into single rooted pieces. The roots of almost all multi-rooted 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 hand piece. 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.

**Key Points:**
- Proper preparation and mindset are crucial
- Small/sharp instruments are the most effective choice
- Gentle elevation and patience are necessary for successful outcomes
- All teeth can be broke down into simple single root extractions
- Dental radiographs are an invaluable resource

**Further reading:**
- Niemiec BA: Dental Extractions Made Easier. Practical Veterinary Publishing. Tustin CA
Interpreting dental radiographs can be daunting, but it is very similar to interpreting a standard boney 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, 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 not 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 fibrocartilagenous 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 furcational 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 on only on surface (i.e. lingual, palatal, or facial) may be hidden by superimposition of bone or tooth. This may resulting in a non-diagnosed 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 multi-rooted 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 non-vital, this development stops secondary to the death of the odontoblasts. Consequently, non-vital 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 (TR’s)**

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 authors 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 boney 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 tumor leaving a decalcified soft tissue filled space in the tumor 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 (eg 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 tumors. Finally, aggressive tumors 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, post-operative radiographs are critical in all cases. In addition, they will serve as a legal document in cases of complications.
Rule #1 Cats are NOT small dogs (or people).
They have unique problems that are not encountered in other species. This coupled with the fact that they have only been domesticated a short time means we know little about them. In reality we have only been treating them with advanced medicine for a few decades. This is really just a blip in the grand scheme of things. There are a few disease processes that they get in their mouths which are generally not described in other species, especially the eosinophilic granuloma complex. In addition, they will commonly develop lip trauma following maxillary canine extraction. This can be a very frustrating problem to the client.

They are less affected by the common canine problems such as endodontic and severe periodontal disease. They do, however, get these problems. They generally respond to the same treatments that work in canines and people (IE root canals for endodontic disease and cleaning and homecare for periodontal disease).

Rule #2 Cats DO NOT read textbooks
They do not follow the nice little rules set out in textbooks for their treatment. This may be due to the fact that we often break rule number one and treat them like dogs (much to their dismay!). However, things that work for one cat may not work for the next one. This may be due to the fact that we are treating multiple disease processes that have the same clinical signs. In addition, cats often have unusual presentations for their oral problems. Often there will be no or subtle signs of oral disease. Be prepared for your initial assessment to be proved wrong.

Rule #3 Cats will almost never stop eating because of their mouth
Patients with the most horrific mouths will generally eat just fine. Cats with numerous FORL’s, cancer, periodontal disease, abscessed teeth, etc. will still generally eat. I feel that this is due to the fact that they do not expect food to be there tomorrow. In general, anorexia in all veterinary patients is generally due to some systemic disease process (renal, neoplasia, infectious). The exception to this rule is L/P Stomatitis. These cats will often not eat. Therefore you must completely work-up a patient prior to assuming the cat is not eating due to oral pain.

PRE-OPERATIVE TESTING
Far too often we hear “my pet is too old for anesthesia”. Sometimes this is from clients, but often they are hearing it from their veterinarian. Generally, there are no specific issues just that the patient is over ten. First, it is known that age is not a disease. In fact, when all other factors are equal (meaning the patient has no other medical problems) age was proven to not be a negative indicator in anesthetic complications. While it does increase the odds that the patient does have some systemic (cardiopulmonary or metabolic) derangement, it does not guarantee it.
Therefore, you cannot tell if a patient is (or is not) an anesthetic candidate until a minimum database is performed.

A. Complete blood panel (renal, hepatic, CBC, T4)
B. Urinalysis (critical for evaluation of renal values in cats)
C. Chest radiographs
   - HCM is often not ausculted
   - Over 50% of patients over 6 have significant findings on chest film
D. Blood pressure

Once the baseline health of the patient is established, you (or we) can determine the appropriate risk: benefit ratio for each patient. Even if there is mild to moderate metabolic derangements, the vast majority of patients would benefit from good oral health. Even patients with severe systemic derangements can be treated, especially if their level of disease is significant.

FELINE TOOTH RESORPTION (TR)
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 remodeling 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 caudal 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.

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 peri-apical 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.

FRACTURED TEETH
This is very similar to canine endodontics. However, true fractures are rare in any teeth except the canines. Teeth with direct pulp exposure are painful and/or infected and require root canal therapy or extraction. Root canal therapy is always recommended if possible as extraction of the maxillary canines is challenging and maxillary canine extraction carries a high risk of lip catching. (see below).

There are two main differences between dogs and cats with regard to endodontic disease. First, the pulp chamber of the canine teeth extends very close to the cusp tip. This means that any fracture (no matter how small) is suspect for endodontic disease. Anesthesia for careful probing and dental radiographs should be performed for any fracture. Secondly, cats will tend to resorb the root apex when a tooth has a chronic infection.

LIP TRAUMA FOLLOWING MAXILLARY CANINE EXTRACTION
In my experience, approximately 1/3 of cats that have maxillary cuspids extracted surgically will develop lip trauma from the mandibular canine. For this reason, we try to avoid extracting maxillary canines when possible preferring to perform root canal therapy or periodontal surgery if indicated. Many of these cats will show no clinical signs, however if the lip is examined, ulcers will be present. These cats are painful and are in need of therapy. Other patients may show mild to severe evidence of discomfort. The options for therapy include coronal amputation and vital pulp therapy or extraction of the offending mandibular canine.

Case report: “Sid” had both maxillary canines extracted elsewhere due to periodontal disease. He presented with a complaint of having difficulty eating and the owner was concerned about a TMJ problem because of the way he moved his jaw after eating. Oral exam revealed significant trauma to the lips secondary to the mandibular canines. The treatment was to perform coronal amputation and vital pulp therapy on the mandibular canines. This is much less painful for the patient and will maintain the strength in the rostral mandible.
EOSINOPHILLIC 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 non-painful, 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.

Histopathology should be performed to confirm the diagnosis. Following confirmation of the diagnosis, a thorough allergy evaluation should be conducted including food trial, flea treatment, +/- allergy testing.

The acute disease process is best treated with systemic corticosteroids; however corticosteroids should NOT be used for long term disease control due to the significant systemic side effects. The typical initial protocol is prednisone 2 mg/kg q 12 hours for 3-4 weeks. Additional options include intralesional triamcinolone (3 mg weekly) or methyl prednisone injections.

Antibiotic therapy is required occasionally to induce remission and/or treat secondary infection. There are also cases that appear to respond to antibiotic therapy alone. Therefore, we initially treat mild cases with antibiotics alone and more severe cases with a combination of antibiotics and corticosteroids. Many cases remain idiopathic, requiring lifelong therapy; options for this include antibiotics and cyclosporine. Fewer side effects may be expected with cyclosporine in comparison to steroids, but there are reports of opportunistic fungal and fatal protozoal infections associated with its chronic use.

CAUDAL STOMATITIS
This is another frustrating oral inflammatory disease. The best description is a severe immune mediated reaction to dental tissues. 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.
A pre-operative blood panel will generally show a marked elevation in globulins (Polyclonal gammopathy) and total protein.

Histopathology is recommended but not required. There have been a few cases with the classic look that were created by another pathology (fungal, Pemphigus). In this case full mouth extractions would be ineffective. Recently bartonella has been implicated as a possible cause of stomatitis. This is due to the high incidence of bartonella in the domestic feline population.

Stomatitis is one clinical sign of bartonella infection; however it is not a typical cause. The other major sign is lymphadenopathy. If you see severe lymphadenopathy with stomatitis, consider testing prior to therapy. Most veterinary dentists do not really think that this is the cause of the vast majority of cases. Treatment is zithromax for 21 consecutive days. In multi-cat households the patient must be isolated or all patients treated. The results are questionable at present and therapy is pricey.

**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 (depomedrol 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 anti-inflammatory effect.

Other immune suppressive such as Imuran, Cytoxan, Gold Salts, Cyclosporine have been used. However, they are all very expensive with numerous adverse side effects (mylosuppression). Cyclosporine is currently the most commonly prescribed immune modulatory drug (other than steroids) for this disease process. However, its chronic use is somewhat expensive and has been implicated in severe fungal and protozoal infections. Starting dose is 5-10 mg/kg. Look for a trough level of about 500 ng/ml on regular basis. In most dentists opinion it is only really effective AFTER teeth are removed. However, it has shown promise in resistant cases.

Laser therapy is not proven at all, most clients and RDVM’s are very unhappy with the long term results. It is very expensive and short term relief only.

**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 post-operatively as well as long term. For extractions to be successful, the
teeth must be COMPLETELY removed. Therefore post-operative radiographic confirmation of complete extraction of the tooth roots is recommended. Following the insurance of complete removal of the teeth, perform aveloplasty to remove the periodontal ligament and smooth rough bony edges. This is typically performed do this 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.

**FELINE JUVENILE (PUBERTY) GINGIVITIS/PERIODONTITIS**

**Definition:** Juvenile periodontal disease is inflammation which occurs soon after permanent tooth eruption. This syndrome can be described in two categories, *feline hyperplastic gingivitis* and *juvenile onset periodontitis*.

**Etiology:** The etiology of this condition is unknown. However, in humans there is a period of increased susceptibility to gingivitis during the pubertal period. A genetic predisposition towards feline juvenile onset periodontitis has been reported in Siamese, Somali, and Maine Coon cats.

**Clinical Features:** *Hyperplastic gingivitis* appears as gingival enlargement and significant inflammation which is confined to the gingiva and begins during the eruptive period of the permanent dentition. Bleeding during mastication and on oral exam are common findings. While occasionally seen in dogs, this condition has a much higher incidence in cats. It is generally a non-painful condition for the patient, and halitosis is a common complaint. If left untreated, it typically proceeds quickly to periodontal disease, which may result in early exfoliation of the teeth. This disease is commonly mistaken for caudal stomatitis. The distinguishing clinical sign is the lack of caudal inflammation in this disease process. As the patient matures, susceptibility appears to subside at approximately two years of age.

In contrast, *juvenile periodontitis* does not involve enlargement of the gingiva and usually leads to the rapid proliferation of plaque and calculus and subsequent inflammation. This in turn results in significant early bone loss, periodontal pocket formation, and furcation exposure. This is generally the worst in around the mandibular first molars. Treatment and effective management of these cases is often exceedingly difficult.

**Diagnostics:** Histopathology (via incisional biopsy) should be considered to rule out other causes of gingival inflammation. Culture and sensitivity testing is generally unrewarding, but may be of value in non-responsive cases. Dental radiographs should be performed to evaluate the quality of the alveolar bone and also for early tooth resorption. Finally, Bartonella testing may be beneficial in some cases, especially in patients who do not respond to traditional management practices.
Management: In the management of both of these conditions, early (9 months of age) and frequent (q 6-9 months) dental prophylaxis (even if only minimal plaque is present) along with strict homecare is critical to decrease inflammation. Ideally, homecare consists of daily brushing, as it is the gold standard of plaque control. Other homecare alternatives include chlorhexidine rinses as well as plaque control diets and treats. In cases where gingival hyperplasia is present, early gingivectomy is recommended to remove pseudopockets, decrease inflammation, and facilitate plaque control (both professional and homecare). Finally, extraction of any significantly diseased teeth is warranted to decrease the degree of inflammation.

Key Points:
• The diagnostic key between caudal stomatitis and periodontal disease is the presence of inflammation in the caudal area.
• All fractured teeth in cats are suspect for endodontic disease.
• Extraction is the treatment of choice for caudal stomatitis and tooth resorption. Dental extractions are critical for proper therapy.

Further Reading:
• Niemiec BA: Dental, Oral, and maxillofacial pathology, a color handbook.(Manson)
• Niemiec BA: Dental Applications in Emergency Medicine (Practical Veterinary Publishing) www.practicalvetpublishing.com
• Bellows, J: Feline Dentistry (Wiley Blackwell)
Challenging extractions are best performed via a surgical approach. Canine and carnassial (maxillary fourth premolar and mandibular first molar) teeth are typically considered “difficult”. However, it is also beneficial for teeth with root malformations or pathology (i.e., ankyloses) and retained roots. A surgical approach allows the practitioner to remove 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. Envelope flaps are created by incising the interdental gingiva and then 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 flap is created by incising the gingiva in the interdental spaces gingiva along the arcade and then releasing the tissue to or below the level of the mucogingival junction (MGJ). The advantages to this flap are:

- Decreased surgical time
- Blood supply is not interrupted
- Less suturing
- Less chance of dehiscence

The more commonly used flap includes one or two 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. Classically, the vertical incisions are created at the line angle of the target tooth, or one tooth mesial and distal to the target tooth. Line angles are theoretic edges of teeth. However, if there is space between the teeth, either a naturally occurring diastema or from previous extraction, the incision can be made in the space rather than carrying to a healthy tooth.

The incisions should be made slightly apically divergent. It is important that the incisions be created full thickness and in one motion. 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 muco-gingival junction.

Following flap elevation, buccal bone can be removed. Again, this author favors a cross cut taper fissure bur. The amount is controversial, with some dentists removing the entire buccal covering. However, this author prefers to maintain as much as possible and starts by removing 1/3 of the root length of bone on the mandible and 1/2 for maxillary teeth. This should only be performed on the buccal side. If this does not allow for extraction after a decent amount of time, more can be removed. If ankylosis is present, a significant amount of bone removal may be required.
Following bone removal, multirooted teeth should be sectioned. Then follow the steps outlined for single root extractions for each piece. After the roots are removed (and radiographic proof obtained) the alveolar bone should be smoothed before closure.

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. This can be performed with a scalpel blade, however a LaGrange scissor allows superior control. 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.

**Maxillary fourth premolar**

The first step when extracting this tooth is to create a gingival flap. Classically this is a full flap with one or two vertical releasing incisors. This will allow good exposure, as well as providing sufficient tissue for closure. However, an envelope flap is sufficient for small and toy breed dogs, as well as cats.

Full flaps are created by making full thickness, slightly divergent incisions at the mesial and distal aspect of the tooth. These incisions should be carried to a point a little apical to the mucogingival junction. Be careful to avoid cutting the infraorbital bundle as it exits the foramen above the third premolar. The flap is then gently elevated with a periosteal elevator.

Following flap creation, buccal bone is removed to a point approximately ½ the length of the root. Next, the tooth is sectioned. The mesial roots are separated from the distal by starting at the furcation and cutting coronally. Next, the mesial roots are separated by sectioning in the depression between the palatal and buccal roots. Another way to visualize this is to follow the ridge on the mesial aspect of the tooth. When performing this step, a common mistake is not fully sectioning the tooth. The furcation is fairly deep, so make sure that you have it fully sectioned by placing an elevator between the teeth and twisting gently. If fully sectioned, the pieces will move opposite each other easily.

Following these steps, extraction proceeds as described in the last lecture for single rooted teeth.

**Mandibular first molar**

In canine patients, these extractions are further complicated by a groove on the distal aspect of the mesial root. In addition, the mesial root is often curved. Finally, in small breed dogs, there is commonly a significant hook at the apex. Moreover, this tooth is the most common place for an iatrogenic mandibular fracture and it is possible to damage the mandibular nerve and vessels. This is much more likely in small and toy
breed dogs, because the roots of these teeth are much larger in proportion to the mandible than large breeds.

Bony resorption can significantly weaken the bone and predispose to a mandibular fracture. It is advised to warn clients of these potential complications. Dental radiographs are required to demonstrate the level of remaining bone. Finally, consider referral for these extractions (or possible root canal therapy).

The first step when extracting this tooth is to create a gingival flap. Classically this is was full flap with one or two vertical releasing incisors. However, this author finds that an envelope flap is sufficient in virtually all cases. Following flap creation, buccal bone is removed. Next, the tooth is sectioned and the extraction proceeds as for single rooted teeth.

**Maxillary Canine**

Maxillary canines are a very challenging extraction due to the significant length of the root. In addition, the very thin (less than 1-mm) plate of bone between the root and the nasal cavity often results in the creation of an oronasal fistula.

Vertical incisions are usually necessary for exposure and closure. At least a distal incision should be performed, and performing a mesial and distal incision will allow for increased tissue for closure.

The distal releasing incision is typically created at the mesial line angle of the first premolar. An exception exists if the first premolar is very close to the canine. In this case, carrying the horizontal component to the mesial line angle of the second premolar is recommended. This is to allow sufficient exposure for bone removal, as the root curves back to over the second premolar.

If a mesial incision is performed, it should be in the diastema between the canine and third incisor. Classically it was made at the line angle of the canine or third incisor. However, in this author’s opinion, the mesial line angle of the canine does not allow sufficient exposure and there is no reason to risk damaging the third incisor and increase surgical trauma. It is critical to fully incise the interdental gingiva to avoid tearing the flap. This is particularly challenging in the area mesial to the canine. Make sure to cut all the way to the bone. Following the creation of the vertical incisions, the flap is carefully elevated. If it is not elevating fairly easily, ensure that the interdental tissue is fully incised.

Once the flap is raised, approximately ½ of the buccal bone is removed. Make sure to remove some of the mesial and distal bone as the tooth widens just under the alveolar margin. After the bone removal, elevate the tooth carefully. Do not torque the crown too much bacularly as this will lever the apex into the nasal cavity. Once the tooth is elevated to a point of being very loose, it can be carefully extracted with forceps. The bone is then smoothed with a coarse diamond bur.

Closure is initiated with fenestration of the periosteum. When this is performed the tissue should stay in position over the defect. If it does not, tension is present and the flap will dehisce. It is critically important to relieve all tension if an oronasal fistula is
present. Close the flap starting at the corners to avoid having to start over if it does not close correctly.

**Mandibular canine**

These are quite simply the most difficult extraction in veterinary dentistry. This is due to the length and curve of the root, the hardness of the mandible, and the minimal bone near the apex. Furthermore, extraction of this tooth will greatly weaken the jaw and further predispose the patient to an iatrogenic fracture either during or after surgery. This tooth often holds the tongue in, and therefore it is not uncommon for the tongue to hang out following the extraction. Finally, the patient loses the function of the tooth. Therefore, it is strongly recommended to avoid extraction of this tooth. Referral for root canal therapy is a much better solution, if possible.

Some authors recommend a lingual approach to this extraction since less bone needs to be removed as to tooth curves lingual apically. However, this author prefers the standard buccal approach. This is because superior exposure is afforded and the flexible buccal mucosa allows for easier closure.

The flap for this extraction is generally triangular with just one distal vertical flap. A horizontal incision is created along the arcade to the mesial line angle of the first premolar. Then a distally divergent vertical incision is created. Next, the flap is carefully elevated and the buccal bone is removed to a point about 1/3 of the way down the root. More bone can be removed if necessary, but be careful with creating a larger flap or taking more bone as the mental nerve and artery exit approximately 3/4 of the way down the root. The tooth is then carefully elevated and extracted. Debridement and closure is as above.

**Extraction of retained roots**

Root fracture is a very common problem in veterinary dentistry. While it seems that removal of retained root tips is a daunting task, with proper technique and training it can be fairly straightforward. The first step is to create a gingival flap. Depending on the anticipated amount of exposure necessary to retrieve the fragments, this can either be an envelope flap or a full flap with one or two vertical releasing incisions. Following flap creation, buccal cortical bone is removed with a carbide bur to a point somewhat below the most coronal aspect of the remaining root. If necessary, the bone can be removed 360 degrees around the tooth, but this author tries to avoid this aggressive approach.

Once the root(s) can be visualized, careful elevation with small, sharp elevators is initiated. Once the tooth is mobile, it can be extracted normally. After radiographic confirmation that the tooth is fully extracted, the bone is smoothed and the defect closed.

**Oronasal fistula repair**

In most cases, the single layer mucogingival flap technique is sufficient to repair ONFs, especially when done correctly the first time. This is the most common surgical treatment used to repair ONFs and therefore will be presented here.
The single layer mucogingival flap is created with either one or two vertical incisions. Depending on the size and location of the fistula as well as presence of the offending tooth, a horizontal interdental incision may also be necessary for successful repair. Proper design of the mucogingival flap will allow maximum exposure of the area for extraction of the tooth (if necessary), debridement of the fistula, and critically important tension-free closure.

Incisions are created with a number 15 or 11 scalpel blade. As described previously, the vertical incision(s) were classically started at the line angle of the teeth. A line angle is a theoretic corner of a tooth. When repairing an ONF associated with a maxillary canine tooth, the distal incision is made at the mesial line angle of the first premolar, and the mesial incision is started at the mesial line angle of the canine (if present). However, it is not necessary to cut over to a line angle if there is a diastema. If the tooth is already absent, the incisions are made at the mesial and distal edges of the fistula.

When making flap incisions, adequate pressure should be placed to ensure full thickness of the soft tissue is incised down to the bone. Any vertical incisions should be created slightly divergent as they proceed apically. Divergent incisions allow for adequate blood supply for the newly created pedicle flap. It is important to choose the location of the incisions to ensure that sutured margins will have adequate bony support and will not lie over a defect.

The mucogingival flap is gently elevated off the bone using a periosteal elevator. Approximately 2-3mm of palatal mucosa is also gently elevated/lifted off the palatal bone so that fresh epithelial edges are created. Any margins of the flap associated with the oronasal fistula should be debrided using a LaGrange scissors or coarse diamond bur to remove 1-2mm of tissue, leaving fresh epithelial edges.

A coarse diamond bur on a high-speed handpiece is used to smooth the edges of the remaining maxillary bone (if necessary) and to remove any epithelial remnants between the fistula and the nasal cavity.

As with any closure in the oral cavity, the key to success is to ensure there is no tension on the incision line. Fenestration of the inelastic periosteum (see previous section on surgical extractions) is performed to increase the mobility of the flap and allow for a tension free closure. This is accomplished by a combination of sharp and blunt dissection with a LaGrange scissors to ensure the overlying mucosa is not damaged. The gingival flap is then placed over the defect so that it remains in position without being held. Once this is accomplished (i.e. no tension is present), the flap is ready to be sutured into place.

Placing a subcuticular layer can improve the chances of healing. A few buried horizontal mattress sutures will help maintain the flap as well as smooth out the incision line. Finally, this layer cannot be licked out by the patient.

Closure is performed as described in previous sections, with the initial sutures placed at the corners of the flap. This will avoid having to resuture the flap if it does not align correctly. This is not necessary if a subcuticular layer has been placed. The remainder
of the flap is then sutured over the defect in a simple interrupted pattern every 2-3 mm using an absorbable suture material. Suture lines should be placed over healthy bone.

Further reading:
• Niemiec BA: Dental Extractions Made Easier. Practical Veterinary Publishing. Tustin CA www.practicalvetpublishing.com
Introduction
A thorough knowledge of gastrointestinal vascular anatomy is required. Gentle tissue handling and careful, accurate suture placement will promote successful enterotomy/anastomosis healing. Gastropexy can be performed prophylactically or following correction of gastric dilatation volvulus (GDV). Failure of healing (dehiscence) most commonly occurs 3-5 days post-operatively. Appropriate surveillance for this potential complication is warranted.

Indications
If the practitioner is performing exploratory laparotomy with some frequency, they will be faced with several indications for enterotomy/enterectomy and gastropexy. Some of these include intussusception, neoplasia, vascular compromise as a result of a foreign body, trauma and feline idiopathic megacolon. GDV is common in deep-chested breeds and gastropexy should be performed at time of derotation to prevent recurrence. Gastropexy can also be performed in a prophylactic fashion.

Technique – Enterotomy
Enterotomy is commonly performed for retrieval of a foreign body or intestinal biopsy. Once the foreign body has been identified and the abdomen has been thoroughly explored to evaluate for any other abnormalities, that portion of the intestine is packed off using laparotomy sponges. In the authors’ experience it is not uncommon for foreign bodies to lodge in the junction between the descending and ascending duodenum at the level of the duodenal-colic ligament. This ligament can be incised to improve mobility of that portion of the intestine. Care should be taken to not compromise the blood supply to the intestine when incising this ligament. Intestinal contents are milked away from either side of the foreign body and Doyen intestinal clamps are placed 5-10 cm away from the proposed enterotomy incision. Care should be taken to ensure the Doyen clamps do not compress the vascular supply located in the mesenteric border of the small intestine. A #15 scalpel blade is used to make a stab incision in a healthy area of bowel. The enterotomy is usually not directly over the foreign body and in an aborad location – the orad portion is usually dilated and may have been traumatized as a result of passage of foreign material. Suction and/or a laparotomy sponge is used to remove any spillage of intestinal contents. The enterotomy is enlarged using Metzenbaum scissors to the desired length and the foreign body retrieved using Allis tissue or Carmalt forceps and immediately passed off the table. For enterotomy closure, the author prefers the use of fine, monofilament, absorbable, long-acting suture material (e.g. polydioxanone, 4-0). Tension must be placed on the incision using the Doyen clamps to ensure approximation of the edges of the enterotomy. Closure is performed using a simple interrupted suture pattern. Longitudinal enterotomies are usually closed in the same orientation. For intestinal biopsy, the author uses 4 or 6 mm skin biopsy punch instruments. The biopsy punch is placed on the anti-mesenteric border and a full-thickness sample obtained by pushing the punch onto a sterile tongue-depressor. Reliable and consistent biopsy samples are obtained using this technique. The biopsy incision is closed transversely in a similar way.
manner as an enterotomy to prevent luminal constriction. Following enterotomy closure, a leak test is performed by injection of sterile saline into a portion of bowel isolated by Doyen clamps or occluded by an assistant’s fingers. The leak test is performed with a 25-gauge needle and 12 cc syringe. If leakage is noted, sutures can be placed accordingly. The Doyen clamps or assistants fingers are removed prior to removing the needle from the intestine. The surgical team’s gloves and instruments are changed following completion of any gastrointestinal tract surgery.

Technique – Enterectomy and Anastomosis
Enterectomy requires a thorough knowledge of gastrointestinal vascular anatomy as this is a key factor in decision-making. When determining the amount of intestine to remove, several factors are considered. For neoplastic masses, generally a margin of grossly healthy intestine (~5cm) on either side of the lesion is required. In a situation of devascularization as a result of foreign body, the practitioner must determine viability based on clinical judgement from intestinal appearance/color, palpation and vascular supply. With experience, this decision-making becomes less challenging. In cats with idiopathic megacolon, an ileo-colic or colo-colic anastomosis are most commonly performed.

Following routine abdominal exploration, the area of bowel in question is packed off from the abdomen using laparotomy sponges. Intestinal contents are milked 10-15cm away from both the orad and aborad side of the lesion. The enterectomy is then planned. Doyen intestinal forceps are placed on the portion of intestine that will remain within the patient and either Carmalt or Kelly forceps are placed on the ends of bowel to be resected. The vascular supply to the proposed bowel to be resected is then visualized. In obese patients this may be challenging and the practitioner may have to rely on palpatung pulses of the jejunal vessels. A window is then made around the jejunal vessels supplying the bowel to be resected and they are double ligated using 3-0 absorbable suture material. In obese patients where the vessels are not visible the swaged end (not the needle tip) of the suture needle is passed around the vessels to avoid inadvertent laceration resulting in nuisance hemorrhage. The arcade vessels, which run along the mesenteric border, are then double ligated at the orad and aborad ends of the proposed enterectomy. These vessels can also be challenging to visualize and the swaged end is preferably passed when ligating these vessels. Once the vascular supply has been ligated, discoloration of the proposed bowel resection will occur rapidly. This will ensure the practitioner has ligated the appropriate vascular supply and the resection has been planned correctly.

Transection of the bowel is then performed at a right angle or with slight angulation at the level of the Doyen clamps. When angulating the bowel incision, the practitioner must understand that the blood supply is originating from the mesenteric border. Therefore, the anti-mesenteric portion of bowel should be shorter than the mesenteric portion to ensure appropriate vascular supply (FIGURE 1). Angulation can also be performed to resolve luminal disparity (luminal disparity will be demonstrated further in lecture).
Figure 1. Intra-operative picture of a jejuno-jejunal intussusception. Dotted lines represent location for enterectomy. Angulation of bowel ends is performed to maximize blood supply to antimesenteric ends.

Suturing of the bowel ends can now commence using a simple interrupted, approximating pattern of fine monofilament, absorbable, long-acting suture (e.g. polydioxanone, 4-0). The most common location for anastomosis failure is at the MESENTERIC border. This is likely a result of the mesenteric fat which obstructs the view of the tissue layers in this region. For this reason, the author routinely places 4-5 sutures in the mesenteric border FIRST. Bites should be ~2-3 mm wide and must incorporate the submucosa. Once the mesenteric border has been sutured, then the ends of the bowel at the anti-mesenteric location can be approximated. The defects are then filled in accordingly by flipping the bowel sides back and forth to close the defect in the opposite side. A simple continuous pattern can also be considered for closure of the enterectomy. Caution must be exercised if using this pattern for closure to ensure appropriate and accurate apposition of the mesenteric border ends of bowel.

A leak test is then performed by digitally occluding the orad and aborad portion of the enterectomy and filling the lumen with sterile saline using a 25 gauge needle on a 12cc syringe. The authors commonly use the 5-5-10 rule, where the bowel is occluded (digitally or with doyen forceps) 5cm orad and aborad from the anastomosis site, and then filled with 10 ml of saline. Once no leakage is confirmed, digital occlusion is released prior to removing the needle. If leakage of saline is visualized, additional sutures may be required. A local lavage of the anastomosis is performed and then the surgical team’s instruments and gloves are changed. The defect in the mesentery is carefully approximated with care taken not to lacerate jejunal vessels. Finally, an omental wrap is then placed over the anastomosis. The author does not suture the omentum as the suture tags “grasp” the omentum.
Some surgeons perform serosal patching of the enterectomy site. Serosal patching involves suturing of an antimesenteric portion of intestine over the enterectomy site to provide an additional support for bowel healing. This technique will be further discussed in lecture.

Technique – Gastropexy
Gastropexy is performed as part of the surgical treatment for GDV following derotation of the stomach to its normal anatomical location. Prophylactic gastropexy is also being performed commonly in predisposed breeds to prevent the potential morbidity and mortality associated with GDV. Numerous methods exist for gastropexy, however, the author prefers incisional gastropexy due to its speed, technical ease and biomechanical strength. An assistant is of great benefit for this procedure. The pyloric antrum is located, which is 5-7cm orad to the pylorus. The pylorus can be identified as a thick, muscular ring in the right cranial abdominal quadrant and may be confused as a gastric foreign body. Practitioners should become familiarized with the pylorus, as in cases of GDV it is found in the left cranial abdominal quadrant and can be readily palpated. It must then be grasped and rotated ventrally back to the right (and anatomically) correct side of the abdomen. A 5-7cm incision is made in the pyloric antrum depending on the size of the dog midway between the greater and lesser curvatures in a parallel plane to the long axis of the stomach. The incision is then created through the seromuscular layer only and the mucosa/submucosa layer will be noted to bulge through this incision. In case of penetration through the mucosa/submucosa layer, the defect is closed primarily using a monofilament, absorbable, long-acting suture prior to continuing with gastropexy. The primary surgeon moves to the left side and an assistant moves to the right hand side of the patient and elevates the body wall using two towel clamps placed through the external rectus fascia. The gastric wall incision is then matched to a location in the body wall caudal to the last rib and in the ventral 1/4th of the body wall. An incision is then created using a #15 scalpel blade through the peritoneum and transversus abdominus muscle in a craniodorsal to caudoventral direction approximating the same length as the gastric seromuscular incision. The craniodorsal aspect of the body wall and gastric incisions are sutured using a monofilament, absorbable long-acting suture (e.g. polydioxanone, 0 – 3-0). At the ventral aspect of the incision, the suture is tied to prevent a purse-string effect and then continued along the caudal aspect of both incisions. Failure of incisional gastropexy is uncommon. Fistula formation has been reported following the use of polypropylene sutures and is not recommended.
INTRODUCTION
Cystotomy is one of the most commonly performed surgical procedure of the urogenital tract for retrieval of cystic calculi, with other indications including ureteral evaluation and biopsy/removal of masses. This procedure will be the focus of the 50-min lecture. Perineal urethrostomy will also be discussed in these notes.

Several key principles should be followed when performing cystotomy in dogs and cats. For example, the urethra should be included in diagnostic imaging prior to surgery and should urethral calculi be present, they should be hydropulsed back into the urinary bladder. There is no reason to perform a dorsal cystotomy despite concerns for urine leakage and/or adhesion formation to a ventral cystotomy incision. A cystotomy incision should be closed using a simple continuous, appositional pattern with a monofilament short-acting suture with FULL-thickness bites of the bladder wall which will ensure the submucosa is captured. Inversion of the urinary bladder as a second layer is not required and contraindicated in cases where there is a thick bladder wall. Post-operative diagnostic imaging is a REQUIREMENT of the procedure to ensure all calculi have been retrieved. Minimally-invasive surgical (MIS) techniques have recently been described for cystic calculi removal.

PREOPERATIVE PREPARATION
A thorough pre-operative evaluation and patient stabilization is performed prior to undertaking surgery of the urogenital tract, especially in cases of urethral obstruction secondary to calculi. The vast majority of animals undergoing cystotomy are healthy and standard protocols for anesthesia will suffice. Caution should be exercised in patients with hyperkalemia and this should be addressed prior to induction of anesthesia.

If pre-operative radiographs (performed under general anesthesia) reveal urethral calculi, retropulsion into the urinary bladder must be performed and urethrotomy avoided. It is the authors’ experience that urethrotomy can be avoided in the vast majority of cases with urethral calculi by retropulsing them back into the urinary bladder. It is of paramount importance to perform retropulsion with the patient under general anesthesia (+/- epidural anesthesia) to allow for urethral relaxation. A schematic description of retropulsion of urethral stones is shown in Figure 1. Briefly, an assistant should place a gloved finger per rectum and occlude the urethra. An appropriately sized urinary catheter is inserted and flushing commenced. As the assistant feels urethral dilation, the finger is removed from the urethra and the sudden jet of fluid allows for urethral calculi to eventually move into the bladder after several flushing cycles. It is important to realize that, as stated previously, the vast majority of urethral calculi can be retropulsed into the urinary bladder avoiding the need for urethral surgery.
Once all calculi have been retropulsed into the urinary bladder (confirmed radiographically), the ventral abdomen is aseptically prepared for surgery. In female dogs and cats, it is ideal to place a urinary catheter (Foley) prior to surgery. In male dogs, the author recommends preparing the prepuce routinely and keeping the prepuce in the surgical field so that the surgeon is able to place the urinary catheter at the time of surgery in the operating room. This allows the surgeon the ability to perform retrohydropulsion several times at their discretion. If the prepuce is draped outside of the surgical field, a non-sterile assistant is required to perform retrohydropulsion and replacing the urinary catheter is challenging.

**OPERATIVE PROCEDURE**

A caudal laparotomy is performed; in male dogs the skin incision is created only to the lateral aspect of the prepuce (parapreputial – right hand side for a right handed surgeon). As stated previously, the author routinely leaves the prepuce in the surgical field to facilitate retrograde lavage of the urethra and urinary bladder. Following preputial incision, subcutaneous dissection is performed to the body wall and the linea alba is visualized. Preputial blood vessels will be encountered during the preputial approach and these can be ligated or cauterized. Once the bladder is visualized, a stay suture is placed in the apex of the bladder using a monofilament suture. This is readily apparent as a small, circular, fibrous scar at the cranial aspect of the bladder. The bladder is then exteriorized from the abdomen and then packed off with laparotomy sponges. If operating as a solo surgeon, the stay suture can be attached to the surgical drapes to maintain cranial tension on the urinary bladder.

A ventral cystotomy is recommended as this location provides the best visualization of the trigonal region where calculi are often found and avoids iatrogenic damage to the ureteral openings and neurovascular supply which are located in the dorsal aspect of the urinary bladder. Previous research has shown that ventral cystotomy is not
associated with an increased incidence of complications such as body wall adhesions and incisional failure resulting in uroabdomen.

Suction is extremely helpful and valuable in this procedure. The ventral ligament of the bladder attaches on the midline and is sharply detached from the body wall. This attachment can be used as a proposed location for cystotomy. A stab incision is then made into the ventral aspect of the urinary bladder. Immediately following stab incision, the suction tip is inserted to empty the bladder of urine and prevent spillage into the abdomen. The cystotomy is then extended to the desired length using Metzenbaum scissors. Readily apparent calculi are retrieved using an atraumatic instrument (e.g. bladder spoon). In some cases, calculi are not readily apparent upon performing cystotomy. This can be a result of calculi falling into the proximal urethra when positioned for laparotomy and as the bladder is exteriorized. At this point the bladder is emptied of visible calculi.

In male dogs a urinary catheter is passed by the surgeon or assistant surgeon in a retrograde manner and flushing with sterile lavage fluid initiated. Additional stay sutures can be placed in the lateral and caudal aspects of the cystotomy incision to improve visualization especially if operating solo. These stay sutures can be connected to the surgical drapes to free the surgeon’s hands for performing lavage and retrieval of calculi. The suction tip is placed in the urinary bladder as suction is being performed to improve visualization by removing lavage fluid. Suction also helps prevent spillage of fluid into the abdomen. The urinary catheter is gradually advanced while flushing with saline and then withdrawn once its visible in the urinary bladder and the procedure repeated several times until the surgeon is confident calculi are not present within the lower urinary tract. The author will routinely perform retrograde flushing several times to be confident calculi are not present in the lower urinary tract.

In female dogs and cats a urinary catheter is placed pre-operatively and gradually withdrawn (with concurrent lavage) by a non-sterile assistant. *This strategy can also be performed for male dogs, however, repeated lavage cannot be performed to ensure a calculi-free urinary tract if the prepuce is draped outside of the surgical field. In female dogs and cats, following retrograde lavage by a non-sterile assistant, normograde lavage can be performed by the surgeon. Normograde lavage should be performed cautiously in male dogs since if calculi are present, they can become lodged at the level of the os penis.

Prior to closure a crushed calculi or a mucosal biopsy should be obtained and submitted for bacterial culture and sensitivity. It is has previously been shown that antibiotics do not need to be withheld until after the bladder mucosal biopsy is obtained and, therefore, standard protocols for administration of perioperative antibiotic prophylaxis should be performed (within 60 minutes of surgical incision and re-dosed every 90 minutes for cefazolin).

**CYSTOTOMY CLOSURE**
Several strategies exist for cystotomy closure. The author usually performs a single layer, appositional closure with a monofilament, rapidly absorbable suture material (e.g. polyglecaprone 3-0). A clear advantage of a double layer inverting pattern has not been demonstrated in recent studies. In fact, a double layer inverting closure may
be challenging to perform in bladders where marked thickening of the wall exists. In fact, in animals with a thickened bladder wall secondary to cystic calculi, the author believes a second inverting layer is contraindicated as this may result in additional trauma to the urinary bladder wall, compromising closure integrity. Full thickness bites of the urinary bladder wall should be taken in order to capture the submucosal layer (holding layer). Ideally, the mucosa is not captured so as to prevent suture exposure within the urinary bladder as this could be a potential for suture-associated calculi. However, it is very likely that exposed suture becomes epithelialized and it is more important to ensure the submucosa is captured during cystotomy closure. The urinary bladder is unique compared to other tissues in that 100% of bursting strength following cystotomy is achieved after 3 weeks. Post-operative radiographs should be performed in all cases to ensure complete calculi removal. Three-view radiographs should be performed including two lateral views with the limbs extended and flexed to ensure a complete view of the urethra. Should calculi be present on post-operative imaging it is much easier to return to surgery to remove retained calculi than to continue some type of medical management.

POST OPERATIVE MANAGEMENT
In the authors’ institution patients are recovered on intravenous fluids overnight. Non-steroidal anti-inflammatory therapy (pending contra-indications) is highly recommended for urogenital surgery. A urinary catheter is not maintained in most cases. Discharge of the patient is performed 24hrs postoperatively. At that time the animal should be urinating normally and may or may not have hematuria present which the owner should be cautioned about. If the animal has not urinated or is straining to urinate this warrants diagnostic investigation as to the cause (e.g. uroabdomen, incomplete calculi removal).

MINIMALLY-INVASIVE CYSTOTOMY
Minimally invasive techniques such as laparoscopic-assisted cystotomy and percutaneous cystolithotomy have been reported for the retrieval of cystic calculi in dogs and cats. Purported advantages of endoscopic-assisted techniques include smaller incisions and reduced tissue trauma, improved visualization of the urinary bladder and the ability to remove very tiny (<1mm) calculi from which are difficult to see at the time of open cystotomy. These tiny calculi may be a nidus for future calculi growth also termed “pseudorecurrence” in human surgery. A comparative study between laparoscopic-assisted and open cystotomy revealed that the minimally invasive technique took significantly longer and was significantly more expensive. However, dogs undergoing laparoscopic-assisted cystotomy required significantly less analgesia than the open cystotomy group.

FELINE PERINEAL URETHROSTOMY
This procedure most commonly indicated for permanent enlargement of the urethral opening in male cats after recurrent urethral obstruction. Medical management should be initiated as in many instances urethral obstruction secondary to feline lower urinary tract disease (FLUTD) can be managed medically.

Once the decision has been made to perform perineal urethrostomy (PU) the practitioner should have in-depth knowledge of relevant anatomy. The most common reason for stricture development at the PU site is failure to create a stoma in the pelvic
portion of the urethra. Instead, a stoma is created in the penile portion of the urethra where the lumen remains much smaller compared to the pelvic portion. Meticulous dissection and atraumatic technique is also of great importance. Finally, ensuring that accurate urethral mucosa/skin apposition occurs is a key surgical principle in preventing stricture at the PU site.

OPERATIVE PROCEDURE
The patient is positioned in sternal recumbency with a slight downward tilt. A towel can be placed underneath the axilla to ensure excessive pressure on the diaphragm is not present. A purse-string is placed in the anus. A tomcat catheter is placed in the urethra to guide dissection. An elliptical incision is created around the base of the scrotum and prepuce. Using a combination of sharp and blunt dissection, the subcutaneous tissue is incised. The ventral attachment of the urethra to the pelvis is identified and transected using Metzenbaum scissors. The penile body is retracted laterally to identify the ischiourethralis muscles. The scissors is placed on either side of its attachment on the ischium and is transected. Incising mid-body through this muscle will result in nuisance hemorrhage. This procedure is repeated on the contralateral side. At this stage, an index finger can be placed ventrally between the urethra and the pelvis to ensure all attachments have been severed. Excessive dissection of the dorsal and lateral muscle attachments is avoided to reduce the risk of fecal and urinary incontinence secondary to neurovascular trauma. Once the dissection is complete the bulbourethral glands should be readily apparent and are located just cranial to the insertion of the ischiourethralis muscles.

A #11 blade is used to incise the urethra over the tomcat catheter at the level of the scrotum. Fine scissors are used to extend the urethrotomy along the dorsal surface of the penile body to the level of the bulbourethral glands. A characteristic “crunch” can be felt when incising the urethra at the bulbourethral glands. The urethral diameter at this level should be wide enough to accept a closed mosquito hemostat to the level of the box locks so that the tips are no longer visible. The urethrotomy should be extended cranial until this is the case.

A monofilament, absorbable, suture material (e.g. polydioxanone - 5-0, 6-0) is most commonly used for PU. The first two sutures are pre-placed to ensure accuracy and are positioned at the 10 and 2 o’clock positions. The dorsal most (12 o’clock) suture is placed ensuring maximal urethral diameter by placing a mosquito hemostat within the stoma and slightly opening it to improve visualization when biting through the urethral mucosa. The pre-placed sutures are tied and urethral diameter tested again. If reduction in diameter occurs, remove the sutures and replace as needed. The urethral mucosa is then sutured to the skin using a simple-continuous pattern on either side of the new urethral stoma. A “draining board” of ~2cm should be left. The distal penile body is amputated prior to skin closure. In the post-operative period, an Elizabethan collar is mandatory for 7-14 days to prevent self-trauma to the surgical site.

Further Reading:
Introduction

Minimally invasive surgery (MIS) is performed with increasing frequency in veterinary medicine. Laparoscopy and thoracoscopy are MIS techniques that provide minimally invasive access to the abdominal and thoracic cavities, respectively.

Benefits

Numerous benefits of MIS exist and include reduced post-operative pain, shorter hospital stay, reduced complications and faster return to normal activity. Additional benefits of laparoscopy and thoracoscopy include improved visualization of the abdominal or thoracic cavities, lower incidence of surgical site infections and reduced surgical stress.

Instrumentation

The basic instrumentation necessary to perform small animal laparoscopy and thoracoscopy can be found in previously published literature. Briefly, a rigid endoscope specific for laparoscopic or thoracoscopic use is attached to a light source to provide illumination within the body cavity. The endoscope is also connected to a video monitor for displaying the camera’s field of view. “Portals” (also termed trocar-cannula assemblies) are used for insertion of the camera and endoscopic specific instruments.

In laparoscopy, a mini-celiotomy approach, termed the modified-Hasson technique, is performed in the sub-umbilical region to allow placement of the first portal. Once this portal is inserted into the abdomen, a mechanical insufflator is used to insufflate carbon dioxide (CO₂) into the abdomen to a maximum pressure of 10-15 mmHg. CO₂ (non-combustable gas) insufflation is required to provide a working space within the abdominal cavity. Following insertion of the camera into the abdomen, additional portals can be placed (depending on the procedure) with guidance from the laparoscope to prevent iatrogenic trauma to abdominal organs. The mechanical insufflator is able to detect an intra-abdominal pressure drop and insufflate CO₂ accordingly. Leakage of CO₂ may occur during instrument exchange through the portals or around the portals if the incision is larger than necessary.

In thoracoscopy, the initial camera portal can be placed in a para-xiphoid location with the patient in dorsal recumbency or in an intercostal location depending on the lesion location/desired working space. Insufflation is not commonly performed during thoracoscopy as working space is created when portals are placed allowing free movement of air into the thoracic cavity. All thoracoscopy patients are mechanically ventilated. For advanced thoracoscopic procedures (e.g. total lung lobectomy) one lung ventilation (OLV) is required to further increase working space. OLV is performed using either a double-lumen endobronchial tube or an endobronchial blocker. Dogs are able to tolerate OLV extremely well in most instances.

Procedures/Indications
Indications for laparoscopy include: Organ biopsy (liver, spleen, gastrointestinal tract, lymph node, peritoneal, pancreas, prostate, and kidney), cholecystocentesis, cholecystectomy, prophylactic gastropexy, cryptorchidectomy, ovariectomy/ovariohysterectomy, cystotomy, adrenalectomy, and gastrointestinal feeding tube placement.

Indications for thoracoscopy include: Organ biopsy (pleura, lung, mediastinum, lymph node), mediastinal mass removal, thoracic duct ligation, lung lobectomy, exploration +/- lavage and debridement, and pericardectomy.

In addition to entirely laparoscopic/thoracoscopic procedures, “assisted” procedures are also commonly performed in human and veterinary surgery. In endoscopic-assisted procedures, a traditional “open” approach is not performed and assistance is provided through a small open incision. This reduces the post-operative morbidity associated with standard “open” surgery. An example of an assisted procedure is laparoscopic-assisted gastropexy. Following placement of a sub-umbilical camera portal and paramedian instrument portal, the stomach is grasped with laparoscopic atraumatic forceps. The instrument portal is enlarged and the stomach exteriorized through this incision. A routine incisional gastropexy is performed. This laparoscopic-assisted technique reduces post-operative morbidity, hospital stay and return to normal function that may be associated with a gastropexy performed via full laparotomy. Additional assisted procedures include: cystotomy for bladder stone retrieval, intestinal biopsy, enterotomy or resection and anastomosis and splenectomy and thoracoscopic-assisted lung lobectomy.

Several of the most commonly performed by the author are discussed below: (several of these procedural videos will be shown during lecture)

**Ovariohysterectomy (OVH) and Ovariectomy (OVE):** Either technique can be performed using laparoscopy, however, OVE is technically easier. If deciding to perform OVE, case selection is important, and should generally be limited to dogs <2 years of age as progesterone priming of uterine tissue has not progressed to an advanced stage. It is important to note that based on research out of Europe, OVE has NOT led to increased incidence of pyometra, uterine neoplasia, endometritis, urinary sphincter mechanism incompetence or mammary gland neoplasia compared with OVH. OVE or OVH is commonly performed at time of prophylactic gastropexy, in mature, large or giant breed dogs or in dogs with coagulation abnormalities (e.g. von Willebrands disease).

**Prophylactic Gastropexy:** Gastric dilatation and volvulus (GDV) remains a life-threatening disease with mortality rates depending on time of presentation to a veterinarian. Prophylactic gastropexy is recommended in predisposed breeds (Great Dane, Standard Poodle, Weimeraner) to prevent GDV and the potential associated mortality/morbidity. When using traditional “open” surgery for prophylactic gastropexy, a full laparotomy incision is required, which can be sizable in large to giant breed dogs and result in morbidity for the patient. Some owners may deem this morbidity to high for prophylactic gastropexy. With a laparoscopic-assisted approach (briefly described above), post-operative morbidity is far reduced compared with “open” surgery, which can make the decision for an owner considering gastropexy much easier.
**Cryptorchidectomy:** The main advantage of laparoscopic techniques for cryptorchidectomy is the improved visualization compared with traditional “open” approaches. A potential devastating complication reported for abdominal cryptorchidectomy is inadvertent prostatectomy. This is a result of an inappropriately small caudal laparotomy incision which precludes clear visualization of the cryptorchid testicle. As with other laparoscopic techniques, the post-operative morbidity and time to return to normal function is considerably reduced.

**Cholecystectomy:** Gall bladder mucocele (GBM) is one of the most common indications for cholecystectomy in dogs. Predisposed breeds include Shetland Sheepdog, miniature Schnauzer and Cocker spaniel. GBM is commonly associated with extra-hepatic biliary obstruction (EHBO) and even gall bladder rupture. In the early stages of GBM, EHBO is not present and cholecystectomy is recommended. Early GBM is often an incidental finding at time of abdominal ultrasound for another abdominal condition. It has recently been shown that hyperadrenocorticism is associated with GBM and so dogs with this endocrinopathy and clinicopathologic evidence of EHBO should undergo gall bladder evaluation with abdominal ultrasonography. Laparoscopic cholecystectomy has been performed by the author in a small number of cases (<20) and represents one of the more challenging laparoscopic procedures. Visualization of the region of the cystic duct is far improved compared to “open” surgery and most cases are discharged from hospital the following day.

**Cystotomy:** Cystic and urethral calculi are a common diagnosis in small animal practice. A laparoscopic-assisted technique for cystotomy allows for minimally invasive removal of cystic calculi from the urinary bladder following retropulsion of urethral calculi. In this technique a urinary catheter is placed and sterile fluid is continually flowing allowing for distension and visualization of the urinary bladder. The laparoscopic portal is inserted into the urinary bladder that is temporarily fixed to the skin and suction used to retrieved calculi. Successful removal of a large number (>50) of stones is feasible in a timely fashion, however, stones larger than 10 mm are challenging to remove. Post-operative radiographs are performed to ensure all calculi have been removed. It is the authors’ impression that dogs undergoing laparoscopic-assisted cystotomy do not have post-operative hematuria likely due to the considerably smaller cystotomy incision (~5mm).

**Abdominal/Thoracic Organ Biopsy:** Endoscopic or endoscopic-assisted methods are frequently used to perform organ biopsy for chronic medical conditions. This limits the morbidity associated with a traditional surgical approach (laparotomy, thoracotomy) that is required for a diagnostic purpose. For cases requiring multiple organ biopsy of the abdomen, the author frequently employs the following strategy: upon insertion of the sub-umbilical camera portal an instrument portal is placed and liver biopsy is performed of multiple liver lobes. A superficial evaluation of the abdomen is performed. The subumbilical portal is enlarged to 4cm and the cannula removed. A wound retractor (see picture in lecture) is placed to allow for exteriorization and palpation of the gastrointestinal tract. Biopsy of the small and large intestine and mesenteric lymph nodes is performed routinely and the mini-laparotomy incision closed. Morbidity associated with this technique is reduced compared to a traditional laparotomy. Limitations to this laparoscopic-assisted method for organ biopsy is the
stomach, proximal duodenum or colon cannot be exteriorized because of their fixed location within the abdomen. Another limitation is that this technique is limited to biopsy of the liver, small/large intestine and mesenteric lymph nodes. Other abdominal organs cannot be visualized/palpated and would require open laparotomy for evaluation.

Pericardectomy: Pericardectomy is indicated for resolving tamponade associated with neoplastic, infectious or idiopathic effusions, as an adjunctive treatment for chylothorax, or for restrictive pericarditis. This is traditionally performed through an intercostal thoracotomy which can have complications related to wound healing and post-operative pain associated with rib retraction for facilitating the surgical approach. Thoracoscopic pericardectomy is an alternative to intercostal thoracotomy and is performed with the patient in dorsal recumbency. A para-xiphoid camera portal is inserted followed by two intercostal instrument portals on each side of patient. A vessel-sealing device is used to perform pericardectomy and a thoracic drainage catheter is placed prior to recovery from anesthesia. With the thoracoscopic technique, most cases are discharged from hospital the following day post-operatively.

Role of Technical Staff
The technical staff play an extremely critical role as part of the surgical team when performing MIS. Specifically to laparoscopy and thoracoscopy, the veterinary technician is required to assist in the surgical procedure and may be required to drive the endoscope and/or other instruments during the procedure. The veterinary technician should have a thorough understanding of the equipment in order to troubleshoot during times of malfunction and to provide instruction to the hospital staff responsible for appropriate sterilization and storage. Without the veterinary technician, surgeons cannot be successful at what they do, however, this is amplified with MIS because of the advanced equipment and requirement for additional assistance to the solo surgeon.

Potential Complications and Limitations
Complications relating to laparoscopy and/or thoracoscopy include hemorrhage, splenic or other organ trauma, poor visualization, need for conversion to an open procedure. It is important to perform appropriate client communication when undertaking MIS procedures and have a thorough discussion with owners about the need for conversion to a traditional approach. If this happens, this should not be deemed a failure, but instead good judgement and in the patient’s best interest. Endoscopic equipment and training is expensive and requires an initial commitment by the surgeon(s) and technical staff. In many cases, the procedural costs to clients are more expensive. Surgeon’s performing MIS techniques must be astute in case selection as that will set them up for procedural success.

Further Reading:
Pain management in the cat presents a challenge due to their peculiar behavior, the cat’s unique metabolism of many drugs, and the lack of licensed analgesic drugs. Recently, many clinical and research studies have been undertaken to understand the pharmacology of analgesics and the unique features of recognition, assessment and treatment of pain in this species.

RECOGNITION AND ASSESSMENT OF ACUTE PAIN IN CATS
Neuroendocrine assays have been correlated with acute pain in cats; however other factors such as anxiety, stress and fear, and even physiological changes during anesthesia and surgery, affect these hormones and adreno-cortical responses as well. With the exception of blood pressure, objective measurements such as heart rate, pupil size and respiratory rate have not been consistently correlated with signs of pain in this species. Hence, subjective evaluation of pain based on behavioral changes is still widely used for evaluation of acute pain in cats. Key behavioral indicators of acute pain in cats have been recently identified. In addition, there has been some recent interest on how changes in facial expressions play a role in pain in cats. Novel pain scoring systems have been recently validated for cats.

TREATMENT OF ACUTE PAIN IN CATS
There has been a substantial increase in the number of publications about the pharmacokinetics of analgesic drugs as well as their antinociceptive, behavior and inhalant anesthetic-sparing effects in the cat. Opioids may not reduce inhalant anesthetic requirements in cats as commonly observed in dogs. Pharmacokinetics (PK) of analgesic drugs has contributed to the development of rationale analgesic protocols. Dose-regimens, routes and intervals of administration have been studied for contemporary and new analgesic drugs. Robenacoxib is a new coxib which possesses analgesic, anti-inflammatory and anti-pyretic effects. This drug has shown to have good safety index in cats since it distributes selectively to inflamed tissues and inhibits COX-2, while sparing COX-1, at clinically recommended dosages. Robenacoxib is licensed for use in cats in several countries.

Subcutaneous administration of classic formulations of hydromorphone and buprenorphine has produced less antinociception or analgesia when compared to the IV and IM injection of the same opioid and dosage regimens. In addition, the oral transmucosal route (OTM) has been used for the administration of sedatives and analgesics with interesting results. Methadone, buprenorphine and (dex) medetomidine have been administered by the OTM and plasma concentrations and analgesic effects have been determined. On the other hand, sustained-release formulations of opioids and local anesthetics may be a promising technique to relief long-term (days) acute pain. A new formulation of buprenorphine (Simbadol) for subcutaneous administration has been released in the USA. This formulation may provide analgesia for up to 24 h after administration.
This lecture highlights different aspects of pain recognition, assessment and treatment of acute pain in cats. The session will provide an overview of current literature and recommendations for pharmacological interventions in acute pain states. Analgesic dose regimens, routes of administration, new analgesic techniques and drug delivery systems, and future trends and challenges in feline pain management are discussed.

Further Reading:
• Giordano T, Steagall PV, Ferreira TH et al. (2010) Postoperative analgesic effects of intravenous, intramuscular, subcutaneous or oral transmucosal buprenorphine administered to cats undergoing ovariohysterectomy. Vet Anaesth Analg 37, 357-366.
The critically-ill cat represents an important challenge for the anesthetist. This session will discuss a step-wise approach for patient stabilization in preparation for general anesthesia as well as anesthetic management for three common conditions.

“BLOCKED” CATS
Cats with urethral obstruction and lower urinary tract disease ("blocked cats") present a common and frequent challenge for the small animal practitioner.¹ They are commonly presented with lethargy, anorexia, dehydration, depression, anuria or oliguria, halitosis, abdominal pain, vomiting and diarrhea. Clinical signs will be ultimately dependent on the severity of uremia and hyperkalemia. Acute renal failure can result. A hematocrit, total protein, dipstick BUN, sodium, potassium and ionized calcium concentrations are important to guide adequate treatment and reduce the risks associated with anesthesia.

Anesthesia can be required for urethral catheterization, perineal urethrostomy and abdominal exploratory surgery (bladder rupture). A list of problems is presented with reduced glomerular filtration rate, hypothermia, hyperkalemia, acidemia and circulatory collapse with weak peripheral pulses. An electrocardiogram (ECG) identifies bradycardia and signs of hyperkalemia; it includes an increased T wave amplitude, decreased R wave amplitude, ST segment depression, decreased P wave amplitude, prolonged PR, QRS and QT intervals and not uncommonly loss of P wave with possible ventricular arrhythmias.

Management of anesthesia includes correction of electrolyte and acid-base imbalances, in addition to severe dehydration. Venous catheterization is mandatory for fluid and electrolyte administration such as 10% calcium gluconate, dextrose, sodium bicarbonate, among others. Acidemia is partially induced by potassium ions moving extracellularly in exchange for hydrogen ions which are buffered intracellularly. Metabolic disturbances will lead to hypovolemia and cardiovascular depression since the resting membrane potential is raised in hyperkalemia, and cardiac automaticity, conductivity and contractility are decreased. Cats with a serum potassium concentration greater than 6 mEq/L should not be anesthetized until hyperkalemia is treated. Warming techniques will prevent and treat hypothermia.

Analgesia is required and NSAIDs are contra-indicated due to their deleterious effects on renal tissue. An opioid (buprenorphine or hydromorphone) can be administered IV.

A step-wise approach to treatment of severe hyperkalemia is suggested herein:

- Calcium gluconate 10% (0.5-1 mL kg⁻¹ IV) is injected over 10-15 minutes. The administration of calcium will increase the membrane’s threshold potential, resulting in increased myocardial conduction and contractility. Calcium will restore the normal gradient between resting and threshold potentials of the cardiac cells and it is
indicated for life-threatening arrhythmia. However, its effects are short-lasting (15-20 minutes) and this therapy does not change potassium concentrations. Calcium is better administered while ECG is monitored.

- A dextrose bolus (0.25-0.5 g/kg IV) is diluted in two parts of saline 0.9% and administered in combination with a bolus of insulin (0.1-0.25 U/kg IV). Insulin will shift potassium to the intracellular compartment. On the other hand, dextrose will induce endogenous release which translocate the potassium intracellularly.

- Sodium bicarbonate (1 mEq/kg IV) is given slowly (10-15 minutes) and will also shift the potassium to the intracellular compartment. Its effects usually last for a few hours and is recommended if metabolic acidosis (pH < 7.1) is observed.

- Rapid fluid resuscitation is required especially if poor perfusion and severe dehydration are present in the absence of cardiac disease. A bolus of balanced isotonic crystalloid fluid such as saline 0.9% is administered at 45-60 mL/kg/h while relief of obstruction relief.

Anesthesia is required for manipulation and placement of an indwelling urinary catheter or an open-end tomcat. However, some cats may tolerate the procedure under sedation with an opioid (buprenorphine) and midazolam, or low-doses of propofol or alfaxalone. It is important to highlight that ketamine is actively excreted by the kidneys in the cat and it is usually contra-indicated. Prolonged anesthetic recoveries after ketamine may be observed in kidney failure. Inhalant anesthesia with isoflurane or sevoflurane is an option when long-term anesthesia (> 20 minutes) is required since intravenous agents may accumulate. In any case, uremia may change the blood-brain barrier permeability facilitating drug overdosing.

A sacro-coccygeal epidural block has been recently described to facilitate urethral catheterization in cats with urethral obstruction. The technique is performed under aseptic conditions and produces anesthesia of the perineal area, penis, urethra, colon and anus. Preservative-free lidocaine 2% (0.1-0.2 mL/kg) is injected for this block. Relaxation of tail and perineal region is normally observed.

**DIAPHRAGMATIC HERNIA**

Diaphragmatic hernia is caused by trauma, high-rise syndrome or attacks. Iatrogenic injuries and congenital peritoneopericardial diaphragmatic hernia will require surgical herniorrhaphy. It can cause significant morbidity and mortality in cats and trauma per se is associated with multiple fractures and organ rupture (spleen or bladder).

Organs that are commonly moved into the thorax include liver, stomach and small intestines.

Physical examination will include areas of borborygmus or silence on thoracic auscultation. A list of problems may include respiratory distress with dyspnea, severe lung collapse and pleural effusion, cardiovascular collapse with hypoxemia, decreased venous return, hypotension and poor tissue delivery, strangulated viscera with tissue necrosis, pain, hypothermia, vagal stimulation, release of endotoxins and re-expansion pulmonary edema.
A step-wise approach for patient stabilization include the administration of 100% oxygen, fluid therapy, analgesics and further diagnostics with laboratorial and radiographic examination. A stress-free induction with pre-oxygenation is paramount in these cases. Quick intubation and initiation of intermittent positive pressure ventilation (IPPV) is strongly recommended. However, IPPV and high peak airway pressures may induce re-expansion pulmonary edema.

Anesthetic management should take in consideration drugs that will not produce cardiorespiratory depression (midazolam, opioids, ketamine). Balanced anesthesia should be employed including the administration of opioids and ketamine infusions. Intercostal blocks are mandatory. Thoracocentesis/chest drain placement may be required in cats with severe pleural effusion. Mortality can be up to 20% in cats after diaphragmatic hernia repair and postoperative care is a major player in these cases. Mortality is mostly correlated with other complications and concurrent injuries. Postoperative analgesia will include opioid and ketamine infusions and interpleural anesthesia with bupivacaine up to 96 hours after surgery. Cats may be placed on an oxygen tent where pulse oximetry and oxygen concentrations are monitored.

**GI EMERGENCIES**

Emergency abdominal exploratory surgery is commonly required due to intestinal obstructions (foreign body, intussusception, neoplasias, megacolon, etc.), GI ulceration, uroabdomen, GI biopsies, etc. Some complications include fluid losses, proliferation of intestinal bacteria and secondary intestinal inflammation, GI perforation, peritonitis, systemic inflammatory response, severe hypotension, hypovolemia and shock.

Clinical findings are variable but tachycardia is observed in hypovolemic patients. Bradycardia can commonly be observed in cats with increased vagal tone. The list of problems includes hyper- or hypothermia, electrolyte imbalances and acid-base abnormalities. Hypochloremia, hypokalemia, hypoglycemia and hyperlactatemia are not uncommon, but again fluid imbalance will vary with condition (vomiting versus diarrhea, dehydration, hypovolemia, septic versus non-septic). Dehydration with cardiovascular collapse including hypovolemia, hypotension, hypoproteinemia requires aggressive fluid therapy.

Severe abdominal pain is treated with opioid and ketamine infusions since NSAIDs are absolutely contra-indicated. The intraperitoneal administration of bupivacaine is recommended at the end of the surgery and it provides postoperative analgesia for up to 8 hours. Epidural anesthesia for abdominal analgesia is an option once hypovolemia and cardiovascular collapse are treated. Anesthetic management should take in consideration drugs that will not produce cardiorespiratory depression (midazolam, opioids, ketamine). Balanced anesthesia should be employed including the administration of opioids and ketamine infusions.

Regurgitation followed by aspiration pneumonia is a common complication after induction of anesthesia. Positioning for induction should take this issue in consideration. Suction should be available and a stomach tube can be used to empty GI contents. Early postoperative management will address oxygenation, analgesic requirements, urine output, appetite, laboratory monitoring, nutrition and TLC.
Further Reading:
DEMystifying NSAIDs and their adverse effects in Clinical Practice

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Non-steroidal anti-inflammatory drugs (NSAIDs) are the most widely used analgesics in veterinary medicine.\(^1\) After the recent introduction of preferential and selective COX-2 inhibitors with improved safety profiles, these drugs became even more popular for their anti-inflammatory, analgesic, and antipyretic effects.\(^1,3\)

Early studies found that the analgesic effects of NSAIDs were secondary to the inhibition of cellular expression of cyclooxygenase (COX) enzymes in cell membranes. There are at least two COX isoforms. COX-1 isoform is a constitutive form of the enzyme found in many tissues that regulates normal homeostasis by producing prostaglandins (PG) in the gastrointestinal (GI) mucosa, and by playing a role in platelet aggregation and renal blood flow. The COX-2 isoform is also constitutively expressed in a range of tissues and organs, including ovarian and renal tissues; but it is primarily induced by damage or tissue injury as a pro-inflammatory enzyme and is responsible for the production of various eicosanoids, which are inflammation mediators and amplify nociceptive input and transmission to the spinal cord.\(^4\)

NSAIDs reveal different levels of inhibition of both COX-isoforms and for this reason may induce adverse effects that are consisted of gastric irritation, development of protein-losing enteropathy, hepatic and renal damage, articular degradation and prolonged bleeding time by prevention of platelet aggregation.\(^3\) Therefore, the clinical relevance of NSAID administration in small animal clinical practice is undoubtedly of utmost importance, especially with its increasing availability and usage. However, their popularity as unsafe drugs and the incautious use of these compounds may compromise pain management in veterinary medicine.

If there is a clear consensus that NSAIDs are effective drugs in the management of pain, there is still a lot of controversy and lack of understanding of their adverse effects. In an attempt to educate veterinarians on long-term NSAIDs administration in cats, a panel of experts has reviewed the scientific literature and provided guidelines for the use of these drugs in this species.\(^5\) Indeed, some NSAIDs are licensed for long-term use in cats in some countries and it is now clear that cats with maladaptive (chronic) pain will benefit of NSAID therapy.\(^6\) However, it is important to say that these individuals should be screened for other conditions and the lowest effective dose should be employed with careful monitoring of side effects. In addition, a thorough systematic review has been recently published on this subject in dogs.\(^3\) The authors aimed to identify, assess, and critically evaluate the quality of evidence of NSAID-induced adverse effects. Overall, it seems that the administration of NSAIDs for the treatment of acute or chronic pain in dogs where labeled dosages and intervals are respected, and close monitoring is performed, is usually safe in the vast majority of cases. Most commonly observed clinical signs after NSAID adverse reactions are vomiting, diarrhea and inappetance.
Fluid therapy and monitoring of blood pressure while avoiding hypotensive states are highly recommended during the intraoperative period in both cats and dogs after administration of NSAIDs. Dogs and cats with hypovolemia and hypotension should not be given an NSAID. Clinically significant renal toxicity or bleeding disorders does not seem to be a risk in healthy patients. Besides their potential detrimental effects on bone healing, the clinical use of NSAIDs in orthopedic surgery is unquestionably required for acute pain management. In addition, NSAIDs are normally not required for long periods after stabilization of a fracture.

When clinical signs of toxicity develop, close monitoring and alternative or symptomatic treatment should be initiated. Individual sensitivity plays an important role in the development of side effects which unfortunately cannot be foreseen. It does, however, emphasize the need of close monitoring of these patients as well as a good client-clinician communication in regarding to the potential of side-effects.

NSAIDs are crucial in the treatment of acute pain as in the perioperative period and are the cornerstone in the treatment of osteoarthritis and other chronic painful conditions as a single agent, or as part of a multimodal analgesia approach.\textsuperscript{3,5-8}

References
**MEDIASTINAL IMAGING**

Greg Starrak, DVM, DACV

**Objectives:** The objective of this lecture is to initially describe the normal radiographic anatomy of the mediastinum. This will then be compared to changes that can be seen with disease processes and variants of normal. Discussion and comparison of computed tomographic imaging of the mediastinum will be used to highlight the anatomic changes. Identification of the structures involved is critical to determining the likely differentials/etiologies as well as an aid to determining how to obtain a diagnosis. Ultrasound examination and a description of the ultrasound appearance of common mediastinal abnormalities will also be discussed. The use of computed tomography for mediastinal imaging will be highlighted as well.

**Normal anatomy.** The mediastinum is comprised of three sections – cranial, middle and caudal. The cranial mediastinum is from the thoracic inlet to the heart and contains the cranial vena cava, brachiocephalic trunk and subclavian arteries, the trachea, esophagus, lymph nodes, lymphatics, nerves and variable amounts of fat. These structures are typically difficult to see (with the exception of the interior of the trachea) as compared to other thoracic structures. This is due to the absence of air within the normal mediastinum, thus reducing contrast. (Soft tissue structures within the lungs are easily identified because of the air surrounding them.) The majority of the tissue within the cranial mediastinum is soft tissue and thus will silhouette with each other on radiographs.

On the radiograph above there is air within the mediastinum due to traumatic cervical injury (a stick penetrating from the oropharynx into the cervical tissues). On the lateral radiograph the structures within the mediastinum can now be identified due to the surrounding gas. The pneumomediastinum continues into the middle and caudal
mediastinum highlighting the location of the aorta, azygous and especially the esophagus. Gas continues to track along the aorta into the retroperitoneal space as well.

The cranial mediastinal structures are primarily in the mid dorsal thorax, central and surrounded by inflated lung. Ventrally the lungs come very close together and then are pushed apart at the level of the cava to the esophagus. This results in two visible boundaries on VD radiographs of the thorax. Below are the same radiograph, one unlabeled, one with the ventral mediastinum highlighted and the third with the boundaries of the dorsal half of the cranial mediastinum outlined. A fourth image has the tracheal and proximal portions of the mainstem bronchi outlined. Note that the trachea is slightly to the right side of midline. In many small dogs this can be exacerbated and there can be deviation to the right that is incidental. However identification of the normal structures and size on the VD radiographs is essential for identification of mass effects in the mediastinum.
The middle mediastinum is comprised of the heart, aorta, esophagus, lymphatics (including the thoracic duct), azygous vein, trachea, tracheal bifurcation and principle bronchi and lymph nodes. The caudal mediastinum divides into three sections — dorsal with the aorta and azygous, middle with the caudal cava and esophagus and ventral with the caudal mediastinal reflection.

**Mediastinal Masses. There are numerous causes as listed below**

Apparent masses can be simply due to fat – extremely common in some dogs especially bulldogs. Will usually be uniform opacity and smooth edge to axial lung borders. Can be quite opaque mimicking soft tissue due to the thickness of the fat on the VD/DV views.

Mediastinal lymphadenopathy (inflammatory or neoplastic) is considered the most common cause of mediastinal masses but they are far from the only cause. Perihilar masses can be the result of regional lymphadenopathy however chemodectomas/heart base tumors, ectopic thyroid tumors, esophageal foreign bodies can all appear similar in radiographic appearance.

Aortic and pulmonic stenosis and PDA’s can result in focal marked enlargement at the cranial heart base mimicking a mass lesion.

Pulmonary masses on the axial margin of the lung can mimic hilar masses, as can marked left atrial enlargement in some cases. Caudal mediastinal masses can be the result of granulomas/abscesses, cysts, esophageal tumors or foreign bodies.

Mediastinal fluid from hemorrhage (coagulopathy, trauma, neoplasia), inflammation (mediastinitis), edema (hypoproteinemia, lymphangiectasia) can cause variable widening (mild to marked).
Superimposition of forelimb musculature can be mistaken for mediastinal disease on lateral views. Rib, spinal and sternal tumors can extend into the mediastinal space.

Esophageal dilation can cause mediastinal mass effect especially in cranial dorsal thorax when associated with aortic arch anomaly. Esophageal foreign bodies are usually at heart base or between heart and diaphragm and secondary esophageal diverticula, tumors or granulomas can form in the cranial and caudal mediastinum. Hiatal and peri-esophageal hernias may be mistaken for caudal mediastinal masses. Esophageal abnormalities are discussed in another lecture.

**Lymph Nodes and Lymphadenopathy/masses.**
The radiograph below is of a dog 2 months after having 200mls of barium sulfate solution tubed into its accessory lung lobe. There has been uptake in all thoracic lymph nodes indicating their normal position.

Lymph nodes: red – sternal, green – cranial mediastinal; blue – tracheobronchial/hilar

There are three localizations of thoracic lymph nodes. All are mediastinal and all therefore are on midline. They are sternal, cranial mediastinal and trachea-bronchial or hilar. The sternal lymph node(s) are typically on midline, dorsal to the 2nd and 3rd sternebra (although they can extend caudal). When enlarged they will be visible as well on the VD view due to focal abaxial displacement of the ventral mediastinum. Cranial mediastinal lymphadenopathy is the most difficult to identify until they are very large. A vague increase in opacity on the lateral view may be visible however rarely are there distinct margins. On the VD radiograph however the dorsal mediastinum normally has relatively smooth even axial margins to the cranial lung lobes and with cranial mediastinal lymphadenopathy it will usually be wider and there may be focal deviations of the axial margins of the lungs (several images will be reviewed in the lecture). With increasing size of cranial mediastinal masses (of whatever origin, see below) there will be progressive abaxial displacement of the cranial lungs and the cranial margins of the lungs will be displaced caudally. (Note on the radiographs below that the cranial lung lobes extend cranial to the first rib in a normal dog, with mediastinal masses as below the lungs do not extend nearly as cranial – often only to the 2nd or 3rd intercostal space. This can be seen on both lateral and VD films.)
Mediastinal mass causing widening of the mediastinum, irregular contour of the axial margins of the cranial lung and caudal displacement of the cranial boundary of the lungs. (normal thorax on right).

Hilar or tracheo-bronchial lymph nodes are present at the heart base. Cranial to the carina they are located ventral to the trachea and primarily on the left side. Caudal to the tracheal bifurcation the lymph nodes are located dorsal to the airways. Enlargement cranial to the bifurcation will result in dorsal deviation of the distal trachea and increased opacity at the heart base. Enlargement of the lymph nodes distal to the bifurcation can result in a vague soft tissue mass effect that can deviate the lobar bifurcation abaxially and mimic left heart enlargement on both the lateral and VD films however it will result in focal ventral displacement of the distal trachea and tracheal bifurcation (actual left atrial enlargement causes dorsal displacement on the lateral view).
Dog with LSA with massive hilar lymphadenopathy mimicking left atrial enlargement with concurrent pulmonary infiltrates caudal dorsal that could be confused for edema.

**Other mediastinal mass effects**

Marked mediastinal widening is seen in this radiograph however in this case it is simply due to fat within the mediastinum. It can be very difficult to differentiate with an actual mass – usually the axial margins of the lung is smooth and the patient will be very obese. Most bulldogs will appear similar to this. The fat has opacity more similar to soft tissue due to the amount of fat present plus summation with other soft tissues.
This patient has marked mediastinal widening with apparent mass lesion plus concurrent pleural effusion and some pulmonary infiltrates raising strong concern for a neoplastic process however in this case it is due to rodenticide toxicity with hemorrhage into the mediastinum.

Advanced imaging of mediastinal disease

Positional radiography – Especially in cases with concurrent pleural effusion it may be very difficult to differentiate mediastinal masses from simple effusion. Horizontal beam radiography could be considered – make the radiograph as a VD projection with the animal in a vertical standing position (it requires a person holding each forelimb so the patient is vertical position) and the tube directed horizontal to the floor. Pleural effusion, if present, will settle dependently and the cranial lung lobes should close together if there is no mass present in the mediastinum. In the radiograph below on the initial VD radiograph there is fluid in the right hemithorax extending through the mediastinal region. On the horizontal beam radiograph the fluid in the right hemithorax has settled dependently and the mediastinal widening is unchanged indicating it is a mass lesion, not simply fluid. The fact that the effusion is unilateral is extremely important as it indicates there is closure of the mediastinal fenestrations. Causes of this include diffuse neoplasia (i.e. carcinomatosis, mesothelioma) or very viscous/highly proteinaceous fluid (FIP, pyothorax, chronic chylothorax).

In cases of marked pleural effusion, often the ventral thorax and boundaries of the heart and diaphragm and pleural surfaces is uniformly soft tissue silhouette. Another method of positional radiography is a horizontal beam (the beam projected horizontal to the table) with the patient in dorsal recumbency. The effusion will settle dorsally due to gravity and the ventral thorax will have much greater/normal conspicuity. This is an effective method of determining the presence of ventral diaphragmatic hernias in trauma cases with pleural effusion and for identification of small amounts of free air in the thorax or abdomen.
Ultrasound Examination of the mediastinum

In a normal patient the mediastinum can not be visualized with ultrasound due to the inflated lungs surrounding the mediastinum. If the mediastinal masses are large enough to displace the lungs or if there is pleural effusion present resulting in atelectasis of the lungs it may be possible to evaluate masses within the mediastinum. It is best to place the patient in dorsal recumency and image the thorax immediately adjacent to the sternum. A small footprint curvilinear probe is usually the most useful probe selection.

Enlarged lymph nodes are usually moderately well defined, relatively homogenous hypoechoic tissue with mild to moderate vascularity. In aggressive lymphomas and conditions such as malignant histiocytosis the mass can be very amorphous with mottled/heteroechoic echogenicity and can extend along the sternum and encircle the cranial aspect of the heart or even extend to the diaphragm. Unfortunately this appearance can also be mimicked by mediastinal hemorrhage with rodenticide toxicity – a variable mixture of fluid and amorphous blood clots that can appear mass like (but without vascular flow). Hilar lymph nodes can sometimes be imaged through the heart during echocardiography and differentiation with heart base masses (chemodectomas) is optimal with echocardiography.
Thymomas are can be variable in size, from relatively small to very large, occupying the majority of the cranial thorax. Thymomas tend to be variably cystic/cavitated with echogenic fluid and tissue striations and can encircle the cranial heart. There is a variable amount of solid tissue wall thickness however the tissue is usually vascular. Cytologically thymomas and lymphoma can be quite similar to differentiating the appearance is beneficial in determining the likely etiology (thymomas usually require surgical excision with poor response to chemotherapy).

Not all mediastinal masses are aggressive disease. Mediastinal cysts are seen, especially in cats, that are benign. On radiographs they appear similar to any other mediastinal mass however on ultrasound they are very thin walled with anechoic fluid. This fluid when aspirated is completely clear and colorless.

Ultrasound guided aspiration of mediastinal masses is usually an effective diagnostic technique however if possible always perform color doppler interrogation of the tissues to determine location of mediastinal vessels.

**Esophogram** – Mediastinal masses may involve the esophagus or may at the least cause significant deviation or distortion of the normal location in the mediastinum. Therefore it is often beneficial to perform an esophagram with barium paste to aid in identification of these changes.

**CT** – Contrast enhanced computed tomography is the optimal modality for evaluation of the mediastinum. Examples of clinical CT imaging will be demonstrated during the lecture.
**MEDIASTINAL IMAGING – THE ESOPHAGUS**

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**Objectives:** The objective of the lecture is to describe methods of imaging the esophagus from the upper sphincter to the stomach using plain radiographs, contrast radiography, contrast fluoroscopy, and ultrasound. The proper method of performing an esophagram will be discussed. Specific conditions that will be demonstrated include cricopharyngeal achalasia, vascular ring disorders, megaesophagus, esophageal strictures, esophageal reflux and esophagitis, esophageal foreign bodies and bronchoesophageal fistula.

**Esophageal Evaluation**

The esophagus is difficult to visualize on normal thoracic and cervical radiography unless it has air or some type of contrast material within it. For that reason it is common to perform a positive contrast esophagram using various types of barium or iodinated contrast. Even if the esophagus is visible with gas dilatation this is not definitive for a megaesophagus syndrome – that requires evaluation of esophageal motility and thus again requires use of contrast esophagram. The most optimal method of imaging for esophagrams is with fluoroscopy however that is rarely available in private practice. With the advent of digital radiography, especially DR technology, it is possible to perform rapid image acquisition to evaluate esophageal function. (While the actual movement cannot be seen, radiographs can be rapidly repeated so as to determine if there is movement of material or evidence of peristalsis). An important concept with static radiography is that if a perceived abnormality is identified it **must** be repeatable.

The normal swallowing process starts withprehension of food within the oral cavity by the tongue, bolus formation and caudal displacement by the base of the tongue. Simultaneously the upper esophageal sphincter relaxes and the epiglottis and arytenoids close. Ingesta enters the esophagus initiating a peristaltic contraction that propels the bolus towards the stomach. At the thoracic inlet there is an increase in compressive pressure on the esophagus and if the bolus is small and the contraction weak, the bolus may stop until another bolus “joins” it. At this stage a secondary contraction is usually initiated and the bolus continues to the stomach. (Recognition of this temporary stasis as normal is important in evaluation of esophageal function.) As well some breeds (bulldogs, shar pei’s, Boston terriers) can have a mildly redundant esophagus at the thoracic inlet resulting in a focal dilation or deviation. As the bolus approaches the stomach, the lower esophageal sphincter relaxes allowing passage to the stomach. Reflux from the stomach back into the esophagus should not occur.

With fluoroscopy I prefer to perform the study in a standing position for two reasons. First it approximates real life ingestion and secondly it reduces the risk of aspiration as compared to performing the study while restraining the patient in lateral recumbency. The study is generally started with liquid barium sulfate solution administered orally into the mouth and the bolus formation and transit is recorded “live”. The study is repeated with barium mixed with soft food and then barium mixed with hard food/
kibble – it is generally recommended to fast the patient for 12-24 hours prior to the study to induce eating.

Real world esophagram
Few clinics have access to fluoroscopy and as such this is a formula for performing the study with static radiography:

INDICATIONS:
1. Esophageal dilation or abnormal opacification of esophagus on radiographs.
2. Regurgitation of undigested food.
3. Acute gagging or retching.
4. Dysphagia.
5. Excessive salivation.
6. Possible foreign body ingestion.
7. Suspected mediastinal disease on survey radiographs – mass effect, increased opacity, hiatal hernia.

CONTRAINDICATIONS: Suspected esophageal rupture – pneumomediastinum. Severe respiratory compromise (due to restraint and potential for aspiration).

CONTRAST MEDIA:
1. Barium sulfate paste. Preferred contrast for esophagram with static radiography, at least for initial examination. Liquid solution tends to pass too quickly and does not coat the mucosa. Barium paste tends to coat the mucosa of the pharynx/larynx and esophagus. (Plus the material is extremely thick and viscous and thus stays within the patients mouth and is not spit all over the xray suite and people performing the study).

2. Barium sulfate liquid mixed with food – first soft/canned food, then mixed with kibble.

3. Water-soluble iodinated oral contrast agent (when perforation is suspected).

TECHNIQUE:
1. Survey radiographs must always be taken first. (This is the same for all contrast exams.) Survey radiographs can be made in normal lateral and VD positioning. For the actual barium swallow, if available I would prefer to perform it with the dog standing and the radiographs made in horizontal beam positioning. A standing survey lateral should also be made prior to the study as well as standard views.

2. Administer the barium paste solution into the buccal cavity to induce swallowing. Dosage varies from 3-5 mls in small dogs and cats to 10+ mls in large dogs. The paste will usually adhere to the mucosal surface (much better than liquid barium solution).
3. Make a lateral radiographic exposure immediately after administering the paste, making sure to include the oropharynx to diaphragm (large dogs may require 2 exposures).

4. The process may be repeated as many times as needed to clearly identify the entire esophagus. It may be useful to try to get exposures during peristaltic contractions. It must be remembered again that this is a dynamic study with static images. There will often be segmental dilation or constriction that is due to a peristaltic wave. **For this reason, any suspected lesion should be repeatable.**

5. It is preferable to perform the study without sedation.

6. Esophageal wall strictures may be present that will allow normal passage of barium liquid or even barium paste. If the study is negative for paste/liquid then the esophagram should be repeated using food mixed with barium solution – initially the soft food mixture, followed by the kibble mixture.

7. Once the lateral study for each section (paste, food) is complete – the process can be repeated with VD radiographs – usually just one radiograph made after administration of barium or possibly more if a lesion is identified.

**FINDINGS:**
There is often mucosal coating of the pharyngeal region and a small amount of contrast may pool in front of the upper esophageal sphincter, however, there should not be pooling of contrast anywhere else in the oropharyngeal region. Abnormal pooling within the oral cavity or in the esophagus is an indication of disease, including neurological, inflammatory and traumatic disorders. While aspiration is considered an abnormality, a small amount of barium aspiration may occur in normal patients.

The normal canine esophagus has distinct, longitudinal folds throughout its length. The distal feline esophagus (caudal to the heart base) has a distinct “herringbone” mucosal pattern. Abnormalities may include focal, regional or complete dilation. Focal and regional dilation is usually related to obstruction, however, it can also be related to regional neuromuscular disease and chronic inflammation (esophagitis, including gastric reflux esophagitis). Some breeds may also have regional dilation due to congenital redundant esophagus (Shar Pei’s and Bulldogs for instance). Obstructive disease can include congenital disease such as vascular ring anomalies, intraluminal foreign bodies, strictures due to previous inflammation or obstruction, mural or extramural mass lesions and gastro-esophageal intussusception. With megaesophagus syndromes (regardless of etiology) or even with severe esophagitis the barium will simply lie within the esophagus and no evidence of active transit will be seen. In cases of chronic disease there is often food/ingesta and fluid within the esophagus and the barium may mix with this resulting in multiple filling defects throughout the dilated lumen.

After the study is complete, if the stomach is filled and if there is concern for reflux or sliding hiatal hernias, a lateral radiograph can be made during compression of the cranial abdomen (remember to collimate!). Unfortunately if there is no reflux or hernia identified that does not rule out the potential for those etiologies still be present. An esophagram can also be used to evaluate the mediastinum, which is very difficult to image otherwise (without a transesophageal ultrasound). Mediastinal mass lesions
will often cause deviation of the esophagus, whereas fluid (due to mediastinitis or hemorrhage) will usually not change the course of the esophagus. Esophageal perforations will usually cause leakage into the mediastinum but occasionally there can be direct communication with the lung parenchyma due to a broncho-esophageal fistula. In this case there will usually be no contrast within the trachea (usually seen with aspiration). Broncho-esophageal fistulas are usually the result of chronic esophageal foreign bodies. With acute perforation of the esophagus with a foreign body, the classic radiographic change seen is pneumomediastinum with possible mediastinal fluid accumulation. However with fistula formation this typically is due to a foreign body with sharp margins in the caudal thoracic esophagus. The foreign body causes marked inflammation which is transmural and the inflammation results in direct inflammation of the adjacent lung pleural surface – usually the accessory lobe. This results in an effective seal between the two serosal surfaces and the perforation occurs after this (a slower degenerative process due to focal necrosis rather than acute tearing). This results in direct communication between the esophagus and lung however the amount of fluid transference is usually low due to the foreign body “plugging the hole”. It will usually result in focal alveolar infiltrates in the accessory lung lobe (this can be seen in the mid caudal thorax with silhouetting of the caudal vena cava), rather than in the ventral lungs expected with aspiration pneumonia. If the foreign body is removed with a scope (or surgery) the actual size of the fistula usually markedly increases resulting in catastrophic pneumonia from the direct open communication of the esophagus to the airways. A clinical case with images will be reviewed.

**Esophageal Ultrasound**
The cervical esophagus is easily visualized on cervical ultrasound however the intrathoracic esophagus cannot be ultrasounded without a specialized transesophageal probe. There is however a short segment of abdominal esophagus, from the diaphragm to the cardia of the stomach that can be imaged with ultrasound. The technique for obtaining the scan will be described as well as images showing the normal appearance of the esophagus, esophageal dilation with reflux esophagitis and the appearance of a hiatal hernia on ultrasound. The identification of the esophagus allows concurrent evaluation of the cardia and lesser curvature of the stomach, which is not typically seen with most abdominal approaches.

The technique involves placing the patient in dorsal recumbency and placing a small footprint curvilinear probe in the most cranial aspect of the abdomen on midline, immediately adjacent to the xiphoid. It requires substantial direct compression towards the spine – the stomach will actually be displaced caudally and the direct image will be of the diaphragm and liver. The esophagus will be a short tubular structure crossing the diaphragm in a caudal direction, dorsal to the liver. It has wall striation similar to small intestine and may contain a small amount of gas. It is usually ventral and slightly to the left of the aorta as it crosses the diaphragm into the abdomen. Angulating the probe caudally slightly should allow you to follow the esophagus into the insertion in the stomach.
Normal cat esophagus entering the cardia of the stomach

13 year old Cairn Terrier with esophageal reflux.
Evidences point to the condition being very chronic in development. Dogs can have syncope. In many cases, however, they present with acute disease even when all conditions such as heart worm disease, some congenital cardiac disorders and as an acute condition with pulmonary thromboembolism. However it is increasing recognized secondary to pulmonary fibrosis (which can be idiopathic or related to chronic inflammation) and especially secondary to chronic left sided cardiac disease - usually chronic mitral valve disease (MVD) in smaller dogs. Chronic MVD can result in chronic pulmonary venous hypertension, with subsequent muscularization of the alveolar arteriole system. Chronic low grade edema can induce regional inflammatory disease that in turn can induce fibrosis. In people chronic airway obstruction is recognized as a cause of pulmonary hypertension and this may have a role to play in dogs as well. Chronic systemic disease, such as Cushing’s syndrome or chronic renal disease, can result in diffuse dystrophic mineralization in the lungs. In fact in many most cases in companion animals it is impossible to definitively determine the inciting cause. Clinically these patients may present with a history of chronic disease (progressive lethargy, slowly increasing respiratory rate, exercise intolerance and even syncope). In many cases however they present with acute disease even when all evidence points to the condition being very chronic in development. Dogs can have

**Objectives:** The objective of this lecture is to discuss the identification of specific pulmonary disease conditions with diagnostic imaging. The first is airway collapse, especially that seen with concurrent pulmonary disease. The next is pulmonary hypertension syndromes which can mimic the radiographic and clinical appearance of left sided congestive heart failure. Finally the use of CT for identification of the primary underlying disease in chronic or mixed etiology pulmonary disease will be discussed through the use of clinical cases.

**Airway collapse:** Redundant dorsal tracheal membranes and cervical tracheal collapse has long been recognized. There has been increasing recognition of intrathoracic airway collapse, especially of the left caudal bronchus in dogs with chronic left heart disease. The degree of intrathoracic airway disease however is probably greatly underdiagnosed/underestimated, especially in small/chondrodystrophic breed dogs. The exact etiology is uncertain however it is probably a combination of genetic predisposition complicated by chronic inflammation. Intrathoracic collapse is frequently seen in combination with left heart disease as a complicating factor and is likely related to low grade/occult pulmonary edema and congestion which is acting as a source of inflammation within the lungs.

Classically thoracic radiographs are made in inspiratory phase of respiration, however intrathoracic airway collapse occurs during expiratory phase of respiration and thus is not seen on normal radiographic studies. Comparison of inspiratory and expiratory phase radiographs will be described as the primary screening method in private practice. Dynamic fluoroscopic evaluation of the airways/lungs will also be shown as well as some images of the appearance with computed tomography.

**Pulmonary hypertension:** Pulmonary hypertension has been recognized in people with chronic respiratory disease for a relatively long time. It is becoming more commonly recognized in companion animals, especially dogs. It has been recognized as a familial disease in terrier breeds, especially West Highland Whites, as a sequella to conditions such as heart worm disease, some congenital cardiac disorders and as an acute condition with pulmonary thromboembolism. However it is increasing recognized secondary to pulmonary fibrosis (which can be idiopathic or related to chronic inflammation) and especially secondary to chronic left sided cardiac disease - usually chronic mitral valve disease (MVD) in smaller dogs. Chronic MVD can result in chronic pulmonary venous hypertension, with subsequent muscularization of the alveolar arteriole system. Chronic low grade edema can induce regional inflammatory disease that in turn can induce fibrosis. In people chronic airway obstruction is recognized as a cause of pulmonary hypertension and this may have a role to play in dogs as well. Chronic systemic disease, such as Cushing’s syndrome or chronic renal disease, can result in diffuse dystrophic mineralization in the lungs. In fact in many most cases in companion animals it is impossible to definitively determine the inciting cause. Clinically these patients may present with a history of chronic disease (progressive lethargy, slowly increasing respiratory rate, exercise intolerance and even syncope). In many cases however they present with acute disease even when all evidence points to the condition being very chronic in development. Dogs can have
moderate pulmonary hypertension without overt disease and patients with normally low activity levels can have quite severe hypertension with no clinical signs until a critical point is reached where they can no longer compensate and have acute and severe clinical presentation of respiratory failure.

In many cases the patient will have radiographic changes that are consistent with left heart failure – cardiomegaly and increased pulmonary opacity that can mimic cardiogenic edema. This is especially true in cases of chronic mitral valve disease where there is marked left atrial enlargement. In cases with pulmonary fibrosis it is usually present in the caudal dorsal lung lobes and is similar in appearance to cardiogenic edema. Various clinical appearances/presentations will be presented. Most patients will have a limited response to traditional cardiac therapy (Lasix, pimobendan, ACE inhibitors), either clinically or radiographically (the pulmonary opacity thought to be edema does not resolve) and the patient requires continuous oxygen supplementation. Diagnosis of the underlying pulmonary hypertension (PH) will usually require echocardiography, specifically identification and accurate measurement of the velocity of tricuspid insufficiency (usually present with any significant degree of PH). Essentially the maximal velocity (V) is squared and multiplied by 4 (modified Bernoulli equation). Anything above 30 to 35 mm Hg is considered abnormal, with 30-50 being mild, 50-70 being moderate and over 70 mm considered severe. Also in any case of suspected pulmonary hypertension the abdomen should be imaged with ultrasound to determine if there is evidence of right heart failure (caval congestion, hepatic vein congestion, hepatomegaly, ascites). In severe/chronic cases there will be right ventricular hypertrophy, right sided volume overload and even septal flattening noted on the short axis evaluation of the ventricles. This will be demonstrated during the presentation.

**The use of computed tomography in pulmonary disease.** Time permitting, clinical cases will be used for demonstration of the efficacy of using CT to diagnose pulmonary conditions including foreign bodies to neoplasia to chronic degenerative disease and mixed etiology lung disease.
ULTRASOUND OF THE GASTRO-INTESTINAL TRACT
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Objectives: The objective of this lecture is to describe the sonographic appearance of changes noted in the ultrasound appearance of the gastrointestinal tract (GIT) secondary to obstructive disease. Secondly I will describe the appearance of various inflammatory and neoplastic conditions, correlating to normal appearance and highlighting the similarities between the two in some cases. Tips will be given as to how to optimize image acquisition in some cases. It is a case-based discussion using real clinical cases.

Functional ileus versus obstructive disease

Ileus is commonly described in veterinary medicine as functional ileus (an absence of GI motility due to underlying physiological disease) and mechanical ileus (obstruction of the GIT usually due to foreign body, intussusception or mass effect). The latter usage is not actually correct as most obstructions, at least in the initial phase, will have marked motility, although it may not result in passage of the obstruction. However for the purposes of this discussion we will continue to use it in the traditional manner.

Ileus in dogs, whether due to functional disease or mechanical obstruction, is a common and sometimes difficult diagnosis. Early detection and treatment is often vital for reduced morbidity and mortality. In regards to imaging modalities it is noted that ultrasound and radiography are complementary. This is especially true with gastric obstructions where gas within the stomach may obscure significant findings. It is also noted however that there can be significant obstruction with minimal radiographic changes (early linear foreign bodies in dogs is a classic example). For this reason even if there is no radiographic changes supporting obstructive disease, I still recommend abdominal ultrasound if there is ongoing clinical disease (i.e. recurrent vomiting, pain on abdominal palpation) or clinical concern.

Stomach evaluation: the stomach should be closely examined for luminal contents – with intestinal or pyloric obstructions the stomach is often quite full with fluid, unless the patient has recently vomited. In relatively acute obstructions and in cases of acute functional ileus the stomach will contain primarily anechoic fluid. The more echogenic debris within the stomach, the more likely it is a chronic obstruction.
Anechoic fluid accumulation due to duodenal obstruction

Chronic obstruction with marked debris accumulation in the stomach

If there is marked gas within the stomach, try rolling the patient to first the left side down and then put your probe on the down or dependent side of the patient (slide it to the edge of the table or focally compress the padded bed to allow your hand and probe underneath the patient). This will allow you to look through the fluid which should settle dependently and evaluate the stomach wall and contents. Do the same with the right side and make sure to clearly visualize the pyloric outflow tract – this will often require imaging through the right intercostal spaces. Foreign material within the GI tract tends to have marked sound attenuation unlike normal ingesta or gas (occasionally this some foreign bodies transmit sound similar to tissue.)

Peach pit in duodenum with complete shadowing.

Plastic FB in stomach with distal shadowing

Try to follow the duodenum out of the pylorus – it will course cranial and slightly dorsal for a few cm then turn abruptly 180 degrees and travel down the right side of the abdomen. It typically crosses over the right kidney and should be very straight (if it has abrupt turns or change in direction look very closely for a linear foreign body) as it extends to the caudal right abdomen where it again turns 180 degrees abruptly at the caudal duodenal flexure (a common location for foreign bodies to lodge due to the
abrupt turn). Observe the duodenum closely – is it dilated, is there motility present. Always evaluate the wall for evidence of thickening or alteration in architecture (more later). Then continue to scan the remainder of the intestinal tract. The optimal method is to continuously trace the intestine from duodenum to ileum (or vice versa) however that is technically difficult. Sweeping the abdomen in a grid pattern will allow you to evaluate the intestine – for obstructions and ileus look to see if there are normal appearing, empty intestines as compared to the dilated duodenum. If there are two populations of bowel it is likely there is an obstruction somewhere however you will have to trace the gut to find it. You can usually find the ileo-colic junction in one of two means. First find the transverse colon caudal to the stomach and follow it down the ascending duodenum, looking for insertion of the ileum (it is often beneficial to look from the mid right side as the ileum tends to insert on the dorsal side of the colon and from a ventral approach the colon gas obscures the junction). In cats this is much easier than in dogs. Another method is to find the caudal border of the right kidney, align it in the middle of your image in a longitudinal plane and sweep gently to midline – the proximal colon, cecum and ileum are generally at this level, before midline. If you see the central mesenteric vessels and lymph nodes you have gone too far to the left - the ilio-colic junction will be to the right (immediately adjacent). If the ileum can be identified and it is empty/normal with dilated proximal bowel this is highly probable to be an obstruction. If the ileum is dilated and fluid filled as well as the proximal intestine, it is likely a functional ileus. If the dilated proximal intestine has obvious or even stronger than normal motility it is very likely to be a recent obstruction. If there are two populations of small intestine and the dilated intestine has no visible peristalsis it is likely a more chronic obstruction (“the gut has given up”). Again, finding the actual obstruction, whether foreign body or mass or intussusception, is the true goal of the examination.

While examining the intestine closely evaluate the surrounding mesentery if the mesentery around dilated bowel or especially around an actual foreign body is very hyperechoic than this is supportive of significant wall inflammation from the passage of the foreign body and would warrant more expeditious exploration. Small perforations in the wall can occur without significant effusion – the mesentery will adhere to the gut and often result in only marked local inflammation with small pockets of fluid, although occasionally small gas bubbles can be seen in the mucosal or even passing through the wall of the intestine. Any evidence of free peritoneal air is an indication for immediate exploratory (unless the patient has had recent surgery). Examples of this will be shown during the lecture.

Linear foreign bodies invariably have material lodged in the stomach (or tongue with a cat) and as such the duodenum is typically involved – if the duodenum is plicated (acutely folding back and forth) then look very closely for a thin, linear, hyperechoic shadowing line running through the intestine. Even if the actual string or foreign body is not seen not seen, the presence of plication with appropriate clinical signs would warrant exploratory surgery. Occasionally plicated or gathered small intestine is seen within the central intestine without involvement of the duodenum. These are usually the result of previous surgery and subsequent adhesions or in some cases due to a luminal mass or stricture that is occluding a foreign body that may have normally passed. Post-operative adhesions and strictures are probably more common than we think and predispose patients to obstruction by material that would not obstruct in a normal patient. These regions of focal plication can be seen in non-clinical cases
occasionally and thus there should be appropriate clinical signs and oral distension of the intestine to determine this as a significant finding.

In regards to intussusceptions, they are usually easily recognizable with a target like appearance on cross-sectional imaging – an outer dilated intestinal wall with another intestine centrally plus mesentery. If possible one should also evaluate the mesentery with color Doppler to determine viability of the tissues.

Plicated duodenum with a small amount of effusion adjacent to the intestine. This is indicative of a linear foreign body.

Cloth foreign material in stomach with intense distal sound attenuation.
Intussusception – cross sectional image. Note the intestinal segment and mesentery within the dilated outer intestine. Color Doppler is used to identify active blood flow within the mesentery.

All three of these images are from the same patient. The intussusception is at the end of the linear foreign body, which can happen relatively often.

**Inflammatory and infiltrative disease of the GIT**

The normal gastric and intestinal wall thickness and appearance is described in many textbooks (I would recommend *Atlas of Small Animal Ultrasonography* 2nd Edition by Dominique Penninck and Marc-André d’Anjou or *Small Animal Diagnostic Ultrasound, 3e* 3rd Edition by John S. Mattoon DVM DACVR and Thomas G. Nyland DVM MS). Intestinal wall thickness in the dog is generally a maximum of 5 to perhaps 6 mm and cats generally 3 mm for the duodenum and the jejunum and 3.5 for the ileum. Comparison the different segments is also important – in a dog the duodenum should be the thickest segment, the jejunum next and the ileum the thinnest or at most similar to the jejunum. In cats the ileum is the thickest, then the duodenum then the jejunum. However it is equally important to observe the wall layering – the mucosa should be hypoechoic and thicker than the hypoechoic muscularis. The outer serosal should be thin and smooth. Evaluation for motility is also important as well as luminal contents. The colon can be traced from the pelvic canal to the ilio-colic junction in cross section allowing for evaluation of at least half of the wall (fecal and gas contents will obscure the far wall in most cases). The colon wall can be quite variable with thickness from 1 to 3 mm depending on the degree of filling (empty is thicker). Sometime discrete layers are visible in the colon however this is usually incidental. Infiltrative disease or inflammatory disease will have many presentations - from focal mass lesions to diffuse wall thickening that can be irregular or uniform. The important factor when noting abnormal wall is to understand its exact location within the gut – is it reachable by a scope or will it take a surgical enterotomy. Is the lesion mucosal or more muscularis or serosal (scope vs surgical).
Infiltrative lesions can have many types of presentation within the intestine and stomach, ranging from subtle wall thickening and change in layer architecture (seen in cats with small cell LSA) to very large masses incorporating the menteric lymph nodes, cecum, distal ileum and proximal colon (seen in cats with lymphoma). The ultrasound appearance of common presentations of lymphoma and carcinoma in the cat and dog as well as more benign polyph masses will be demonstrated. Ulcerative lesions in the stomach and intestine (some benign, some related to neoplasia) will be shown, as well as apparent mass lesions secondary to mural hemorrhage that spontaneously resolved. Another example is the appearance of small cell lymphoma and eosinophilic enteritis in cats can be essentially identical.

In regards to inflammatory disease, the typical appearance is of wall thickening however in some chronic cases, especially in cats, the walls can actually thin with mild luminal dilation and markedly reduced motility – clinical cases will be shown as well. A similar appearance with wall thinning and mild luminal dilation with absent motility can be seen with an acute shock syndrome in the intestine (secondary to shock syndromes, severe blood loss, trauma or septicemia). Some inflammatory diseases, especially PLE and lymphangiectasia, can cause thickening with marked increase in echogenicity of the mucosal, sometimes with linear hyper-echogenicities. However recent eating of a fatty meal can result in a similar appearance due to fat in mucosal lacteal. In cases of peritonitis, the small intestine may have a crimped/spastic appearance with thickened and irregular serosa. The take home point in this discussion is that rarely are lesions seen on ultrasound pathognomonic and tissue sampling (biopsy, FNA) is required for a diagnosis, however with careful examination the extent of the lesion (unifocal vs multifocal vs diffuse) and location within the gut will help in determining the next procedural step.

Thickened colon in cat due to FIP
Focal gastric mass – leiomyoma with small central gas foci indicating small
| Thickened, hyperechoic mucosal layer in jejunum with markedly hyperechoic mesentery, typical of PLE | Circumferential, hypoechoic, vascular mass in the antrum of cat stomach - lymphoma |
**Objectives:** The objective of this lecture is to describe a technique that will allow practitioners to acquire some basic cardiac parameters (atrial size, LV size) and evaluate the heart for conditions such as cardiomyopathy, pericardial effusion, cardiac neoplasia. The purpose is to enable identification of disease conditions that require immediate attention and possibly stabilization prior to referral for complete cardiac workup. This technique is not intended to replace a proper echocardiographic exam by qualified individuals – i.e., a cardiologist. It will also include discussion of indications for further evaluation of the heart using ultrasound with emphasis on abdominal ultrasound findings and radiographic findings. There will also be discussion and examples of ultrasound evaluation of the lungs and pleural cavity.

**Findings on abdominal ultrasound that would indicate need for further evaluation of the heart and thorax.** Any time abdominal effusion is identified it is worthwhile to evaluate for pleural effusion with ultrasound. If there is any evidence of enlargement of the caudal vena cava (greater diameter than the adjacent abdominal aorta) or hepatic veins (greater diameter than paired portal veins) and/or any evidence of hepatic congestion/enlargement, with or without concurrent ascites, one should evaluate the heart for evidence of cardiac tamponade (pericardial effusion) or right heart failure. The exception to this is if the patient has been sedated with dexmedetomidine (it frequently results in enlargement of the caudal cava and even hepatic veins and can alter several cardiac parameters).

If there is evidence/suspicion of any neoplasia in the abdomen I would recommend a quick evaluation of the pleural surfaces - essentially just run the probe up and down the intercostal spaces after wetting down the hair coat. The lung should be a bright/shiny reflective surface with hypoechoic tissue visible. If hypoechoic tissue or material is visible it will either be parietal pleural masses, lung infiltrates or effusion. The appearance of lung infiltrates will be demonstrated. I would also recommend brief evaluation of the heart for any evidence of pericardial effusion or heart masses, especially if hemangiosarcoma is suspected.

In cases of FUO (fever unknown origin) or CUO (cancer of unknown origin – radiographs of the thorax reveal numerous pulmonary nodules typical of metastatic disease with no identifiable underlying disease) I recommend at least brief evaluation of the heart for evidence of pericardial effusion (pericarditis or neoplasia), endocarditis (usually mitral or aortic valves) or cardiac neoplasia. Occasionally there can be significant cardiac neoplasia without pericardial effusion – for instance there can be right atrial hemangiosarcoma that grow within the right atrium but do not extend into the pericardial sac – when they rupture and bleed they simply bleed into the pulmonary arterial supply, seeding the lungs (thus the metastatic lesions). In regards to endocarditis – if there is evidence of multiple small infarcts in abdominal organs, especially the renal cortex, evaluation of the heart is warranted. Ultrasound images of these conditions will be demonstrated.
Differentiation of cardiogenic vs non cardiogenic edema can be difficult in many cases. A brief examination of the heart that allows for accurate determination of the left atrial size can be the critical determining factor – this can be absolute size from various charts/textbooks or simply ratio of the left atrium to aorta.

Differentiation of pericardial effusion versus dilated cardiomyopathy can sometimes be difficult but is easily differentiated with brief ultrasound examination.

(Note that ultrasound examination of the heart in a patient with a heart murmur but no clinical signs and unremarkable thoracic radiographs is not included in this list.)

**Acquisition of basic cardiac measurements and subjective evaluation.** Proper echocardiographic technique is relatively difficult with an initial “steep learning curve” and requires a specialized table with approach to the thoracic window from the ventral side – the placement of the probe and the orientation with the patient is not actually visible – it all depends on remote hand-eye coordination. It also generally requires specialized cardiac probes (sector or phased array probes) that the majority of people purchasing ultrasound machines for private practice cannot justify purchasing (often 8-10,000$ per probe). The acquisition technique used in this lecture is based on acquiring images from a left parasternal approach resulting in a typical “left long axis view” – the appearance of the heart on this view can be seen in multiple articles and textbooks and will be described in the lecture. However, rather than having the patient in left lateral recumbency it is based on having the patient in dorsal recumbency, such as when the patient is having an abdominal ultrasound. The patient is rolled to the left side down (30 to 45 degree angulation) and the left thoracic wall is palpated for the point of maximal cardiac intensity. This is usually the apex of the heart/ventricles (think of a VD radiograph where the heart naturally tips to the left – if you rotate the patients sternum to the left there will be mediastinal shift that way and in most cases, especially in dogs, the heart apex will rest against the thoracic wall). You simply place your probe on the thoracic wall at the PMI and point the probe towards the opposite shoulder or between the shoulder and the spine. If nothing else it will allow for identification of pericardial effusion however in most cases with even minimal practice it will result in adequate visualization of the heart in a 4 or 5 chamber view. Depending on the quality of the image and the ability to scroll back through images (even basic machines should do this) you should be able to acquire the 2D size of the left and right atrium, the aortic diameter and the left ventricular diameter in diastole (maximal diameter) and systole (minimal diameter) and from that the fractional shortening (LVD – LVS / LVD). If the image quality is adequate you should also be able to evaluate the size and appearance of the tricuspid, mitral and aortic valves for any evidence of endocarditis and even possibly prolapse. The method of acquiring these parameters will be described in the lecture, as well as identification of cardiac masses.

Optimization of the ultrasound machine settings and probe selection will be discussed. This is often overlooked in private practice but can make a substantial difference in the diagnostic quality of the exam. Most machines probably do not have dedicated cardiac probes however in most cases a curvilinear probe, especially a small footprint curvilinear probe, will be adequate. Most probes are multiple frequency and the available frequency will determine the depth of penetration (the lower the frequency the greater the penetration however resolution will decrease). For instance with a large dog you will need at least 10 to 12 cm depth penetration (and possibly more) and

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**COMPANION ANIMAL DIAGNOSTIC IMAGING**

Differentiation of cardiogenic vs non cardiogenic edema can be difficult in many cases. A brief examination of the heart that allows for accurate determination of the left atrial size can be the critical determining factor – this can be absolute size from various charts/textbooks or simply ratio of the left atrium to aorta.

Differentiation of pericardial effusion versus dilated cardiomyopathy can sometimes be difficult but is easily differentiated with brief ultrasound examination.

(Note that ultrasound examination of the heart in a patient with a heart murmur but no clinical signs and unremarkable thoracic radiographs is not included in this list.)

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Thus will typically require a frequency less than 5 mHz. For a small dog or cat you would like a higher frequency – perhaps 5 to 7 mHz for a small dog (6 to 10 cm depth) and 7 mHz or higher for a cat (depth of 4 to 6 cm). Again every machine will be different and “playing” with the combination of frequency and depth to obtain the best image is required.

Also machines typically have “presets”. If you are lucky enough to have a cardiac preset then use that as a starting point, although even with a cardiac preset I would consider trying to optimize the image. If there are only abdominal presets then there are several modifications that can be done to optimize the image. (Abdominal presets are usually much less than optimal for cardiac imaging). Once the depth of the field of view (and frequency to match) is set, then reduce the width of view - on curvilinear probes the image fans laterally away from center and the degree or width of the fan can be altered from very wide to very narrow focus. Most abdominal setups will have the width of view maximized – for cardiac evaluation reduce it to a size that just images the heart. This will increase the speed of image acquisition (frame rate) and reduce artifact from the surrounding lung. Next turn the “power” knob to close to maximum and reduce the gain control until the blood in the chambers is almost black. This will aid in a higher contrast image. There is usually a control called “dynamic range” – decrease the dynamic range for cardiac imaging as compared to abdomen (reduces the grey scale to again make things more black and white).

Turn off the various image processing settings on your machine. You can turn them on one at a time after to see if the image is improved, however in most cases the image processing will slow the frame rate and for cardiac imaging you need the fastest frame rate possible. These processing controls will have different names depending on the brand of ultrasound machine but are usually the same principle in each group and may include:

1. Persistence - this is frame averaging and should be 0 or off with cardiac.
2. Compound imaging (different names include CrossXBeam, SonoCT, XView, ApliPure) – combines multiple images from slightly different planes – again generally slows the frame rate too much.
3. Speckle reduction - reduces influence of weaker signals – makes the image cleaner in most cases but again may substantially slow the frame rate. Other names used XRes, MView, SonoHD, SRI, Adaptive speckle reduction and SRF. Initially turn it off, get a good image and then turn it on to see if it has a beneficial effect on the image – if it is not obvious, leave it turned off.
4. Harmonics – this involves transmitting and receiving at different frequencies. It may or may not be beneficial depending on the individual machine – turn it off with everything else then turn it on by itself to see if it improves the image – if a definite improvement is not seen leave it turned off.
5. Line density – keep low to keep frame rate high.

Machines will typically have several different “grey maps” to choose from (differences in the degree of black and white and number and range of the grey scale). It is best to try all the different maps to see what gives you the best image (very subjective evaluation admittedly).
Once you have tried all the various combinations save it as a custom preset (all machines should allow some custom presets) – name the preset after the type of animal you are scanning and the type of procedure – i.e. large dog cardiac. Then the next time you look at a large dog heart you can simply go directly to this preset. (This can also be done with abdominal imaging – one preset is rarely optimal for every type and size of animal, however most people never alter individual settings to optimize the image, let alone save those settings.) Once practitioners become familiar with the controls and image manipulation they could apply these techniques to all the ultrasound imaging done – i.e. optimize and save the settings for a very large dog abdomen or cat thyroid examination.
VETERINARY IMAGING – CAUSES OF DIAGNOSTIC FAILURE

Greg Starrak, DVM, DACVR
Associate Professor Diagnostic Imaging, Western College Veterinary Medicine

1. Poor quality radiographs. “A poor radiograph is at best totally useless and at worse totally misleading”. Dr. Peter Suter.
   a. With digital radiography there is reduced influence of improper exposure parameters on diagnostic quality, however it is still a possibility.
   b. Poor positioning is by far the most common reason for poor quality radiographs and in return diagnostic mistakes. Common positional mistakes through actual cases will be presented.

2. Equipment and/or work environment failure.
   a. Monitor quality – get the best quality monitor you can afford. Medical grade monitors for radiography are the best however they are very expensive. Newer commercial off the shelf monitors (COTS) are better than ever, with 4K monitors (2560 x 1600) with high contrast available. However the biggest differentiating factor is brightness or lumen. Dedicated radiology monitors have lumen brightness in excess of 1000 CD (candle) or nit, with most commercial monitors less than 300. There are “professional” class COTs that have higher brightness. I recommend at the very minimum 300 CD and preferably 350 CD. Brightness is important as it determines the level of both whiteness and blackness on the screen and thus the degree of difference between the two (more shades of grey). Set brightness at maximum on the monitor – adjust contrast to effect – usually around 60-70%. Avoid 16:9 screens – good for movies not radiographs.
   b. Reading environment – find a quiet, dark room with minimal ambient light and no distractions to read your radiographs in. Due to their high luminescence radiology monitors can be viewed in a lighted room however the commercial monitors can be enhanced by reduced ambient light.
   c. Environmental distraction (Doc you gotta look at this right now!), time constraints and fatigue.
   d. Lack of diagnostic protocol – scattered approach to diagnostic image review. These latter two issues can be resolved by setting aside a time of the day to review your radiographs without interruption in a controlled environment (quiet, dark, alone) and then reviewing the studies in a structured/organized manner.

3. Observer error
COMPANION ANIMAL DIAGNOSTIC IMAGING

a. Not recognizing an abnormality, mistaking a normal variant for an abnormality, over-interpreting radiographic changes. “Over interpretation” of normal variants and age related changes are one of the most common problems seen in veterinary medicine. Part of the issue is we only radiograph animals when they have a clinical problem in many cases and thus everything we see is considered a clinically significant change.

b. Observer error can improve with continuing education but also the proper library – for a small animal clinic I would recommend at least the following textbooks for diagnostic imaging: Thrall, Textbook of Veterinary Diagnostic Radiology. Dennis, Kirberger, Barr and Wrigley, Handbook of Small Animal Radiology and Ultrasound. Thrall and Robertson, Atlas of Normal Radiographic Anatomy and Anatomic Variants in the Dog and Cat. Evans, Millers Anatomy of the Dog (or similar anatomy textbook).

c. A clinic can build its own file of radiographic normal. For limbs always consider the contralateral limb as a control. In patients with a high probability of developing a certain condition (i.e. Cavalier King Charles spaniel or toy poodle for mitral valve disease and possible heart failure) consider radiographing the patient prior to the development of clinical disease and save the images as a control study for when the disease does occur. Radiographing clinically normal animals will also help to improve your recognition of incidental variants and age related changes, as discussed in 3.a.

d. Secondary read – whether it is a colleague in the same clinic or sending out to a radiologist can greatly enhance diagnostic accuracy.

4. Improper interpolation with history and other clinical findings.

   a. Using the imaging study to confirm the differential diagnosis you have already determined.

   b. Premature closure – a historical or clinical bias that results in you seeing what you want and not completely evaluating the study on its own merits and determining a proper differential list.

This is a very common cause of interpretation error and primarily missed findings in clinical practice. For instance – you have a busy day, a dog that was vomiting yesterday but has not vomited for 12 hours. It palpates normal but is ADR and depressed. You radiograph the abdomen to determine if there is any evidence of gastric or intestinal obstruction or abnormalities. The GIT is a normal size with no obstructive pattern – that answers your question and concern and the patient is treated for enteritis. However if you had taken time to closely evaluate the entire study you probably would have identified a subtle decrease in serosal detail indicating peritoneal effusion that would warrant further work up. Another example would be an older GSD with pain in the lower lumbar spine – you see this all the time – you make a lateral radiograph of the lumbar spine/pelvis and identify LS spondylosis and make a diagnosis of LS.
disease. However critical examination of the radiographs would reveal a lytic lesion in the body of L4 due to an early developing tumor.

5. Satisfaction of Search Error. You find one abnormality and then stop looking and miss other findings that would be of marked clinical significance. Perform and read the entire study. Diagnostic ultrasound is the modality in which this is most commonly seen. Actual clinical cases will be used to demonstrate this.

6. Historical bias. If you have missed something in the past (been burnt) you may have a predisposition to over-reading that condition on future cases. This is especially common with thoracic radiographs in cancer patients. If you have missed subtle pulmonary lesions in the past you are likely to over-interpret pulmonary osteomas and end-on blood vessels as metastatic lesions.

7. Limitations of the modality. Especially with radiography and ultrasound but even with advanced cross sectional imaging (CT and MRI) there can be significant pathology that may not be recognizable with that modality at that time. One must recognize the potential and consider alternative procedures (follow up films, other modalities) based on the overall clinical findings. For instance – a 9 year old golden retriever is presented with RF lameness you localize to the proximal right humerus. Radiographs are unremarkable – do you simply write it off as “muscle strain” or do you consider the potential of occult osteosarcoma which is simply not visible with radiographs at this time and re-radiograph the patient in one week? Ultrasound is again extremely prone to this as most people do not even realize they may be missing something and if the images are interpreted by a second party they can only interpret what was imaged at the time of the exam. If the operator did not actually image a region then you have no idea if there was an abnormality in that region (ultrasound is truly unique in this manner from other imaging modalities). For instance – if no abdominal lymph nodes are seen, that does not mean they are normal, it simply means that they were not seen.
RUMINANT CAMELID MEDICINE

CAMELID HERD HEALTH

Mélanie Boileau, DVM, MS, ACVIM

Facilities and Environmental Management
Llamas and alpacas are adapted to the high plains of South America, with cool, dry winters and mild, dry summers. This makes parts of North America challenging for their survival. Heat stress is a major health concern in the southern United States, and handling and housing of animals in consideration of the Heat Stress Index (HSI) is important.

The formula and interpretation guidelines for the HSI are: HSI = % relative humidity + temperature (°F)
- HSI less than 120, handling is considered safe
- HSI between 120 and 150, unnecessary handling should be avoided
- HSI greater than 160, handling is considered dangerous and animals should be closely monitored

In areas where high HSI occurs throughout the year, air-flow and shade provision are of utmost importance. In cold winters, straw bedding and wind-breaks should be provided. In summer, however, straw bedding closes the thermal window, which in camelids is the ventral thorax and abdomen, and does not allow for heat dissipation. In the summer, sand, concrete or open pasture under shade and with fans or misting devices make good housing.

The hierarchy within camelid herds makes it imperative that shelters be sufficiently large, with large enough openings that all animals are able to enter without harassment. It is easy to design and provide adequate pasture and shelter for the healthy population of animals on the farm. On the other hand, hospital, maternity, and quarantine facilities should be well designed and constructed to be separate from each other and from the main herd groups.

Any diseased animals should be isolated from the remainder of the herd and managed separately ideally through quarantine. All animals leaving the premises and coming in contact with other mammals should be placed in quarantine. This includes new additions to the herd, transient breeders or boarders that leave the farm, and returning show animals. In general, recommended quarantine period is 30 days. Producers should organize the facility so that the animals can be moved through on an all-in-all-out basis. Biosecurity procedures to be followed when entering and leaving contaminated areas should be used.

Stocking density is generally recommended to be five to seven camelids per acre full-time to allow for parasite control and meeting nutritional needs. Quality and quantity of forage and dung-pile management are factors to include in stocking density recommendations.

Nutrition
In general, camelids should be supplied approximately 1.5% of their body weight in dry matter for maintenance, with pregnant, lactating, or growing animals requiring highest
dietary energy and crude protein requirements (Table 1). Llamas and alpacas are grazers and browsers. With fermentation in all three forestomach compartments, camelids are more efficient than other ruminants at thriving on low-to-moderate quality forage. Palatable camelid- or sheep-labeled mineral mixes should be offered in every herd and actively monitored for consumption. Cattle, goat, or horse mineral should not be offered to camelids because of the risk of copper toxicity.

Table 1. Suggested Camelid Feeding Groups Based on Physiologic State and Nutrient Requirements.*

<table>
<thead>
<tr>
<th>Group</th>
<th>Physiologic state</th>
<th>Feeding plan</th>
<th>Dietary guidelinesa</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nursing dams with crias</td>
<td>Lactationb</td>
<td>Feed best quality forages</td>
<td>60-70% TDN</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Energy/protein supplementation</td>
<td>12-14% crude protein</td>
</tr>
<tr>
<td>Weanlings ≤ 1.5 year</td>
<td>Growthb</td>
<td>Feed best quality forages</td>
<td>55-65% TDN</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Energy/protein supplementation</td>
<td>14-16% crude protein</td>
</tr>
<tr>
<td>Males &gt; 1 year</td>
<td>Maintenanc eb</td>
<td>Low requirements unless</td>
<td>55-60% TDN</td>
</tr>
<tr>
<td></td>
<td></td>
<td>working; low-to-moderate quality forage</td>
<td>8-10% crude protein</td>
</tr>
<tr>
<td>Pregnant females (1-8 months)</td>
<td>Maintenance</td>
<td>Low requirements; ensure no loss of BCS &amp; adequate protein, minerals and vitamins</td>
<td>50-55% TDN</td>
</tr>
<tr>
<td>Pregnant female (9–11 months)</td>
<td>Pregnancy h,c</td>
<td>Moderate-to-high forage quality Supplement w/ mineral &amp; vitamins</td>
<td>55-70% TDN</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>10-12% crude protein</td>
</tr>
<tr>
<td>Breeding females</td>
<td>Maintenance</td>
<td>Low-to-moderate requirements;</td>
<td>50-55% TDN</td>
</tr>
<tr>
<td></td>
<td></td>
<td>maintain BCS</td>
<td>8-10% crude protein</td>
</tr>
<tr>
<td>Obese females</td>
<td>Submaintenance</td>
<td>Low requirements; low-quality forages with mineral/vitamin supplement unless pregnant</td>
<td>40-50% TDN</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>8-9% crude protein</td>
</tr>
</tbody>
</table>


a Palatable camelid- or sheep-labeled mineral mixes should be offered in every herd and actively monitored for consumption. White (loose) salt should be used when trace minerals are included in a supplement. b These feeding groups require higher amounts of trace minerals and vitamins, preferably delivered by a supplement. c Dietary energy and crude protein content may need to be increased further in late pregnancy if dry matter intake drops < 1.5% of body weight. BCS: body condition score.

Internal Parasites

Like any other animal, camelids are faced with exposure to internal and external parasites. These can be relatively benign or cause severe disease. Where each camelid ends up on this spectrum of severity depends on a number of factors, including type of parasite, number of parasites, and host immunity. Except in very rare cases, we cannot eliminate parasites. Therefore, our primary jobs are to promote immunity and prevent large-scale exposure. We attempt to manage parasitism to reduce or eliminate health effects. Most common internal parasites are listed in Table 2. A complete review of the topic has been described elsewhere (Ballweber (2009); Cebra (2014); Walker (2015)).
Table 2. Common internal parasites in camelids.

<table>
<thead>
<tr>
<th>Gastrointestinal tract</th>
<th>Liver flukes</th>
<th>Respiratory tract</th>
<th>Nervous system</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Eimeria punoensis</em>, <em>E. alpaca</em>, <em>E. lamae</em>, <em>E. macusaniensis</em>, <em>E. ivitae</em></td>
<td><em>Dicrocoelium dendriticum</em></td>
<td><em>Dictyocaulus viviparus</em>&lt;sup&gt;d&lt;/sup&gt;</td>
<td><em>Parelaphostrongylus tenuis</em></td>
</tr>
<tr>
<td><em>Cryptosporidium parvum</em>, <em>C. ubiquitum</em></td>
<td><em>Fasciola hepatica</em></td>
<td><em>Oestrus ovis</em>&lt;sup&gt;e&lt;/sup&gt;</td>
<td><em>Cephenomyia hominivorax</em>&lt;sup&gt;e&lt;/sup&gt;</td>
</tr>
<tr>
<td><em>Giardia duodenalis</em></td>
<td><em>Fasciola magna</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Haemonchus contortus</em>, <em>Ostertagia, Teladorsagia</em>, <em>Trichostrongylus</em>, <em>Camelostrongylus</em>, <em>Marshallagia</em>&lt;sup&gt;b&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Cooperia, Nematodirus</em>, <em>Trichostrongylus, Lamanema, Cooperia</em>&lt;sup&gt;b&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Oesophagostomum, Capillaria, Trichuris, Strongyloides, Monezia</em>&lt;sup&gt;c&lt;/sup&gt;</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> Stomach (C3) strongyles, <sup>b</sup> Small intestinal strongyles, <sup>c</sup> Large intestinal strongyles, <sup>d</sup> Lungworms, <sup>e</sup> Nasal bots

**Screening for Internal Parasites**

Camelids are not uniform in their parasite loads. The old tenet is that 10 to 20% of the animals have 80 to 90% of the parasites. These animals usually reflect those that have lower immunity (the old, young, and infirm), or have higher exposure (outcasts, dirt-eaters, crias). If the burden is high enough, these animals will show characteristic signs and should be screened for parasites. If the burden is more moderate, it is harder to identify these animals. We recommend regular parasite screenings. How often they occur depends on the specifics of the herd. Generally, if no animals are showing clinical disease (pale mucous membranes (MM), low body condition score (BCS) or underweight for age and size), examining feces from around 20% of the animals should provide reasonable surveillance. This may miss the intermittent heavy shedder, but all animals with a previous history of heavy shedding should be added to or included in the 20%. With most parasites, we try to quantify the fecal egg count, an estimate of the concentration of worm eggs in the feces. In doing so, we hope that these egg counts correlate well to the number of worms or protozoa in the intestine, and hence the risk of disease. Unfortunately, this is not always the case. Certain parasites cause disease before they shed large numbers of eggs or larvae (i.e., *E. macusaniensis*, *P. tenuis*).

Proper monitoring of internal parasites in camelids can be challenging, especially in larger herds. Ideally a fecal exam should be done on each animal before any anthelmintics are administered; this can be impractical in a large herd. It is tempting to do group fecals to “test” a group/pen. Owners like that approach as it will cost less for testing. They must be educated as to why pooling fecal samples is a poor choice and waste of money/time. Every animal has a different immune system and can be
exposed to the same parasites with different abilities to fight off the parasites. Unfortunately, no one test optimally finds all parasites (Table 3). Many tests completely miss certain parasites. Thorough breaking up of pelleted feces is key for accuracy.

Table 3. Diagnostic Methods to Detect Internal Parasites in Camelids.

<table>
<thead>
<tr>
<th>Diagnostic test</th>
<th>Parasite spectrum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Direct smear</td>
<td>Motile protozoa (Giardia); most types of worm eggs at high concentrations</td>
</tr>
<tr>
<td>Saline or zinc sulfate flotation</td>
<td>Most strongyles</td>
</tr>
<tr>
<td>Sucrose flotation (Sheather’s sol.)</td>
<td>Protozoa, strongyles</td>
</tr>
<tr>
<td>Double centrifugation technique</td>
<td>Protozoa, strongyles; best way to find small # of worm eggs</td>
</tr>
<tr>
<td>McMaster counting chamber</td>
<td>Most types of worm eggs at high concentrations</td>
</tr>
<tr>
<td>Sedimentation / Flukefinder</td>
<td>Fluke eggs</td>
</tr>
<tr>
<td>Baermann</td>
<td>Lungworms</td>
</tr>
<tr>
<td>Immunological methods</td>
<td>Enzyme Immunosorbent tests (<em>Cryptosporidium &amp; Giardia</em>), ELISA (liver flukes)</td>
</tr>
</tbody>
</table>

**Internal Parasites Control - Key Points**

Environmental management is an important component in parasite control in all species. As stated above, stocking density less than five to seven camelids per acre full-time should allow for parasite control and meeting nutritional needs. The establishment of dung piles by camelids provides some resistance to internal parasite transmission. Dung piles should be cleared at least weekly or biweekly, and more often when they occur around feeders or are sheltered. Pasture rotation or intensive grazing can be a useful tool from a nutritional and parasite standpoint, improving land usage and reducing larval burdens across land. BCS and MM color check should be performed on all animals within a herd monthly throughout the year to ensure timely intervention. Internal parasite screening (as described above) should take place at least quarterly. Drug resistance can be detected in a variety of ways, such as the fecal egg count reduction test (comparing premedication egg counts to those 10 to 14 days later), or the larval development assay. It is not currently recommended that deworming (Table 4) take place at routine intervals because of the development of anthelminthic resistance.
Table 4. Common anthelmintics used in camelids.

<table>
<thead>
<tr>
<th>Anthelmintic</th>
<th>Dosage</th>
<th>Route</th>
<th>Spectrum</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albendazole&lt;sup&gt;b&lt;/sup&gt; (Valbazen)</td>
<td>10 mg/kg once; can repeat in 5-7d</td>
<td>PO</td>
<td><em>Fasciola hepatica</em>&lt;br&gt;<em>Monezia</em>, GIN, lungworm</td>
<td>Do not use if pregnant or &lt; 50 lbs. Can cause liver failure young crias or if given for multiple days.</td>
</tr>
<tr>
<td>Doramectin (Dectomax)</td>
<td>0.4 mg/kg</td>
<td>SC</td>
<td>GIN, lungworms, mange mites, sucking lice</td>
<td>Not effective on <em>Trichuris</em> and <em>Monezia</em>.</td>
</tr>
<tr>
<td>Fenbendazole&lt;sup&gt;b&lt;/sup&gt; (Panacur/Safeguard)</td>
<td>20 mg/kg q24h x3d 50 mg/kg q24h x5d</td>
<td>PO</td>
<td>GIN, lungworms, <em>Trichuris</em>, <em>Nematodirus</em>, <em>P. tenuis</em>, <em>Monezia</em>, <em>Giardia</em></td>
<td>Safe in all ages (high dose included) and if pregnant.</td>
</tr>
<tr>
<td>Ivermectin (Ivomec)</td>
<td>0.2 mg/kg 0.4 mg/kg</td>
<td>SC</td>
<td>GIN, lungworms, mange mites, sucking lice</td>
<td>Not effective on <em>Trichuris</em> and <em>Monezia</em>.</td>
</tr>
<tr>
<td>Moxidectin (Cydectin drench)</td>
<td>0.4 mg/kg</td>
<td>PO</td>
<td>GIN, lungworms</td>
<td>Use in crias &gt; 4 months. Moderate margin of safety.</td>
</tr>
<tr>
<td>Oxfendazole (Synanthic 22.5%)</td>
<td>19.4 mg/kg</td>
<td>PO</td>
<td>Lungworms, GIN, <em>Monezia</em></td>
<td>Metabolized to fenbendazole.</td>
</tr>
<tr>
<td>Pyrantel pamoate (Strongid Paste)</td>
<td>8.5 mg/kg; 2Tx q10d 18 mg/kg q24h x3d</td>
<td>PO</td>
<td>GIN Tapeworms, GIN</td>
<td>Not to be used with levamisole. Moderate margin of safety.</td>
</tr>
<tr>
<td>Levamisole (Prohibit)*</td>
<td>8 mg/kg 6 mg/kg</td>
<td>SC</td>
<td>GIN</td>
<td>Last resort. Narrow margin of safety. Poor efficacy against <em>Trichuris</em> and lungworms</td>
</tr>
<tr>
<td>Clorsulon</td>
<td>7 mg/kg</td>
<td>PO</td>
<td><em>Fasciola hepatica</em></td>
<td></td>
</tr>
<tr>
<td>Praziquantel</td>
<td>50 mg/kg</td>
<td>PO</td>
<td><em>Dicrocoelium dendriticum</em>, <em>Monezia</em></td>
<td></td>
</tr>
<tr>
<td>Sulfadimethoxine</td>
<td>110 mg/kg q24h x10d</td>
<td>PO</td>
<td>Coccidia</td>
<td></td>
</tr>
<tr>
<td>Amprolium (given as 1.5% sol)</td>
<td>10 mg/kg q24h x10d</td>
<td>PO</td>
<td>Coccidia</td>
<td></td>
</tr>
<tr>
<td>Ponazuril</td>
<td>20 mg/kg q24h x1-3d</td>
<td>PO</td>
<td>Coccidia</td>
<td>Most effective for <em>E. macusaniensis</em></td>
</tr>
</tbody>
</table>

<sup>a</sup> Given once, unless otherwise noted, <sup>b</sup> GIN: gastrointestinal nematodes

**Routine Care**

*Body Condition Score:* Once weaning has occurred at approximately 4 to 6 months, BCS becomes more important than body weight. Animal weighing should be a routine procedure for camelid owners and done when using anthelmintics or other
pharmaceuticals to ensure accurate dosing. The author use a 1 to 10 scale, with 1 being emaciated, 10 being obese, and 5 being ideal.

**Shearing:** The single most effective time for husbandry intervention is in the spring, when shearing should occur in all camelid herds. Shearing provides protection of the animal against heat stress, along with harvest of fiber. Heat stress directly affects health, productivity, and male and female fertility. Because the thermal window for heat dissipation in the camelid is the ventral thorax and abdomen, a “barrel clip” or shear of only the trunk of the animal, is an acceptable means of preventing heat stress, although full-body clipping is preferred. It is recommended that this be completed before May 1 in the southern areas and June 1 in the northern areas of North America. Shearing should not be delayed until later in the summer in northern climates, to provide adequate time for blanket regrowth before the fall and winter.

**Toenail Trimming:** At the time of shearing, the feet and teeth should be examined and trimmed as needed. Camels have a toenail around a soft foot pad, rather than a hoof, and this toenail should be trimmed flush with the soft pad using small shear-type foot-trimming. Well-trimmed toenail showing the “V” shaped nail and proper relation (alignment) to the foot pad. After this yearly trim, owners should examine feet periodically, as some animals, particularly those on soft ground or with a corkscrew nail conformation, may need to be trimmed two to three times per year.

**Dental Care:** The teeth of all animals should be examined at shearing. The lower incisors grow continuously (alpaca only – not in llamas) and inferior prognathism, or underbite, occurs commonly in camelids. The lower incisors should just meet the rostral end of the superior dental pad and may be trimmed to this length using a diamond dremel-type bit and tool, or powered incisor trimmers designed for camelids, or obstetric wire. Next, the three pairs of fighting teeth, or upper fourth incisor and upper and lower canine teeth, should be evaluated. These erupt around 2 to 2.5 years of age in most animals and should be trimmed, particularly in males, to prevent camelids from injuring human beings and other animals. These may be trimmed using diamond dremel bits or obstetric wire, removing the sharp point and leaving 2mm to 4mm of the crown above the gum line to prevent tooth abscession. Side cutters or other manual cutting tools are strongly discouraged because of the high risk of tooth fracture. Forced extraction of these teeth is also discouraged, particularly of the mandibular canine, because of its proximity to the mental nerve and third incisor and risk of mandibular fracture.

Molar teeth do not continuously grow in camelids. The mandibular cheek teeth extend 3- to 4 mm medial (lingually) to the maxillary teeth, resulting in normal points on the crowns lingually on the mandibular teeth and buccally (cheek side) on the maxillary teeth. These points develop normally and should not be floated unless there is evidence of oral laceration, ulcerations, stomatitis, dysphagia (dropping food, chewing only one-sided instead of “figure 8”), and/or weight loss. Routine floating of camelid cheek teeth is strongly discouraged.

**Vaccination**
When making vaccine recommendations to camelid owners, veterinarians must remember that they are using these products in an extra-label manner, without
manufacturer liability. Additionally, most of the studies published demonstrate only serologic responses to vaccines, providing no known protective antibody level or challenge data. It is advisable to discuss risks and potential benefits of all biologics and pharmaceutics thoroughly with owners when designing herd-health protocols. With the exception of Clostridium perfringens type C, D and Clostridium tetani toxoid (CDT) and perhaps rabies, all vaccines listed herein are only recommended when there is documented evidence of that disease etiology in the herd and where vaccine is relied upon only to support management controls.

Toxoid vaccination against CDT is the current “core” vaccine recommended for camelids. Crias are immunocompetent at birth and, therefore, active immunization may be attempted during the neonatal period. Vaccination of the healthy cria against CDT, using full dosages of toxoid vaccines, should occur at 48 to 72 hours after birth, with a booster administered 2 weeks later. The duration of immunity for CDT vaccines is not known, but annual vaccination is recommended. It may be advisable to administer this vaccine 4 to 6 weeks before expected parturition to provide tetanus protection for birth-associated trauma and to increase colostral immunity for these pathogens. The author recommends that, if a husbandry procedure (i.e., castration) or injury occurs that would place an animal at risk for tetanus, a tetanus toxoid booster should be given at the time of the procedure if the animal’s last tetanus toxoid has occurred more than 6 months previously. If that animal’s tetanus toxoid was more than a year previously or is not known, a tetanus antitoxin should be administered at the time of injury. In regions where snakebite or liver flukes are common, which predispose to other serious clostridial infections (C. septicum, C. novyi and C. haemolyticum), a seven- or eight-way Clostridium vaccination may be considered. If they are used, it is recommended to recheck the injection site one week after vaccination for a local adverse reaction.

Rabies virus is a concern in most areas of North America and the spitting behavior of camelids increases the concern of zoonotic transmission of this disease. There have been sporadic cases of cameld rabies encephalitis in North America, with outbreaks in Peru reported. Transmission from alpaca-to-alpaca via biting has been demonstrated. The use of a large-animal-labeled rabies vaccine should be performed at full dose annually.

Leptospirosis has been diagnosed in llamas and alpacas and causes both reproductive and renal disease. In herds or areas where leptospirosis is an endemic disease, vaccination with multivalent bovine vaccines may be performed. There is evidence of short-lived immunity in alpacas vaccinated against Leptospira and it is recommended that high risk farms vaccinate females before breeding and again at mid-gestation. This vaccine may need to be given up to four times annually in endemic areas where wildlife and contaminated water-source exposure cannot be controlled. Another potential abortive agent, Chlamydophila (formerly Chlamydia), isolated from infected fetal materials, may be vaccinated against, with good serologic responses to sheep labeled vaccines.

Camelids housed near domestic and exotic equids are at risk for encephalomyelopathy caused by Equine Herpesvirus-1 and Eastern Equine Encephalitis virus. This risk is greatest in the presence of exotic equids, and in such situations, killed EHV-1
vaccination should be considered quarterly and in high-risk areas for Eastern Equine Encephalitis, killed vaccines may be advisable.

West Nile Virus has been reported in camelids, however the relative risk appears to be low. Vaccination against West Nile virus should begin 2 to 3 months before the start of vector (mosquitoes) season, and boosters every 2 to 3 months should be considered. It is not known how annual revaccination should be scheduled, but it should probably occur 3 to 6 weeks before peak exposure.

Vaccination of camelids against BVDV is not currently recommended because of potential interference with diagnostic testing and lack of efficacy or safety information.

**Further Reading:**
Management of neonatal camelids requires knowledge and understanding of normal peripartum events and how to correctly identify abnormalities so that the potential for complications can be minimized. It is important to evaluate all newborn crias for the presence of congenital defects so that action may be taken where necessary to improve the welfare of affected animals. Close monitoring is recommended for all crias so that, if necessary, prompt treatment may be undertaken. Premature crias should be identified as soon as possible as this group is particularly at risk of morbidity.

Management of the Pregnant Female Cameld
Ensuring the health of neonatal camelids begins with ensuring that pregnant dams reach their due dates in optimum condition. This is important for ensuring a problem free periparturient period and also for enabling the female to produce sufficient colostrum of adequate quality, and milk for the growing cria. On the 1- to 10- scale, pregnant and lactating females should be maintained at a body condition score (BCS) of 5.5 to 6 at all stages. Consideration should be given to the location of the birthing area. This should ideally be conveniently located to increase the amount of observation time in case of dystocia. The area should be clean so that crias are born into an uncontaminated environment. A clean grassy pasture is the ideal location. If this is not available, females could be moved into a clean, straw-bedded stall when parturition is imminent, immediately afterwards.

The length of gestation in camelids can vary considerably. On average, it is around 343 days (11.5 months) but this is highly variable. In most cases, gestation length tends to be significantly longer in llamas and alpacas giving birth in the spring than in the fall. Stage one labor (preparatory phase) may last from 1 to 6 hours in camelids. Indications of this stage include separation from the rest of the herd and lack of interest in eating, vocalization by humming, restlessness, frequent visits to the communal dung pile, and sitting in sternal recumbency with the hind legs out to the side. Stage two labor (expulsion of the cria) typically only lasts 20 to 40 minutes, but may take up to an hour. If active straining without progression is observed for 15 minutes in a camelid, the female should be evaluated for malpresentation of the fetus. Crias are delivered in an anterior presentation, dorsosacral position, with head and front limbs extended. Stage three labor (expulsion of the placenta) normally takes place within an hour of delivery of the cria, but the placenta should be expelled within 4 to 6 hours maximum. In the rare case of a retained placenta, prostaglandin and oxytocin therapy can be initiated. Cloprostenol can be given at 250 mg intramuscularly (IM) and oxytocin used at a dose of 10 IU IM every 4 to 6 hours until the placenta is delivered. Larger doses of oxytocin or more frequent dosing may cause myometrial spasm or invagination of the tip of the uterine horn resulting in signs of colic. Camelid dams will not consume their placenta.

Routine Care of the Healthy Newborn Cria
Assuming normal delivery of a term cria in a clean environment, management of the newborn consists in two stages: first observational, then hands-on. Dams and crias should be left alone as much as possible to begin with so that bonding can occur, but it is best to step in early in inclement weather or if a maiden female is being
overwhelmed. Dams typically show very little interest in the newborn cria until the cria has latched onto a teat for the first time and starts nursing. They will not lick the cria, but will generally stand over the cria until it is ambulatory and may vocalize by humming. The newborn cria is covered in a thin, translucent epidermal membrane that is attached at the mucocutaneous junctions. The function of this membrane is unknown but it is thought that it may aid lubrication in the birth canal and also act as a windbreaker in the immediate postpartum period. It usually rubs off during the first few hours of life as the cria dries off.

Healthy crias usually stand within 1 hour, walk within 2 hours, and nurse successfully within 2 to 4 hours after birth. Crias normally nurse frequently, two to three times per hour in the neonatal period, and often for less than a minute. They will spend only a short amount of time on each teat before moving on to the next. A cria that is seen to be nursing frequently and for long periods should alert the observer to the possibility that the dam has insufficient milk for the demands of the cria.

If it is cold or wet outside, crias should wear a fitted cria coat or be brought inside with their dams to reduce the risk of hypothermia. A stall or pen that has been freshly bedded with straw is ideal for this purpose. Heat lamps are useful for providing a small warmed area.

Once bonding has occurred between the dam and cria, the cria should be weighed and the umbilicus dipped in an antiseptic solution. Birth weight should be taken using an electronic scale and recorded. Normal alpaca crias should weigh at least 5.5 kg (12 lbs) and llama crias 7 kg (15.5 lbs), although average weights are typically closer to 7 kg (15.5 lbs) and 9 kg (20 lbs) for alpaca and llama crias, respectively. Their expected growth rate is 0.25 to 0.5 lb/day for alpaca cria and 0.5 lb to 1.0 lb/day for llama crias. Similarly to other large animal neonates, a cria’s umbilicus should be dipped with a disinfectant soon after birth. Consensus on dipping solution, frequency, and dilution factor, however, is lacking. Suggestions vary among investigators and include 2% to 3% or 7% iodine tincture, 0.5% chlorhexidine solution (preferred), povidone-iodine solution, 1:1 povidone-iodine and glycerin, and Lugol’s solution. Recommended dipping frequency varies from one to three times daily within the first 24 hours and up to 72 hours after birth. Ligature placement increases the chance of umbilical abscess formation since normal drainage is prevented. In healthy crias, rectal temperature, heart rate, and respiratory rate range from 100 to 102°F, 70 to 100 beats per minute, and 10 to 30 breaths per minute.

**Meconium Impaction**

Meconium is typically passed around 18 to 24 hours of age. Meconium impaction often occurs in dehydrated or hypothermic crias who show decreased to absent nursing and persistent tenesmus in the face of normal urination. A warm soapy water enema (10 ml–12 ml for alpaca cria, 12 ml–24 ml for llama cria) should be administered using a soft, flexible, small-diameter catheter. It can be repeated if meconium is not passed within 30 to 60 minutes after the first enema. Although infrequent, cases not responding to warm soapy enema may benefit from receiving a retention enema, similar to that used in foals with meconium impaction (except for total volume administered, i.e.1/5 of a foal dose). The cria is physically restraint, ideally in a standing position. A foley catheter is inserted ~ 3 cm in the rectum, the
balloon is inflated (use 0.9% NaCl) and left in place for at least 30 to ideally 45 minutes. Retention enema recipe is as follows: 4 grams NaHCO₃ + 1.6 grams of acetylcystein (= 8 ml of a 20% solution (200 mg/ml)) + 40 ml of water. Although infrequent, refractory cases involving impaction within the spiral colon may require intravenous fluid and, in last resort, oral administration of corn syrup (10 ml–15 ml by mouth, every 12 hours: osmotic laxative). The use of Fleet enemas available in human pharmacies is not recommended. The author has seen several cases of peritonitis-induced proctitis and hyperphosphatemia associated with repeated administration of this product in newborn camelids.

**Colostrum**
Crias are born severely hypogammaglobulinemic because of the diffuse epitheliochorial nature of placentaion in camelids. Ensuring adequate colostrum intake (10% of body weight in 24 hours) is vital to acquire passive immunity. Failure of passive transfer of immunoglobulin (FPT) has been demonstrated to be a major cause of neonatal mortality in alpacas. If a female has insufficient milk, then frozen camelid colostrum can be used, or goat, cow, or sheep colostrum as alternatives. Llama milk has been shown to have higher sugar content (6.5%) and less fat (2.7%) than the milk of other domestic ruminants and it appears that either goat or cow milk is the most suitable alternative. This can be either fresh or frozen. Frozen colostrum keeps for up to one year in the freezer. In general, it is best to avoid powdered colostrum supplements or replacers as these have been shown to be inadequate substitutes for maternal colostrum in many studies in other species.

**Bottle-Feeding**
If bottle-feeding a neonatal camelid, it is advisable to aim to feed 10% to 15% of the cria’s body weight over 24 hours, divided into feedings every 2 hours initially. A commercial camelid milk replacer (Wombaroo Alpaca Milk Replacer) is available. Feeding small amounts frequently more closely simulates nursing behavior and reduces the potential for overfeeding resulting in milk entering the first stomach compartment (C1). Larger volume feedings that are safe in calves appear to result in a higher occurrence of C1 acidosis in crias. Bottle feeding is preferred, although tube feeding may be necessary, initially. Repeated tube feedings may cause esophagitis.

Beserk male syndrome also called aberrant male behavior is most common in male, typically bottle-fed cria (llama > alpaca) or cria with close (and continued) interaction with humans for extended periods of time. The cria will imprint on humans and see them as part of its herd. Once puberty sets, the male instinctively tries to establish dominance in the herd, which unfortunately includes the owners. Most cases become difficult and somewhat unsafe to handle. In intact male, castration may attenuate the extent of the behavior some but will not make it disappear (usually irreversible). Beserk male syndrome can be prevented by limiting human contact with crias for the first 2 to 3 months of age. If bottle-feeding is needed, it should be done while in contact with other herd mates. This behavior is not the same as undisciplined or “bad mannered” llama which is often curable.

**Other Considerations**
In selenium-deficient areas, crias should receive Bo-Se (0.5 ml for alpaca cria, 1 ml for llama crias, subcutaneously (SC)). To prevent hypophosphatemic rickets, vitamin D
(usually in vitamin A, D, and E mixture) should be administered at a dosage of 1,000 IU/kg SC, twice at 3-month intervals (ex: November and February), especially for dark-coated fall-born crias. Please note that injectable vitamin D given at higher dosage to young crias has been reported to be fatal in some cases.

**Vaccination**

Toxoid vaccination against *Clostridium perfringens* types C and D and *Clostridium tetani* (CDT) is the current core vaccine recommended for camelids. Crias are immunocompetent at birth and, therefore, active immunization may be attempted during the neonatal period. Vaccination of the healthy cria against CDT, using full dosages of toxoid vaccines, should occur at 48 to 72 hours after birth, with a booster administered 2 weeks later. The duration of immunity for CDT vaccines is not known, but annual vaccination is recommended. It may be advisable to administer this vaccine 4 to 6 weeks before expected parturition to provide tetanus protection for birth-associated trauma and to increase colostral immunity for these pathogens.

**At-Risk Crias**

Crias are most at risk of complications during the first 2 to 3 weeks of life. Approximately 35% of crias will develop diseases (mainly diarrhea or other infectious diseases) between birth and weaning, and close to 2% will die before weaning. It is therefore recommended to weigh each cria daily for the first 2 to 3 weeks of life so that problems can be detected early on and treated before the onset of severe disease. Close observation of neonatal crias is also important to detect the subtle signs of illness. Following this initial period, the frequency of weighing can be reduced to weekly for the next few months, and then less frequently.

Crias thought to be at risk of FPT should be tested to assess the adequacy of passive transfer of immunity. Other potential risk factors for FPT include evidence of prematurity or dysmaturity, dystocia or C-section birthing, maternal factors (poor mothering, maiden female dam, poor udder development, mastitis, underweight or obesity), inclement weather conditions that may result in hypothermia, and congenital defects that may prevent effective nursing.

There are various methods described for the determination of passive transfer status in crias. These include serum total solids measurement (using a refractometer), total protein and globulin concentrations (automated biochemistry analyzer), sodium sulfite turbidity test (SST), and radial immunodiffusion (RID). In healthy, well-hydrated crias, refractometer readings of total solids less than 4.5 g/dl are consistent with FPT, while readings greater than 5.5 g/dl is consistent with adequate passive transfer. The most accurate determination of the adequacy of passive transfer is achieved using a RID.

The disadvantage of this test is that it takes 24 hours to perform. A commercial camelid-specific IgG test is available (Triple J Farms, Bellingham, WA, USA). In the United States, an IgG test showing a concentration of 800 mg/dl is required by insurance companies to insure a cria before it reaches 3 months of age. The ideal time for blood sampling crias to assess adequacy of passive transfer is 48 to 72 hours of age.

**Premature and Dysmature Crias**
Based on the available data it seems reasonable that any cria born at less than 335 days of gestation could be termed premature. Dysmaturity is the term used to describe crias born at normal gestational age but having indications of prematurity. These crias are as much at risk of morbidity as premature crias. Clinical signs of prematurity are the same as those in other species and include low birth weight, unerupted incisors, floppy ears or ears that are bent backward, poor or absent suckle reflex, and silky short hair coats. Tendon laxity may result in overextension of the carpal and fetlock joints so that crias may be seen walking on their fetlocks. Crias may also have a rubbery covering on the nails of the feet and a thicker epidermal membrane that persists for longer than normal. Premature crias tend to be weak and may have difficulty standing or holding their heads up to nurse because of poor muscle development. They are at risk for hypothermia because their thermoregulatory control is poor and they are prone to hypoglycemia. Respiratory system immaturity may result in poor lung expansion caused by lack of pulmonary surfactant and this also reduces oxygen transfer across the alveoli. This, together with bradypnea, results in hypoxia. The intestinal mucosa may also be inadequately mature in premature crias resulting in inefficient absorption of colostral antibodies. Recognition of the clinical signs of prematurity is vital in newborn crias as they may deteriorate rapidly. Therapeutic measures include oxygen administration, intravenous-fluid therapy with dextrose-containing fluids, plasma administration since FPT is common, and provision of warmth. Tendon laxity in premature crias may resolve spontaneously once the newborn cria starts weight bearing and the ligaments and tendons strengthen from use. In severe cases, splinting of the forelimbs may be necessary from the elbow to below the fetlocks. However, these must not be placed for too long as this may actually worsen the problem.

Congenital Defects in Crias

Many congenital defects have been reported in camels. It is important to be able to recognize these defects promptly to alleviate suffering in affected individuals and to minimize the emotional trauma to the owner or breeder. Certain defects, such as severe facial deformities (i.e., maxillofacial dysplasia, choanal atresia (CA)) represent a significant welfare issue and affected individuals should be diagnosed and euthanized as soon as possible, when appropriate. Other defects require prompt diagnosis so that therapeutic measures may be instigated as soon as possible (i.e., vulvar deformities resulting in an inability to urinate). It is also necessary to consider the potential breeding implications following diagnosis of a congenital or hereditary condition in camels. Not all congenital defects are hereditary, some may be induced by teratogens or viral infections, but once present in an animal, they may still be passed on. Therefore, no animal with a defect should be bred. Unfortunately, hard data concerning the heritability of many of the congenital conditions seen in camels is incomplete making decisions concerning the parents of affected individuals particularly difficult.

Choanal atresia - this is a condition in which the oronasal membrane fails to open during embryogenesis (at the choanae) because a membranous or osseous defect prevents air from passing through the nostrils to the nasopharynx. Some crias compensate for this by mouth breathing while the majority exhibit signs of dyspnea shortly after birth. Hypoxia develops rapidly resulting in collapse. A characteristic feature is that affected crias suck air into their mouths, ballooning the cheeks. They
then squeeze the cheeks to force air around the soft palate and over the epiglottis. Expiration requires similar effort to force air around the soft palate and out of the mouth. Affected crias will struggle to nurse effectively and aspiration pneumonia is a common complication. Diagnosis may be achieved by passing a soft, round-ended catheter up each nostril; an obstruction at the level of medial canthus is supportive of this diagnosis. Contrast radiographs or computer tomography scan will confirm, but they are not usually necessary when failure to pass the catheter is repeatable and consistent. Surgery is possible at referral centers but should only be considered in animals that are stable, or in unilateral cases.

**Plasma Administration**
There is probably no need to administer plasma to crias that are healthy, have marginal passive transfer and are housed in a large pasture with minimal pathogen exposure. Decision to transfuse must be taken in the case of a sick or septic cria, premature cria, valuable cria or at the owner’s request.

A commercial camelid (llama) plasma is available (Triple J Farms, Bellingham, WA, USA). Recommended plasma dose in crias is 20 to 40 ml/kg. However, in sick/septic cria, 2 or more units of plasma (~300 ml/unit) may be needed to achieve IgG levels over 1000 mg/dL. Plasma should be thawed slowly by immersion in warm water. Active heating with microwave or other means must be avoided to prevent denaturing of proteins. Recipient should have a jugular intravenous (IV) catheter placed aseptically. Thawed plasma should be warmed up ideally close to the body temperature of the cria and attached to a filtered administration set. Recommended rate of administration are as follow: 5 ml/kg/hour for the first 15 to 20 min then increase the rate to 10 ml/kg/hour. Total amount should be administered within 3 hours. Recipient cria should be monitored very closely for transfusion reaction especially in the first 15 to 20 minutes of plasma administration. Clinical signs suggestive of an allergic reaction include but is not limited to: facial edema, hives, rise in body temperature, fever, tachycardia, tachypnea, dyspnea, and collapse. If present, plasma transfusion should be discontinued. Flunixin meglumine (1 mg/kg IV) may decrease the severity of adverse reaction.

Intraperitoneal (IP) administration of plasma is another option for correcting FPT in neonatal cameldids. This technique offers a time-efficient method for administration of plasma without the need to catheterize the jugular vein. However, plasma should not be administered IP in cria with gastrointestinal problems or those overly septic (due to chances of peritonitis). **Equipment needed:** clippers, aseptic prep kit (chlorhexidine and alcohol), metal teat canula (5 cm in length), sterile gloves, IV administration set, 2% lidocaine, no.15 scalpel blade and a 3 or 6 ml syringe. **Procedure:** In cria < 30 days, left flank (point adjacent to the costochondral arch of the last rib and at the junction of the ventral and middle thirds of the abdominal wall) is preferred site for IP canula placement. After sterile prep and lidocaine infiltration, tent skin and make incision with 15 blade. Placement of the teat canula into the peritoneal cavity is achieved by steady pressure. Intraperitoneal position of the canula should be ensured prior to infusion of any plasma (free end is movable, suction applied to verify that viscera have not been penetrated). Once the plasma transfusion is completed, the metal canula is removed and skin incision is closed with a surgical staple or tissue glue.
Further Reading:

Behavior
Llamas and alpacas are herd oriented animals and have a distinct social structure including a dynamic command hierarchy. When camelid herds are moved to a different location, a member of the group is removed, or members of different groups in different pastures are mixed, a period of reorganization occurs. As such, high stocking density due to housing space limitation is likely to create social stress. Overall, llamas’ behavior is similar to the one of goats; they are interactive with humans and inquisitive but stoic. Alpacas act similarly to cattle and sheep; they are less interactive with humans, fairly calm but will flee if a perceived threat is present.

Capture Techniques
When performing group activities such as annual vaccination and deworming, these procedures can be easily and readily performed in small groups of animals. Small group settings (< 10 alpacas, < 5 llamas) lessen the stress of individual handling and may help to prevent stress-induced problems (peracute third compartment (C3) ulcers, abortion, premature births, etc.). Ideally, the farm facilities should be used to create a series of enclosures such as pens or corrals so that the entire group can be captured in total, and then smaller groups separated off for procedures and interactions. Using laneways leading to a smaller paddock and ultimately to a catch pen (no larger than 3 x 3 m [10 x 10 feet] square and 1.2–1.5 m [4–5 feet] in height) makes catching easier and more appealing for the animals. The use of 1.2 meter (4 foot) herding poles allows handlers to communicate with camelids from a distance, which is easier and safer for the animals. Tying a rope or using a nylon tape attached to the corner of a paddock allows handlers to create a temporary fence or laneway leading to a smaller area. Alternatively, the rope can be suspended between two people and used to gradually reduce the area of containment. Ropes or herding tape is not suitable for trapping animals in a corner. Herding tape is only useful for creating a temporary path to a safe handling area. A handling area with pens of various sizes, including 2.7 x 2.7 m (9 x 9 feet) and 1.2 to 1.5 m high (4–5 feet), which is useful for catching, haltering, restraint-free injection, and other management tasks; 2.7 x 0.9 m (9 x 3 feet), which is useful for trimming toenails; and 2.7 x 8.2 m (9 x 27 feet), which is useful for initial lead training, allows handlers to work with containment instead of using restraint. Owners who do not have a proper setup but who own trailers can use a trailer as a handling area.

Camelids flight zone is similar to what described for cattle. The point of balance is 90% to the shoulder – if the handler is behind this line, the animal moves forward; if in front of the line, the animal moves backward. To capture a single animal, a lead rope may be draped across the back of the animal with the goal of forming a loose loop around the neck. The length of rope is then shortened until the handler’s arms can be encircled around the base of the neck. Then the arms are moved forward along the neck until positioned behind the head. An appropriate sized camelid halter (with lead rope) is fitted over the nose by first approaching the head with the halter below the jaw line.

Halter Placement
A properly fitted alpaca halter has the muzzle portion of the halter encircling the jaws in the caudal third of the nasal passages and fitting securely around the head.

Basic Handling
In the absence of a halter, head and neck are grasped and pulled firmly against the handler’s body for manual restraint. Control can be maintained using minimal restraint by grasping the mandibles with one hand and simultaneously placing the other hand behind the poll of the head and the uppermost portion of the neck. An ear squeeze can be used to gain additional control in uncooperative patients. During standing restraint, the tail may be grasped at the base to facilitate restraint (relaxation can often be achieved by gently rotating the tail in a circular motion). Finally, a cursory oral exam can be done by placing the poll of the head in the crook of the elbow and then inserting the fingers into the cheek.

Overall, camelids do not like their distal limbs touched (will commonly lie down in the process!). To facilitate feet/distal limb examination the animal can either be halter tied to a post temporarily (should not be left unattended), placed into a suitable manual camelid chute, or in the worst case scenario, administered mild sedatives.

Local Blocks
Similarly to other species, lidocaine is used to provide local anesthesia in camelids (local infiltration lasts 60 to 90 minutes). The toxic dose for lidocaine 2% in llamas and alpacas is 4 mg/kg (9 ml/100 lbs or 10 ml/50 kg of body weight). Lidocaine dose for caudal (sacroccocygeal) and lumbosacral epidural is 1 ml/100 lbs and 1 ml/20 lbs, respectively.

Injection Sites
Intravenous: The jugular vein is most commonly used although lateral saphenous and cephalic veins may also be used. The jugular vein is most easily injected in either the upper one-third (high venipuncture) or lower one-third of the neck. The mid-portion of the neck is easily accessed, but the carotid artery lies closer to the jugular vein in this region. Thus, caution should be exercised for mid-cervical injections. At the high (proximal) site, the skin is thinner than the mid-cervical region, and the vein is superficial and somewhat separated from the carotid artery. At the low (distal site), the carotid is close to the jugular, and there is increased risk of intra-arterial injection. However, the landmarks for puncture of the jugular vein can be easily palpated, and the vein is larger in this location. For both sites, the head and neck should be held upright and the animal well restrained. The operator should palpate the transverse processes of the cervical vertebrae and the trachea medially. The jugular groove lies between these two structures, and the vein is rarely visible, particularly when the animal is not clipped. The operator occludes the jugular groove using their thumb, bridging between the transverse process and the trachea, waiting to allow the vein to fill. The vein is then balloted with the opposite hand to confirm position and fill. The needle is inserted at a 45- to 60-degree angle into the skin. After the needle is in the soft tissues of the neck, the plunger is held with negative pressure as the needle is advanced and inserted into the vein.

Intramuscular: The neck (small volume), semimembranosus/semimembranosus muscles at a location one hands-breath ventral to the point of the ischium may be
used. Alternatively, smaller volume of drugs (< 5 mls) can be administered in the triceps muscles (5 to 10 cm proximal to the point of the olecranon).

Subcutaneous: Injection can be done anywhere into the loose tissues at the base of the neck, behind the elbow along the ribcage, or alongside of the sternum cranial to the forelimbs.

**Sedation & Tranquilization**
Clinically, alpacas demonstrate more resistance to anesthetic drugs, and llamas are most susceptible to their effects. In general, llamas require 33% less drug on a body-weight basis as compared with alpacas to achieve a similar effect.

**Sedation Protocols Commonly Used in Camelids**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Species</th>
<th>Dosage range</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butorphanol</td>
<td>Llama</td>
<td>0.025 – 0.1 mg/kg</td>
<td>IV, IM, SC</td>
</tr>
<tr>
<td></td>
<td>Alpaca</td>
<td>0.05 – 0.2 mg/kg</td>
<td>IV, IM, SC</td>
</tr>
<tr>
<td>Xylazine</td>
<td>Llama</td>
<td>0.05 – 0.4 mg/kg</td>
<td>IV, IM, SC</td>
</tr>
<tr>
<td></td>
<td>Alpaca</td>
<td>0.1 – 0.5 mg/kg</td>
<td>IV, IM, SC</td>
</tr>
<tr>
<td>Acepromazine</td>
<td>Llama or Alpaca</td>
<td>0.02 – 0.05 mg/kg</td>
<td>IV, IM, SC</td>
</tr>
<tr>
<td>Diazepam</td>
<td>Llama or Alpaca</td>
<td>0.05 – 0.2 mg/kg</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Ketamine stun</td>
<td>Llama or Alpaca</td>
<td>Butorphanol: 0.05 mg/kg</td>
<td>IV</td>
</tr>
<tr>
<td>(B:K:X combination)</td>
<td></td>
<td>Xylazine: 0.1 mg/kg</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ketamine: 0.2 mg/kg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Llama</td>
<td>Butorphanol: 0.1 mg/kg</td>
<td>IM</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Xylazine: 0.2 mg/kg</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ketamine: 0.4 mg/kg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Alpaca</td>
<td>Butorphanol: 0.15 mg/kg</td>
<td>IM</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Xylazine: 0.3 mg/kg</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ketamine: 0.6 mg/kg</td>
<td></td>
</tr>
</tbody>
</table>

**Field Injectable Anesthesia**
Prior to general anesthesia and to decrease the likelihood of regurgitation, bloat and aspiration pneumonia, adult llamas and alpacas are to be held off feed for 24 to 48 hours and not allowed to drink water for 8 to 12 hours. Nursing crias less than 2 months of age may be held off milk and water for 6 to no more than 12 hours.

When overseeing general anesthesia under injectable anesthetic drugs, one must ensure proper patient positioning. Patient maintained in lateral recumbency should be placed on adequate padding and positioned with head and neck extended (maintain patency of upper airways). In addition, the patient’s nose should be placed lower than the larynx (allow saliva and/or rumen content to flow out the oral cavity). Lastly, its down forelimb should be pulled forward, the latter to prevent radial nerve paralysis.
### Injectable Anesthesia Protocols Commonly Used in Camelids

<table>
<thead>
<tr>
<th>Drug</th>
<th>Species</th>
<th>Dosage range</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ketamine stun (B:X:K combination)</td>
<td>Llama</td>
<td>Butorphanol: 0.05 - 0.1 mg/kg</td>
<td>IV, IM</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Xylazine: 0.2 – 0.25 mg/kg;</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>: 0.3 – 0.35 mg/kg</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ketamine: 2 – 2.5 mg/kg</td>
<td>IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>: 3 – 4 mg/kg</td>
<td>IM</td>
</tr>
<tr>
<td></td>
<td>Alpaca</td>
<td>Butorphanol: 0.05 – 0.1 mg/kg</td>
<td>IV, IM</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Xylazine: 0.25 – 0.3 mg/kg</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>: 0.4 – 0.5 mg/kg</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ketamine: 2.5 - 3 mg/kg</td>
<td>IV</td>
</tr>
<tr>
<td></td>
<td></td>
<td>: 4 – 5 mg/kg</td>
<td>IM</td>
</tr>
<tr>
<td>Ketamine – Diazepam</td>
<td>Llama</td>
<td>Diazepam: 0.15 mg/kg</td>
<td>IV, IM</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ketamine: 2.7 mg/kg</td>
<td>IV</td>
</tr>
<tr>
<td></td>
<td>Alpaca</td>
<td>Diazepam: 0.2 mg/kg</td>
<td>IV, IM</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ketamine: 3.6 mg/kg</td>
<td>IV</td>
</tr>
</tbody>
</table>

### Reversal Agents Commonly Used in Camelids

<table>
<thead>
<tr>
<th>Xylazine Reversal Drugs</th>
<th>Species</th>
<th>Dosage range</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yohimbine</td>
<td>Llama or Alpaca</td>
<td>0.125 mg/kg</td>
<td>IV or IM</td>
</tr>
<tr>
<td>Tolazoline</td>
<td>Llama or Alpaca</td>
<td>1 – 2 mg/kg</td>
<td>IM recommended</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>IV: administer VERY slowly</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(emergency situation only)</td>
</tr>
<tr>
<td>Atipamezole</td>
<td>Llama or Alpaca</td>
<td>0.125 mg/kg</td>
<td>IV or IM</td>
</tr>
</tbody>
</table>

### Aftercare
After the conclusion of the procedure, the animal should be closely monitored until it is fully aroused and able to maintain sternal recumbency/posture. Food and water should not be offered until the patient can stand up and walk around without falling. This will decrease the risk of aspiration, esophageal choke, or bloat.

### Further Reading:
Thin Ewe or Thin Doe Syndrome: An Investigative Approach

Jeffrey Wichtel BVSc, PhD, Dip ACT

Chronic wasting, with or without obvious signs of disease, is a common presenting complaint in older ewes and does, often leading to reproductive failure and early culling. A variety of disease and management factors, singly or in together, can lead to chronic weight loss. A logical series of investigative steps can facilitate coming up with a plausible diagnosis and action plan.

Affecting an entire group?

Where all or most of a group is affected with chronic weight loss, the most likely factors will include nutrition and/or parasitism.

1. Sub-optimal nutrition. Forage analysis is often not performed for small ruminant flocks. Producers may be unaware of the low nutritional value of their forage or pasture. This can lead to inadequate energy and protein intake. Some loss of condition during the first weeks of lactation, especially in dairy breeds or highly fecund flocks, is to be expected, but should not result in BCS less than 2/5. Late pregnancy, lactation, and very cold weather can increase requirements; pregnant females often cannot consume enough poor quality forage to support their needs. Inadequate bunk space and/or wide variation in live weight may result in a subset of the group not competing well for the ration. Finally, failure to provide continuous access to an appropriate balanced trace element mineral supplement for sheep (no added copper) or goats can lead to failure to thrive. Selenium is most likely micronutrient deficiency in non-supplemented animals in many parts of the country (1mg supplementary Se / head / day will usually meet requirements).

2. Gastrointestinal parasitism. The most common GI parasites leading to chronic weigh loss in adult ewes and does are the trichostrongyles, especially Haemonchus and Teladorsagia. Larvae of these worms require a pasture phase so will not be a concern in total confinement operations. Winter hypobiosis is a common feature; signs of clinical parasitism can be seen in winter-confined ewes and does arising from larval burdens obtained on pasture during the previous fall, leading one to ask, “where did these worms come from”? Morbidity and mortality will depend on the larval challenge, the species of parasite, the physiologic state of the animal, and the genetics of the host. Animals that are pregnant or lactating, underfed (especially protein) or stressed for other reasons, are more susceptible. Other than weight loss, parasitized animals will tend to show some of the following signs: agalactia, failure to rear lambs, diarrhea (not Haemonchus), anemia (mainly Haemonchus), bottle jaw, and sudden death. Fecal egg counts (FEC) on 10 randomly identified animals per group provide an indication of the parasite load. In general, FEC correlate well with worm burden in ewes and does during the grazing season, however in the winter they are not usually representative. Degree of anemia is useful in identifying the burden of Haemonchus (see FAMACHA guidelines). Post mortem examination and worm counts are definitive.
Affecting individual(s)?
Where one or a few individual(s) are affected with chronic weight loss, the most likely factors will include social issues, dental disease, and/or chronic infectious diseases.

1. Social Issues. Sheep and goats are sensitive to the social stress associated with frequent changes in groupings. Mixing of animals of highly disparate age, live weight, and/or production stage (young growing lambs with older ewes, for instance) will lead to competition for feed. Overcrowding will occur when pen space is less than 10-20 sq. feet per female. Feeder space should be 16-18 inches per adult.

2. Dental Disease. Tooth problems are common in sheep greater than 6 years of age. It is not uncommon to see incisor loss, leading to “broken mouth” and in extreme cases “gummy” ewes (absence of incisors). Depending on the source of forage, this can lead to low intake and weight loss. Check the teeth of every ewe you examine. The reasons for excessive tooth wear, periodontal disease, and tooth loss in sheep are not known but may be related to forage quality, soil ingestion, and/or trace element deficiencies.

3. Chronic Infectious Diseases. Approach to the diagnosis of chronic infectious disease is challenging. Although the issue is usually a flock problem, often just a few individuals are clinically affected at the time of investigation; other infected animals are at a pre-clinical stage of the disease, or have already been culled early for a variety of reasons, such as infertility. For every clinically affected individual, assume 10 sub-clinically infected animals. Consider the following diseases once you have ruled out the non-infectious causes outlined above.
   a. Caseous lymphadenitis (CL). *Corynebacterium pseudotuberculosis* infection results in abscesses of lymph nodes. The progression of this disease is slow: months to years. If only peripheral lymph nodes are affected, severe weight loss is not likely, but these abscesses will often discharge sticky pus, thus acting as a source of infection for flock mates. Internal (mediastinal, abdominal) lymph nodes, when infected, can lead to chronic weight loss and a variety of other signs. In a study of cull ewes in Alberta, over 50% were serologically positive for CL, and over 20% had abscesses. In a Quebec study, 25% of abscessed ewes had only internal abscesses; thus presence of enlarged peripheral lymph nodes cannot be relied upon as the sole diagnostic criterion. *C. pseudotuberculosis* is a long-lived organism in the environment. Transmission occurs from ruptured abscesses, coughing, wounds at time of shearing, and at feeders (this is why nodes around the head, neck, and shoulder are more often affected). Little transmission occurs before weaning. A history of ill thrift in older females with enlarged nodes suggests CL, and diagnosis can be confirmed using clinical examination, with bacteriologic culture if required. Blood tests are characterised by poor sensitivity and specificity, and cannot easily differentiate infected, exposed and vaccinated individuals, but can be used for flock screening.
   b. Persistent lentivirus infection (Maedi-Visna / ovine progressive pneumonia [OPP] in sheep, and caprine arthritis and encephalitis [CAE] in goats). MVV and CAEV are the small ruminant lentiviruses, leading to progressive, mononuclear inflammatory lesions in the lungs, joints,
udder and central nervous system. Most lentivirus-infected animals are asymptomatic, but remain persistent carriers. Primarily transmitted from dam to offspring through colostrum. Contact transmission is rare, but possible.

i. Clinical CAEV in goats is characterized by polyarthritis (big knees) and emaciation. About 20% of infected animals become clinical. In an Ontario study of 30 dairy and 15 meat flocks, using a CAEV ELISA, 90% of flocks (dairy) and 53% (meat) had ≥1 seropositive animal; 80% dairy goats and 17% of meat goats tested positive for antibodies to CAEV. A Spanish study showed that daily milk yields were higher in seronegative (1.96 L) vs. seropositive (1.77 L) does.

ii. Maedi Visna / OPP in sheep. The virus is found in monocytes in the bone marrow. When those cells mature, they move to tissues and organs and lymphocytes are recruited to try to kill the virus. Damage occurs to those tissues and organs, leading to labored breathing and emaciation caused by progressive pneumonitis. Indurative mastitis, decrease in weaning weights, and premature culling are noted. MVV mechanisms of transmission (most to least likely): respiratory secretions; colostrum (20% of cases); milk; in utero; semen; blood from dirty needles or surgical equipment. There is a long incubation (> 3 years); MVV infects the host for life. Of Ontario flocks, 40% have at least one positive. Typically 20% of the flock is infected. Ontario has a successful voluntary accreditation program.

c. Johne’s Disease (paratuberculosis), infection with *Mycobacterium avium* subspecies *paratuberculosis* (MAP). Sheep can get infected with the sheep (OJD) & cattle (JD) strains, while goats tend to have the cattle strain. This infection is prevalent in all regions, especially in goat herds, resulting in a serious industry problem; however the exact prevalence unknown. Infection causes granulomatous enteritis, leading to malabsorption of nutrients and protein loss. MAP may survive more than 12 months in the environment. Infection is transmitted mainly by adults to lambs via feces (teat/udder contamination), while feeders, bedding, and flock additions are other sources. A slowly progressive disease, there are no signs until at least 2 years of age. Unlike cattle, less than 30% of infected sheep have diarrhea: weight loss is the typical presentation. Signs can be associated with stresses such as those occurring around lambing and kidding. Terminally, one sees emaciation, anemia, and bottle jaw. Pre-mortem tests are not sufficiently reliable to be used in a test-and-cull program; do not rely on a pre-purchase test results from an individual animal to rule out a JD carrier. Tests available include: ELISA; fecal PCR (hspX gene); fecal culture, and histopathology. If suspected in the herd or flock, test 10% or more of the flock using ELISA, fecal PCR, or fecal culture, including the oldest or thinnest animals. You can effectively pool samples (<5 animals per pool). Test accuracy is higher in clinical animals: use ELISA or fecal culture/PCR. Histopathology and culture of the ileum and mesenteric lymph nodes gives a definitive diagnosis.
**The Thin Ewe / Doe Syndrome: Summary of Investigative Steps**

1. Visit farm
2. Examine affected animals
3. Assess BCS in light of production cycle
4. Individual(s) or group affected?
5. Assess environment, facilities & social issues
6. Rule out all non-infections causes of weight loss (social, nutritional, dental)
7. Assess parasite load (fecal) & control measures
8. Submit terminal animals for post-mortem, review historic diagnostic results
9. Perform culture, serology, fecal culture or PCR if indicated
10. Plan corrective interventions
Ewes in late gestation are susceptible to a wide range of disorders and diseases that influence ewe performance and the success of the lambing period. Through a series of steps, veterinarians can assess the management of gestating ewes, and can use this information to encourage improved nutritional and general management. In the best-managed ewe flocks, producers and veterinarians monitor disease and productivity, and set goals; these are the prerequisites for any preventative health program.

**Assessing Flock Performance and Goal Setting**
What should be measured in a commercial meat flock? (goals vary with production system, breed, etc.)

<table>
<thead>
<tr>
<th>Metric</th>
<th>Target</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pregnancy %:</td>
<td>95% of ewes exposed to ram</td>
</tr>
<tr>
<td>Abortion %:</td>
<td>&lt; 5% of pregnant ewes</td>
</tr>
<tr>
<td>Lambing %:</td>
<td>90% of ewes exposed to ram</td>
</tr>
<tr>
<td>Reproductive proportion:</td>
<td>1.5 – 2.5 lambs / exposed ewe / year</td>
</tr>
<tr>
<td>Stillborn %:</td>
<td>&lt; 2% of total lambs born</td>
</tr>
<tr>
<td>Weaned %:</td>
<td>95% of lambs born alive</td>
</tr>
<tr>
<td>Marketed %:</td>
<td>93% of live lambs born alive</td>
</tr>
<tr>
<td>Marketing proportion:</td>
<td>1.2 – 2.3 lambs marketed per ewe per year</td>
</tr>
</tbody>
</table>

Other items that should be monitored include: ewe turnover (should be < 20% per year); ewe mortality (should be < 5% per lambing); lamb average days to market; average age at first breeding; and average ewe body condition at critical times (lambing, mating). Recording and collating the data are the first requirements for comparing flock performance against these industry-based benchmarks. Where there are deviations from these expected targets, assessment of the flock should encompass environmental, nutritional and disease risk factors.

**Advising on Flock Nutrition**
Suboptimal nutrition in gestating ewes is likely to result in a range of metabolic disorders and poor lamb survivability. In assessing nutrition of the flock, consider the breed and its fecundity (Rideau Arcott or Suffolk cross?), parity of the group (are these pregnant ewe lambs or adults?), and the feeds and facilities available. Do the ewes need to maintain or gain condition during gestation? Does the producer employ an accelerated lambing system (e.g., each ewe lambs 3 times in 2 years) or just once-per-year? To assess nutritional management effectively, first one needs to get on the farm.

**Pregnancy diagnosis and Body Condition Scoring**
Pregnancy scanning using real time ultrasound at 45-90 days of gestation permits calculation of pregnancy rate and, when compared to lambing rate, permits an estimation of pregnancy loss. Estimation of fetal numbers permits grouping by litter size and body condition if desired. Finally, it also provides an opportunity for the veterinarian to get on the farm and discuss nutritional and general management of the flock at this critical time in the production cycle.

Body condition at lambing or kidding is critical, and should be between 3.0 and 3.5 on
a scale of 1-5. Over-fat females (>4.0) are susceptible to vaginal prolapse and dystocia. Thin females are at risk of pregnancy toxemia, small weak lambs, and poor colostrum quality. The latter leads to poor passive transfer and loss of lambs to infectious disease in the neonatal period. For accelerated lambing systems, farmers need to fine tune the ration so that the BCS is not less than 2.5 at weaning; there is little time to regain condition before the next breeding cycle because it takes up to 6-8 weeks for ewes to gain 1 body condition unit. Playing catch-up is not effective: excessive feeding of females in late pregnancy to correct sub-optimal body condition will have little effect on BCS, and risks oversized offspring and dystocia.

Feeds and Ration Balancing
The feeds available (energy, protein, mineral) need to be inventoried, and samples of the forages (hay, silage) should be sent to the lab for assessment for nutritional quality. Forage quality is the basis of all ruminant nutrition, and all flocks should have forage tested annually. Ewes rations should be based on 1.5-2 lbs. / day good quality forage. As performance expectations increase, it becomes more critical to obtain good quality forage on which to base the ration: there is a limit as to how much concentrate can compensate for poor quality forage. The ewes will sort through “stemmy” forage and most of the forage may end up as bedding – this should be monitored so true dry matter intake can be estimated.

You can use book values for protein and energy content of grains and supplements. The best reference for sheep and goat nutrition is Nutrient Requirements of Small Ruminants (The National Academies Press, 2007). Major variation occurs in the palatability, protein and energy content of forages. In response to this variation, one must adjust choice of grain and protein supplement. Use the published tables to look up expected dry matter intake for the size and fecundity of ewe group in question, and then calculate the energy requirements for that class of ewe. Feed a grain complement to balance the energy requirements. Finally adjust protein by adding a protein supplement if needed, and check that provision of free choice mineral at least meets label recommendations.

The keys to good management of pastures are fencing, regular moving of the group, and worm control. Pastures should be grazed before they get too long and rank (graze at no higher than 7 inches). If the pasture growth is excessive, recommend that paddocks be dropped out of rotation for forage harvesting so the remainder stay in good growing condition. The best pasture managers use portable electric fences and frequent moves to maintain pastures between 2 and 7 inches length. Growing market lambs indoors removes a major source of parasite larvae from the pastures and makes worm management in the ewe flock easier. Ewes must not be allowed to return each day to the same loafing areas near the home barn; instead, they must be made to fully use the pastures of the entire farm.

Nutritional Problem Solving for Gestating and Lactating Ewes

Pregnancy Toxemia
Pregnancy toxemia is seen in late-pregnant females; affected ewes are initially dull, lag behind, and exhibit grinding of teeth, labored breathing, frequent urination, unsteadiness, walking in circles, or pushing against solid objects. At later stages, they are recumbent. Risk factors include inadequate nutrition during last month of
gestation: insufficient energy density of ration; insufficient protein in ration; multiple fetuses; insufficient feeder space; too fat (≥4.0 BCS) coming into late gestation; environmental stress that stops ewes from eating for a period of time, or inadequate shelter. Grouping for customized nutrition is useful: ewes carrying singles should gain 4 kg; those carrying twins should gain 7.5 kg. Shearing before lambing increases the dry matter intake of the ewe and leads to better lamb survival and growth rates. Early recognition and treatment of pregnancy toxemia is important: affected animals usually respond to 60 cc propylene glycol PO BID followed by improved nutrition.

Vaginal Prolapse
Risk factors for vaginal prolapse include multiple fetuses; poor feeder design (too much crowding); eating on slope; poor hay quality leading to an extra-full rumen; low BCS (<2.0) or high BCS (increased fat in abdomen); phytoestrogens (red clover, 2nd cut alfalfa); genetic predisposition; short docking; older ewes; and hypocalcemia.

Ewe Pre-lamb Vaccination Program
One month prior lambing, all ewes should receive an 8-way clostridial vaccine, primarily to protect lambs from Clostridium perfringens C & D (pulpy kidney) and Clostridium tetani (tetanus). Optionally, where caseous lymphadenitis (CL) is a problem, a Corynebacterium pseudotuberculosis vaccine can be used on the same schedule. All replacement ewe lambs should have a sensitizer and booster before 6 months of age per label instructions.

Infectious Abortion
Our goal is to have an abortion rate < 2%, with a trigger >5%. If there is between 15% and 70% of ewes aborting, clustered in time, this characterizes an abortion “storm” in a naïve flock. If there is between 5% and 10% ewes aborting each lambing period, with first lambers and introductions most affected, this characterizes an endemic infection. This rate of abortion is often accepted as “normal” (but it’s not!). Typical history includes, during the last weeks leading up to expected lambing start date: bloody tails during pregnancy; premature dead or weak lambs; full-term weak or stillborn lambs; macerated and mummified fetuses; abnormal / thickened placenta. An unusual number of non-pregnant ewes (due to reabsorption) may also be part of the syndrome. As examples, Chlamyphilia abortus is transmitted via oral / nasal contact with aborted tissues, infected newborns, and vaginal secretions. In an outbreak, employ isolation of affected ewes and strict disposal of aborted tissues. In contrast, Toxoplasma is excreted in cat feces, especially in kittens. Controlled by limiting kitten population, keeping spayed females only, and keeping cats out of feed. Controlling rodents helps, as they are a reservoir of toxoplasma organisms.

If the typical rate is < 5%, the producer should maintain a closed flock, or, exercise diligence when purchasing, and quarantine new arrivals. If the rate is > 5%, this should trigger a diagnostic work-up. Diagnostic lab submissions should always include placenta. The producer should reduce opportunities for transmission, including to humans. Suppression with antimicrobial drugs (tetracycline) should be considered. Vaccination for Chlamyphilia and Campylobacter is recommended. There are no toxoplasma vaccines in Canada.
Weaned lambs are susceptible to a variety of disorders related to nutritional management. There are several “over-eating” disorders that can affect groups of growing lambs: rumen acidosis (clinical and sub-clinical), polioencephalomalacia (polio), and enterotoxaemia (pulpy kidney), and abomasal bloat. Other diseases with nutritional risk factors include urinary calculi, and copper toxicity.

**Rumen Acidosis**
Many nutritional diseases can be traced to mild to severe rumen acidosis. This disorder is most common in feedlots, but can also occur on pasture. Affected lambs are off feed, bloat, and may have loose manure. More advanced cases are bloated, depressed and recumbent. Rumen acidosis follows accidental engorgement, or a purposeful but too rapid shift to a more energy-dense ration. It may also be associated with animals temporarily kept away from feed (for procedures or weaning); engorgement can occur once these hungry animals are given access to the feed bunk.

A regular feeding schedule should be maintained. Aggressive grain feeding for fast gains can be safely employed (up to 85% concentrate can be fed if good quality hay is available, see Table 1), however all transitions between rations must be gradual. Wheat should be avoided in such rations. Forage intake of growing lambs is often overestimated; they tend to sort through forages and much of the forage may end up as bedding. Bunk space and social issues are important: all animals should be given equal access to a delivered feed, and animals differing greatly in live weight should not be kept in the same feeding group. Initial management of rumen acidosis should include backing off of the “hot” feed, and providing good quality leafy hay. Access to rumen buffers (e.g. free choice bicarbonate) will help protect grain-fed animals from acidosis. Lambs should not be weaned until they are well adapted to creep feed.

**Polioencephalomalacia**
Polio is an acute thiamine deficiency due to over-growth of thiaminase-producing bacteria in rumen. More rarely, it may be induced by high sulfur intake. It is most often caused by a sudden change in diet -- rumen acidosis is an underlying cause (see above). Early in the progression of the disorder, lambs present as for mild rumen acidosis: off feed, mildly bloated, with or without loose manure. More advanced cases are found recumbent and paddling, with opisthotonus. Treatment of animals that are still ambulatory may halt the progression of the disease, but neurologic deficits may persist in survivors. Thiamine hydrochloride administered intramuscularly at 10 mg / kg twice daily for 2-3 days, dexamethasone, and other supportive therapies are indicated. Steps taken to reduce the prevalence of sub-clinical rumen acidosis will reduce the risk of polio.

**Enterotoxemia / Pulpy Kidney**
Lambs affected by pulpy kidney are typically found dead. Death is caused by enterotoxins produced by the proliferation of *C. perfringens* type D. Clostridial spores of *C. perfringens* are ingested but conditions in the normal gut discourage growth and elaboration of toxin. Ingestion of excess fermentable carbohydrate in the ration (grain...
or pasture) leads to changes in pH of the gut, producing an environment suitable for toxin elaboration. The largest lambs with the highest feed intake are often typically the first lambs to succumb. Pulpy kidney is easily preventable with multivalent clostridial vaccines. One month prior lambing, all ewes should receive an 8-way clostridial vaccine, to protect lambs up to 4 months of age. All replacement ewe lambs, and market lambs retained past 5 months of age, should have a sensitizer and booster before 6 months of age per label instructions.

Table 1. Typical lamb rations

<table>
<thead>
<tr>
<th>Lamb Weight</th>
<th>Concentrate Allowance</th>
<th>Concentrate Mix</th>
<th>Target Protein %</th>
</tr>
</thead>
<tbody>
<tr>
<td>65 lbs</td>
<td>2 lbs</td>
<td>3:1</td>
<td>15%</td>
</tr>
<tr>
<td>80 lbs</td>
<td>2.5 lbs</td>
<td>9:1</td>
<td>12%</td>
</tr>
<tr>
<td>110 lbs</td>
<td>3 lbs</td>
<td>grain only</td>
<td>10%</td>
</tr>
</tbody>
</table>

Free choice hay (timothy or similar)
Salt 1% DM intake
Balanced mineral providing 0.5 mg Se /lamb/day
No added copper

Urolithiasis
Urolithiasis (water belly) occurs in male lambs at any time from 2 weeks of age until lambs attain market weight. Several routine management steps can be taken to avoid urolith formation: access to plentiful palatable fresh water; feeding at least 1% salt in diet (DM basis); and ensuring a ration Ca:P of about 2:1. Legume hays (alfalfa, clover, etc.) are good sources of calcium and promote the desired Ca:P ratio. In addition, feeding roughage will increase salivation and rumination, which will increase the amount of phosphate excreted in the urine. Cereal grains (corn, barley, etc.) have low calcium-to-phosphorus ratios: 1:4 to 1:6. Therefore, rations containing cereal grains need to be balanced with other feeds or mineral sources to form a complete ration that has the proper ratio of calcium and phosphorus. Extra calcium is well tolerated by sheep, so where rations are unbalanced, they can be counterbalanced by adding ground limestone (not dicalcium phosphate!).

Free choice salt and minerals may be adequate to prevent urinary calculi in male goats and sheep but intake may vary between animals. However, delivery of mineral in a mixed ration ensures consumption. When feeding textured feeds or mixed rations (e.g. whole grain + pellets), you need to make sure the animals are not picking at certain
feed ingredients. The use of ammonium chloride at a level of 0.5 percent of the total diet will help to acidify the urine and prevent the formation of calculi. Most commercial lamb and meat goat diets contain ammonium chloride, as well as the proper ratio of Ca:P.

**Copper Toxicity**
All breeds are susceptible to Cu toxicity, but some breeds are highly susceptible, Texel being the most common example. The requirements for Cu are estimated to be 7-11 mg Cu/kg DM, however Texel sheep have been poisoned on rations with as little as 14 mg Cu/kg DM, and no other known source of Cu intake. Sheep should get no Cu supplement unless a Cu deficiency has been confirmed by postmortem findings, including estimation of Cu concentration in liver tissue.

Where Cu toxicity has been confirmed using liver Cu concentrations, the next step is to identify the sources of excess Cu. These often include cattle or other livestock feeds not formulated for lambs, mineral licks or supplements with added Cu, or Cu-contaminated pasture (as a result of application of hog manure, for instance).

Cu antagonists can be added to the diet for groups that have been exposed to high copper diets, or groups that appear to be at risk of toxicity on seemingly safe rations. Antagonists can be safely fed for 4-6 weeks; Mo, S, Zn and Fe are employed singly or in combination, and should be formulated in a complete feed under advice of a nutritionist. If fed to excess, or for long periods, Mo toxicity and/or Cu deficiency can occur. For prevention, avoid feeding any rations or minerals not formulated for sheep.
The potential value of hematology and plasma chemistries in equine practice is well documented by numerous case series reports and experimental studies. The maximal use of hematology and chemistries are to help with the diagnosis, clinical management and/or prognosis of the ill or poorly performing horse. In cases where the diagnosis can be arrived at from the history, signalment, and clinical exam, and the prognosis can be accurately predicted, hematology/chemistry testing may not be the best use of the client’s financial resources. During the past few years several point of care instruments (mostly for blood gases, lactate, glucose and chemistries) have become available for use in equine practice. These instruments allow almost immediate stall side results for many analyte measurements. Additionally, at least 3 companies have marketed small, automated bench instruments for equine hematology and chemistry measurements. These instruments have proven reliable and provide quick and valuable laboratory information without using a reference laboratory. The stall side testing and small bench top testing facilitates an improved quality of care for horses and in many practices area sound investment. The information on hematology and plasma/serum chemistries in this handout refers mostly to adult horses. Responses in foals may differ and will be mentioned in the presentation.

Abnormally high values (spurious polycythemia) are most commonly seen in horses with abdominal pain and/or colitis or rhabdomyolysis. The high values are caused by intravascular volume depletion and splenic contraction. A retrospective study on equine abdominal pain found horses with HCT (PCV)> 50% are at increased risk of death. HCT and plasma protein concentration should be used along with clinical findings such as mucus membrane color, heart rate, blood lactate, response to treatment and peritoneal fluid analysis in predicting prognosis for abdominal pain. Monitoring of HCT% and plasma protein concentration is routine for most equine medicine cases and will frequently provide both diagnostic and prognostic information. Along with clinical findings and measurement of BUN, PCV often correlates with intravascular volume depletion and acute dehydration involving loss of extracellular fluids (e.g. diarrhea, sweating, etc.). When the PCV is abnormally high and plasma protein abnormally low, one should consider intestinal disease and dehydration. PCV and plasma protein are minimally affected by water deprivation in healthy horses since most of the water loss is intracellular loss! On rare occasion, horses have absolute polycythemia, most commonly associated with neuroendocrine neoplasia or hepatic disease. Race horses may normally have resting HCT between 35-44% but should be increased immediately following race. A moderately low HCT (21-26%) is most commonly a result of a chronic inflammatory disease. In many cases, plasma protein will be elevated indicating increased globulin production from chronic antigenic stimulation and/or elevated acute phase proteins! If the inflammatory disease involves the bowel (encysted small strongyles or inflammatory bowel disease), the total protein is often abnormally low. Lower hematocrits (<20%) may occur from hemolytic or hemorrhagic disorders. For hemolytic diseases, the HCT%, along with heart rate, clinical signs, pVO₂, blood lactate, and persistence of the hemolytic process
can be used to determine the need for transfusion. There is no single HCT number that serves as a “transfusion trigger” with a range from 9-20%. Low HCT will not be seen with acute hemorrhage; in fact, animals may die from acute hypoxia/hypotension caused by acute hemorrhage but have a normal HCT. Regarding crystalloid therapy in the anemic patient, this therapy is indicated in most horses if there is evidence of dehydration/poor perfusion. Although the fluid therapy will decrease the PCV, it will not decrease the total number of RBCs and may improve oxygen delivery! Severe nonregenerative anemias are rarely seen, but may result from adverse reaction to erythropoietin injections (red cell aplasia), Fell pony syndrome, and rarely from cytotoxic drug reactions or neoplasia. MCV would be expected to be low in these cases. When blood is spun in a micro hematocrit tube, the plasma should always be examined for icterus, hemolysis and lipemia! Lipemia is common in sick ponies and miniature horses and can be rapidly fatal due to hepatic lipidosis. Myoglobin, due to its small size and rapid filtration in the glomerulus rarely causes discolored plasma. The size of the buffy coat can provide an idea of the white blood cell count.

Platelet counts can help determine the severity of an illness and can provide a laboratory clue for neoplasia. Thrombocytopenia can be a laboratory clue for diseases such as anaplasmosis! Horses with severe systemic inflammation/coagulopathy may have thrombocytopenia (usually in the 40-70,000 range), increased D-dimers and low anti-thrombin III levels; fibrinogen is often normal or sometimes even high. Horses with marked thrombocytopenia (< 20,000) usually have drug-induced or neoplasia related (immune) thrombocytopenia. In the horse, another cause of “reportedly” low platelet count is pseudothrombocytopenia which is a result of platelet clumping and can be seen on a blood smear. If the platelet count is low and there is no clinical evidence to support the finding, submission of a sample in a citrate tube is recommended. Some of the small bench analyzers may underestimate platelet counts.

The neutrophil count and morphology are one of the most valuable laboratory tests in equine internal medicine. High neutrophil counts (neutrophilia) can be used, along with acute phase protein measurements, (e.g., high serum amyloid, and low serum iron and high fibrinogen and globulin concentrations) to help determine if an inflammatory disease is present and, to some extent, the duration of the inflammatory response. Common causes of a mature neutrophilia include inflammatory diseases without endotoxemia or severe systemic inflammation, excitement and either physiologic stress or corticosteroid administration. If band neutrophils and/or toxic changes are present, this is highly suggestive of a bacterial infection and supports some degree of systemic inflammation. One mechanism for this is the causative relationship between sepsis, increased TNF, and early release of neutrophils into circulation. Increased adhesion activity associated with sepsis results in margination of neutrophils and resulting neutropenia. Although there is no evidence-based publication to confirm such, this author believes that most horses that develop laminitis secondary to systemic inflammatory illness undergo either a “left shift”, and/or have toxic changes in neutrophils 12-36 hours prior to the clinical signs. Neutropenia with a left shift and/or toxic changes is frequently observed in acute bacterial infections and/or endotoxemia. In horses with acute colitis and/or severe systemic inflammation, a change from neutropenia to neutrophilia is commonly observed during the first 3-4 days of the illness. Although this is generally thought to be a favorable response (less margination of neutrophils and less systemic
Inflammatory mediators), I am not aware of published data to support this. Persistent neutropenia may be seen in some horses in association with drug administration, (e.g., TMP-S, NSAIDs). Acute viral infections may cause severe neutropenia with variable lymphocyte counts, but left shift and toxic changes are usually absent. Lymphocyte, monocyte, and eosinophil counts are of less value to the equine practitioner although some diseases (e.g. EHV-5) associated pulmonary fibrosis have a consistent lymphopenia (along with elevated fibrinogen). Foals with EHV-1 infection are persistently leucopenia (low number of both neutrophils and lymphocytes). Premature foals with normal or high neutrophil counts generally have a better prognosis than premature foals with neutropenia. It should be noted that some healthy foals have neutrophil counts below the normal range for that age foal. Remember most normal laboratory ranges are age sensitive and based upon 95% of the normal population being within that range. On that rare occasion when eosinophilia is present, diseases of either the skin or intestines should be considered.

An important analyte that should be measured when inflammation is suspected is plasma fibrinogen. Plasma fibrinogen is an acute phase protein and a component of the coagulation pathway. Increases in fibrinogen may occur with either local or systemic inflammatory responses with elevations occurring as quickly as 24-36 hours. Although this measurement is of clinical value, other measurements, such as abnormal neutrophil numbers, low serum iron, and increased serum amyloid occur more quickly following inflammation and should be used along with plasma fibrinogen in determining presence or absence of inflammation and response to therapy. Fibrinogen can be measured by automated system using the VetScan VSpro in less than 15 minutes and is portable for out of the hospital use. It can also measure prothrombin time (PT) and partial thromboplastin time (PTT). Another method for measuring fibrinogen is using microhematocrit tubes and using the refractometer. Two tubes of the same blood sample are tested: one after spinning; and the other after spinning, heating for 2 minutes at 58° C, and spinning again. The difference between the two is the fibrinogen. Example: If the first tube reads 7.2 g/dL and the heated tube reads 7.0 g/dL, this equates to 200 mg/dL of fibrinogen. Certain diseases tend to have very high plasma fibrinogen concentrations, (e.g. abdominal abscess, septic physitis). Total plasma protein is an important measurement in clinical practice. Horses with inflammation, either acute or chronic, often have elevations in plasma protein with the greatest elevations occurring in horses with chronic infection (abscission). Abnormally low total protein concentration is most commonly associated with enteric loss or hemorrhage (PCV should be decreased concomitantly when there is subacute or chronic hemorrhage). Protein-losing nephropathy and “third spacing” of protein, (peritonitis) is rare in the horse. Additionally, liver disease in the horse rarely causes low total protein; albumin may be decreased, but this is often offset by increased globulins! The plasma protein concentration can be used as a guide to when colloid therapy is needed. In critically ill adult horses, P.P. < 4.5 may be an indication for colloid therapy?

Serum or plasma chemistries are used to detect electrolyte abnormalities, organ system disease, inflammatory markers, and some metabolic diseases. Hyponatremia is common with diarrheal and some urinary system diseases. The finding of hyponatremia, hypochloremia and neutropenia in a colicky/febrile horse or foal is suggestive of colitis/enteritis. The degree of hyponatremia in foals is important to
establish as too rapid correction of the hyponatremia may result in neurologic signs and irreversible CNS disease. This author believes that too rapid correction of hyponatremia should be considered but the condition may not be as common in foals as is reported in children. I don’t hesitate to increase the Na 10-15 mEq/L in a couple of hours if the foal is severely dehydrated and the hyponatremia is believed to be of less than 3 days duration. Hyponatremia and hypochloremia are also present with acute or chronic renal failure, ruptured bladder and severe myopathy/myositis. These finding may also be present with severe edema, peritonitis and or body cavity effusion. Another cause of marked hyponatremia and hypochloremia in foals < 10 days of age is hydroureter; an interesting and as yet unpublished syndrome. With ruptured bladder (in foals) and severe muscle disease, the serum potassium is often elevated and in foals with ruptured bladder it may be a serious management problem. With acute or chronic renal failure plasma potassium concentration is variable, generally either normal or high. When it is high and does not drop with fluid therapy (most fluid therapy causes a drop in K concentration), oliguric or anuric renal failure should be considered. Of course, another cause of hyperkalemia in Quarter horses is HYPP. The “dash board” effect on chemistries is not as dramatic in horses as some other species with glucose (decreasing) being the only consistently dramatic finding when samples are carried in the truck all day before submission. Increases in K and phosphorus may occur when the sample is hemolyzed. Low potassium is most common in foal diarrhea, often reaching a life threatening level! Although anorexic horses may maintain normal serum potassium, their total body potassium is always low. Only 1.5% of body potassium is in plasma and for every 1 mEq/l decline in K below normal range, a 10-15% loss of total body K (this could be equal to 225 grams for a 500 kg horse) should be considered! Large K deficits are best replaced by oral administration. Hyponatremia is rarely a problem in equine medicine except in the treatment of neonatal foals with high Na containing fluids. Both sick and healthy foals maintain higher levels of aldosterone than do adult horses and even prolonged use of plasmalyte (Na=140 mEq/L) etc. may cause hypernatremia in foals. I therefore rarely use hypertonic saline in neonatal foals.

Bicarbonate measurement is very useful in determining the metabolic acid/base status of the horse or foal and the need for either enhanced fluid therapy (perfusion) or less commonly bicarbonate administration. Low bicarbonate is by definition a metabolic acidosis (if the pH is also low then there is a metabolic acidosis with acidaemia). The most common cause of a metabolic acidosis is titration of HCO3 by lactic acid or other strong anions that are not routinely measured. L-lactic acid can be indirectly measured by measuring L-lactate. The point of care measurement of lactate is of great value in the immediate evaluation of critical care horses and foals. The magnitude of the elevation in lactate is not as important prognostically as the lactate measurement following aggressive treatment, (if lactate concentration does not go down following resuscitation with fluids etc. then the prognosis is often guarded or worse). A lactate value above 6.0 mEq/l has been associated with outcome in horses with large colon volvulus. Other strong anions such as sulfates ( most commonly elevated by renal dysfunction) may also accumulations and can cause a metabolic acidosis. The anion gap [(sodium + potassium)- (chloride + bicarbonate)] is usually elevated (normal, 15 mEq/L) if there is an increase in lactate or sulfates and this magnitude often gives us a clue as to the severity of the disease and the required therapy. The anion gap may not be increased in spite of increased in unmeasured anions if albumin values are low such
as commonly occurs in horses with colitis! Hypoalbuminemia can cause a mixed metabolic acidosis (increased lactic acid/lactate) and mild metabolic alkalosis (increased bicarbonate) with a normal calculated anion gap. The important clinical message is fluids (crystalloids and ideally colloids) are still needed to combat both the lactic acidosis (poor perfusion most likely) and the hypoalbuminemia in those horses. Extremely severe and sometimes persistent metabolic acidosis is most common in foals with diarrhea. Many of these cases require bicarbonate therapy in order to correct the acidosis suggesting excessive loss of bicarbonate or d-lactate production rather than titration with L lactic acid. Metabolic acidosis may also be caused by excessive administration of chloride. When administering bicarbonate to foals, serum potassium must be monitored very carefully!! A common cause of hyperchloremic metabolic acidosis with a normal anion gap is large volume of NaCl treatment. Hypochloremic metabolic alkalosis with elevated anion gap is common with excessive sweating, (e.g., myopathy, exhaustion), diarrhea and renal failure; severe cases may develop metabolic acidosis. Bicarbonate should rarely if ever be administered to these horses. Some knowledge of the importance of strong ions (Na, K, Cl, albumin) is important for understanding metabolic acidosis/alkalosis but I personally do not believe calculation of a strong ion difference is necessary in clinical cases.

The most common causes of decreased plasma calcium are hypoproteinemia, acute gastrointestinal disorders and/or endotoxemia. Ionized calcium is often normal in horses with low albumin and in most cases no calcium therapy is recommended. Ionized calcium can be quickly measured using an I-STAT. With acute gastrointestinal disease and low ionized calcium treatment with diluted calcium may be used in hopes of improving intestinal motility and cardiac contractility. Lactation tetany, synchronous diaphragmatic flutter and tetany associated with acute gastrointestinal disease should be treated with both calcium and magnesium as both are low in these cases. Magnesium and phosphorus are commonly abnormal in sick horses but their importance in many cases is not known. Low serum magnesium is common in many horses with systemic inflammatory disease unless they are severely azotemic. I generally do not treat with magnesium unless there are muscle tremors etc. Hypermagnesemia may occur from administration of excessive magnesium sulfate to azotemic (dehydrated) horses in which case paralysis can occur. Phosphorus is often very low in critically ill horses but specific treatment is rarely given.

Blood glucose determination, measured within 1 hour or on separated plasma samples should be routine in sick foals as hypoglycemia is common. Although sick horses (other than foals) rarely have hypoglycemia, they may have a nutritional need for glucose supplementation, especially pregnant mares! High blood glucose is common in “colicky” horses and horses with primary hyperammononemia (where glucose of >250 mg/dl, lactate > 10 mEq/L and correspondingly low bicarbonate are expected). There is some correlation between magnitude of the hyperglycemia horses with colic and outcome. Persistently high blood glucose is also present in many horses with Cushing syndrome, but is rare with equine metabolic syndrome unless they have received grain/molasses within the hour! Horses with hyperlipidemia syndrome and triglycerides > 1500 mg/dl have been reported to have a poor prognosis although I have found that it is not as important how high the triglycerides are initially but instead, do they decrease significantly with the initial treatments? This concept is similar to using blood lactate or cardiac troponin I as prognostic measurements in
horses with shock. In some cases the triglycerides decrease remarkably fast and the miniature horses/ponies quickly recovers! I have seen ponies go from > 2000 mg/dl to less than 100 within 24 hours. Fasting plasma insulin and seasonal ACTH concentrations have been excellent predictors of metabolic diseases in the horse. Plasma leptin might be a future test to add to the metabolic profile testing. Dynamic challenge test for both Cushing’s disease and equine metabolic syndrome are thought to be better than baseline testing.

Muscle disease is best detected by elevations in CK and/or AST (if it has been 2-7 days since the possible muscle disturbance. Poor performing horses, especially fillies, that have abnormally high AST as the only abnormality on a full chemistry panel should be strongly considered to have a myopathy. Horses suspected to have chronic intermittent rhabdomyolysis should be jogged for 10-15 minutes and a CK sample taken 3 hours later to help confirm the myopathy. QHs with PSSM often have very high CK and AST when clinically affected, while draft horses and Warmbloods may have only modest elevations in some cases. Horses with PSSM often have resting CK and AST in high normal or slightly increased range.

Renal function tests include BUN and creatinine. Differentiating prerenal azotemia from primary renal dysfunction is best done by combining history, clinical exam, PCV/ P.P. and other chemistry values, urinalysis (specific gravity) and speed of return to normal values following fluid therapy. The BUN:creatinine ratio may also provide some information that is helpful in separating pre-renal azotemia from primary renal failure. Thoroughbreds generally have lower creatinine concentration than Quarter Horses or Warmbloods, and this should be considered when evaluating renal function, particularly if the horse is being treated with nephrotoxic drugs; ie. a creatinine of 1.8 mg/dl (still within published normal range for the horse) in a Thoroughbred being treated with an aminoglycosides might be highly significant (decrease in GFR and toxicity). Marked elevations in serum creatinine in a horse with a severe myopathy is a result of a decreased GFR and not abnormal release of creatinine from damaged muscles. Newborn foals with serum/plasma creatinine values > 2.2 mg/dl generally suggest that placentitis was present!

Liver disease is detected by measuring liver enzymes in the serum. These enzymes may be from hepatocellular origin (SDH, AST, GLDH) or biliary (GGT, ALP) in origin and are often helpful in determining the location of most severe pathology in the liver. Consideration of half-life and liver specificity of the enzymes should also be duly noted in the evaluation. GGT is the most sensitive enzyme for detecting serious liver disease in horses but it test disease and not function. Its decline often lags behind the other enzymes during recovery from hepatic injury and greatest elevations occur with biliary diseases. ALP is of limited value in the horse, commonly increased above reference adult values in growing foals and in a variety of organ disorders in all ages. Liver function test include direct bilirubin concentration, indirect bilirubin (not specific for liver as hemolysis and fasting also cause elevations), prothrombin and partial thromboplastin times, ammonia, urea, fibrinogen and total bile acids. Bile acids are increased prior to the other function test in the progression of most equine hepatic diseases. Bile acids are increased (10-22 µmols/l) due to anorexia and some normal foals < 6 weeks of age have higher concentrations than the normal adult range. With chronic non-infectious liver disease, finding bile acid values greater than 30 umol/l is a
poor prognostic finding. Only serum bile acids and blood ammonia are elevated in the rare cases of portosystemic shunts in a foal. Measurement of blood ammonia is important in horses with liver failure, hyperammonemia associated with gastrointestinal disease or in some Morgan foals with acute cerebral signs and in some foals with meconium impaction. To my knowledge, only one of the veterinary bench machines measures ammonia; we use that equipment to measure approx. 40 samples per year. If blood is separated immediately after collection and plasma (EDTA or heparin)/serum frozen the blood ammonia can be measured at an outside laboratory the following day. Blood ammonia increases if left at room temperature.

Elevations in GGT without other hepatic enzyme/function abnormalities are also associated with poor performance in race horses and some have speculated this relationship is related to overtraining. GGT elevations do not occur with pancreatic disease; serum and peritoneal fluid amylase and lipase are increased with acute pancreatitis.

POINT-OF-CARE DIAGNOSTICS

**Definition:** “Point-of-care testing” is diagnostic testing performed at or near the patient. These analyses are important and, in many cases, essential in evaluating the emergency or critical care equine patient. Point-of-care devices are oftentimes portable and are used patient-side in a hospital setting or on the farm. **Practice Tip:** The primary advantage of point-of-care testing is the ability to obtain immediate results, allowing adjustments in patient treatment and minimizing the need to send and await sample results from a clinical pathology laboratory. Most tests must be conducted at temperatures of approximately 18° to 30° C (64° to 86° F). Although this temperature range is recommended, samples tested at temperatures as low as 50° did not result in noticeable erroneous values.

**Blood Gases and Blood Chemistries**
The I-STAT system has been extensively used during the past several years with accurate and uniform results on blood sodium, potassium, chloride, creatinine, BUN, ionized calcium, glucose, acid-base measurements and troponin. Hct is sometimes falsely low.

**Glucose**
A veterinary glucometer, AlphaTRAK, is validated for healthy and critically ill horses and foals. This glucometer uses 0.3 μL of whole blood with results in 25 seconds. Assure Chronimed, Inc. and Accu-Chem are two other instruments used to measure blood glucose. These test instruments appear to be accurate at predicting severity of hypoglycemia but may not have the same accuracy in reporting the level of hyperglycemia. Glucose also can be measured with the i-STAT.

**Lactate**
Increases in blood lactate are used in equine critical care as a marker of disease severity and prognosis.

Lactate measurement can be performed with a point-of-care device – Accutrend - validated in critically ill horses. This lactate meter uses less than 20 μL of whole blood or plasma and provides results within 60 seconds.
Lactate can also be measured quickly (1 minute), accurately, and inexpensively using the Lactate Pro.

Whole blood or cellular samples (i.e., peritoneal fluid) should be measured within 30 minutes, or the lactate may increase from sample storage. It is recommended that plasma lactate be used more reliably to evaluate trends in serial lactate measurements.

Transient elevations in lactate are commonly seen in early life in neonatal foals; however, decreases to adult levels are expected by 24 to 72 hours.

_Elevations in lactate are present in most critically ill horses; the initial value and the change in lactate concentration following appropriate treatment/resuscitation can both indicate prognosis!_

**Cardiac Troponin**

Elevation of cardiac specific troponin I (cTn-I) is an indicator of myocardial injury, and changes in the protein provide diagnostic and prognostic information. Cardiac injury in horses can be secondary to: myocarditis, sepsis, rattlesnake envenomation, cantharidin intoxication and other causes of multi organ system failure.

**Serum amyloid A**

Serum amyloid A (SAA): An acute phase hepatic derived protein can be measured at referral laboratories or with point of care instruments awaiting further validation.

The advantages of SAA are:

- Very low values in normal horses/foals (0 to 30 mg/L).
- Dramatic elevations (often 100×) within 6 hours, peaking at 24 to 36 hours after acute local or systemic inflammation.
- Samples are very stable at room temperature or when refrigerated for several days.

SAA increases with tissue trauma (e.g., postsurgery), but SAA is not increased with recurrent airway obstruction (RAO).

Levels in joint septic joint fluid are higher than non-septic inflammatory joint fluid SAA does not appear to be useful as a screening test for _R. equi_.

_There are some clinical pathologic differences between foals and adult horses and between horses and other equines; the reader is referred to the references below for more information on age and species differences._

NEWER DISCOVERIES IN EQUINE GASTROENTEROLOGY

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One of the newer discoveries in equine acute gastrointestinal disorders has been the increase in reports of coronavirus disease in horses. All risk factors are not know but season (cooler) is one, duration of fecal shedding (there is some nasal shedding) is generally 2 weeks, and methods of spread is believed to be fecal. What is apparent is that adult horses are commonly affected in many areas of the United States. Broodmares are less likely to be affected possibly because of a high incidence of non-clinical coronavirus in foals? Anorexia, fever, and leukopenia are pronounced. Colic and diarrhea are sporadic or rare. Although there are isolated cases on a farm, outbreaks are common. Diagnosis is by clinical signs and PCR testing of feces. This beta-coronavirus has at least two different strains both of which are detected by the currently available PCR. Serologic testing has been performed on herd outbreaks. Treatment is mostly supportive fluids, antipyretics that have low intestinal toxicity, gastric protectants , when appropriate. Mortality is low but 3 to 5 days may lapse before the horses begin to eat. On rare occasion, an infected horse with diarrhea may die of fatal neurologic signs (intestinal hyperammonemia). It might therefore be prudent to monitor blood ammonia in affected horses and if levels approach abnormal range; treatment with lactulose and or neomycin might be indicated.

Another recent finding in horses with acute colitis has been the association of Clostridium perfringens type C and the disorder. This disease caused by the beta toxin (CPB) of the organism has been well-described in young foals (<7 days of age) but only recently reported in older equines. Prior antibiotic therapy does not appear to be a risk factor.

Diagnosis is by ruling out other causes of enteritis/colitis, culturing the feces anaerobically and testing for Clostridium perfringens isolates tested by Multiplex PCR for Clostridium perfringens toxin genes. A commercial capture ELISA can also be used on the feces of horses and appears to be sensitive. Diffuse or more commonly segmental red lesions occur in the bowel. Small intestinal lesions are most common distal to the duodenum. Small intestinal contents are red-brown while large bowel contents in adult horses were not hemorrhagic. Mucosa necrosis is commonly full thickness with submucosal thrombosis. Treatment would presumably include intravenous penicillin, orally administered metronidazole, and Clostridium perfringens type C antitoxin given slowly and carefully IV (after low-dose Banamine) or subcutaneously and orally. Supportive therapy for septic shock would be needed. As with any infectious and inflammatory colitis, the use of NSAIDs could be detrimental to intestinal repair, while the use of misoprostol might have beneficial effects. A role for type A Clostridium perfringens in acute hemorrhagic and necrotizing enterocolitis of neonatal foals and sometimes horses has long been suspected but incompletely characterized. It has recently been suggested that type A C. perfringens produces both enterotoxin (CPE) and pore-forming toxins such as netF that may synergistically cause severe diarrhea in very young foals. A beta-2 toxin (CPB2) has been incriminated in adult horses with some association of diarrhea, CPB2 and the use of gentamicin. CPB2 may be commonly found in healthy foals.
Clostridium difficile continues to be a problematic cause of acute severe colitis and toxemia mostly in adult horses receiving antibiotics. Clostridium difficile diarrhea in adult horses is often peracute and may be fatal even with early intensive therapy. Gas distention of the colon and ileus are common findings. A low number of healthy adult horses may harbor the organism (pathogen or non-pathogenic). The disease in young foals, which may be more likely to harbor the organism than are adults, is not well understood. Newborn foals commonly develop Clostridium difficile diarrhea without any history of antibiotic therapy. Is it possible that an unestablished microbiome is the predisposing factor to Clostridium difficile diarrhea in neonatal foals? Metronidazole remains the specific antimicrobial treatment of choice in most cases. In foals metronidazole should be dosed 10 mg/kg q 8hrs due to delayed elimination of the drug in foals. A hyperimmune plasma product, from horses, was shown to lessen disease in experimentally-infected rats.

Perhaps the most excitement in equine gastroenterology has been the research into the equine microbiome and transformation therapy for Clostridium difficile, antibiotic associated dysbiosis and many other infectious causes of colitis. Dramatic changes in the faecal microbiota of diarrheic horses can be a result of microbiome dysbiosis or overgrowth of equine enteric pathogens. Use of metagenomics has shown significant changes in the microbiome following feed withdrawal, anorexia, change in feed, and antibiotic therapy. In our hospital we have been using fecal or cecal transformation in the above listed cases for three years with what we believe is clinical success (manure can return to normal consistency within 14 to 24 hours in some cases). We prefer to use cecal contents from a healthy orthopedic research horse that is scheduled for elective euthanasia and has been fed a roughage diet. A negative fecal for Salmonella is required. Cecal contents can be given immediately after collection or can be frozen for future use. The cecal contents are strained through a metal strainer prior to administration or freezing. Generally, 1 to 2 quarts of content is given via nasogastric tube every 12 to 24 hours for 1 to 3 treatments. Horses that are being transfaunaed are treated with omeprazole for at least 24 hours prior to the initial transfaunaation. If cecal (fresh or frozen) are not available, we use fecal contents taken from the rectum by palpation. A large draft horse, tested every two weeks for Salmonella, and used as our resident blood donor, is our choice for fecal donor. Changes in faecal microbiota prior to foaling preceded and appeared to be associated with post foaling colic. Associations between Firmicutes and Proteobacteria and development of colic were noted but more work is required to determine how or if we can “correct” the fecal microbiome. An increase in nonesterified free fatty acids prior to and just after foaling and a decrease in ionized calcium 2-4 weeks postpartum were also associated with postpartum colic in mares. Sorting our cause and effect in that study is difficult.

Newer findings in the diagnosis and treatment of abdominal pain in horses include the ability to diagnose right sided colon displacements with ultrasound (in addition to history and clinical examination), and data showing medical treatments are extremely successful in left colon displacement and moderately successful for left colon displacements to right. The point of care for testing of lactate in both blood and peritoneal fluid and in some degree cTnI in blood is the decision for surgery in horses with abdominal pain. The decision as to the use of perioperative and post-operative antibiotics in horses undergoing colic surgery has not been resolved but a general
consensus may be perioperative antibiotics with immediate discontinuation of antibiotics in some cases if the surgery is deemed “clean surgery”. The use of Buscopan® has now expanded to its use in meconium impactions in foals. Esophageal and gastric motility disturbances are frequent in Friesian horses and most equine veterinarians immediately consider genetic motility disturbance in horses of this breed that are examined for esophageal choke, gastric impactions or colic. Friesians and draft horses are predisposed to primary gastric impactions. Horses with recurrent nephrosplenic entrapment of the large colon could be treated with Mesh or suture obliteration of the nephrosplenic space to prevent recurrence. Medical treatments or rolling are highly effective in treating the disorder; phenylephrine should be used cautiously or not at all in older horses. Horses that have colic surgery and then need to undergo a second laparotomy (mostly due to persistent gastric reflux/ileus) have a low survival rate, and for those that do survive a high incisional infection and hernia rate.

The overall prognosis for long-term survival in horses with a presumptive diagnosis of inflammatory bowel disease (IBD) appears to be fair, and the initial response to anthelmintic and systemically administered corticosteroid therapy could be a useful prognostic indicator. A markedly low peak xylose (or glucose) concentration in absorption testing may be associated with a less favorable prognosis, supporting the continued use of this test. Ultrasound findings that strongly suggest infiltrative bowel disease are focal or diffuse thickness of the small intestine of 5 mm or more. An interesting talk by David Freeman at the AAEP suggested that when horses are off feed (decreased gut fill) their volume of IV fluids to maintain hydration may not be a great as once thought (monitor urine S.G); this would likely not be true if there is excessive loss through reflux or diarrhea.

Lastly the discovery of equine hepatitis viruses may explain Theiler’s disease and many other liver diseases in the horse. Hopefully Theiler’s disease will soon be a disease of the past!

**Further Reading:**
NEUROLOGIC DISORDERS IN HORSES – WHAT IS YOUR DIAGNOSIS?

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TEMPOROHYOID OSTEOARTHROPATHY
Temporohyoid osteoarthropathy is a relatively common neurologic disorder in the horse. Since 2000, we have examined 26 horses with this condition. The etiology of this arthritic disease is unknown and although previous otitis interna/media is generally blamed there only limited evidence to support this hypothesis. Only a small number of cases have active infection (mostly gram positive cocci and one case Aspergillus) of the inner ear area. The disease is most common in middle age horses, youngest that we have examined being 4 years of age (rarely younger horses have been reported) , and approximately 40% have bilateral disease, although the clinical signs are almost always unilateral. This may very well be an osteoarthritic disease in the horse with risk factors such as cribbing, prior infection etc. The signs are variable with some horses having behavioral signs associated with being ridden or while eating; these are likely signs of pain. Acute onset of neurologic signs are believed to occur when the fused T-H joint fractures. Fractures may be associated with sudden movements of the head and have sometimes been reported in horses following passage of a nasogastric or after dental procedures. Neurologic signs are those associated with cranial nerve VII and/or VIII dysfunction. Diagnosis is made by clinical signs, endoscopy of the gullet pouch, radiographs or computed tomography. CSF can be inflammatory in approximately 25% of cases and in a rare case, septic (consider Staphylococcus aureus). Treatment goals are directed towards decreasing inflammation and infection, if present, and preventing complications, of which exposure keratitis due to facial nerve paralysis is perhaps the greatest complication! Approximately 50% of the horses return to use with medical treatment (anti-inflammatorys and antibiotics). Unfortunately, we are not able to predict clinical response at the time of the initial diagnosis! Some horses may require many months for nerve function to return. We have routinely recommended a ceratohyoidectomy for both patient comfort, to prevent further fractures at the fused T-H joint(s) and improved recovery times. Most horses are deaf on the side of the lesion and this is likely to persist even with apparent clinical recovery.

BOTULISM
Botulism in horses seems to have an increase in incidence associated with the more common practice of feeding “round bales” of hay. Diffuse neuromuscular weakness occurs in all cases with dysphagia being present in most cases of type B botulism which is most common in the Northeast U.S. If a dead animal is in the feed type C may occur and type A has been associated with moldy grass clipping in the west. In the adult horse the disease is caused by ingesting the preformed toxin in decaying forage and rarely is it caused by wound contamination. Botulism in 3-8 week old foals due to ingestion of the organism and sporulation in the bowel lumen, “shaker foal syndrome” is common in those areas that also observe type B botulism in adults. Antitoxin should be administered early in the course of the disease, feeding and fluids should be administered via nasogastric tube and antibiotics (not aminoglycosides, procaine penicillin or tetracycline) administered as treatment for aspiration pneumonia. Adult horses that are recumbent and cannot rise have an extremely low prognosis.
EQUINE PROTOZOAL MYELITIS

I think we would all agree that equine protozoal myelitis (EPM) has been for the North and South American equine practitioner a difficult and contentious disease. Our ability to properly diagnose the disease has not been good and many questions remain regarding proper treatment. Only with an understanding of epidemiology of the disease, diagnostic tests, pharmacokinetics and pharmacodynamics of the anti/protozoal medications and from clinical experience can we make the most educated recommendations for treatment and prevention of the disease.

The diagnosis of EPM is based upon knowledge of: prevalence of the disease in a geographic area, the most common clinical signs, risk factors for the disease, awareness of sensitivity and specificity of diagnostic tests, in addition to ruling out other disorders and response to treatment. The disease should only exist in horses exposed to opossums which are in an area from lower Maine to southern Florida on the east coast. Racing and show horses are at greatest risk. There is no proven breed predisposition, although Standardbreds were reportedly more common in 2 studies. The disease incidence peaks at 4-6 horse years of age and rarely affects horses less than 1 year of age. Infection and clinical diagnosis is less common in the winter months in the more northern states. Adverse events and shipping within last 60 days appear to be additional risk factors. The clinical signs include ataxia in 95% of cases, muscle atrophy in approximately 40% of cases, cranial nerve deficits in less than 12% of cases, seizure in 6% and some mild change in mentation in many cases. The ataxia is mostly tetra ataxia (70%), hind limb ataxia 20% with a very low percentage having coccygeal signs. Ataxia and/or atrophy is asymmetrical in approximately 69% of cases. Cranial nerve dysfunction is asymmetrical in virtually all of the cases with frequency of lesions being greatest in cranial nerve 7 followed by 5, 8, 12, 9, 3 in that order. The most common and confusing differential for EPM is cervical compressive myelopathy (CCM). An understanding of risk factors, clinical signs and diagnostic tests for CCM (see below) are helpful in distinguishing EPM from CCM. The SAG 2, 3/4 ELISA assay has improved diagnostic testing for the disease but only if both serum and CSF are tested. I would recommend using historical information, clinical impression (including response to therapy) with ELISA 2,3-4 serum:CSF ratio testing for diagnosis. The only U.S. Food and Drug Administration approved and available drugs for treating EPM are Ponazuril (Marquis®), Diclazuril (Protazi®), ReBalance® (pyrimethamine /sulfadiazine, PRN Pharmacal). Both ponazuril and diclazuril are triazine anti/protozoal agents effective in-vitro and in-vivo against a large number of apicomplexa organisms. These drugs have a long T ½ requiring several days to reach maximum serum and CSF concentration, both are static drugs and have low CSF to serum ratio (1:20). An inadequate dose of ponazuril may explain some poor responses and an estimated relapse rate of 9%. Alternatives to triazine treatments include the addition of pyrimethamine, a cidal anti/protozoal drug with better CSF to serum ratio (>25%) but lower bioavailability and higher toxicity and decoquinate, a 4-hydroxyquinoline, that is not Food and Drug Administration approved. Prognosis is somewhat related to: severity of the clinical disease, early and aggressive treatment and of course having the correct diagnosis. Prevention should focus on decreasing exposure to opossum feces (closed feed containers, protected hay storage). In endemic areas intermittent treatment during high risk periods are used. Racing and western performance horses have been found to be at increased risk of EPM. Intermittent treatment of at risk horses has been
evaluated as a means of preventing CNS infection with *S. neurona*. Administration of dicyclazuril daily does have fair to good efficacy in preventing infection. Although a relatively uncommon cause of protozoal myelitis in comparison to *S. neurona*, *Neospora hughesi* should also be considered.

**CERVICAL COMPRESSIVE MYELOPATHY**

Diagnosis of cervical vertebral compressive myelopathy (CVM), also called cervical stenotic myelopathy, continues to be an area of investigation. Intra-vertebral ratios on plain cervical films has proven to be the screening test of choice for most clinics, but sensitivity and specificity of the test are variable between clinics/radiologist reading the films, and there is poor correlation between the site of the abnormal ratio and the lesion site. Since the site of cord compression is often inter-vertebral due to cystic, soft tissue or instability compression, rather than intra-vertebral in horses, many cases with CVM will be not diagnosed by intra-vertebral ratios. We have even questioned the accuracy of the 50% reduction in the dorsal myelographic column for diagnosis of CVM. Accuracy of this criteria seems to vary with vertebral site, C5-C7 being most accurate. Measurement of the reduction in dural diameter at the inter-vertebral sites in comparison to adjacent mid-vertebral body dural diameter might be more accurate for compression in the mid-cervical region. For horses >3 years of age, Levine has found older Warmbloods to be at highest risk of CVM. Risk factors including breed predisposition and clinical signs for CVM have recently been reviewed. In addition a large multi-center study looking at the signalment and diagnostic features of CVM has been recently completed. Horses with CVM were younger (median 2 years) than control horses. Thoroughbred, Warmblood, and Tennessee Walking Horses were significantly over-represented in the CVM group. Horses with a history of racing or English performance were significantly more likely to be affected than those used for Western/pleasure activities. Asymmetric gait deficits (71/166) and cervical hyperesthesia (44/91) were common in CVM. The most frequent necropsy lesions were stenosis and articular process osteophytosis. Agreement between radiography and necropsy was between 65% and 71%. Agreement between myelography and necropsy was between 67% and 78%. The agreement between findings of necropsy and imaging was only moderate, indicating that accurate diagnosis with these techniques can be challenging. One of the more interesting finding in equine neurology is that horses with cervical facet osteoarthritis may have stumbling gaits without involvement of the spinal cord or at least the myelogram is considered normal (could be lateral compression). A fair percentage of these horses have improved gaits following facet injection with corticosteroids. Cervical pain in horses has become a common diagnosis for an abnormal stiff, stumbling gait and cervical facet injections with corticosteroids have allowed return to function in many horses.

**EQUINE HERPESVIRUS-1**

Equine herpesvirus-1 (EHV-1) is, for multiple reasons, a problematic infectious disease in the horse. EHV-1 may cause several different clinical syndromes, some of which result in epidemics of disease following the arrival of an infected horse on the premise. The virus can also be maintained in carrier animals, which may then result in either self-disease (i.e. abortions) and/or spread at some later point in time. Recent studies have shown that carrier horses given very large and repeated doses of dexamethasone will have viral reactivation at only low levels and no or limited transfer of the virus to in contact horses. This helps explain why we have rarely had EHV-1 outbreaks or spread
in hospital housing although stressed or corticosteroid treated horses are commonplace and undoubtedly some are carriers. Spread of the virus is mostly horse to horse as it’s persistence in the environment is relatively short.

Transmission is typically via infected nasal secretions, although fetal membranes and placental fluid from the aborted fetus are also highly infective. After direct contact, the virus first colonizes the nasal mucosa and then replicates in regional lymph nodes by 1-3 days. This is followed by viremia for 3-14 days (depending upon the strain). From day 2 or 3 post-infection to at least day 7, there should be high virus load in nasal secretions. With high virus loads and the appropriate host (mostly older horses with low levels of cytotoxic T lymphocyte response to EHV1) there might be sufficient transmission of EHV-1 from lymphocytes to CNS endothelial cells to cause CNS disease. Outbreaks of neurologic disease, associated in many situations (83%) with a mutation in the polymerase gene, the mutant virus’s suppression of chemokines and cytokines, and high levels of viremia resulting in CNS vasculitis, retinal vasculitis and spinal cord disease. There appears to be an age predilection for EHV1 myelitis. Adult horses > 4 years are the great majority of cases. There are rare reports in yearling and young adults. In one report older mares were more likely to be affected with the neurologic disease. Older horses tend to get higher levels of viremia than younger horses regardless of infection with neurotrophic or “non-neurotropic” strains. A minority, cases of neurologic disease are sometimes associated with the “non-neurotropic” strain. Outbreaks are most common at boarding stables, show facilities, and seemingly slightly less common at racetracks. Diagnosis is based upon characteristic clinical signs, including hind limb ataxia and paresis with less common or less severe involvement of the front legs, brain or brainstem. CSF is generally xanthochromic, elevated protein but in most cases no pleocytosis. Nasal swabs and whole (EDTA) blood should be submitted for PCR.

If the disease is suspected, ring quarantine and repeated testing or isolation of all horses for 3 weeks after the last fever is recommended. There is no definitive proof to my knowledge to suggest that a superior vaccine is available but frequent vaccination should be performed in at risk horses! Treatment is mostly supportive, although antiviral treatments (valacyclovir 25 mg/kg PO q8-12h or ganciclovir 2.5 mg/kg IV q8-12h) are often used. Aspirin and/or Clopidogrel are used to decrease small vessel thrombosis and in rapidly progressive or severely affected horses corticosteroids are administered. Attention to bladder or lest commonly fecal retention dysfunction is required.

**PARELAPHOSTRONGYLUS TENUIS**

*Parelaphostrongylus tenuis* infection in young horses is a recently reported neurologic disease of horses. In areas of North America that routinely diagnose P. tenuis in small ruminants and camels, infection in the horse should be considered and will be seen! The clinical sign of acute onset scoliosis is nearly diagnostic for the equine disease. Affected horses have been 6 mo-3 years of age. The parasite appears to have a migratory predilection in the horse for the dorsal gray column of the cervical and sometimes thoracic cord. If the lesion extends three or more vertebral segments, acute onset scoliosis with the head deviated away from the lesion side will occur. Hypalgesia to analgesia will be found on the affected (convex) side over the affected
segments. There may be very mild ataxia and paresis of the limbs on the affected side. In the few cases we have performed CSF collection and evaluation on, the results were unremarkable, unlike *P. tenius* in small ruminants and camelids. The signs are characteristic enough in horses in an endemic area to make the diagnosis. Initially, there is no pain in manipulation of the neck, but after several weeks, arthritic changes develop. Treatments have included high doses of ivermectin and steroids, but lesions have been so advanced that successful treatment does not seem possible. Some clients have worked with physical therapists to provide exercises and braces in hopes of a return to normalcy, but this has been uniformly unsuccessful.

**PRIMARY HYPERAMMONEMIA**

Primary hyperammonemia is a not uncommon cause of acute and fulminant encephalopathy in horses often associated with mild colic 12-24 hours previously. This disease is caused by marked increase in enteric ammonia production which overwhelms the detoxification capacity of the normal liver (liver enzymes and function are normal). Blindness, dilated pupils, propulsive walking, head pressing are the most common signs. Blood ammonia, lactate and glucose are very high and bicarbonate is low. Treatments include: sedation if needed to control the horse, oral administration of neomycin and/or lactulose to lower ammonia production and crystalloid therapy with added KCL. Horses are generally better in 24 hours or have progressed to coma during that time. Survival rate is approximately 50%.

**ACUTE HEPATIC ENCEPHALOPATHY**

Treatments of liver failure or liver disease will vary depending on cause. Treatments for hepatic encephalopathy (HE) revolve around decreasing enteric- derived neurotoxins (primarily ammonia), decreasing cerebral edema, correcting glucose, electrolyte and acid-base abnormalities, maintaining perfusion and oxygenation to the brain and other vital organs. Horses with HE can have propulsive cortical signs which may require sedation in order to properly attend the horse and prevent injury to the horse or humans. A low dose of detomidine (5-10 μg/kg IV) may suffice. It is important to not overly sedate a horse with HE as that might cause excessive lowering of the head in the standing horse and promote cerebral edema in addition to the potential negative effects on the brain, liver, and other organ perfusion. Diazepam should not be used in horses with HE as it will induce astrocyte swelling and worsen HE. *Clinical experiences to support this comes from a foal with PSS developing seizure immediately following diazepam tranquilization to place a catheter and from the report on phosphine toxicity where 3 horses were reported to develop neurologic signs soon after receiving the drug. Ideally, sedatives should be avoided in HE. The next stepwise treatment for HE would be to correct intravascular fluid, electrolyte, and glucose abnormalities. A normal or slightly high sodium content fluid with additional potassium chloride (20 mEq/L) added is an acceptable initial crystalloid therapy for HE. Supplemental potassium is generally recommended since the horses with HE are anorexic and would most likely be deficient in total body potassium (TBP) and hypokalemia is known to increase proximal tubular ammoniogenesis with the increased ammonia being returned to circulation and worsening the symptoms of HE. Hypokalemia can also have adverse effects of other organ systems (e.g., cardiovascular). One of the most important goals in the treatment of HE is to reduce in blood and CSF ammonia concentration. The primary means for reduction of blood/CSF ammonia is to reduce the production or absorption of ammonia from the gut. Therapeutic options include...
neomycin (10 mg/kg PO q8h) or another poorly absorbed antibiotic. It may be preferred to combine neomycin with lactulose. Neomycin decreases ammonia production via its effect of microbial population and decrease in ammonia producing bacteria. Lactulose, a poorly absorbed carbohydrate, decreases ammonia when its metabolism in the large bowel results in increased H+ production and conversion of some ammonia ions (NH₃) to poorly absorbed NH₄⁺ (ammonium) salts. Orally administered antibiotics should not be prolonged beyond 1-3 days to lower the risk of antibiotic-associated diarrhea. It would be ideal if the treatments softened the stool (a cathartic effect) without causing diarrhea. Other oral treatments that have been used to lower enteric ammonia production include probiotics and in humans the antimicrobial rifaximin. Probiotics, prebiotics, and symbiotics (probiotic with lactulose) may all have some efficacy in treating HE by modulating the gut flora. They may decrease ammonia production and endotoxin absorption but they are not proven to be beneficial in HE studies. Feeds with small amounts of carbohydrates and high branch chain amino acid should be given every 2-4 hours. Mannitol or high sodium containing fluids can be used for suspected brain edema. Treatments to decrease neuroinflammation and disruption of the BBB are mostly unproven but include N-acetylcysteine, minocycline, neurosteroids such as progesterone or allopregnanolone and hypothermia. Horses with acute liver failure should receive supportive treatment for systemic inflammation and possible bacterial translocation. Hepatic lipidosis treatment are: treat the predisposing disorder, fluids including dextrose and potassium, enteral nutrition if possible and regular insulin (0.1-1.0 IU/Kg) (or 0.4 IU/kg q24h Zn insulin) as needed for hyperglycemia.

**EQUINE MOTOR NEURON DISEASE**

Equine motor neuron disease (EMND) may have decreased in incidence or reporting but sporadic cases exist. Recent work continues to explore the strong link between anti-oxidant deficiencies, i.e., tocopherol and pro-oxidant excess in the pathogenesis of the disease. Deficiencies in vitamin E may cause abnormal permeability in the CNS blood-brain barrier which may play a role in the pathogenesis of this oxidative disease. Another oxidative disorder of the equine CNS (neuronal axonal dystrophy or degenerative myelopathy) has been recently reviewed and we sporadically see cases in young horses some of which must have heritable components.

**GUTTURAL POUCH MYCOSIS**

Mycotic disease of the gullet pouch may cause acute onset of neurologic signs (mostly dysphagia or epistaxis. The close relationship between gullet pouches, cranial nerves, and sympathetic structures make neurologic abnormalities due to diseases of the gullet pouches (especially mycosis) possible. Surgical ligation of arterial supply to the diseased site is the preferred treatment. Medical treatment provides some benefit but if dysphagia is present many months may be required before there is return of function.
SPIROCHETE DISEASES IN THE HORSE-LYME AND LEPTOSPIROSIS

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Borrelia burgdorferi sensu stricto is a common infection in adult horses in many areas of the US and the world. The greatest unknown regarding B. burgdorferi infection in horses is the incidence of clinical disease! The high seroprevalence found in many areas and the lack of scientific information on both the incidence and clinical signs of Lyme disease have perhaps made Lyme disease the most controversial equine infectious disease. There are many other unknowns for B. burgdorferi infections and Lyme disease in horses including: what are the clinical findings, how can Lyme disease be confirmed both ante mortem and post mortem, interpretation of B. burgdorferi serologic test in clinical practice, what are the preferred antibiotics for treating B. burgdorferi infection, and is there an appropriate use of canine approved vaccines in horses. Although the list of unknowns is considerable, some evidence-based information is available on B. burgdorferi infection in horses that may serve as a guide for interpreting serologic test, confirming Lyme disease, selecting antimicrobials for treatment and expected efficacy of vaccination.

PREVALENCE OF INFECTION AND GEOGRAPHIC DISTRIBUTION
Prevalence of B. burgdorferi infection is not well documented in horses with the only published prevalence studies suggesting 77%, 45%, 60%, 60%, 15% prevalence in adult horses in the mid–Atlantic,1 New England,2 New York,3 Minnesota,4 and Northwestern5 states, respectively. It should be noted that these figures may be biased as some horses were likely tested based upon some suspicion of infection. The six-month incidence of new infection is reported to be 8% in horses in New York State.6 Those of us who practice in endemic areas know the seroprevalence is high, even if we cannot provide the precise percentage. The geographic distribution of infection (seropositive horses) might be expected to be similar to that published for both human and canine Lyme disease. Although it is unproven, adult ticks are likely responsible for most of the Borrelia infections in horses, whereas nymph stages of the tick are responsible for a high percentage of human infections. CDC maps for human Lyme disease (30,000 cases/year) likely underestimate the endemic areas for horses and dogs since horses and dogs are more likely to have attachment of infected ticks than humans. Serologic studies in unvaccinated horses might indicate what CDC human Lyme maps would look like in future years. It is well documented that Ixodes ticks are geographically spreading and nearly 50% of all continental United States counties have Ixodes ticks, although B. burgdorferi infections remain more common in those areas with large populations of white footed mice and other small rodents serving as reservoirs of the organism.7 Based upon canine and equine serologic surveys for B. burgdorferi, a high adult equine seroprevalence (>25%) would be expected in all or most New England and mid-Atlantic states and in some areas of Minnesota and Wisconsin, a moderate seroprevalence 6 to 15% in the surrounding states and into southern Ontario and along the California coast.1-5,8

SEROLOGIC TESTING FOR B. BURGDORFERI
There are several serologic tests available for B. burgdorferi. A list of tests include: the
indirect florescent antibody (IFA) test, Western Blot, whole cell kinetic ELISA, point of care C6 ELISA, and a multiplex ELISA measuring antibodies to outer surface proteins A, C and F. Ponies exposed to *B. burgdorferi* infected ticks developed detectable whole cell k-ELISA antibodies 5–6 weeks after exposure, with antibody titers steadily increased for 3–4 months and remaining high until euthanasia 9 months later. In infected ponies that were treated with antibiotics, the ELISA often declined after 4 weeks of antibiotic treatment but unless the ELISA continued to decrease to a negative value, infection was still present. Western Blot testing is thought to be more specific than ELISA testing and in human medicine ELISA screening and WB follow up (two step testing) or WB testing (using both IgG and IgM) alone remain the recommended test for human Lyme disease. WB tests generally detect antibody against several outer surface antigens including those used in the Lyme multiple ELISA. These are several articles suggesting that the C6 ELISA could replace the two step (ELISA/WB) testing in humans. The IDEXX C6 point of care ELISA has good sensitivity and excellent specificity for measuring *B. burgdorferi* antibodies in horses. The published sensitivity in horses was 68% for experimentally infected ponies but if the samples collected less than 3 months after tick exposure are removed from the equation, the sensitivity increases to 75%. There are no serologic tests, other than the IgM based test, that claim to consistently detect early (<2 months) infections. Available evidence from pony and dog experimental treatment studies suggest that *B. burgdorferi* infected horses that are successfully treated with antibiotics should have a negative C6 ELISA 2–4 months after completion of successful treatment, although this might vary depending upon duration of infection prior to treatment. There is no published data on the multiplex ELISA results following experimental infection in horses, but there is good evidence in other species and from retrospective evaluation of field infected horses to indicate a temporal pattern of change in OspC and OspF antibody in association with infection. The OspC antibody is believed to increase within 2-5 months after infection and then returns to a negative range regardless of infection status. OspC is, therefore, a good marker for recent (3-5 months) infection, but on its own is not suited to measure the response to treatment. Recognition of early infection is important if treatment is being considered since treatment of early *B. burgdorferi* infection is believed to easier (shorter treatment and higher success) than when treatment is delayed. Horses with more chronic infections generally have elevations in OspF and this antibody titer likely remains high as long as the horse is infected. An unknown question is: are horses with persistently high OspF or C6 antibody for several months chronically infected or could it be prolonged antibody production without infection? We have no evidence to suggest anything other than chronic infection. OspF antibody, like C6 antibody, might be used as markers of chronic infection and treatment success (negative antibody). High levels of OspA antibody are believed to most commonly result from vaccination, but a small number of unvaccinated horses have persistently elevated OspA antibody. The significance of a high OspA antibody in those horses is unproven but some horses with neuroborreliosis and in one confirmed Lyme uveitis case, only an elevation in OspA antibody was present. Comparison of serum and CSF serology can be somewhat useful in diagnosing neuroborreliosis based upon the concept of intrathecal specific antibody production with CNS infection. The multiplex Lyme assay results cannot be viewed exactly like *Sarcocystis neurona* ELISA ratios because for *B. burgdorferi* multiplex antibody measurements the serum is diluted. Generally, CSF results on the multiplex assay should be 3 or more times greater than serum levels to suggest intrathecal antibody production (personal
observation). A marked increase in antibody ratio (CSF:Serum) for only 1 of the 3 antigens may be more significant that an uniform increase for all three. It should be noted that in a few confirmed cases of Lyme disease, horses have been seronegative in serum but positive in other fluid (CSF, aqueous humor). A case of equine neuroborreliosis and common variable immunodeficiency has been reported.18

**CLINICAL DISEASE**
There is strong evidence for a very large number of horses being infected with *B. burgdorferi*, but we have absolutely no clue how many actually have clinical disease! The only well documented and published cases of equine Lyme disease in North America have been in horses with uveitis, neuroborreliosis, or pseudolymphoma.17-20 Clinical signs observed in horses with neuroborreliosis have included: acute to subacute onset of reluctance to lift the neck to proper height, “root signature” signs, muscle wasting of the neck, top line and gluteal muscles, hyperesthesia in these areas, spinal cord ataxia and less commonly signs of peripheral nerve disease, decreased tail and anal tone. Dysphagia and other cranial nerve signs have also been observed (Dr. Amy Johnson, personal communication). In confirmed cases of equine Lyme disease fever has often been absent. Generalized stiffness, lameness, and hyperesthesia are the most commonly incriminated clinical signs of equine Lyme disease in equine practice but causative proof of Lyme disease in these cases is absent.21 Suspect Lyme cases, which are mostly sport horses, rarely have severe joint effusion or single leg lameness. Tendon sheath inflammation and bursitis have been rarely associated with *B. burgdorferi* infections. The commonly observed clinical response to doxycycline or minocycline in field cases supports the diagnosis of Lyme disease but improvements in clinical signs may be a result of anti-inflammatory properties of those drugs rather than specific treatment of Lyme disease.22,23 A dual effect (antimicrobial and anti-inflammatory) of tetracyclines in treating Lyme disease in primates has also been reported.24 Ponies experimentally infected with *B. burgdorferi* consistently had lymphocytic/histiocytic reaction in the skin near the tick bite, regional lymph nodes, and a lymphocytic infiltrate mostly around synovial nerves and blood vessels of all synovia. In some ponies, meninges, nerve roots and peripheral nerves had similar mononuclear inflammation. These experimental finding could support the most common clinical observations associated with Lyme disease in practice but this remains unproven.9,10,21,25 With seroprevalence for *B. burgdorferi* very high in many geographic areas and clinical signs often relatively subtle and likely not proven to be due to *B. burgdorferi* infection there is likelihood that equine Lyme disease might be an over diagnosed disease.

**DIAGNOSIS**
It is clear that positive antibody test, clinical signs and response to treatment do not provide sufficient basis for the diagnosis of Lyme disease. Instead, diagnosis should be based upon a combination of: clinical findings, ruling out other diseases that may cause similar findings, serologic testing, cytology or histopathology results on affected fluids and tissues, antigen testing if possible and response to treatment in some cases. The sensitivity and specificity of antigen or organism detection tests such as immunohistochemistry, silver staining, fluorescent *in situ* hybridization and even PCR are sometimes not well validated in veterinary testing laboratories.

**TREATMENTS**
Treatment of *B. burgdorferi* infection in horses has for the past 15 years has consisted mostly of orally administered doxycycline or minocycline. These antimicrobial selections seemed reasonable based upon their known success in treating Lyme disease in humans, *in vitro* susceptibility profiles and ease of administration and safety when given to horses for several weeks.\(^{26,27}\) Upon closer examination, there are at least two very different variables regarding the use of these drugs in horses versus humans for the treatment of *B. burgdorferi*. Firstly, the duration of infection prior to treatment is likely much shorter in humans that in horses making successful treatment more difficult in the horse. Secondly, the absorption of doxycycline and minocycline are 95% and 100% in humans, respectively, while in horses the absorption is approximately 16% to 26%, respectively.\(^{28-30}\) Peak serum levels following a 200 mg dose of either drug range from 2.3 to 7.5 µg/ml in humans while in horses given 10 mg/kg of doxycycline or 4 mg/kg minocycline peak serum levels are less than 0.75 µg/ml.\(^{28-30}\) Peak synovial fluid concentrations of the drugs are similar to plasma concentrations in horses although doxycycline trough concentrations in synovial fluid are higher than serum trough levels.\(^{31}\) Minocycline appears to have substantially better penetration of the aqueous and CSF in horses than does doxycycline although levels are less than peak serum levels.\(^{30}\) The tetracycline class drugs, which are time dependent antimicrobials, have an MIC\(_90\) for *B. burgdorferi* of 0.25-0.8 µg/ml.

Therefore, in horses at the current dosing recommendations, tissue and body fluid concentrations would often be below the MIC\(_90\) for *B. burgdorferi*. This pharmacokinetic and MIC data may help explain the transient decrease in serum antibody following doxycycline treatment of four *B. burgdorferi* experimentally infected ponies but elimination of the organism and negative serology occurred in only 1 of the 4.\(^{10}\) In addition, field infected horses when treated, even long term, with either minocycline or doxycycline often have only modest or no decrease in *B. burgdorferi* serologic titers.\(^{32}\) Conversely intravenously administered tetracycline (6.6 mg/kg) reaches high peak serum levels and may maintain trough levels above MIC\(_90\) for *B. burgdorferi*\(^{33}\) which might explain oxytetracycline’s success in eliminating the organism in the experimental pony infection study.\(^{10}\) Horses that have been infected for many months prior to treatment may not have the same successful response following oxytetracycline treatment as what occurred in the experimental pony study.

The potential for nephrotoxicity, thrombophlebitis, and the practical difficulty in daily, long-term administration have prevented oxytetracycline from becoming a standard of care treatment. Other options for treatment in horses include parenteral administration of a beta-lactam antibiotic, several of which have excellent *in vitro* sensitivity against *B. burgdorferi*. Cefiofur at a daily IM dose of 2.2 mg/kg for 28 days was able to eliminate *B. burgdorferi* from 2 of 4 experimentally infected ponies (all placebo treated ponies remained infected).\(^{10}\) The crystalline form of the drug is approved to be administered intramuscularly on days 1, 4, and then weekly and will maintain serum levels >0.22 µg/ml throughout the treatment period and for at least 6 days following end of treatment.\(^{34}\) Cefiofur MIC\(_90\) against *B. burgdorferi* is 0.08 µg/ml (unpublished data). It is unknown if tissue and body fluids levels would be maintained above the MIC. Potassium penicillin administered intravenously could be used, but the practical aspects of long term, 3 or 4 times daily treatments would be difficult except in a hospital and treatment expense would be considerable. Penicillin has a reported MIC\(_90\) of approximately 4 times that of the tetracyclines.\(^{35}\) *B. burgdorferi* resistance has
been demonstrated to trimethoprim sulfas and enrofloxacin, two commonly used oral antibiotics in the horse.

Most in vitro testing is performed on viable, motile B. burgdorferi as they are readily available for testing and assumed to be the Borrelia stage most commonly causing disease. A cystic form of B. burgdorferi has been associated with chronic Lyme disease in humans (although not proven) and this form of B. burgdorferi is known to have a very high resistance pattern to the macrolides, tetracycline and B-lactam antibiotics that are commonly used for treatment of acute infections. Although motile B. burgdorferi are reported to be resistant to metronidazole in vitro, it has moderate in vitro sensitivity for the cystic form of the organism. Some equine veterinarians have used metronidazole as treatment for chronic B. burgdorferi infections when other antibiotics did not provide a suitable clinical response or decrease in serum antibody. There is currently no proof of clinical efficacy in humans and horses for metronidazole in treating acute or chronic B. burgdorferi infections.

VACCINATION
There is good evidence in horses, humans and dogs that a high level of OspA antibody prevents the transfer of B. burgdorferi from the infected and attached tick (complete protection). Some canine Lyme vaccines, when administered to horses, will result in a marked level of antibody production but the antibody T½ appears short and more than yearly vaccination would likely be required to provide protection from infection. A large number of canine vaccines have been administered to horses in the past 5 years and without reported adverse effects. Although there is good evidence of efficacy and reasonable field evidence of safety, importance of the vaccine for equine practice can only be answered when a determination of incidence of clinical disease is established. The value of adding OspC antigen to an OspA antigen vaccine is unknown; there are genomic strain variations in the OspC antigen. FDA equine approved and effective tickicides or tick repellants for horses are sorely needed.

LEPTOSPIROSIS
Leptospirosis is caused by a highly invasive spiral bacterium of the genus Leptospira. The infectious agent is capable of infecting both humans and animals, including the horse. There are at least 22 recognized species of Leptospira that have been further classified into three major subgroups: pathogenic, intermittently pathogenic and nonpathogenic (saprophytes), comprising >250 serotypes (“serovars”) based primarily on the immunological characterization of surface lipopolysaccharide (LPS). Serovars are sometimes classified as causing host-adapted infection or incidental host infection. Host-adapted strains seldom cause clinical disease in their maintenance host, infection and shedding are prolonged, and the serologic response following infection is relatively low. Conversely, incidental host serovars are more likely to cause clinical disease in a nonmaintenance host, be associated with a marked serologic response following infection, and be shed only briefly by the host.

In North American horses, Leptospira interrogans serovar Pomona type kennewicki is the prominent incidental (pathogenic) serovar for horses. In Europe, important equine strains are Leptospira kirschneri serovar Grippotyphosa, strains duster (Western Europe) and moskva (Eastern Europe). In South America, L. interrogans
icterohemorrhagiae and copenhageni are important strains. *L. interrogans* serovar Bratislava is considered by most researchers to be the host-adapted serovar of the horse and although horses may have high serum antibody titers against serovar Bratislava, it is generally considered to be nonpathogenic to the horse. One explanation for the high serum titers to Bratislava is a high cross reactivity with *Leptospira interrogans* serovar Pomona type kennewicki on the microscopic agglutination assay (personal observation). A variety of wildlife including raccoons, striped skunks, opossums, and red and grey foxes have been shown to host serovar Pomona and multiple strains of *L. Pomona* kennewicki.

**PATHOGENESIS**

*Leptospira* infection most often occurs following contact with *Leptospira* contaminated environmental reservoirs (water, soil), urine or reproductive fluids. The bacteria are believed to penetrate mucosal membranes or enter transdermally through wet or abraded skin. Motility of the organism is thought to be important in penetrating mucosal membranes and crossing cell layers. Upon penetration of membranes, the bacteria adhere to extracellular matrix and host cells. Virulent leptospires are resistant to complement and to killing by neutrophils in nonimmune hosts. Bacteremia may follow within 2-20 days after initial mucosal contact and fever may occur during this acute phase, which generally lasts 2-7 days. Although *Leptospira*, like other Gram-negative bacteria, contain lipopolysaccharide (LPS) it has low endotoxin activity and fever is not believed to be associated with the endotoxin. Bacteria are disseminated from the blood to organs of trophism. A primary lesion of acute leptospiremia is damage to endothelial cells of small blood vessels, inhibition of coagulation and inflammation within the trophic tissues. Once specific antibodies are produced, complement and antibody rapidly eliminate the organism from the blood and tissues other than the kidney, eye, CNS, and reproductive tract. A second phase of the disease may occur later in some of those privileged sites and may be driven by the host immune response more than virulence of the organism.

**CLINICAL SYNDROMES**

Pathogenic *Leptospira* infections in the horse appear to have organ trophism for the kidney, eye, female reproductive tract and, less commonly, the lung. Infection may result in placentitis and abortion, neonatal jaundice, acute renal failure or hematuria, and, most commonly, recurrent uveitis. Acute, fatal respiratory distress with pulmonary hemorrhage, thrombocytopenia, jaundice in combination with acute renal failure has been reported in five 1- to 3-month old foals in Europe. The serovar involved in the respiratory cases was not confirmed and I am not aware of confirmed similar cases in North America.

**REPRODUCTIVE TRACT INFECTION**

*Leptospira interrogans* serovar Pomona abortions may account for approximately 13% of bacterial abortions in mares in endemic regions, although incidence varies considerably between years. The reason for the yearly variation in incidence of abortions is not clear but rainfall may play a role. Serovar Pomona type *kennewicki* is responsible for most of the *Leptospira* abortions in North America, but serovars Grippotyphosa and Hardjo have also been reported. A single genetic variant of *Leptospira interrogans* serovar Pomona type kennewicki was found to be associated with the majority of mare abortions in Kentucky. Most abortions occur after 8 months of gestation, and rarely, a live foal may be born ill in association with fetal
leptospirosis.² *Leptospira* may be found in the placenta, umbilical cord, kidney, and liver following abortions. Lesions include placentitis (edema and areas of necrosis in the chorion) that does not involve the cervical star. Microscopic lesions include necrosis and calcification of the placenta.¹⁴ Placental disease may occasionally result in the mare developing hydramnios.¹⁵ Macroscopically, the fetal liver may have yellow discoloration and microscopic disease includes a multifocal necrosis with lymphocytic/plasmacytic infiltration of hepatic portal triads and sometimes giant cell hepatopathy. ¹⁴ Tubulonephrosis and interstitial nephritis may be detected in the kidney of the aborted fetus along with lung hemorrhages and myocarditis. Inflammation of the umbilical cord, funisitis, may be recognized by diffuse yellowish discoloration.¹⁶ Although more than one mare on a farm may abort because of *Leptospira* infection, epidemics of abortions are unusual. A substantial proportion of infected mares deliver healthy foals. This may be explained by timing of infection or a mild degree of placental and fetal pathology and efficacy of the acquired immune response in the infected fetus. It is likely the balance between degree of damage in the placenta and fetus and efficacy of the fetal immune response that is critical to the final outcome.¹⁷ The presence of specific agglutinating antibodies in the infected fetuses implies the infection occurred several days prior to the abortion. In support of this is that very high *Leptospira* antibody titers are found at the time of abortion in the infected mares. Mares that abort their fetus will most commonly do so 2-4 weeks after infection.¹⁷ Aborting mares and other recently infected horses are believed to shed *L. interrogans* serovar Pomona in the urine for approximately 2 to 3 months.¹⁸ A small number of horses on a farm with one or more *Leptospira* abortions may develop uveitis weeks later.

**ACUTE RENAL FAILURE**
Occasionally, *Leptospira* Pomona causes fever and acute renal failure in horses.¹⁹,²⁰ The kidneys become swollen as a result of tubular necrosis and tubulo-interstitial nephritis, and urinalysis may reveal hematuria and pyuria without visible bacteria. Fever has been reported to be persistent for 17-25 days if effective antibiotic treatment is not administered; the animal generally remains bright, alert and eating until azotemia develops.²⁰ On rare occasions, multiple weanling or yearling horses may be affected with fever and acute renal failure following *Leptospira* Pomona infection. Young horses appear to be more likely to have fever and fulminant clinical disease (renal failure, respiratory disease) associated with marked leptospiremia.² Horses with persistent leptospiremia and fever will have very high level of *Leptospira* antibody at the time of azotemia. Chronic renal failure caused by *Leptospira* infection has not been documented in the horse.

**RECURRENT UVEITIS**
The most important clinical disease associated with *L. interrogans* serovar Pomona infection in adult horses in North America and *L. kirschneri* serovar Grippotyphosa in Europe is equine recurrent uveitis (ERU).²¹,²² ERU is believed to be an immune-mediated disease frequently involving antibody against certain *Leptospira* antigens, specifically the LruC outer membrane protein, which cross-reacts with tissues of the lens, cornea, and possibly retina.²³ In North America, approximately 50% of ERU is associated with leptospirosis.²⁴ Live *Leptospira* organisms can be found in the aqueous or vitreous fluid of many horses with *Leptospira* associated recurrent uveitis.²⁵,²²,²⁶ High concentration of antibody against *L. interrogans* serovar Pomona in the aqueous humor, compared with serum titers, also suggests persistent local antigenic
stimulation. Survival of the organism in the face of high ocular antibody may indicate an absence of cells or molecules (e.g., complement) involved in bacterial clearance, suggesting an ocular immune privilege similar to that of the central nervous system. Recurrent episodes of the disease may be related to a Th1 and Th17 response of autoreactivity following mimicry and intermolecular or intramolecular epitope spreading or both.

Genetic factors are likely involved in the disease process, helping to explain why only some horses infected with *Leptospira* develop uveitis. Appaloosas, Warmbloods and some other breeds are thought to be genetically predisposed and specific MHC markers on ECA1 (Appaloosa) or ELA-A9 (Warmbloods) microsatellite are strongly associated with the disease. The prevalence of ERU is unknown but reports suggest that 1% or greater horses will develop the disease during their lifetime. *Leptospira*-associated uveitis may cause corneal, anterior chamber, and posterior chamber disease. Therefore, clinical findings may vary from corneal edema or other changes, to clinically quiet retinal lesions observed on fundus examination, to dramatic recurrent and progressive painful uveitis. Chronic disease may cause cataracts, retinal degeneration, or even glaucoma. There is a high frequency of blindness, globe loss and loss of function in horses with ERU.

**PULMONARY HEMORRHAGE AND PNEUMONIA IN FOALS**

A syndrome of acute respiratory distress, interstitial pneumonia with hemorrhage into alveoli and thrombocytopenia has been described in 1-5 month old foals in association with leptospirosis. The offending serovar was not determined but *Leptospira* antigen was found in tissues (lung was not tested) and tested foals had positive serology for *Leptospira*. This syndrome has not been documented in North America.

**DIAGNOSIS**

Diagnosis of *Leptospira* abortion is best accomplished by PCR of aborting tissue or fluids or fluorescent antibody testing (FAT) of the placenta, umbilical cord, fetal liver, or fetal kidney. The sensitivity and specificity of the PCR and FAT in these fluid or tissues are nearly 100%. Examination of silver-stained kidney samples in horses with renal disease is not highly accurate because there may be false-negative and false-positive findings, likely a result of non-pathogenic serovars. Polymerase chain reaction (PCR) testing is preferred for evaluation of fluids, such as urine, ocular fluids, fetal fluid and blood. Collection of a urine sample following furosemide administration may improve sensitivity of PCR, darkfield staining, or culture testing. The microscopic agglutination test is the gold standard for serologic detection of *Leptospira* antibodies. Either IgG or IgM will cause agglutination of the live organisms used in the test. ELISAs for detection of antibodies against immunodominant epitopes of outer membrane proteins of pathogenic *Leptospira* have been evaluated in horses and have similar sensitivities and specificities to MAT. A point of care lateral flow ELISA is available for testing dogs (SNAP Lepto Test IDEXX). *Leptospira* LPS, although minimally toxic, is highly antigenic and after acute infection antibody titers to *Leptospira* develop within 7 days and high titers within 2 weeks (unpublished data from author). Therefore, marked increases in serum antibody titers often accompany *Leptospira* abortions or acute renal failure, but serum titers may be low in horses with recurrent uveitis because of the chronic and localized nature of infection. Acute *L. interrogans* serovar Pomona infections often cause marked increases in antibody titers to several
serovars (especially icterohemorrhagiae and Bratislava) but these non-infecting serovar titers usually decline more quickly than the titers to the actual infecting serovar. Interestingly, an aborted fetus does not have the cross reactivity or is not as pronounced as what occurs in horses with recent infection. The reason for this difference is not known but could be related to a predominance of IgM in aborted fetal fluids. A combination of serology, culture, and PCR testing of aqueous fluid may be the only way to confirm Leptospira-associated uveitis. In ERU, the organism is most commonly found in the vitreous rather than aqueous fluid, which somewhat limits the practical application of ocular fluid PCR testing.

**TREATMENT**

Systemic administration of antimicrobials is indicated for horses with fever and acute renal failure caused by leptospirosis. Ticarcillin, penicillin, and enrofloxacin have been used successfully to treat horses with acute renal failure. Other antimicrobials to which Leptospira may be sensitive include ampicillin, cephalosporin, tetracycline, and doxycycline. Trimethoprim-sulfa and chloramphenicol, two commonly prescribed oral antibiotics, are not effective. Attempts to decrease urine shedding in mares following Leptospira abortion by administering oxytetracycline, penicillin G, and streptomycin were surprisingly ineffective in one report. Fluid therapy is indicated as supportive treatment of acute renal failure. If polyuria does not ensue soon after fluid therapy is started, furosemide and administration of drugs that may affect intrarenal hemodynamics should be administered (e.g., dopamine - I know there is no proof of efficacy in a large population of humans but it seemingly works in many large animals with ARF in my experience). Treatment for acute renal failure caused by Leptospira infection is often successful.

A variety of treatments for ERU, such as corticosteroids and cyclosporine implants have been used in hope of decreasing the inflammatory response, but these have generally provided only temporary relief and most affected horses either become blind or undergo enucleation because of intractable and persistent pain. Active Leptospira infections are present in many horses with ERU in some geographic areas and it might seem prudent that an effort should be directed toward treating the possible infection. Unfortunately, antimicrobial treatment of ocular leptospirosis may not be easy because the blood-ocular barrier inhibits movement of antimicrobials from plasma into the eyes. Even with inflammation, some interference from the blood-ocular barrier may persist. In a study of healthy horses, doxycycline could not be detected in the aqueous humor after a five-day treatment with a dosage of 10 mg/kg given every 12 hours. Minocycline administered at 4mg/kg orally twice daily to healthy horses will result in detectable, albeit low, concentrations in aqueous. Enrofloxacin after repeated intravenous dosing with 7.5 mg/kg results in a peak concentrations in aqueous humor of approximately 0.32 μg/mL and minimal inhibitory concentration (MIC) and minimal bactericidal concentration for enrofloxacin is 0.05-0.30 μg/mL against L. interrogans serovar Pomona strains. Other antimicrobials that may reach therapeutic concentration in the eye and could be effective against Leptospira organisms are intravenously administered oxytetracycline or penicillin. Topical antimicrobials may reach adequate concentrations in the aqueous humor but typically diffuse poorly into the vitreous humor. There is no good data to show that antimicrobial use has been successful in eliminating Leptospira from the eye. There is some concern the organism develops a biofilm in the eye which may make
antimicrobial therapy ineffective. Intravitreal antibiotic injections have anecdotally been reported to control uveitis in horses. Vitrectomy with gentamicin lavage has been used successfully in Europe in treating Leptospira associated uveitis.

PREVENTION
Acutely infected horses and mares aborting from Leptospira infection should be isolated for 14 to 16 weeks or the urine should be tested by PCR to determine whether the mare is shedding the organism. Limiting exposure to stagnant water and to potential maintenance hosts of the organism may help in the control of leptospirosis. This can be very difficult to do in equine husbandry as horse feeds (hay, grass) and water are frequently contaminated by rodents or wildlife that may be shedding Leptospira organisms. Vaccination of horses against L. interrogans serovar Pomona using off-label vaccines has historically been performed on farms with endemic abortions or a high rate of uveitis. Numerous canine or farm animal–approved Leptospira vaccines have been used in horses. Serovar specific vaccination against leptospirosis has been highly successful in many species providing protection against serovars that cause incidental host infection. Although Leptospira endotoxin has little toxicity it is very important in the development of immunity as Leptospira antibodies are agglutinating and opsonic to the LPS specific serovar. Following natural infection, serovar specific antibody along with complement will rapidly eliminate Leptospira from all tissues except from the brain, eyes, and kidneys. Proper vaccination and antibody production prevents bacteremia and therefore, should prevent the organism from invading the eye, CNS, kidney or reproductive tract. There has been concern that vaccination of infected horses with a whole-cell vaccine could theoretically cause ERU (although this has only rarely been observed and could not be demonstrated in one study) but Leptospira vaccination of horses with uveitis is not recommended. There is currently one vaccine approved for use in horses, Lepto EQ Innovator® (Zoetis). It is a killed Leptospira interrogans serovar Pomona type kennewicki, whole cell bacterin. The product is labeled for vaccination of healthy horses 6 months of age or older as an aid in the prevention of leptospirosis caused by Leptospira interrogans serovar Pomona. The vaccine has demonstrated safety in foals as young as 3 months of age and in pregnant mares during the second trimester. Efficacy of this product was demonstrated utilizing an intraperitoneal leptospiiral challenge model. Vaccinated horses did not develop leptospiremia or leptospiuria as compared to controls. The duration of immunity of this product has not been determined. One limitation to the vaccine is that it would not protect against Leptospira kirschneri serovar Grippotyphosa infection. Although Leptospira interrogans serovar Pomona type kennewicki is undoubtedly the predominant Leptospira equine pathogen in North America, L. Grippotyphosa would be second.

ZOOONOSIS
Leptospirosis in humans can vary in severity according to the infecting serovar of Leptospira, and the age, health and immunological competence of the patient. It ranges from a mild, influenza-like illness to a severe infection with renal and hepatic failure, pulmonary distress, and death (the classical Weil's disease). I could find no data on zoonotic potential of Leptospira interrogans serovar Pomona type kennewicki; it should be considered a potential zoonotic serovar but of unknown importance. All Leptospira are easily and quickly (within 1-2 minutes) killed by common disinfectants and drying.
Immune-mediated anemia, thrombocytopenia, and neutropenia are sporadic diseases in the horse. The immune disease process may result in decreased production of bone marrow derived cells or, more commonly, enhanced peripheral destruction of the cells. In many of the immune cytopenia disorders, only one cell line is affected while in other disorders two or all three of the cell lineages may be affected simultaneously. The immune process may be the result of primary causes, i.e., autoimmunity disorders that are often idiopathic, or secondary causes associated with infectious agents, drugs or neoplasia. Newborn foals may develop alloimmune cytopenias from colostral acquired antibodies, while isoimmune reactions (an immune response to foreign antigens from members of the same species) may occur from blood transfusion. The immune reaction causing the cytopenia is most often considered a cytotoxic type II antibody mediated response. Diagnosis of immune mediated cytopenias should include a thorough history and clinical examination, routine CBC and more advanced immunological testing when needed. The primary treatments for immune-mediated anemia, thrombocytopenia, or neutropenia include removal of any offending drug or infectious agent, immunosuppressive therapy when indicated and supportive care such as transfusions, if indicated.

The signalment and clinical signs often provide a major clue in the diagnosis and cause of the immune mediated anemia (IMA). For example, foals affected with neonatal isoerythrolysis (NI) are very young foals usually born to multiparous mares and the disease is most common in mules. Clinical signs of immune mediated hemolytic anemia are variable depending upon the severity and speed of onset of the anemia, and presence of secondary systemic disease or organ system failure such as disseminated intravascular coagulation, renal failure, etc. Horses and foals with severe IMA will appear weak, with marked tachycardia and tachypnea resulting from hypoxia. Icterus is generally noticeable if the immune-mediated anemia has been ongoing for one day or more. If the hemolysis is intravascular, which is common in many equine IMA disorders, the urine may be a light or dark red (hemoglobinuria) color. Neurologic signs may occur in foals with NI if the hemolysis is severe and bilirubin concentration increases to 20 mg/dl or greater and bilirubin toxicity (kernicterus) develops.

In horses with immune-mediated anemia caused by neoplastic diseases, weight loss, anorexia, fever, or other signs directly attributable to the anatomical location of the neoplastic disease are common signs. Evans syndrome is a rare hematological disease commonly defined as Coombs-positive hemolytic anemia and immune thrombocytopenia.

Immune-mediated anemia is a result of antibody mediated destruction of Red blood cells (RBCs) in either the bone marrow or peripheral destruction of the RBCs. Peripheral destruction of RBCs appears to be the most common cause of cell loss in equineIMA. Immune-mediated hemolytic anemia is most often caused by the binding of antibodies to the red blood cell surface. These antibodies may be produced as a
primary autoimmune disorder (autoantibodies) resulting from colostral (alloantibodies) or blood product transfusion (isoantibodies) antibodies, or from idiopathic causes. Secondary immune mechanisms in association with lymphoma, hemangiosarcoma, bacterial antigens (Streptococcus sp., Clostridium perfringens, and Rhodococcus equi), parasitic (uncommonly Thelerea equi or Babesia caballi may cause a Coombs positive immune anemia), viral causes (equine infectious anemia) or drug administration may also cause IMA. Drug-induced immunocytopenia may occur from one or more mechanisms and there is generally no relationship between the dose of the drug and the reaction. Penicillin may be an exception as there is evidence of dose related immune hemolytic anemia. Some drugs such as penicillin may act as hapten binding to cell membranes or act as neoantigens which result from the interaction of the drug with cell membranes causing the production of drug-dependent antibodies. Intravascular hemolysis can occur when there is an antibody mediated complement activation process. Extravascular hemolysis occurs from an antibody mediated phagocytosis with Fc, IgG-, or IgM opsonization and phagocytosis (Salama 2009). In horses, IgG appears to be the most common primary antibody associated with immunocytopenias, although IgM may be the predominant causative antibody in some cases (Wilkerson 2000). Immune mediated anemia may be caused by either warm agglutinating antibody, that reacts strongest at 37 C, or cold agglutinating antibody (agglutination of RBCs is greatest at lower temperatures). Although there is not a large volume of documentation in the horses, warm agglutinating antibodies are thought to be mostly associated with IgG while cold agglutinating antibodies are likely IgM. The spleen is thought to be the major site of RBC removal in immune-mediated anemia.

In aplastic anemia, the bone marrow is the site of cell destruction and all 3 cells lines are often affected causing pancytopenia. Aplastic anemia may result from immune mediated causes or myelophthisic causes (infiltration most commonly with neoplasia). Drug induced aplastic anemia/pancytopenia and bone marrow infiltration with lymphosarcoma are the most common causes of aplastic anemia in horses. Other common causes include toxic, neoplastic or drug-induced pancytopenia. Immune mediated anemia (red cell aplasia) has been reported in horses receiving injections of the first generation human erythropoietin (Epojen); administration of the product caused production of cross reacting antibodies against native erythropoietin in some horses resulting in a life-threatening erythroid hypoplasia.

The initial diagnostics for suspected immune-mediated anemia should be basic testing to determine if the immune disorder has caused decreased production of red blood cells or if the problem is caused by either increased intravascular lysis or phagocytosis of the cells by the reticuloendothelial system. When there is no evidence of a regenerative process (increased RBC, MCV, and RDW) of the erythroid cell line, a problem with decreased marrow production (aplastic anemia) should be suspected. Pure Red cell aplasia is generally not caused by an immune process unless there has been prior administration of human erythropoietin with subsequent autoantibodies produced against innate erythropoietin. There will be no discolored plasma (hemoglobinuria or marked jaundice) or discolored urine with bone marrow origin anemia; MCV will be low and RDW normal.

If the IMA is caused by increased RBC destruction, the plasma may be pink or at least jaundiced; urine may be red (hemoglobinuria) if the anemia is due to intravascular
hemolysis. MCV will often be increased due to release of larger than normal RBCs from the bone marrow. Although horses with regenerative anemia do not exhibit reticulocytosis on normal Wright’s stain, reticulocytosis can be detected with certain automated hematology machines. Another commonly used diagnostic test to confirm IMA is the direct Coombs’ antiglobulin test that detects immunoglobulin on the RBC surface. With primary or secondary IMA, circulating RBCs can become coated with IgG or IgM antibody. The direct Coombs’ test is used to detect these antibodies or complement proteins that are bound to the surface of red blood cells; a blood sample is taken and the RBCs are washed and then incubated with anti-horse IgG, IgM and complement antibody (also known as "Coomb’s reagent"). A positive Coombs’ reaction is the agglutination or lysis of RBCs upon visual inspection, confirming that antibodies are bound to the RBC surface. Agglutination occurs because Coombs’ reagent antibodies bind to multiple RBCs causing cross linkage. A false negative may occur with excessive antigen or antibody (prozone effect).

If autoagglutination is noticed initially in the blood tube, Coombs’ testing or addition of the species-specific antiglobulin is not required. If there is no gross agglutination in the blood tube, which is frequently the case with warm agglutinin antibody reactions, Coombs’ test is recommended to detect agglutination with the addition of antiglobulin or hemolysis after complement is added. Some antibodies such as Aa and Ca are mostly agglutinating antibodies while Qa is a hemolytic antibody and might not be detected without the addition of complement to the assay. Cold agglutinating antibody is frequently auto agglutinating at 4 C or more and would therefore not require Coombs’ testing. For IMA, the use of flow cytometry to detect the percentage of antibody coated cells can be helpful in the diagnosis and, in some cases, monitoring of the response to therapy. Osmotic fragility testing of RBCs can be used as an additional aid in the diagnosis of IHA although infectious and toxic causes of hemolytic anemia will also result in increased fragility when RBCs are place in hypotonic saline solutions. Historical information on drug administration and other pertinent information as discussed above should be considered along with the above laboratory testing. With extravascular hemolysis, the spleen may appear enlarged on ultrasound examination but this is a subjective finding.

The primary treatments for IMA include removal of any offending drug, immunosuppressive therapy when indicated and supportive care such as transfusions when needed. For drug-induced (secondary) IMA the duration of time to recovery following removal of the offending drug is variable. Immunosuppressive treatments may be indicated when anemia is progressive and/or life-threatening. The two most commonly used immunosuppression drugs for IMA are corticosteroids and azathioprine. Corticosteroids have effect by controlling both the cell- and antibody-mediated immune responses. Corticosteroid treatment is most effective for warm agglutinin reactions. Dexamethasone is generally the preferred corticosteroid in horses based upon field response reports, although there are no controlled studies comparing different corticosteroids. Starting doses of dexamethasone are generally 0.05-0.1 mg/kg SID, IV, or IM. Doses are high as 0.2 mg/kg have been required in a few horses. When doses higher than 0.1 mg/kg are needed, the success rate for eventual corticosteroid response is less. For horses that do not respond to corticosteroids or cannot be given high doses of the drug due to metabolic disease, Cushing’s disease and risk of laminitis, azathioprine is an alternative treatment. There are several reports
of successful azathioprine treatment for IMA when corticosteroid therapy had failed. Azathioprine is a purine analogue and acts as a prodrug for mercaptopurine, inhibiting an enzyme that is required for the synthesis of DNA. The recommended dose is 0.3 mg/kg q24h PO. Duration of treatment is generally one week at this dose if there is a good response, followed by a tapering dose or interval during the following days or weeks with continued monitoring for evidence of relapse of the immune-mediated disease. Although azathioprine is an immunosuppressive drug and could cause bone marrow suppression, adverse effects in the horse have been rarely reported even when used for several weeks. Horses with IMA and extravascular phagocytosis that do not respond to corticosteroids and azathioprine could be treated by splenectomy; it is important to rule out predisposing disorders such as lymphoma prior to recommending the procedure.

Supportive care for IMA includes blood transfusion (either whole blood or packed red cells) when required to maintain adequate global delivery of oxygen to tissues. Need for transfusion can be determined by clinical findings along with measurement of HCT, blood lactate, and venous oxygen concentration and saturation. Deciding upon a choice of RBC donor can be problematic since autoagglutination of RBCs may already exist in the IMA patient, making cross matching somewhat more difficult. For NI foals, washed RBCs of the mare or choosing a gelding of the same breed are good donor selections when a cross match is not feasible. Since NI cases are frequently due to Aa or Qa antibody, having a preselected donor horse free of Aa and Qa antigen and antibody is desirable but it can be difficult to find horses with this profile. Additional antigens such as Pa, Dg, Ka or Qb may occasionally be the causative antigen in NI. Fluid therapy with an isotonic crystalloid may also be required if the patient is hypovolemic or hypotensive. Although crystalloid therapy will lower the HCT, it does not decrease the total number of RBCs. Without a blood transfusion, crystalloid therapy could theoretically be harmful when the HCT is <8% as the viscosity of blood may become so low that it cannot maintain adequate capillary pressure. Mean post-transfusion survival of cross-match compatible allogenic RBCs is reported to be 39 days.

Complications from IMA include renal failure and laminitis. NI foals receiving multiple transfusions may develop liver disease, possibly related to iron overload. NI foals also appear to be susceptible to sepsis, possibly associated with immunosuppression from the hemolytic disease, the transfusion or both. Dogs with IMHA are reported to be prothrombotic associated with increased release of tissue factor, but publication evidence for this in horses could not be found.

Neonatal isoerythrolysis can be prevented by implementing the following: do not use colostrum from mares that have previously received blood transfusions or from mares that have previously had an NI foal. The mare can be tested for specific isoantibodies and the results compared to the blood type of the stallion, or the colostrum can be mixed with the foals RBCs immediately after birth to determine if an immune reaction (agglutination) is present. If the foal has NI or is determined to be at risk of NI then the foal should not be permitted to nurse the mare for 36 hours.

Immune-mediated Thrombocytopenia
Drug-induced thrombocytopenia is most common in horses and foals that have been treated with trimethoprim sulfa within the five days prior to the onset of clinical signs.
Another common problem is isoimmune thrombocytopenia in newborn foals. Most foals affected by alloimmune thrombocytopenia do not have NI. Alloimmune thrombocytopenia will often develop in the subsequent foals from the mare, even when sired by a different stallion.

The most common clinical signs associated with drug-induced immune-mediated thrombocytopenia are bleeding from the nostril and petechial and ecchymotic hemorrhages of the mucus membranes. Less commonly, swollen joints (hemarthrosis) or large subcutaneous swellings (hematomas) can be noted. These clinical signs may not be observed in many of the cases unless platelet counts are below 30,000 platelets/µl. Neurologic signs (due to bleeding into the central nervous system) become more likely when platelet counts decrease to <10,000. Foals with alloimmune thrombocytopenia have ecchymotic hemorrhages of mucus membranes, hematoma at the site of venipuncture and a dermatitis around the eyes, mouth and sometimes over the elbow region. Although the platelet counts in the affected foals are sometimes less than 2,000/µl, life-threatening bleeding is rare.

In horses with immune-mediated thrombocytopenia caused by neoplastic diseases, weight loss, anorexia, fever, or other signs directly attributable to the neoplastic disease are common signs. Horses with *Anaplasma phagocytophilum* infection often have platelet counts less than 60,000/µl but clinical findings associated with the thrombocytopenia are rare. Evans syndrome, a rare hematological disease commonly defined as Coombs’-positive hemolytic anemia and immune thrombocytopenia, was reported in a mare with less than <14,000 platelets/µl and a PCV of 9.5%; the mare died from cerebral hemorrhage.

Immune-mediated thrombocytopenia is most often caused by the binding of antibodies to the platelet surface most commonly causing peripheral destruction of platelets. There may also be a component of impaired platelet production with antibodies targeting megakaryocytes in the marrow (Khan 2010). The immune thrombocytopenia may be classified as primary (when there is no underlying cause or secondary when associated with drug therapies, infectious diseases or known immunologic disorders. Trimethoprim sulfa administration appears to be the most common drug associated immune thrombocytopenia in the horse. Although common in humans, heparin administration does not appear to be a common cause of drug induced thrombocytopenia in horses. Excessive removal of platelets may also occur associated with absorption of immune complexes onto their surfaces, often referred to as “innocent bystander” cause. This may occur with bacterial (*Anaplasma phagocytophilum*), viral infections (EIA), or immune reactions from vaccines, etc. A primary, alloimmune thrombocytopenia occurs in neonatal foals (NAIT) due to absorption of colostral autoantibodies. The disease may occur in foals of various breeds or in mule foals. Specific alloantigens associated with equine NAIT have not been proven, although several platelet membrane glycoproteins with polymorphism are known to occur and may serve as potential alloantigens. Removal of antibody-covered platelets by the reticuloendothelial system in the spleen is thought to be the most common site of phagocytosis.

Peripheral destruction of RBCs and platelets appears to be most common cause of cell loss in equine immune-mediated anemia or thrombocytopenia. Immune-mediated
hemolytic anemia or thrombocytopenia is most often caused by binding of antibodies to the red blood cell and platelet surface. Secondary immune mechanisms in association with lymphoma, hemangiosarcoma, bacterial antigens (Streptococcus sp., Clostridium perfringens, and Rhodococcus equi), parasitic (uncommonly Theileria equi or Babesia caballi may cause a Coombs’ positive immune anemia), viral causes (equine infectious anemia) or drug administration may also cause immune mediated anemia and or thrombocytopenia. The spleen is thought to be the major site of both RBC and platelet removal in immune mediated anemia and thrombocytopenia.

Bleeding, either internally or externally or both, along with petechial and ecchymotic hemorrhages are the most common clinical signs of IMT in the horse. Fever is usually only present if an infectious disease such as anaplasmosis, EIA, or babesiosis is associated with the disease. Treatment with trimethoprim sulfa within the past 5-14 days would be supportive of a drug-induced immune thrombocytopenia. Concurrent neoplasia should be considered as a cause of IMT if drug associated and infectious causes can be ruled out. With both extravascular hemolytic diseases or immune-mediated thrombocytopenia, the spleen may appear enlarged on ultrasound examination but this is a subjective finding.

There are no highly accurate diagnostic tests to confirm immune mediated thrombocytopenia. Regeneration is often pronounced though, resulting in increased MPV (megakaryocytes) and reticular staining with new methylene blue which detects RNA in megakaryocytes; these finding can help support a diagnosis of IMT. Megakaryocytes may also be seen on peripheral blood smears suggesting enhanced regeneration. Flow cytometry can be used to detect the percentage of antibody covered platelets, although if the platelet count is extremely low the interpretation becomes more difficult. Excessive consumption of platelets due to a septic or systemic inflammatory induced coagulopathy can be ruled out by observation of clinical signs and laboratory findings suggestive of disseminated intravascular coagulation. Pseudothrombocytopenia is very common in healthy horse samples and can confirmed as the cause of a low platelet count by submitting blood collected in a citrate tube in addition to the absence of clinical signs or other platelet indices findings associated with immune thrombocytopenia.

The decision to treat IMT should be based more on evidence of clinical bleeding than on platelet count alone. Immunosuppressive treatments may be indicated when thrombocytopenia is progressive and/or life threatening. Horses with extremely low platelet counts, e.g. <10,000 μl, might best be treated even without evidence of hemorrhage since extremely low platelet counts have been associated with life threatening intradural bleeding in other species. The two most commonly used immunosuppressive drugs are corticosteroids and azathioprine. The use of corticosteroids is the mainstay of first line therapy. The two most commonly used corticosteroids are prednisolone and dexamethasone. Corticosteroids act through inhibition phagocytosis and antibody synthesis, improve platelet production and possibly increase microvascular stability. The average time for response to successful corticosteroid treatment is reported in humans to be 1-3 days. A similar response time has been seen in horses with doses of dexamethasone of 0.1 mg/kg daily. Dexamethasone is generally the preferred corticosteroid, based upon field response reports, although there are no controlled studies comparing different corticosteroids.
Starting doses of dexamethasone are generally 0.05-0.1 mg/kg SID, IV, or IM. Doses are high as 0.2 mg/kg have been required in a few horses. When doses higher than 0.1 mg/kg are needed, the success rate for eventual corticosteroid response is less. Treatment is generally continued until the platelet count returns to normal, at which time corticosteroid treatment is slowly tapered. In horses with IMT, intravenous injection of corticosteroids with a 20 gauge-needle is preferred, and if performed a traumatically and the vein compressed after treatment for 1 to 2 minutes, visible hematomas are rare. For drug-induced anemia and thrombocytopenia, the duration of time to recovery following removal of the offending drug is variable. For horses that either do not respond to corticosteroids or cannot be given high doses of the drug due to metabolic diseases such as Cushing’s disease and metabolic syndrome, azathioprine is an alternative treatment. Azathioprine is a purine analogues and acts as a prodrug for mercaptopurine, inhibiting an enzyme that is required for the synthesis of DNA. The recommended dose is 0.3 mg/kg q24h PO. Duration of treatment is generally one week at this dose. If there is a good response to the 0.3 mg/kg dose, a tapering dose and/or interval is administered during the following days or weeks with continued monitoring for evidence of relapse of the immune-mediated disease. Although azathioprine could cause bone marrow suppression, adverse effects in the horse have been rarely reported even where used for several weeks. There are reports of successful azathioprine treatment for immune-mediated hemolytic anemia immune-mediated thrombocytopenia when corticosteroid therapy had failed. If IMT is life-threatening, platelet contents <10,000/μl or serious bleeding noted, azathioprine and corticosteroids could be used simultaneously until the platelet counts have risen to 40,000/μl or more and clinical bleeding is resolved. Other therapies that have been used to treat refractory IMT include vincristine and plasma exchange. Administration of plasma or concentrated globulin may result in non-specific blockade of the reticuloendothelial system by the Fc portion of the infused IgG molecules. Whole blood transfusions or platelet-rich plasma can be administered but rarely causes a dramatic increase in platelet count. Platelet transfusion are not recommended unless the platelet count is <20,000/μl and there is evidence of clinical bleeding. Thromboelastography measurement of maximum amplitude could be used to determine strength of clot if additional information is required regarding need for transfusion. Centrifuged platelet rich plasma would be ideal for transfusion but, in practice, collection of citrated blood in plastic bags, followed by allowing the RBCs to settle and then removing the plasma (which will contain most of the platelets) is a practical option for platelet transfusion. Glass bottles cannot be used for blood collection intended for platelet transfusion because the glass activates platelets. If the transfusion cannot be immediately administered then plasma should be stored at room temperature and not refrigerated, as this will have a negative effect on platelet function. Platelet survival after transfusion might be 4-5 days.

Prevention of drug-associated anemia and thrombocytopenia is difficult due to its sporadic incidence. There is usually no direct relationship between the given dose of drug and the reaction. Antibodies against these drugs may persist for years and previous affected horses should not receive the drug associated with the disorder.

Immune-mediated Neutropenia
Immune-mediated neutropenia may be seen concurrently with immune disorders that also cause thrombocytopenia or anemia. Neutropenia is relatively common in foals.
with alloimmune thrombocytopenia and is sometimes seen with isoerythrolysis. Neutropenia would be expected to increase risk of infection and clinical signs would depend upon the body site that is infected and fever is expected.

Neonatal alloimmune neutropenia generally improves within the first three weeks as the maternally derived antibodies decline. Treatment is generally conservative, with early antimicrobial treatment of infections and in some cases prophylactic antibiotic administration. Granulocyte colony stimulating factor (3-10 μg/kg IV or S.Q. q 24 with tapering dose) can be administered to increase production and release of neutrophils from the bone marrow and may help prevent secondary infections. If the immune mediated neutropenia is believed to be secondary immune-mediated disease or autoimmune but not alloimmune processes then corticosteroids might be indicated. Corticosteroid administration in IMN is controversial and may not be as effective as it is in immune mediated anemia and thrombocytopenia.

**Purpura Hemorrhagica**

Edema associated with purpura hemorrhagica is most common in the limbs and ventral abdomen and often moderately painful to the touch. Edema can form elsewhere in the body, causing respiratory distress (laryngeal swelling and pulmonary edema), colic, heart failure (distress and trembling), or myositis (stiffness). Fever and petechiae of mucous membranes occur in approximately 50% of cases. Often, the horse has a history of respiratory infection or exposure to *Streptococcus equi* (most frequent) or *S. zooepidemicus* in the preceding 2 to 4 weeks yet, in other cases, no incriminating infectious agent can be found. Diagnosis is based on a complete blood cell count (CBC), measurement of creatine kinase (CK) and aspartate aminotransferase (AST), platelet count, measurement of serum immunoglobulin A, and serologic testing for serum streptococcal M protein antibody and immune complexes (performed at Gluck Equine Research Center, University of Kentucky). A skin specimen from an edematous area obtained with a 6-mm Baker biopsy punch can be submitted in formalin to confirm vasculitis. Detection of immunoglobulin deposition is rare, and submission in special medium (Michel’s) or snap freezing is recommended if a biopsy is to be performed. The biopsy specimen should NOT be harvested from an area over an important structure (e.g., a tendon). In most cases, the biopsy does NOT help diagnosis or management of the case. Mature neutrophilia occurs, and CK and AST levels frequently are elevated with or without signs of myositis. A normal platelet count >90,000 cells/ml is expected. This can be helpful in separating purpura hemorrhagica (with fever and leg edema) from anaplasmosis where fever, thrombocytopenia and sometimes leg edema are present. An elevation in plasma protein measurement is usual, as are elevated immunoglobulin A levels and a high antibody response to streptococcal M protein in some horses. However, a high antibody response to streptococcal M protein also occurs in some healthy individuals. Severe proteinuria and even hematuria occur in some patients. Severe myopathy, mostly involving the pelvic limbs, may also occur in some horses.

Equine viral arteritis (EVA), equine herpesvirus 1 or 4, equine infectious anemia, immune-mediated hemolytic anemia, piroplasmosis and *Anaplasma phagocytophilum* infection are some rule-out diseases for the clinical signs. Treatment of purpura is corticosteroids - administer dexamethasone (0.04 to 0.16 mg/kg IV or IM q24h), continue at the clinical response dose for 2 to 3 days after signs abate then decrease
the dosage over 7 to 14 days. Clinical signs may recur as the steroid dosage is decreased or withdrawn. If corticosteroids are contraindicated, *plasma exchange* can be tried. In mild cases, corticosteroids may NOT be needed. Administer aqueous penicillin (22,000 IU/kg IV q6h) or penicillin procaine (22,000 IU/kg IM q12h) during steroid therapy. Provide supportive care as needed.

**Immune and/or Respiratory Pathogen Associated Myositis**

Immune or respiratory pathogen associated myositis has two or three distinct clinical forms; a chronic immune mediated muscle wasting disorder in mostly Quarter Horses often associated with recent *Streptococcal equi* or other respiratory pathogen exposure, a more severe and rapidly progressive myopathy also mostly in Quarter horses with active Strep. Equi infection, and an acute infarctive purpura myositis. The chronic immune mediated myositis causes rapid muscle wasting over the top-line and stiffness. Muscle enzymes are usually increased although the increase may only be moderate. Treatment with corticosteroids generally provides a favorable response. The myositis that occurs in association with active Strep equi infection caused a marked increase in muscle enzymes, marked pain and rapid muscle wasting over the back. The horses are often treated with penicillin but prognosis is generally poor and corticosteroids could be tried. A per-acute infarctive myositis occasionally occurs in adult horses and may be a form of purpura hemorrhagica that causes severe lameness and muscle swelling of one or more limbs. Horses are often so painful that they appear as a colic case. On clinical examination some muscles of the limbs are noticeably swollen and other signs of purpura (petechiations) may be found. Prognosis is poor in the cases but with aggressive corticosteroid treatments and penicillin, survival is possible.

There are many other immune mediated diseases in horses that are well described in current text books; including Recurrent Airway obstruction (RAO, Heaves), Equine Recurrent Uveitis, and numerous skin disorders (brief mention of these will be included in the lectures). Inflammatory bowel disease and chronic active hepatitis may both have an immune component to the disease but this is not well documented in the horse. Glomerulonephritis is an immune mediated disorder causing organ failure.

**Anaphylaxis**

Type I hypersensitivity reactions can occur from a broad range of antigens (insects, plants, topical medications, inhaled antigens or administration of foreign proteins). IgE is produced after exposure to a specific antigen – a process known as sensitization or priming. IgE binds nearly irreversibly to the FcεRI receptors on mast cells in tissues to create an antigen-specific receptor for the mast cell. While no reaction occurs during priming, once the IgE is bound to the mast cells, subsequent exposures result in antigen binding to mast cell-bound IgE, triggering mast cell degranulation and release of histamine and other inflammatory mediators. These mediators trigger an increased vascular permeability, resulting in rapid edema formation and signs of inflammation. In addition bronchoconstriction may occur. Eosinophils are recruited to the site of the edema and inflammation and go through a process of degranulation and production of inflammatory cytokines as do mast cells. The degranulation and histamine release are responsible for the rapid onset of the reaction, but the inflammatory mediators produced after degranulation are responsible for the duration. Urticaria, increased heart and respiratory rate (with bronchoconstriction) and edema are the characteristic
clinical signs. These are a result of the degranulation of IgE-primed mast cells. The most concerning reactions associated with anaphylaxis are respiratory obstruction from edema or bronchoconstriction and systemic hypotension. Urticaria develops rapidly and generally resolves in the same rapid fashion without medical intervention, although in more severe cases or if there is evidence of more systemic anaphylaxis, a glucocorticoid injection may be helpful. If the causative antigen is known, removal of the agent from the horse’s environment if possible is recommended as additional contact will incite repeat reactions. In some drug induced cases, an anaphylactic reaction that is not a result of previous sensitization and antigen-antibody reaction may occur from an immediate “triggering” of the complement-kinin system caused by some part of the drug.

Acute Systemic Anaphylaxis - Anaphylactic reactions are most frequent with intravenous administration of vaccines, occasionally penicillin or other antibiotics, selenium, plasma, whole blood, phytonadione (vitamin K), and other vitamins and minerals.

Treatments
Mild Forms: Mild forms of anaphylaxis cause urticaria and minor increases in respiratory rate. These may be simply treated with any of the following antihistamines: Diphenhydramine: 0.5 to 1.0 mg/kg IM or slowly IV, Doxylamine succinate: 0.5 mg/kg slowly IV, IM, or SQ q8 to 12h Pyrilamine maleate: 1.0 mg/kg slowly IV, IM, or SQ or Tripelennamine: 1.1 mg/kg IM or SQ.

Administer all antihistamines slowly intravenously because excitement and hypotension are occasional adverse effects. These adverse effects are rarely seen when antihistamines are administered intramuscularly or subcutaneously. Alternatively, but not simultaneously, administer epinephrine (1:1000) intramuscularly, 5 to 8 mL/450-kg adult, because when antihistamines and epinephrine are used together, antihistamines potentiate the effect of epinephrine on vascular resistance.

Severe Forms: Administer epinephrine, 3 to 7 mL (1:1000 undiluted) slowly IV to a 450-kg adult. Epinephrine may be administered intramuscularly in less severe cases at the same dosage or 2 times this dosage intramuscularly for severe anaphylaxis. Epinephrine by the intratracheal route, 5X IV dosage, may be used when intravenous access is not possible or limited. Provide patent airway if needed by intubation. This is important when laryngeal edema is severe. Intubation is also of some benefit in managing pulmonary edema when the upper airway is edematous and compromised. Stridor may not appear until 80% or more of the upper airway is obstructed. For pulmonary edema administer furosemide (1 mg/kg IV) and a corticosteroid. Although of no demonstrated clinical benefit, dexamethasone, 0.2 to 0.5 mg/kg, frequently is administered to prevent delayed edema formation in the lungs, pharynx, etc. Use plasma or hetastarch as an oncotic volume expander if pulmonary edema is believed to be progressive. If other fluids are needed for hypotension, administer hypertonic saline solution, 4 mL/kg. Administer intranasal oxygen.
The News Hour will include information on the revised consensus statement on inflammatory airway disease of horses. It would appear that the term “equine asthma” is now appropriate when describing both inflammatory airway disease (IAD) and recurrent airway obstruction (RAD) in horses. Distinguishing between IAD and RAD can be difficult unless there are signs of increased respiratory effort (RAD). Regarding the etiopathology of IAD in young performance horses, further evidence has been reported to suggest an association between *equine rhinitis A* infection and the disorder. Equine multinodular, pulmonary fibrosis is a well-described syndrome (mostly progressive) in adult horses and find equine herpesvirus-5 (EHV-5) (q PCR) on bronchoalveolar lavage (best) or a combination of whole blood and nasal swab being positive are supportive of the diagnosis.

Corticosteroid treatment is believed to increase the risk of laminitis in horses. This was not found to be the case for prednisolone, but that information may not be applicable when more insulin dysregulating corticosteroids (dexamethasone, triamcinolone).

Diagnosis of equine metabolic syndrome has been based upon both phenotype of the equine and laboratory evidence of insulin dysregulation. A recent report reveals that an enteroinsular axis involving incretin hormones and glucose-dependent insulino tropic peptide may play a role in the elevated serum insulin in horses with EMS. The insulin dysregulation obscured in EMS cases may not be purely peripheral insulin-resistant, but may involve intestinal incretin response permitting increased absorption of glucose and/or decreased first pass hepatic clearance of glucose. This further confirms that a modified glucose or starch oral challenge tests measuring both glucose and insulin are preferred for diagnosing equine metabolic syndrome (EMS).

Hematuria caused by urethral rents in horses is a well-characterized syndrome. Thirty-three cases were recently reviewed and corpus spongiotomy surgery was found to be successful in resolving the clinical signs.

Growing antimicrobial resistance to horse pathogens is a concern for us all. In one study encompassing 13 years, the resistance to streptococcal isolates to enrofloxacin went from 0% to 63%.

A retrospective case-control study on 108 horses with strongyles and 215 other horses with fever but without clinical signs of strongyles were reviewed. Some febrile horses were infected with *Streptococcus equi* and were carriers in spite of not showing clinical signs of the disease. Guttural pouch endoscopy and lavage with PCR should be performed to identify *Streptococcus equi* carriers, although sensitivity is not 100%.

Strangles cases were more likely to have markedly elevated fibrinogen and total protein values. Forty percent of clinical cases were still carriers 40 days later.

*Klebsiella pneumoniae* is a retentive, common, and often severe disease in adult horses. It may be particularly common after mechanical anesthesia. Nearly one-half of
the isolates are multidrug resistant.

Atrial fibrillation is the most important cardiac disease in performance horses. It can be successfully treated by either drug or electrical shock treatment, although recurrence is frequent. Difficulty in the conversion and mitral regurgitation are known to be risk factors for recurrence. A low left atrial fractional area change on echocardiographic examination is also a risk for recurrence and, if present, sotalol (2 mg/kg PO BID) may be included to help prevent recurrence.

Effective cryotherapy is known to decrease the incidence of laminitis in horses. The effectiveness of the cryotherapy varies by method used.

*Corynebacterium pseudotuberculosis* may occasionally cause disease in horses in eastern North America. Lymphangitis is only one of the syndromes associated with the disease.

A virulent form of *Anaplasma phagocytophilum* is known to cause disease in horses, humans, and dogs in northeastern United States (and likely Canada).

Lymphoma and melanoma are some of the most problematic neoplasias in the horse. Diagnosis (lymphoma) and treatment of both are difficult.

**Further Reading:**
- Janvier V, Evrard L, Cerri S, et al. Ultrasonographic findings in 13 horses with

THE DOG DAYS OF DATA

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Introduction
The relationship between veterinary medicine and technology is woven into the history of the profession. In the 1900s, there were approximately 8000 practicing veterinarians within North America and 80% of the workload was equine. Our dependency on horses was causing major challenges within cities and with “horse pollution” increasing daily, many were concerned that we would drown in horse manure. However, this never became as significant concern as projected, as the invention of the automobile fundamentally changed the way that we transported people. The invention of the automobile had another consequence, fewer veterinarians were needed for the care of horses. By 1920, only 10% of the veterinary activity was generated from horses.1

In 2016, we are entering a similar period of veterinary transformation, driven by the acceleration and convergence of technology. The next decade will see the veterinary experience become unbound from the brick and motor clinic and force practitioners to reassess how they build relationships, deliver information, and provide services and products for a digital, connected, and empowered pet owner.

Acceleration of Technology
Today, we are in time of unprecedented technological growth. It may not be as apparent on a day to day basis, but consider the incredible changes we have witnessed over a 20 year period. Today, mobile phones are not only ubiquitous but also more powerful than a supercomputer was only two decades ago. We have witnessed the complete digitalization of music and photography and, advances like the sequencing of the human genome, have gone from “science-fiction” to reality.

How is this possible? How can companies like Kodak, which at its peak, employed 150,000+ employees with revenues in excess of $15 billion, effectively disappear within the industries they created, while others, like Instagram, a company in the same space, succeed with a fraction of the size of the workforce? More importantly, what are the implications of this pace of change on animal health?

An underlying principle that can help explain this change is the Law of Accelerating Returns, crafted by Ray Kurzweil, which was based on an understanding of Moore’s Law.

Moore’s law is the observation that, over the history of computing hardware, the number of transistors in a dense integrated circuit doubles approximately every two years. The law is named after Gordon E. Moore, co-founder of the Intel Corporation, who described the trend in his 1965 paper.3

This observation is critical to our understanding of technology as it explains why our computers are able to get smaller, more powerful, and faster over such a short period of time. This exponential growth is, by its nature, both quick and difficult to predict.
Our brains are accustomed to thinking linearly, and making projections in an exponential world is difficult for the majority of humans. However, Kurzweil was able to not only comprehend these exponential changes, but extrapolate this thinking through the Law of Accelerating Returns. One of the key findings from Kurzweil’s work is that any technology that becomes information enabled, is able to ride this exponential growth curve. This is truly disruptive and, in practice, can be illustrated through the process of mapping the human genome.

In 1990, the US Government began the Humane Genome Project, a mega project that attempted to sequence the human genome. They predicted that it would cost 3 billion dollars and take approximately 15 years to complete. By 1997, researchers had only sequenced 1% of the genome and many were calling the project a complete failure. However, Kurzweil understood what the other experts did not, that the mapping was following an exponential growth curve and while progress seemed slow to date, each year, tremendous progress was being made. Ultimately, the project was completed in 2001, under budget.

Examples like that of the Human Genome Project highlight two very important things, that this exponential pace of change can be seen in areas outside of what most would consider to be “technology” and even those within a particular space are often unable to visualize and predict exponential change.

We are entering a world in which more health information is going to be generated away from the hospital than within it. The tools to capture this data will become increasingly sophisticated and less expensive, and we will have the ability to interpret this information much more effectively and efficiently than ever before.

**EXPONENTIAL CHANGE IN ANIMAL HEALTH**

There are three areas where we are seeing exponential technologies transform veterinary practice.

1. Data Generation - Where, When, How
2. Data Analysis and Interpretation
3. Explosion of Service Delivery

**INTERNET OF THINGS**

In early 2014, the CEO of Cisco declared that the Internet of Everything (IoE) was a $19 trillion opportunity and estimates predict that we will grow from the 8 billion connected devices that we have today, to over 50 billion by 2020.

The internet is an interconnected system of computer networks that allows people to collaborate with each other from all over the world. Today, we access the internet via not only our desktops, but from our phones, tablets, and other mobile devices. As sensor technology becomes cheaper, smaller, and more powerful, these same capabilities will become embedded in more and more objects, cars, fridges, objects around the house, and, yes, even our pet products. This continuous flow of data, from sources outside of the clinic, will provide owners and practitioners with a more comprehensive and objective picture of pet health.
Over the past 12 months, we have seen over a dozen new “connected” pet companies sprout up measuring everything from activity, to drinking and eating behavior, to scratching, heart rate and respiratory rate.

Large initiatives announced by Hills and Merial, also emphasize that IoT is not just a fad, but rather evolving to be another component of understanding pet health and providing care.

However, there are still gaps that exist in the connected care experience. Owners expect their veterinarians to be able to provide actionable insights from these data points, while veterinarians are struggling with how to incorporate these trends into patient treatment and care plans.

The profession will need to build additional value on top of these tools to provide patient centered insights and context.

**ARTIFICIAL INTELLIGENCE**

Artificial intelligence is currently all around us. Our coffee makers that can start at a specific time each morning or our navigation systems in our cars all represent forms of artificial intelligence that many people do not consider to be particularly spectacular. However, AI is becoming increasingly sophisticated and democratized which is allowing us to do things never before possible. This is a complementary set of technologies to those that encompass the IoT AI can allow us to leverage the volume of information brought forward through sensor technologies and make those disparate data streams actionable.

Google, Facebook, Microsoft, and IBM all have developed open tool sets that allow teams to leverage powerful machine learning capabilities in the cloud. Whether it’s predictive analytics, machine vision, or language classification, it’s clear that data in and of itself is not going to be enough, but rather deriving personalized, actionable insights for both practitioners and owners.

We are seeing 1st generation products and services coming to life in the veterinary space. LifeLearn’s Sofie application utilizes IBM Watson’s APIs to provide decision support to companion animal practitioners while IDEXX is layering in machine learning capabilities into its diagnostic equipment to assist with laboratory interpretation.

A critical piece of this conversation is how we can layer in these machine learning capabilities to truly improve patient care and practice workflow.

**DECOUPLING OF SERVICE DELIVERY**

The entire veterinary care model is built around an in-person experience. We have centralized locations with specialized equipment and expertise where we provide and charge for products and services.

However, we are no longer limited to the in-clinic experience as our only channel for service delivery. The rise of the on-demand and sharing economies has been startling as companies such as Uber and AirBnB dominate traditional players by offering a better experience at often lower cost for consumers.
We are not immune from the convenience and affordability that these models can provide. There are several telemedicine options available for pet owners ranging from Skype-like calls to platforms built as an extension of traditional practices. Defining and controlling the scope of care through these experiences remains a regulatory and medical challenge. However, the consumer demand is palpable as seen through the investment dollars flowing to alternative care platforms.

Veterinary equipment often sits underutilized and new mechanisms will emerge to leverage the infrastructure of hospitals. Key to this will be how information flows to and from practices to create a continuous experience. Another important question will be the relationship to the client and how that responsibility and accountability transfer between parties.

The “digital veterinary practice” offers distinct advantages in terms of cost and convenience, but in and of itself is not a holistic solution to the decreasing patient visits practices are experiencing today. We will need care models which leverage the benefits of the online and in-person experiences and offer integration of both avenues of care.

PREPARING OUR PRACTICES FOR EXPONENTIAL CHANGE
These are only a few examples of the technologies which are going to transform the veterinary practice. While an initial reaction may be one of anxiety or fear, there are several reasons why these tools will allow us to build more robust and connected experiences with our clients and patients.

Leveraging sensors will allow us to more effectively and objectively monitor patient health, even when they are not directly in our sights. The evolution of decision support tools will provide practices with additional perspectives and insights to reduce the likelihood of medical errors during care. Last, the ability for veterinarians to build a digital relationship with clients will open up new business models for practices while strengthening our bonds with both owners and pets.

Throughout this session, we will evaluate strategies to ensure that your practice thrives in this period of change, instead of being overwhelmed by it.

References:

TECHNOLOGY IN THE TRENCHES: POINT-OF-CARE TOOLS THAT HELP YOU BE A BETTER YOU

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Introduction
As we progress through 2016, veterinary medicine’s front lines remain some of the least tech-savvy settings on the planet. Why is that? I think it’s partially due to a misunderstanding of technology in medicine.

Often the technology that gets the most coverage comes from the futurists, those touting the next big thing: Google Glass or 3D printing, for example. These “things” are often abstract and not yet obviously applicable to our daily lives. Futurists have their place, but what’s lost in an overly futuristic outlook is a host of practical tools that are already available to deploy in practice, sure to make your team and your patient’s lives better.

This lecture will detail some of today’s digital point-of-care tools offering efficiency, workflow, or cost-saving advantages over their analog counterpart. Every item here has been successfully tested and employed in the veterinary real world. My hope is you will find at least one new technology to embrace.

Go Mobile with a Digital Bulletin Board
Most veterinarians don’t have a 9-5 desk job. We’re a mobile breed often performing our job outside of hospital walls, or in relief shifts at various hospitals, or traveling from patient (or herd) to patient. But we all maintain our select group of go-to references we use in daily practice. Often these are stored in a books with dog-eared corners or on a bulletin board at your desk.

Enter the Digital Bulletin Board. This is one of my favorite examples of a very practical use of simple technology, essentially turning your analog bulletin board into a pinch-to-zoom mobile bulletin board always with you on your phone or tablet.

To make your own, simply scan your favorite references into digital formats and package them together using free online collage-building software. Save that collage as one jpeg (photo) file and voila.

A complete guide to building this, for even the least techy-savvy veterinary professional, can be found at www.vmdtechnology.com/digital-bulletin-board.

Use Google Voice to Take Back Your Voicemail
Google Voice is probably the least-known Google service, but one of the best. This free service transcribes voicemails and allows you to receive your voicemail and text messages on your computer (including in your email). I set Google Voice to handle all
of my voicemail boxes (cell, office, and home). Now they all come to one central place: my email, transcribed and with an audio file, for easy sorting and delegating.

The other powerful way to use this service is to get a free Google Voice phone number and use it as your second cell number. Give it to select clients (or anyone) who may need to reach you, but who you may not want to have your primary cell phone number. The Google Voice dashboard allows you to set which phone this rings (if any), at which time, and set specific voicemail greetings based on who is calling you.

Find more at https://www.google.com/googlevoice.

**Evernote and Google Drive Store Documents Smarter**

The days of folders on a hard drive or in-hospital servers are numbered, mostly because this strategy is fraught with limitations, risk, and higher cost. Cloud storage is a much better, safer, cheaper solution. Ask yourself: would you pay more for an inferior product in any other part of your life?

My recommendation is to utilize two very simple solutions: Google Drive and Evernote.

I use Google Drive for storage of any saved medical articles or documents that I create, replacing my folder tree on my computer’s hard drive. Google Drive actually simultaneously keeps files and folders on your traditional hard drive(s) in sync with a drive on their website or mobile app. Update files and folders in one location and the others update instantly. This gives you peace of mind and allows access anywhere. Google Drive’s other benefits stem from its robust search capability and its collaboration tools using Google Docs instead of Microsoft Word and Google Sheets instead of Microsoft Excel.

Evernote is another powerful cloud storage tool that expands on these offerings in a different way. I use Evernote for personal note storage (to-do list, ideas) and as my digital filing cabinet to keep my personal and business life completely paperless. While you could use Google Drive for both tasks, I feel that Evernote is better for this because it allows you to combine different file types into one “Note” (for example combining a photo of your broken lift table, notes about the customer service call, and a scanned PDF of the bill to fix it).


**Chrome Remote Desktop Extends Your Range**

If your veterinary practice software is currently only viewable on your office computer, there is now a simple solution: Chrome Remote Desktop. Google provides this service for free and unlike most competing options, it is remarkably functional, fast, and simple to use.

Simply use Chrome as your web browser on your computer at work and on your computer at home (you must log into your Google account in Chrome on both
computers). Install Chrome Remote Desktop on both computers and secure it with a PIN. You are now two clicks away from using your work computer from home (and vice-versa). This is also a good option when working from the road (house call). Simply connect any device (check out the Chrome Remote Desktop mobile app) to the internet and you are able to operate your work computer containing your practice software.

Find out more: https://support.google.com/chrome/answer/1649523

**Slack Keeps Your Team Connected**

Who can argue with a product touting the tagline “Be less busy”? Slack is a group instant message tool built for the office setting. It is relatively new but taking the corporate world by storm. It is free and extremely simple to use. In the veterinary setting, Slack is very useful for communication between the front desk and the rest of the clinic, allowing discreet communication at the point-of-care. If your team doesn’t have assigned computer workstations in the clinic, you can install Slack on each computer and set up a username based on the name of that workstation (“lab computer”). Each team member can also have their own username for use on their mobile device, providing easy communication within and outside of the hospital.

Check out https://slack.com/is.

**The Magical Eye-Fi Camera Card**

If your team uses a digital camera for in-clinic photos, the Eye-Fi card is an elegant little device that will save time and effort. This SD card pops into your camera, but unlike other cards, it has a secret: it can connect to your Wi-Fi network and instantly beam newly taken photos to your computer.

If you use your cell phone for clinic photography instead, be sure to set up a Google Photos or Flickr to automatically upload photos off your phone and into the cloud or your hard drive for easy access and use. Google Photos now allows you to set up an album that anyone on your team can add photos to throughout the day.


**Twitter for the Veterinary World**

Twitter is technically a social media tool. But I view it as one of the more powerful technologies at our disposal that is grossly underutilized by the veterinary world.

The way I recommend using Twitter for your practice needs is two-fold:

1. **Team communication**: Twitter is a powerful in-hospital communication tool that is fun and informative for your staff. Create a Twitter username (aka “handle”) and set it as private (restricting followers to those you approve). Then you can require employees to make accounts and “follow” you so they get instant practice updates on their smartphones – a way to kick that overwhelming email habit you are constantly fighting and foster a more responsive staff.
2. **News Ticker**: Use your Twitter account to follow industry organizations, leveraging Twitter as a news ticker for your veterinary life.

Find a guide to getting started, including a starter list of who to follow (such as @VINNewsService) at: [http://www.vmdtechnology.com/amazing-powerful-twitter/](http://www.vmdtechnology.com/amazing-powerful-twitter/)

**Mobile Apps to Improve Your Medicine**

No point-of-care technology discussion would be complete without reviewing helpful mobile apps. I’d recommend these as my top 4 medical apps:

1. **Plumb's Veterinary Drugs**: Full disclosure: I admit biased here as part of the team that built this product. But arguably nothing is more widely used for veterinary referencing than Plumb’s Drug Handbook. Plumb's is finally available in a digital format accessible across all devices that is constantly updated (no more editions). Find it at [www.plumbsveterinarydrugs.com](http://www.plumbsveterinarydrugs.com).

2. **Target Manual**: You are probably familiar with the Target Antimicrobial Reference Guide. If not, this handbook provides the best antibiotic quick-reference I’ve found. Thanks to various sponsors, it is now available as a free mobile app.

3. **Vet Calculator Plus (VetCalc+)**: This app offers practical calculator tools such as fluid rates, CRIs, dilutions, and emergency drugs.


4. **Epocrates**: This app is a human medical go-to reference that provides some very useful veterinary tools such as looking up information on human-only medications, drug interactions, and pill identification tools. Find it at [http://www.epocrates.com](http://www.epocrates.com).

**Conclusion**

Admittedly, this list only skims the surface of the available point-of-care technology tools. But even with the implementation of just one of the aforementioned techniques, you can save your team time, money, improve patient care and client service, and make life more enjoyable for everyone involved.
CULTURE: EVERY PRACTICE HAS ONE...BUT IS IT THE ONE YOU WANT?

CULTURE

Appreciative Inquiry Exercise:
What rich, descriptive words regarding being “valued” resonated with you?
_____________________________________________________________________________
_____________________________________________________________________________

Why?
_____________________________________________________________________________
_____________________________________________________________________________

What is your “Why”?

WHY: a purpose, a cause or a belief; a “guiding principle”
_____________________________________________________________________________
_____________________________________________________________________________

HOW: actions we take to realize those beliefs
_____________________________________________________________________________
_____________________________________________________________________________

WHAT: the results of those actions
_____________________________________________________________________________
_____________________________________________________________________________

“People don’t buy what you do, they buy why you do it”-Simon Sinek
What values best represent you?

Working individually, select the four to six words that best represent the fundamental nature of who you are and circle them.

<table>
<thead>
<tr>
<th>Honest</th>
<th>Cheerful</th>
<th>Competent</th>
<th>Motivated</th>
<th>Clear</th>
<th>Client-Centered</th>
</tr>
</thead>
<tbody>
<tr>
<td>Friendly</td>
<td>Honest</td>
<td>Teamwork</td>
<td>Reliable</td>
<td>Dedicated</td>
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<td>Respectful</td>
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<td>Friendly</td>
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<td>Loyal</td>
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<td>Community Oriented</td>
<td>Collaborative</td>
<td>Sincere</td>
<td>Responsible</td>
<td>Continually Improving</td>
<td>Sensitive</td>
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Other Descriptive Terms:_________________________________________________________________

Your Final Values:

____________________  ___________________  ___________________

____________________  ___________________  ___________________

How would your values align with those of your teammates?

_____________________________________________________________________________

_____________________________________________________________________________

_____________________________________________________________________________

How do your values align with your hospital “Why” and Core Values?

_____________________________________________________________________________

_____________________________________________________________________________

_____________________________________________________________________________

Why do they or don’t they match?

_____________________________________________________________________________

_____________________________________________________________________________

_____________________________________________________________________________
"HOW HEALTHY IS YOUR HOSPITAL?"

The Leadership Team

Who are the leaders in your practice?

_____________________________________________________________________________
_____________________________________________________________________________
_____________________________________________________________________________

How do you choose your leadership team?

_____________________________________________________________________________
_____________________________________________________________________________
_____________________________________________________________________________
_____________________________________________________________________________

Trust

The team members in my hospital trust each other. True or False

Ideas to build trust in my hospital:

_____________________________________________________________________________
_____________________________________________________________________________
_____________________________________________________________________________
_____________________________________________________________________________
_____________________________________________________________________________

Shared Values

In written format, the leadership team has every member of your team answer the following question:

What are the top 3 words YOU would use to describe the personality of ____________ Animal Hospital?

The words are then combined into common terms (example: educator and teacher). From these words, the team chooses the top 4-6 shared values that best describe the organization’s identity as shared by the team. These words define the organization and are used to govern
every aspect of the hospital, from hiring and firing to personnel development and business strategy.

Creating Consistency

1. Pick 2-3 services that your hospitals promote well. Why do these succeed? What could be even better yet?

SERVICE#1: __________________________________________________________
________________________________________________________
________________________________________________________________
________________________________________________________________
________________________________________________________________

SERVICE#2: __________________________________________________________
________________________________________________________
________________________________________________________________
________________________________________________________________
________________________________________________________________

SERVICE#3: __________________________________________________________
________________________________________________________
________________________________________________________________
________________________________________________________________
________________________________________________________________

2. How does your hospital promote effective communication:

Within the team?

________________________________________________________________
________________________________________________________________
________________________________________________________________

To the clients?

________________________________________________________________
________________________________________________________________
________________________________________________________________
What is a Financially Healthy Hospital?

Understanding and Growing Key Financial Performance Indicators

Performance Metrics-Activity One

Midas Touch Veterinary Hospital has an average transaction charge of $175.00, while Average Joe Animal Hospital has an average transaction charge of $125.00. What are some reasons that Midas Touch Veterinary Hospital has a higher average transaction charge?

Performance Metrics- Activity Two

Midas Touch Veterinary Hospital employs three full time veterinarians. Dr. Superstar has an average client transaction (ACT) of $225.00, Dr. Upandcoming has an ACT of $190.00 and Dr. Learning theropes has an ACT of $165.00. What are some factors that impact the variability of the ACT’s for the three doctors?
Performance Metrics- Activity Three

How can a practice increase its new client numbers?

Performance Metrics- Activity Four

What are some possible reasons for the downward trend in transactions?

Why did Medical Transactions/FTE DVM increase?

How can practices increase the average number of transactions/FTE?
Welcome to the 2016 CVMA Convention in Niagara Falls. 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.

The Personal – your home and auto group insurer. Together, The Personal and the Canadian Veterinary Medical Association give you access to all the advantages of group insurance.

Get a quote and compare! 🚗 + 🏡
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 e.g. allergic skin disease, endocrine disease etc. 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
1. To review the latest treatment options available for otitis externa
2. 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 maybe 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. 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, anti-inflammatory agents and anti-microbials, some of which may be the only treatment required.

Multiple ear medications are available and many contain antibiotics, antifungals and anti-inflammatory agents simultaneously. Antimicrobials for canine OE are most often selected empirically based on otic cytology. 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. A topical ticarcillin preparation was found to successfully treat Pseudomonas otitis in a small numbers of cases. Tromethamine edetate disodium dihydrate (Triz-EDTA® Aqueous flush, Dechra Veterinary Products, Kansas, US) is commonly used as an adjunct therapy for dogs with Pseudomonas otitis. It has been documented that flushing the canal with Triz-EDTA® 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 maybe best selected when there is scarce pus within the ear canal. For acute Pseudomonas OE, polymyxin 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 can aid in decreasing inflammation. 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 (extrapolymeric substance, EPS) made of carbohydrates, proteins and DNA. This protects the bacteria from dessication, 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 Triz-EDTA® used in combination with
an antimicrobial, can reduce the MIC and minimum bactericidal concentration (MBC) for certain antibiotics against biofilm embedded bacteria.²⁰

Summary including 5 KEY treatment tips:

1. In cases of OE with stenotic ear canals, anti-inflammatory therapy with oral prednisone or dexamethasone maybe needed for 7-14 days before otoscopic examination or treatment with topical medication is successful.¹ Most cases of OE will benefit from either topical or oral anti-inflammatory treatment with glucocorticoids.
2. Ceruminolytic ear cleaners should be used for cases with excessive cerumen accumulation.
3. 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.
4. 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. 0.5ml for medium dog, 1.0ml for large dogs.
5. Always address underlying etiology.

References/Suggested Reading


“I’VE TRIED X, Y, & Z...WHY IS NOTHING WORKING?” COMMON REASONS FOR DERMATOLOGIC TREATMENT FAILURES

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Adjunct Professor, Atlantic Veterinary College, Charlottetown, PEI

Overview of the Issue
Frustration can arise when treatment instituted for a particular dermatologic disease fails to lead to clinical improvement. Other cases may initially improve, but then show a decline in their condition. Secondary infections, development of another disease or adverse drug reactions can all lead to presumed treatment failure. In these cases of “treatment failure”, diagnostic steps should always be revisited, however, further diagnostics are also likely needed.

Objectives of the Presentation
1. Review common reasons for treatment failure
2. Review steps to take should treatment fail

Key Diagnostic steps:
In any case where you are faced with a patient who is not responding to what you believe is appropriate therapy, there are multiple steps to take to find out why therapy is not working. First and foremost, client compliance must be checked. The correct dose and frequency of administration must be verified with the owner. If the treatment failure involves topical therapy, the owner should be asked how they have been applying or using the topical to make sure application is adequate and correct.

In allergic patients with previously controlled pruritus, who re-present for pruritus +/- dermatologic lesions, cytology should always be performed to check for the presence of a secondary infection. Secondary infections (bacterial pyoderma and Malassezia dermatitis) are common reasons for perceived treatment failure as they mask the full effects of therapy and necessitate higher doses of glucocorticoids and anti-inflammatories to control pruritus. Demodecosis or other ectoparasites must also be ruled out via skin scrapings as they can lead to a worsening of clinical signs in a previously stable patient. An animal who becomes immunocompromised or is receiving high doses of glucocorticoids can develop demodecosis.

Antibiotic resistance has received much attention over the past few years with the incidence of MRSP increasing dramatically during this time. Any animal with a bacterial pyoderma not responding to an appropriate dose and selection of antibiotic, should have an aerobic bacterial culture performed.

Doses of medications and length of treatment should also be revised when dealing with a case of treatment failure. For example, an animal receiving glucocorticoids for atopic dermatitis is presented for non-pruritic hair loss. This hair loss may, in fact, be due to the long-term use of glucocorticoids as opposed to the atopic disease.
**Approach to treatment failure:**

Treatment failure is most often reported when there is a lack of response to glucocorticoids or cyclosporine in a presumed atopic dog. If this occurs, there could be an underlying food allergy and therefore it is imperative to perform an adequate novel protein (or hydrolyzed diet) restricted diet trial. The owner must be informed that no treats, table scraps, pilling vehicles or flavoured medications can be used during the diet trial. The diet trial should involve a diet with a novel protein that the animal has not been exposed to previously and should last for 8-12 weeks to determine whether an improvement is noted.

Cutaneous adverse drug eruptions can lead to a worsening of clinical signs. For example, a dog with a secondary bacterial pyoderma due to allergic skin disease, receives a cephalosporin antibiotic and then develops further dermatologic lesions consisting of erythema, alopecia etc. This animal could be exhibiting signs of a cutaneous adverse drug reaction as opposed to a flare-up of allergies. If pruritus occurs primarily in one location where topical products are applied, one must also consider a contact dermatitis.

In an animal previously responsive to glucocorticoids but now the beneficial effect is waning, consider steroid tachyphylaxis (“steroid fatigue”). The steroid can be switched to another glucocorticoid e.g. oral dexamethasone, Methylprednisolone.²

If there is a lack of response to a correct dose of cyclosporine, cyclosporine levels can be checked. However, in atopic dogs there does not appear to be a correlation between blood concentrations and clinical response.³ In an atopic patient, if there is a minimal response to glucocorticoids and cyclosporine and cytology, skin scrapings etc are all negative, one must consider either a case of pruritus refractory to both glucocorticoids and cyclosporine or an incorrect diagnosis. At this point diagnostic steps should be retraced and revised if necessary. Skin biopsies sent for histopathology can also be a great way to help eliminate and diagnose. Immune mediated disease can usually be diagnosed via histopathology. Other dermatological diseases, such as cutaneous epitheliotropic T cell lypoma, can present with clinical signs similar to atopic dermatitis but are non responsive to conventional anti-inflammatory therapy. If histopathology is consistent with atopic dermatitis then more aggressive immunosuppressive therapy may be needed in these refractory cases.

Prior to beginning systemic immunosuppressive therapy, all patients should have full bloodwork performed (complete bloodcount and serum biochemistry) as well as a urinalysis. Azathioprine has been used in certain cases to treat refractory canine atopic dermatitis at a dose of 2-2.5 mg/Kg once daily.⁴ Further bloodwork is also recommended every 2-4 weeks after starting therapy due to the potential adverse effects such as myelosuppression and hepatic toxicity (increase in ALT, ALP).

Pruritus can originate from regions other than the skin. Pruritus maybe associated with pain or can also be neuropathic. Studies are lacking to document efficacy of the following medications for treatment of pruritus but could be considered in specific cases. Gabapentin is used in humans to treat neuropathic and uremic pruritus.⁵ Side effects include sedation and ataxia. Maropitant is a neurokinin-1 receptor antagonist that inhibits substance P. This medication can have an anti-pruritic effect.⁶ Maropitant
is licensed to prevent and treat vomiting in dogs as a dose of 2 mg/kg once daily. This
dose has also been suggested to decrease pruritus. Oclacitinib, a Janus Kinase
inhibitor, inhibits the function of pro-inflammatory and pruritogenic cytokines.
Preliminary studies show that treatment with Oclacitinib decreases pruritus fairly
rapidly in dogs suffering from atopic dermatitis (currently not licensed in Canada).⁷

Summary including 5 KEY “TAKE HOME” POINTS:
1. In any case where treatment is not efficacious, cytology and skin scrapings
   should be performed and client compliance should be verified.
2. Consider potential antibiotic resistance in a patient with bacterial pyoderma,
   confirmed on cytology, which is unresponsive to antibiotics.
3. Taking skin biopsies and sending for dermatohistopathology can provide more
   information in cases of treatment failure.
4. In certain pruritic cases consider non-conventional therapy or changing the
   type of glucocorticoid prescribed.
5. In a pruritic patient non responsive to therapy for atopic dermatitis, consider
   food allergies, sarcoptes or cutaneous epitheliotropic T cell lymphoma as an
   underlying etiology.

References/Suggested Reading
1. Weese JS, van Duijkeren E. Methicillin-resistant Staphylococcus aureus and
   Staphylococcus pseudintermedius in veterinary medicine. Vet Microbiol. 2010;
   140: 418-429.
2. Miller WH, Griffin CE, Campbell KL. Dermatologic Therapy. In: Miller WH, Griffin
   CE, Campbell KL. Muller and Kirk’s Small Animal Dermatology 7th ed. St Louis,
3. Cotes MES & Swerlick RA. Practical guidelines for the use of steroid-sparing
4. Favrot C, Reichmuth P, Olivry T. Treatment of canine atopic dermatitis with
   1625-1634.
   and safety of oclacitinib for the control of pruritus and associated skin lesions
TREATMENT OF SECONDARY INFECTIONS IN VETERINARY DERMATOLOGY

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Overview of the Presentation
Secondary infections can be frustrating to treat due to underlying etiologies causing inflammation and increased susceptibility to infection. Resistant bacterial infections add to this frustration due to fewer treatment options available. These infections, when present, can mask the benefits of anti-inflammatory therapy.

Objectives of the Presentation
1. To review topical and systemic treatment options for Malassezia dermatitis
2. To review topical and systemic treatment options for both susceptible and resistant bacterial pyoderma
3. To review treatment options for recurrent bacterial pyoderma

Secondary infections due to bacterial and yeast species are a common sequela of underlying skin disease. Inflammatory mediators, disrupted barrier function, moisture along with other factors, all predispose an individual to developing a secondary infection. If left untreated these secondary infections can lead to treatment failure.

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.

Diagnosis of Malassezia dermatitis
Malassezia dermatitis is often under diagnosed and plays a large role in dermatologic disease. The most common cause of yeast dermatitis and otitis in dogs and cats is Malassezia pachydermatis; which is a part of the normal flora in these same individuals. Clinically a Malassezia dermatitis will present with erythema, brown debris around the nail bed or on the skin, alopecia, signs of self-trauma and crusting. This author tends to prefer acetate tape preparations as the cytology of choice for diagnosing Malassezia dermatitis.
When to perform a bacterial culture and susceptibility
A bacterial culture and susceptibility should be performed:
1. If rods are noted on the skin in large numbers
2. If there is a deep pyoderma (furunculosis)
3. If there is a mixed infection
4. If a resistant bacterial population is suspected

A resistant bacterial population should be suspected if:
1. Animal has been on the correct dose of antibiotics for an appropriate length of time (3-4 weeks) and there is no resolution of the lesions.
2. Animal has been on multiple courses of antibiotics that it previously responded to, and it is now not responding

Treatment duration
Treatment of a superficial bacterial pyoderma or a Malassezia dermatitis 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
1. Antibacterial ointments/creams (applied daily)
2. Antibacterial shampoo (5-10 minute contact time q 2-7 days)

Systemic treatment
*S. pseudintermedius* is a ß-lactamase producer therefore antibiotics resistant to this enzyme must be selected. Cephalosporins (Cephalaxin, cefpodoxime, cefovecin), Amoxicillin-clavulanate, 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 pyodermas, 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
1. Human Interferon alfa-2b – given orally at 1000 IU/m/day
2. Staphage Lysate® (Delmont Laboratories) – Autogenous staphylococcal bacterin, S. aureus phage lysate.
   a. Combat potential bacterial hypersensitivity
   b. 0.5ml, SQ, twice weekly for 10-12 weeks (Induction)
   c. 0.5 ml, SQ q 7-14 days (maintenance)

Treatment options for Malassezia dermatitis

Topical treatment
1. Antifungal ointments/creams (applied daily)
2. Antifungal shampoo (5-10 minute contact time q 1-7 days)
3. Antifungal sprays (applied daily)
4. Antifungal wipes (applied daily)

Systemic treatment
There are only rare reports of resistance to Malassezia species therefore resistance does not need to be taken into account when a treatment is selected. Oral azoles such as ketoconazole and itraconazole are effective against Malassezia and there is also evidence to support the use of oral terbinafine. Prior to azole therapy, bloodwork should be considered to verify there are no systemic abnormalities that would prevent therapy.

When treating either a bacterial or Malassezia dermatitis, therapy should continue for a minimum of 3-4 weeks and, at this time, cytology should be repeated to verify the infection has fully resolved.

Summary including 5 KEY take home points:
1. Cytology is the diagnostic tool of choice for secondary infections.
2. Bacterial cultures should be performed if there is suspicion of a resistant bacterial species e.g. no response to appropriate therapy, persistence of a bacterial pyoderma despite therapy.
3. Both bacterial and yeast dermatitis should be treated for a minimum of 3-4 weeks and cytology should then be repeated to determine that the infection has resolved.
4. Topical and systemic options are available for treatment of secondary infections
5. If secondary infections are recurrent in nature, consider an underlying pathology as the trigger for the infections.

Further Reading:
- Hendricks A, Schuberth HJ, Schueler K, et al. Frequency of superantigen producing *Staphylococcus intermedius* isolates from canine pyoderma and

CANINE ATOPIC DERMATITIS: ALLERGY TESTING AND IMMUNOTHERAPY

Dr. Vincent Defalque, Diplomate of the American College of Veterinary Dermatology

Definitions and prevalence
Canine atopic dermatitis is a genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features associated with immunoglobulin E antibodies most commonly directed against environmental allergens. It is becoming increasingly common in everyday practice, affecting up to 10–30% of the entire canine population.

A second entity called atopic-like dermatitis is now recognized. It is defined as an inflammatory and pruritic skin disease with clinical features identical to those seen in canine atopic dermatitis in which an immunoglobulin E response to environmental or other allergens cannot be documented.

Causes
An aeroallergen is an airborne substance which can trigger an allergic reaction in a sensitized patient. House dust mites (Dermatophagoides farinae and D. pteronyssinus) and storage mites (Tyrophagus putrescentiae, Acarus siro and Lepidoglyphus destructor) are the most important environmental allergens worldwide. The presence of these mites (or their allergens like Derf15 et Derf18) in the bedding, skin/hair coat, household and dry food of dogs has been confirmed. The tiny pollens of wind-pollinated plants are also implicated. Pollens of insect-pollinated plants (too large to remain airborne) may also pose a risk. Mould spores and epidermals (e.g. cat dander, bird feathers) are less frequently involved.

Pathogenesis
An older paradigm implied an immunological defect (inside-out theory) involving both immunoglobulin E mediated/immediate hypersensitivity (type I) and cell-mediated/delayed hypersensitivity (type IV) reactions. More recently, a primary defect in the skin barrier function has been recognized (outside-in theory).

There are 4 routes of exposure to environmental allergens (percutaneous +++ > inhalation, ingestion, conjunctival).

Favrot’s diagnostic criteria
The diagnosis of canine atopic dermatitis relies primarily on the patient’s signalment, clinical signs and disease history and not on results of laboratory tests (see below). Diagnostic criteria published in 2010 by Dr Favrot can be used in practice as an aid to diagnosis of canine atopic dermatitis while keeping in mind that these criteria are not absolute. They have a sensitivity 85% and a specificity of 79%. In other words, up to one of five cases could be misdiagnosed if these parameters were to be applied strictly! However, with proper rule-out of ectoparasites and skin infections, the specificity of the criteria increases markedly.

Combination of any 5 out 8 criteria:
1. Onset of clinical signs under 3 years of age (>< cutaneous adverse food reaction, at any age)
2. Dog living mostly indoors
3. Glucocorticoid-responsive pruritus
4. Pruritus without macroscopically visible lesions at onset
5. Affected front feet
6. Affected ear pinnae
7. Non-affected ear margins (> scabies)
8. Non-affected dorso-lumbar area (> flea allergy dermatitis)

Diagnostic methods in veterinary medicine
Two main types of environmental allergy tests are used:

1. The intradermal test screens ‘in vivo’ for the presence of allergen-specific immunoglobulin E bound to the surface of dermal mast cells and their ability to degranulate upon exposure to an allergen (visualization of immediate wheal-and-flare reaction, type I hypersensitivity).

2. The allergen-specific immunoglobulin E serum test screens ‘in vitro’ for the presence of, and measures the level of allergen-specific immunoglobulin E circulating in the peripheral blood.

Neither test is perfect! In other words, there is no allergy test with a specificity and sensitivity of 100%. Veterinarians can use either allergen-specific intradermal or immunoglobulin E serological tests (or a combination of both) to identify the offending environmental allergens as there is no clear evidence that the response to allergen-specific immunotherapy is superior using allergens selected by intradermal or serum tests. There is consensus among veterinary dermatologists to say that environmental allergy tests cannot be used for the initial diagnosis of canine atopic dermatitis, or to differentiate between atopic dermatitis and other causes of pruritus. These tests are never a good substitute for a thorough dermatological work-up! Many normal and atopic patients exhibit positive reactions with either test, thereby markedly decreasing their specificity. Using a serum test or intradermal test as a primary criterion for diagnosing atopic dermatitis could, therefore, lead to misdiagnosis. Negative test results do not rule out environmental allergies because all appropriate allergens may not have been included in the panel.

Environmental allergy tests should only be used for the 3 following reasons:

1. To document whether the skin disease is associated with allergen-specific immunoglobulin E (in other words to differentiate between canine atopic dermatitis and atopic-like dermatitis).

2. To implement allergen-avoidance measures.

3. More importantly, to select allergens to be included in allergen-specific immunotherapy preparations. Environmental allergy test results must be interpreted in light of the history, seasonality of the pruritus and the patient’s environmental allergenic exposure. In other words, the allergens identified must perfectly fit seasonal exacerbation patterns and there must be likely exposure based on the history and geographical location.

Allergen-specific immunotherapy

Allergen-specific immunotherapy involves the administration of gradually increasing quantities of specific allergens to patients with immunoglobulin E-mediated conditions until a dose is reached that is effective in reducing disease severity from natural exposure.
The exact mechanism(s) of action by which immunotherapy achieves improvement of clinical signs is still unclear. One can reasonably assume that the hypothesis of blocking antibodies are solely involved is an oversimplification and that no single mechanism explains the efficacy of immunotherapy in dogs.

The time to efficacy (i.e. how quickly immunotherapy works) and treatment duration (i.e. the exact total duration of immunotherapy before being able to determine treatment efficacy) are currently unknown in veterinary medicine.

In recently published clinical practice guidelines for the treatment of canine atopic dermatitis, because the onset of clinical benefit might not appear for several months, the current consensus among veterinary dermatologists is that:

- Immunotherapy must be continued for at least 1 year to properly evaluate efficacy.
- Anti-inflammatory drugs can be given temporarily, as needed to maintain good quality of life until such time as the immunotherapy is judged to be effective.

Despite a lack of evidence based on randomized controlled trials, results of open uncontrolled studies and a wide body of global observation by veterinary dermatologists suggest that subcutaneous aqueous immunotherapy is efficacious for the treatment of canine atopic dermatitis. It is expected that between approximately 60% and 80% of dogs with atopic dermatitis that have been treated with subcutaneous immunotherapy for six to twelve months will exhibit an improvement in clinical signs and/or a decrease in anti-inflammatory medication use. The response rate for dogs treated with sublingual immunotherapy that previously failed to respond adequately to subcutaneous immunotherapy could be substantial.

At this time, there appears to exist no superiority of a particular immunotherapy protocol over other ones (subcutaneous: traditional, rush or low-dose; sublingual) in terms of efficacy.

Immunotherapy should not be presented to the owner as a curative treatment.

**Considerations when selecting subcutaneous vs. sublingual immunotherapy**

- Schedule and convenience
- Aversion to needles
- Patient cooperation
- History of adverse reactions due to subcutaneous immunotherapy
- Treatment failure with subcutaneous immunotherapy
FOOD ALLERGY IN DOGS AND CATS: COMPARATIVE ASPECTS AND EVIDENCE-BASED RECOMMENDATIONS

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Classification, pathogenesis and symptoms
In human medicine, "food allergy" refers to adverse food reactions involving a clearly demonstrated humoral response (mediated by immunoglobulin E). Other food hypersensitivities involve cellular immune responses (mediated by T lymphocytes). Food intolerance, by contrast, is a non-immunologic adverse food reaction. The exact mechanisms of food allergy/intolerance are not fully understood in pets. Contrary to people, these reactions can be quite delayed in time, usually taking days to a few weeks to develop (delayed-type hypersensitivity). Therefore, the all-encompassing term "cutaneous adverse food reaction" is preferred in veterinary medicine.

Cutaneous manifestations (pruritus, otitis, and skin infections) are often accompanied with digestive signs (vomiting, diarrhea, flatulence, increased fecal frequency) and are rarely outgrown.

Causes
A trophallergen (food allergen) is a usually harmless heat-stable, water-soluble glycoprotein of animal or vegetal origin which can trigger an allergic reaction in a sensitized patient.

The main commercial pet food ingredients are animal proteins and grains. When animals are slaughtered, muscle tissue and a few organs are used for human consumption, while the remaining parts of the carcass (by-products) are used in pet food. By-products are submitted to a thermal process known as rendering that separates fat from bone and lean parts to obtain meat and meat-and-bone meals that are the main source of animal proteins in pet foods.

Only a small variety of foods (mainly animal proteins and a few grains) are clearly implicated in dogs and cats according to the current veterinary medical literature. Adverse reactions to chicken, beef, dairy products and wheat account for over 75% of all the reported cases in dogs. In this species, adverse reactions to chicken egg, soy, lamb, fish and corn are reported much less frequently, while reactions to rice and duck are rare. In cats, adverse reactions to chicken, chicken egg, beef, dairy products and fish account for over 90% of all the reported cases.

Debunking the myth of grain and gluten allergy
Some pet food companies or salespeople warn owners against corn, wheat or soy for marketing reasons. They are looking for ways to distinguish themselves from their competitors by developing a mythology about why these grains are harmful to pets. In reality, there are very few confirmed cases of grain-related cutaneous adverse food reactions in dogs and cats. There are no published reports of gluten (a protein found in wheat) allergy-induced skin disease in dogs and cats. Gluten sensitivity is rare in dogs. It cause an enteropathy in Irish setters, Samoyeds and soft-coated Wheaten terriers. It has not been reported in cats.

Diagnostic methods in veterinary medicine
Definitively diagnosing a cutaneous adverse food reaction in dogs and cats remains a challenge. Results can be ambiguous! Definitive dermatohistopathological diagnosis of this skin disease is not possible. Both intradermal and allergen-specific immunoglobulin E serum testing for food allergens are unreliable. Endoscopic methods (observation of reactions on gastric or colonic mucosa after direct injection or application of food extracts) may be helpful but are invasive (need for general anesthesia and expensive equipment). Unfortunately, the only currently reliable method to identify patients with cutaneous adverse food reactions is to perform an elimination diet trial for a sufficient time while controlling concurrent allergies and secondary infections. This is easier said than done! The term "elimination diet" is preferred to "hypoallergenic diet" because any reduction in allergenicity or clinical reactivity at which point a diet could be considered hypoallergenic is arbitrary.

The 3 steps of a food trial

Step 1 - Elimination diet
Slowly introduce the elimination diet! Requires strict owner compliance: the diet is to be fed exclusively! No treats (unless compatible), rawhides, edible toys and bones. No flavored medications, preventatives, supplements and toothpaste.

Step 2 – Challenge
Once patients are symptom-free, reintroduce the previous food (that was thought to cause the symptoms). If clinical signs return within 14 days, an adverse food reaction is confirmed! This does not differentiate between mechanisms of adverse food reactions (immunologic vs. non-immunologic) but establishes a relationship between certain ingredients and observable clinical signs. This step may be unacceptable to some owners (optional?).

Step 3 – Sequential provocation
This is the only way to identify the exact offending allergen(s). Feed elimination diet until pruritus resolves. Add 1 suspected offending ingredient at a time for up to 14 days, in order to provoke the reaction again. Clinical reactivity is ruled out if there has not been a reaction within 2 weeks. Examples: chicken, beef, lamb, fish, soy, corn, wheat.

Repeat sequence.

This step may be unacceptable to some owners (optional?).

Duration of food trial, control of pruritus and appropriate timing
There are variable recommendations in the veterinary literature regarding the length of time before seeing clinical improvement once a patient is placed on the elimination diet, with some authors recommending a 12-week trial. In reality, it is rare for a food-allergic patient to not show measurable improvement within 4 to 6 weeks, so 6 weeks is a reasonable recommended length. If pruritus is decreased by at least 50% during this time, a diagnosis of adverse food reaction can be presumed. Whenever no clinical improvement is observed during the food trial, an adverse food reaction is ruled out and other diseases such as atopic dermatitis or non-allergic pruritic disorders may be suspected. Requiring owners and patients to struggle on for 12 weeks without seeing improvement in clinical signs can cause many owners to lose faith with the entire application.
process, leading them to abandon the food trial and possibly seek out a different opinion. When starting an elimination diet trial, clinical signs of gastro-intestinal disease will usually resolve within 2 weeks. Cutaneous clinical signs will usually take longer to improve. It may require more than six weeks for the maximum improvement to be seen, but at least the patient is improving during the process, which provides encouragement to continue the trial. During the food trial, it is important to minimize the other causes of pruritus that will interfere with the ability of the owner and veterinarian to determine the success or failure of the food trial. Monitoring for and treating secondary infections (bacterial pyoderma and Malassezia dermatitis) is therefore necessary. These infections are often the reason a food trial is being performed in the first place, so it is not uncommon to treat the patient with appropriate antimicrobial therapy for the first half of the food trial. Further counseling is then needed to ensure the medications are not administered in a noncompatible treat. Even oral/topical antipruritic therapy may be required during the first half of the food trial. A food trial is best performed during the winter months (when the patients are only exposed to house dust and storage mites) if there is a warm weather seasonal exacerbation in the history of the patient.

**Debunking the myth of the silver bullet**
There is no foolproof "works every time" or "one size fits all" diet. Choosing the best diet to feed a patient with a suspected food allergy requires careful and detailed questioning of owners regarding previous and current diets, treats and flavored products. While you take your history, use an internet search engine for each diet in order to find out the nature of the animal proteins and grains that are included. Once that information is known, you must choose a diet that:
1) consists of proteins/grains to which the patient has not had (or very little) exposure in the past (which means it should not contain the more commonly used ingredients).
2) has minimal chance of cross reactions with previously fed proteins.
   - Chicken-chicken egg- turkey-duck-goose-fowl-quail- pheasant
   - Beef-veal-lamb-goat-sheep-buffalo-bison-milk-cheese-butter-deer-venison-elk-moose
3) will be eaten by the patient.
4) will be readily fed by the owner (cost?).

Because of all these factors, diets containing rabbit, kangaroo and, occasionally, fish are the novel protein diets of choice for most patients with suspected food allergies. One should be confident that the manufacturer of the food has truly kept the food limited to what is stated on the label and not allowed contamination with other feeds or proteins.

Hydrolyzed protein diets are also available, with hydrolyzed chicken- and soy-based foods being the most common. Several published studies have reported the majority of patients fed hydrolyzed diets have improvement in clinical signs, even if they are allergic to the parent protein. Yet other studies show up to 50 percent of patients with food allergies flare or fail to improve while eating a hydrolyzed diet. A recent evidence-based report summarized all the various (and sometimes conflicting) articles on the subject and concluded hydrolyzed diets should not be used if a patient could potentially be sensitized to the nonhydrolyzed protein (also known as the parent or native protein).
**Over-the-counter diets**

Pet owners often express an interest in using over-the-counter (OTC) commercial diets sold through retail outlets for the purpose of a food trial. They prefer this option over home-cooked or veterinary diets because of price and convenience. Veterinarians may sometimes acquiesce to such client requests. The selection is often based on the name of the product (venison and potato for example), which ensures nothing more than the product will contain at least 3% of those ingredients.

The assumption is often made that if a food allergen is not named in the ingredient list, then the product does not contain it and, therefore, is a suitable diet for a food trial. The difference and value in product quality control between veterinary therapeutic and OTC commercial lines of pet foods is not fully understood.

The discrepancies between enzyme-linked immunosorbent assay (ELISA), polymerase chain reaction (PCR) and microscopic analysis results and the product labels are key to understanding why OTC diets may not be suitable for a food trial. The presence of unwanted ingredients in these diets is most likely due to cross contamination. This contamination may occur during manufacturing in a wide variety of ways. Pet food manufacturers typically use standards of quality control appropriate for the intended use of the product. OTC diets are not intended for use in diagnostic elimination trials. The companies making veterinary therapeutic diets designed specifically to be used in elimination trials use sophisticated levels of ingredient and product quality control measures to prevent food allergen contamination from the initial handling of raw ingredients to the final stages of packaging.
Veterinary neuro-ophthalmology is the transition between neurology and ophthalmology and relies heavily on neuroanatomy and neurophysiology. As an intermediary, neuro-ophthalmology has typically been a neglected area of study from both neurologists and ophthalmologists. For the purpose of providing a simple but thorough overview of neuro-ophthalmology so that observations and testing can be made and performed in a simple way in daily clinical practice, I have broken up this seminar into four basic areas that include: 1. the visual sensory system (retina – visual cortex), 2. the autonomic nervous system (pupillary function, lacrimation), 3. the neuromuscular system (neural control of the eyeball, eyelid, and third eyelid position and movement), and 4. the trigeminal somatic sensory system (pain sensation) of the eye and adnexa. A short discussion will be made regarding the three most commonly encounter clinical neuro-ophthalmic conditions; anisocoria, strabismus, and blindness.

One of the most important aspects of identifying and subsequently localizing an ophthalmic lesion is derived from a thorough history. When concerns regarding vision deficits are raised, identifying a pattern with respect to environment (new/familiar), lighting conditions (bright/dim), as well as rate of speed (fast/slow) when deficits occur can be extremely helpful. Performing a thorough ocular examination can also help to determine if a vision deficit is functional (visual sensory system failure) or mechanical (image cannot reach brain secondary to a mechanical obstruction – eg cataract, opaque ocular medium, etc). In clinic vision testing can be achieved through obstacle (maze) testing in both scotopic and photopic conditions.

1. The visual sensory system is the transmission of light energy converted to electrical energy by the retina to the visual cortex via the optic nerve (CN II) and optic radiations where the signal is received and interpreted. The oculomotor nerve (CN III) is involved in this system but only as the efferent response to CN II activity and not for proof of receipt of an image into the visual cortex. In-clinic testing of CN II and III include examination of pupil size and symmetry, the swinging flashlight test, and dark adaptation tests. Cognition of a visual stimulus is difficult to assess but can be inferred to be intact when there is a positive menace response (cortical), visual cliff and visual placing reactions, and positive visual field test responses. A negative menace response does imply that the patient is blind as lack of a positive response may be indicative of youth, mechanical obstruction (opaque ocular media), CN VII deficit (inability to blink), or simply being a cat! The dazzle reflex by definition is without cognitive control and is far less reliant on the need for a clear ocular medium. A positive dazzle reflex supports an intact visual sensory system and a negative dazzle reflex supports blindness provided CN VII and III are intact. Defects in the visual sensory system are suggested by anisocoria and/or vision deficits affecting one eye or both. Disease processes that can affect the visual sensory system at the level of the retina include PRA, SARD, or retinal atrophy. At the level of the optic nerve, optic neuritis, trauma, or compressive lesions can result in vision deficits or blindness, and at the level of the brain can include inflammation or tumor formation. More sophisticated testing includes electroretinography (specific to retinal function) and visual evoked potentials (VEP’s).
After ruling out the globe, a thorough cranial nerve examination can often localize the point of injury or inflammation to within the optic nerve(s), optic tract(s), or brain.

2. The autonomic nervous system of the eye is responsible for lacrimation and in part for pupillary function. The clinical signs most commonly associated with defective autonomic control of the eye include anisocoria (mydriasis or miosis), keratoconjunctivitis sicca, elevation of the third eyelid, ptosis, and enophthalmia. Most commonly, ophthalmic abnormalities related to the autonomic nerve system are attributed to Horner’s Syndrome, however the potential for cavernous sinus syndrome should not be ignored if Horner’s Syndrome cannot be confirmed or corneal sensitivity is absent. Additional testing for the presence of Cavernous Sinus Syndrome involves testing of CN’s III, IV, VI, V1, and V2.

3. The oculomotor system controls position and movement of the globe (CN III, IV, VI, VII), eyelids (CN III, VII), and third eyelid (sympathetic NS). All extraocular muscles are innervated by CN III with the exception of the lateral rectus (CN VI) and the superior oblique (CN IV) giving rise to the commonly remembered phrase LR6 SO4. Unilateral or bilateral clinical signs present as strabismus, ptosis, corneal ulceration, enophthalmia, or exophthalmia. Globe movement, range of motion (ROM), vestibulo-ocular reflex (VOR, oculocephalic reflex, doll’s eye), saccadic system testing, and examination of ocular alignment are used to examine the extraocular muscles. By producing eye movements in the direction opposite to head movement, the doll’s eye reflex is stimulated by the vestibular system and functions to stabilize images on the retinas (yoked vision) during head movement, thus preserving the image on the center of the visual field(s).

Range of Motion involves duction (the ability of one eye (fellow eye is covered) to rotate while viewing an object when the head is motionless) and version (= the extent of globe rotation while both eyes are viewing an object and the head is motionless). Any limitations must be noted and muscle paresis (neurogenic) vs mechanical restriction (fibrosis, bony orbital fracture’s with entrapment) considered. A restrictive limitation to motion is identified when the globe cannot be moved by forced duction (traction) tests. Paretic limitations are identified when the globe is found to be moveable by forced duction testing. Lesions of paresis can be supranuclear (eg cerebral cortex, brainstem) or peripheral (eg infranuclear). Differentiation of supranuclear and infranuclear disease is performed via VOR testing. Muscle weakness or mass effect to cause strabismus can also be determined by examining ocular alignment via the corneal light reflex (divergence, convergence) or torsion/vergence alignment (esp in cats).

4. The trigeminal somatic sensory system is responsible for pain sensation of the eye and adnexa. While the efferent response to stimuli is manifested through cranial nerve VII, it is cranial nerve V and its ophthalmic (VI) and maxillary (V2) branches that innervate the face (V1-2), eyelids (V1 =medial canthus, upper eyelid. V2 = upper and lower eyelid), and cornea (V1). These branches can be tested to aid in the localization of a lesion. Clinical signs of reduced facial, corneal, or eyelid sensation may result in a reduced palpebral reflex which is driven by corneal sensation and subsequent corneal ulceration.
All neuroophthalmic abnormalities are uncommon when ranked by the caseload array of a busy general practice, however several specific conditions do arise with surprising frequency. These include anisocoria, strabismus, and blindness.

Anisocoria is by definition ‘inequality in the size of the pupils of the eyes’, a simple concept until asked, ‘which pupil is abnormal?’ or ‘is CN II or CN III affected’? CN II is responsible for afferent stimulus to the brain and CN III is the efferent response from that stimulus. Mydriasis suggests blindness secondary to loss of an afferent signal (via CN II) to the optic chiasm and as such, absence of the efferent response (via CN III) of pupil constriction to the contralateral eye (consensual PLR). Presence of a contralateral PLR supports CN II function of the mydriatic eye and in turn supports either a lesion in the ipsilateral eye that is located in the ciliary ganglion or the pre-tectal nucleus (where CN III originates), or a post-ganglionic restriction to pupil response (physical restriction by synechia/advanced iris atrophy, or pharmacologic impairment to constriction). Determination of CN II lesions caudal to the lateral geniculate nucleus can be determined indirectly via the menace response (cortical) or dazzle reflex (subcortical), or directly via VEP testing. Miosis suggests a lack of sympathetic tone to the ipsilateral pupil provided the consensual pupillary light reflex is intact (supporting ipsilateral CN II function). The swinging flashlight test in both dim and bright light will determine which is the affected eye and if you are dealing with a CN II or CN III deficit. One differential for a visual miotic eye without evidence of physical or pharmacologic constriction is Horner’s Syndrome. Horner’s Syndrome is a constellation of clinical signs caused by a break/irritation in one of three segments of the sympathetic chain originating in the brain and travelling to the eye via the neck and ear. The causes of such a break/irritation can be numerous and include mild middle ear infection (most common), head trauma, neck/brain injury or disease, or are idiopathic. The clinical signs commonly seen in dogs and cats include miosis, ptosis, enophthalmia, and elevation of the nictitans. Uncomplicated Horner’s syndrome signs typically resolve spontaneously within 4-6 months. Horner’s syndrome is not painful or blinding however, some visual impairment can be noted if the third eyelid is markedly elevated or the condition is bilateral.

Strabismus may be congenital, acquired, or positional. Congenital strabismus is typically bilateral and can be inherited (Siamese/Himalayan cats) or secondary to fetal malformation and hydrocephalus. Acquired strabismus often presents acutely and unilaterally and suggests trauma that resulted in damage to one of the extraocular muscles (usually the medial rectus muscle), or displacement of the globe secondary to orbital fracture. Slowly progressive unilateral strabismus is much more likely to be the result of globe displacement by neoplasia, abscess, or cellulitis. When bilateral and slowly progressive, extraocular muscle paralysis or fibrosis (eg Shar Pei progressive juvenile esotropia) is suspected. The muscle(s) of interest can be determined by performing range of motions tests usingduction and version testing. Paralysis/paresis and entrapment (via fibrosis) of the muscle(s) in question can be differentiated by active and passive forcedduction tests while moving the globe towards and away from the abnormal direction of gaze. Positional strabismus (doll’s eye) is a sign caused by lesions to the afferent vestibular pathways and only appears when the head is moved in certain planes, and corrected when the head is moved back to its former position. Lesions in the afferent vestibular pathway can be demonstrated in small animals by elevating the muzzle. Normally, this causes a corresponding dorsal retraction of the
eyes. In patients with lesions to the afferent vestibular pathway, the eye in the affected side will remain in its frontal position when the muzzle is elevated, giving the appearance of a ventral strabismus.

When presented with a case of ‘blindness’ it is important to first determine if the animal is truly blind (negative dazzle reflex), has impaired vision (secondary to advanced lenticular sclerosis, partial retinal detachment, cloudy media), or is mechanically obstructed by opaque ocular media (advanced corneal pigment, cataract, lipid aqueous). Patients with any of the above conditions may present clinically in the same way. Clinical history and a good ocular examination can rule out a mechanical obstruction to vision and a negative dazzle reflex will usually confirm the lack of an adequate afferent light signal (via CN II) to the brain. When possible, examination of the fundus will provide insight into the presence of retinal degeneration or atrophy as well as some cases of optic neuritis. Unless very advanced, a direct and consensual PLR is often still present to some degree with PRA and even slowly developing SARD. The ipsilateral direct and contralateral consensual PLR will be absent in cases of optic neuritis but will be present when the light is directed into the contralateral eye. Some clear differences between PRA, SARD, and optic neuritis can be identified clinically.

<table>
<thead>
<tr>
<th>Disease</th>
<th>Are both eyes affected?</th>
<th>Rate of onset</th>
<th>Retinal changes</th>
<th>ERG testing</th>
<th>Etiology</th>
<th>Is patient systemically ill?</th>
</tr>
</thead>
<tbody>
<tr>
<td>PRA</td>
<td>yes</td>
<td>slow</td>
<td>yes</td>
<td>low-flat</td>
<td>inherited</td>
<td>no</td>
</tr>
<tr>
<td>SARD</td>
<td>yes</td>
<td>fast</td>
<td>not initially</td>
<td>flat</td>
<td>?</td>
<td>possibly</td>
</tr>
<tr>
<td>Optic neuritis</td>
<td>yes/no</td>
<td>fast</td>
<td>no</td>
<td>normal</td>
<td>acquired</td>
<td>likely</td>
</tr>
</tbody>
</table>

Intracranial disease must always be suspected in cases where multiple cranial nerves are affected. When visual and multiple cranial nerves are affected, a high degree of suspicion for cavernous sinus syndrome is raised. When blind, suspicion for meningioma is increased. Definitive identification is accomplished with advanced imaging using CT/MRI and while chemotherapy therapy is often initiated, such tumors will eventually claim the life of these patients.
“Hey Doc, how well does my dog see?”

Such a simple question for which the answer is, well….not simple. Visual ability of normal animals is seldom discussed in the veterinary literature. It is a subject rich in uncertainty of testing methods and our own biases towards the importance of vision in all animals. What is known is that vision is the collective summary of a) the ability to perceive light and motion, b) visual perspective, c) field of view, d) depth perception, e) visual acuity, and f) colour vision. Each of these subjects will be explored.

The world as we know it is geared for human vision. Nearly everything man-made is geared towards how we would perceive it…from the pictures we hang on our walls to the toys be buy our pets. How different the world must be for dogs. I like to use the analogy that life on earth for a dog might be similar to what life would be like for use on a planet where scent is the strongest cue. Without significant training, we certainly could not make our way to work in the morning based on our senses of smell and hearing alone. Perception of light and motion are fundamental aspects of vision that are augmented by perspective, field of view, depth perception, acuity, and colour vision. I am hoping to provide a better understanding of how dogs and cats use their eyes, and that in turn, a better appreciation of what information is being gathered by senses other than vision.

There is a paucity of published information regarding vision in animals however, there are several excellent review articles and it is from these that much of this seminar is based. I am hoping to dovetail these previously published works with visual examples. For additional information, I encourage you to review the reference list below.

a. Sensitivity to light, motion, and flickering lights
- dogs are not strictly diurnal or nocturnal but rather have an arrhythmic photic existence.
- cats and dogs are more sensitive to light and motion than humans
- dogs are generally less adapted than cats and do not possess a vertical slit pupil but are still much better than humans
- cat minimum light detection threshold is ~ 6 times less than humans

Adaptations to dim light
- tapetum lucidum (L. bright tapestry)
  - cats - reflects up to 130 times more light than human fundus
  - choroidal tapetum cellulosum = 9-20 cell layers thick, zinc cysteine (dogs) and riboflavin +/- zinc (cats)
  - located superiorly in the eye to brighten the view of the darker ground (while the inferiorly located darkly pigmented non-tapetal fundus reduces light scattering from a bright sky).
Increases retinal sensitivity by reflecting light back through the photoreceptor layer twice.

- May represent selective visual adaptations associated with feeding behaviour.
- Cats – fluorescent shift - tapetum shifts short wavelengths (450 nm, blue) to longer wavelengths (520 nm) closer to rhodopsin’s maximal sensitivity to brighten a blue-black night sky and enhance contrast.
  - Larger cornea – more light enters eye.
  - Central retina (25°) is dominated by rod photoreceptors (vs cones domination (colour and bright light vision) in humans).

Rhodopsin
- Photopigment specific to rods.
- Increases in sensitivity to light for up to an hour.
- Peak sensitivity to light wavelengths of 506-510 nm function well in dim light (humans=496 nm).
- Takes up to an hour to regenerate after exposure to bright light (a common finding in animals adapted for dim light conditions). Humans regenerate rhodopsin much quicker.

**Sensitivity to moving objects**
- Greater than sensitivity to stationary objects – an obvious evolutionary survival skill.
- In bright light – humans (cone rich fovea) detect moving objects 10-12 times better than cats.
- In dim light - domestic mammals (rod dominant) detect moving objects better than humans when the object is viewed peripherally, or when the object moves at a speed to which the retina is attuned.
- In peripheral visual field, most dogs and cats ignore stationary objects but reflexively chase them if they move.

**Flicker Fusion Frequency (FFF)**
- Frequency at which flickering light appears as a constant illumination (Hz = cycles/sec).
  - Human rod FFF = 20 Hz, dogs FFF = 20 Hz.
  - Human cone FFF = 50-60 Hz, dogs FFF = 70 to > 80 Hz, some birds >100 Hz.
- Provides insight into the functional characteristics of the rods and cones.
- Can be roughly correlated with the speed with which retinas can up-date an image (ie how quickly an animal moves through its environment).
- Varies with light intensity and wavelength of the stimulating light.

**b. Visual Perspective**
- Influenced by:
  - Height of eye above the ground (dogs highly variable vs cats very similar).
  - Could this be why cats have evolved to jump up onto higher surfaces?
  - Behavioural traits (leaping in air while searching for objects).

**c. Visual Field (VF)**
- The area that can be seen by one eye when it is fixed on one point.
- Wide VF allows for better ability scan the horizon.
- Varies by breed (eg brachycephalic (short nose, lateral eyes) vs mesocephalic (long nose, forward eyes).
-total VF - average dog = 240-250°, cats = 200°, humans = 180°
-monocular VF - ave dog = 135-150° (120° ipsilateral VF, a 15-30° contralateral VF)
-dogs have a larger VF (60-70°) than humans, but a smaller degree of binocular than humans

d. Depth Perception
- stereopsis (binocular depth perception) enhances VF
- stereopsis occurs when two eyes view an object from slightly different vantage points and the resulting image is fused into a single image.
- depth perception is likely greatest when viewing straight ahead vs down (nose blocks much of the view)
- double vision - if the two images are not fused
- most dogs = 30-60° of binocular overlap < cats and humans (140°)
- monocular animals can still perceive depth from other cues that include: relative brightness, contour, light and shadow, object overlay, linear and aerial perspective, density of optical texture, and motion parallax.

e. Visual Acuity
=the ability to see details of an object separately and unblurred
-dependent upon the
  i. optical properties of the eye
  ii. ability of the retina to detect and process images (usually the limiting factor)
  iii. the brain to interpret the images received.

e.i. Optical Factors in Visual Acuity
-refractive error (RE) = improper focus of light on the retina secondary to cloudy or opaque media (cornea, aqueous humor, lens, vitreous)
- requires correction with glasses – humans only
- extent of RE is expressed as D (diopters) = 1/f (focal length, m)
- vertical slit pupil (cats) – reduces light scatter to focus light on the retina
- posteriorly located lens (relative to humans) – produces a smaller brighter image on the fundus
- astigmatism = when the ocular media fails to focus parallel rays of light uniformly on the retina
- requires accommodation if objects at different distances are to be seen with equal clarity
- uncommon in dogs and cats
-emmetropia – most dogs and cats
-mycopia (near sightedness) – light is focuses in front of the retina
- brachycephalics, German Shepherds, Rottweiler
- not all dogs are myopic. Other sensory clues (smell, sound) may help to characterize near objects.
- older dogs trend towards myopia secondary to nuclear sclerosis. (= presbyopia = loss of accommodative ability).
-hyperopia (far sightedness) – light is focuses behind the retina
-accommodation = adjustable focusing by altering the curvature of the lens surface or moving the lens anteriorly
- limited in dogs and cats (dogs <2-3 D (50-33 cm), cats 4D (25 cm)
-aphakia = absence of a lens
  -results in severe hyperopia (-14D) and reduced visual acuity to 20/800 or worse
- aberrations
  -spherical
  -of the lens. Results in uneven bending of light waves across the convex optical surface.
  -dogs have negative spherical aberrations in the peripheral lens to compensate for the positive aberrations of the peripheral cornea.
  -chromic
  -light of short wavelengths (blue) is focused in front of light from long wavelengths (red). Significance of this in dogs is not known.

e.ii. Retinal Factors in Visual Acuity
- convergence = photoreceptor:ganglion cell ratio
  -increased convergence results in increased light sensitivity but reduced acuity
- retinas with excellent resolving power have:
  -high # ganglion cells:photoreceptors
  -large # ganglion cells and optic nerve fibers
  -high density of photoreceptors
- cats (and likely dogs) = 4:1 (cone:ganglion cell ratio) vs 1:1 in the fovea in primates.
- peripheral retina (all species) has fewer ganglion cells than in the center to result in reduced visual acuity in the peripheral VF.
- peripheral retina contains ganglia with larger dendritic fields (for better light detection?)
- topography = photoreceptor distribution (humans = fovea, dogs and cats = visual streak (VS))
  -VS
    - oval zone, tapetum, slightly superior and temporal to the optic nerve.
    - tapetal location enhances vision in dim light, degrades visual acuity secondary to scattering
    - high concentration of rhodopsin
    - oval shape
      - short temporal and longer nasal extension
      - may promote binocular vision and facilitate scanning of the horizon.
- domestic dogs - two different types of visual streaks (large, pronounced and smaller, moderately pronounced), both forms can occur within the same breed.
  - variation in appearance of the visual streak may be secondary to breeding programs that place little selective pressure on maximizing visual performance.

e.iii. Estimates of Visual Acuity
- Snellen Fraction = a method for describing visual acuity in the human fovea (expressed in feet)
- typical human 20/20 (patient/average “normal” person)
- estimates of visual acuity vary as they have been obtained by various methods (behavioural testing, measurement of visual evokes cortical potentials, pattern electroretinography, assessment of optokinetic responses, or using repetitive patterns and expressing visual acuity in cycles of alterations per degree of visual field)
- typical dogs 20/75 (dog discerns object at 20’ that normal person could discern at 75’)
- typical cat is between 20/100-200
- cognitive vision evaluation – menace response, following an object (cotton ball/laser)
- crude tests because they test the motion sensitivity of almost the entire retina and positive responses may still be present with visual acuity <20/400

f. Colour Vision
- dogs, and to a lesser extent cats, have colour vision
- dogs
  - see blue and yellow (“red-green colour blind”)
  - wavelengths at the two ends of the blue (429-435 nm)-yellow (555 nm) spectrum are most saturated colours.
  - intermediate wavelengths are less intensely coloured
  - dogs have fewer cones and less colour saturation with the spectral neutral point shifted towards the blue end of the spectrum (480 nm) vs humans spectral neutral point is a greener (505 nm) region of the spectrum.
- service animals
  - require training of other cues – position, relative brightness, smells, taste, textures
  - dim light, colour vision is less important in most animals because low light available to stimulate cone photoreceptors.
- dogs have been reported to be able to differentiate perfectly among closely related shades of gray that are indistinguishable to the human eye.
- cats have a limited capacity for colour vision if the stimuli are large and differ greatly in colour.

Further Reading:
- Campbell FW, Green DG. Optical and retinal factors affecting visual resolution. J Physiol (Lond) 1965; 181:576-593.
Most ocular conditions interrelate which is why eyes are tested so broadly. All eyes should be tested for intraocular pressure, fluorescein stain, and Schirmer tear test (the minimum data base) where indicated. If you are uncertain if you have a uveitis or glaucoma, please send case for tonometry (another local clinic, emergency clinic). All cases being treated with a topical steroid should have the cornea stained for the presence of an ulcer first.

The following information is provided to serve as a guide. The medical treatment plans provided are considered initial and what I believe would be safe starting points unless otherwise indicated. All medical conditions can change quickly rendering the initial treatment plan inappropriate or even harmful. Frequent rechecks are recommended. Always consult with an ophthalmologist if you are unsure of any ocular changes that may be developing or to discuss long-term care. There are no ‘blanket’ recipes or regimens for treating eye conditions.

Common signs associated with most ocular abnormalities that warrant examination include the following:
- increased or persistent conjunctival hyperemia (redness)
- increased or persistent blepharospasm (squinting)
- increased or persistent epiphora (tearing)
- increased or persistent rubbing/scratching at the eye
- persistent mydriasis or miosis.

- Ulcer = A break in the corneal epithelium that exposes the underlying corneal stroma.
- Superficial ulcer = an ulcer that involves loss of epithelial tissue. Generally not an emergency however, can progress quickly, especially in brachycephalic breeds therefore must be treated appropriately and rechecked frequently.
- Stromal ulcer = an ulcer involving corneal tissue below the epithelial layer. Superficial or deep.
- Fluorescein stain is retained in the base of the ulcer.
- Descemetocele = loss of all stromal tissue and no fluorescein stain is retained at the base of the ulcer. Emergency.
- Melting ulcer = a complicating component of any corneal ulcer. During the normal corneal healing process, proteases and collagenases are produced that help remove devitalized cells from the cornea. Corneal epithelial cells, fibroblasts, PMN leukocytes and some bacteria or fungi produce proteases and collagenase. These enzymes contribute to the progressive breakdown and rapid “melting” of the corneal stroma. A "melting" cornea can slough away in a matter of hours and the eye can rupture if treatment is delayed. Melting ulcers and deep ulcers are considered emergencies. These ulcers may require intensive treatments for several days until healing begins. Often, inflammation of structures inside the eye
accompanieds deep or melting corneal ulcers, and this can be as vision-threatening as the corneal disease. With healing, melting ulcers and deep ulcers can result in noticeable scarring. If these scars are large, visual impairment may result.

**Clinical signs:** lacrimation, blefarospasm, photophobia, conjunctival hyperemia, corneal edema, ± miosis and aqueous flare

**Anatomy:** Epithelium covers the outer surface of the normal cornea. The epithelium is heavily innervated, provides a water barrier to the eye, and measures approximately 8-15 cell layers thick. The middle layer of the cornea, stroma, is made up almost entirely of precisely organized collagen fibers, has fewer nerves, and makes up approximately 90% of the corneal thickness. The Descemet’s membrane is the basement membrane for the endothelial cells and becomes thicker with age because it is continuously produced. The endothelial cells are hexagonally shaped cells that are approximately 2500 cell/mm². Endothelial cells increase in size and decrease in number with age. Centrally the cornea is 562 ± 6.2 µm (approximately 0.5 mm) thick, and peripherally is slightly thicker. Corneal thickness increases gradually with age. Corneal sensitivity is highest in dolichocephalic skull types, followed by mesaticephalic, and lowest in brachycephalic skull types. The central cornea is most sensitive, followed by the nasal, temporal, dorsal, then ventral corneal regions. Dogs with diabetes have reduced corneal sensitivity.

Injuries to the cornea are usually the result of trauma secondary to mechanical injury or abrasion of an unhealthy cornea by the eyelids. Any corneal ulcer can become infected with bacteria, fungus, or both. A shallow ulcer, when treated appropriately usually heals quickly and with minimal scarring. Ulcers that become infected may be more difficult to treat. In these cases, the ulcer may become very deep or the cornea may start a process called “melting”.

When ulcers don’t heal it is because they are [Indolent, Infected, or the Inciting cause is still present (remember I.I.I)]

**Indolent Ulcers (Spontaneous Chronic Epithelial Defects (SCCEDs))**
(also known as: canine recurrent erosions, refractory corneal ulcers, Boxer ulcers, nonhealing erosions, persistent corneal erosions, recurrent epithelial erosions, idiopathic persistent corneal erosions).

Indolent ulcers occur in older animals secondary to a reduced ability of epithelial tissue to stick to the underlying stromal tissue. These ulcers are superficial and not infected. I like to think of indolent ulcers as being mechanical and as such, they are treated mechanically. Without proper treatment, these ulcers can become infected and deepen. Treatment options (all with proparacaine applied) include: a) debridement (use dry cotton tipped applicator’s (CTAs) to debride loose epithelial tissue from the cornea. Do not roll CTA’s), b) Diamond Burr keratotomy (to smooth the edges of the debrided epithelium), c) grid/striate keratotomy ((dogs only) ± collagen shield and temporary tarsorrhaphy – using a 25 ga needle (bevel is pointed up), three directions of superficial scratches are made over the ulcer and edges of ulcer. If no blood vessels are present in the cornea, extend the scratches over the limbus. Dog
may be sedated if anxious (dog or vet)), d) keratectomy +/- collagen shield and temporary tarsorrhaphy (refer these).

My typical starting off point for indolent ulcers:

Debride the cornea following topical anesthesia.

<table>
<thead>
<tr>
<th>Topical antibiotic (usually Tobrex) solution (not ointment)</th>
<th>TID</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muro 128 ophthalmic solution (5% hypertonic saline)</td>
<td>TID</td>
</tr>
<tr>
<td>Atropine 1% / Mydriacyl (cats)</td>
<td>1 drop after debriding</td>
</tr>
<tr>
<td>Metacam (dogs) if safe</td>
<td>SID x three days</td>
</tr>
</tbody>
</table>

Recheck ulcer in 10-14 days.
  - If healed: continue with Muro 128 TID for 1 week, then BID for 1 week
  - If smaller ulcer: continue with both medications and recheck in 10-14 days
  - If ulcer same/bigger: consider re-debridement or grid/striate keratotomy

E-collar on at all times until healed.

What to do, what not to do for indolent ulcers

**DO**
- topical antibiotic solution – not ointment
- prescribe pain control – if necessary
- E-collar

**Do NOT**
- debride the cornea every 3 days (give the cornea at least a week between debridement)
- use Gentamicin ophthalmic solution – slows healing
- prescribe serum unless ulcer is deep/melting (it is not harmful, just overkill)
- prescribe a topical anti-inflammatory or send home with a topical anesthetic
- trust the dog not to scratch/rub eye

**Stromal Infected Ulcers (deep/melting)**

Deep ulcers or melting ulcers may sometimes require surgery in addition to medical therapy to prevent rupture of the eye. These surgeries usually involve placement of a flap of conjunctiva to provide a vascular supply as well as tectonic support. These permanent flaps actually incorporate into the cornea at the site of the ulcer, forming a dense scar in a matter of weeks.

Treatment
- Generally speaking, the deeper the ulcer, the thinner (clearer) the treatment. (ie, ointment (thickest) – gels – oils – suspensions – solutions (thinnest).
• Some antibiotics ie. fluoroquinolones (eg Ciprofloxacine, Marbofloxacine, Moxifloxacine, Ofloxacin) kill the protease producing bacteria. These are not first line antibiotics.
• Serum helps stops the “melting” process. Serum contains α-2 macroglobulins and α-1 proteinases which inhibit the proteases and collagenases.

What to do, what not to do for melting ulcers
DO
- topical antibiotic solution – not ointment
- use a combination of narrow and broad spectrum topical antibiotics
- prescribe a mydriatic unless there is a sealed laceration
- prescribe pain control
- E-collar
Do NOT
- debride the cornea unless indolent
- do not use gentamicin ophthalmic or prescribe a topical anti-inflammatory
- trust the dog not to scratch/rub eye
- leave it for a week
- do third eyelid flap if planning to refer

My typical starting off point for superficial stromal ulcers:

<table>
<thead>
<tr>
<th>Topical antibiotic (eg Tobrex, Fucithalmic acid)</th>
<th>TID-QID</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muro 128 ophthalmic solution</td>
<td>TID-QID</td>
</tr>
<tr>
<td>Atropine 1% / Mydriacyl</td>
<td>1 drop to effect</td>
</tr>
<tr>
<td>Metacam (dogs) if safe (and/or Tramadol)</td>
<td>SID if painful</td>
</tr>
</tbody>
</table>
E-collar on at all times until healed.
          Recheck ulcer in 3-7 days.

My typical starting off point for deep stromal or melting ulcers:

<table>
<thead>
<tr>
<th>Topical antibiotic (eg fluoroquinolone)</th>
<th>q1-4h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cefazolin 100 mg/ml IV solution</td>
<td>q1-4h</td>
</tr>
<tr>
<td>Serum – autologous or homologous</td>
<td>q1-4h</td>
</tr>
<tr>
<td>Atropine 1% (dog) / Mydriacyl (cat)</td>
<td>1 drop TID until dilated then to effect</td>
</tr>
<tr>
<td>Cephalexin (dog) or Clindamycin (cat)</td>
<td>BID-TID</td>
</tr>
<tr>
<td>Metacam (dogs) if safe, buprenorphine (cat)</td>
<td></td>
</tr>
</tbody>
</table>
E-collar on at all times until healed.
          Recheck ulcer in 1-2 days.
Serum collection

Some bacteria (beta hemolytic strep., pseudomonas) contain stromalysins (enzymes that rapidly breakdown “melt” the stroma of the cornea). Serum contains alpha (α)-2 macroglobulins (large proteins) what inhibit the action of stromalysins (ie, they decrease the enzymatic collagen breakdown of the cornea).

Many medications will also stop stomalysin activity so why use serum? It’s free, easily available, easily stored.

What you’ll need:

To collect blood: HEALTHY donor (same dog (autologous serum) or healthy donor of same species ((homologous serum)). In an emergency you can use dog serum on a cat (heterologous serum).

Serum (red-top) tubes, 10 ml (± separators)
20 ga vacutainer needles and tube holders
or IV catheter or 20 ga needles with 12 ml syringes

To harvest serum: 1 ml syringes (many)
18 ga, 2.5 cm needles
syringe caps

1. Obtain permission from the donor dog owner after explaining what the serum will be used for.
2. Fill each red-top (serum) tube ½-3/4 full. For big dogs (>lab) you can take 100 ml blood. IV fluids (maintenance rate) is recommended.
3. Let clot fully before spinning, spin blood
4. Draw off serum into 1 ml syringes and cap. The serum yield will vary with hydration status of donor.
5. Stir residual blood clot with a wooden stick (CTA), repeat steps 2 and 3
6. Label bag of syringes and freeze for up to 6 months. Unfrozen syringes can be stored in fridge for up to 5-7 days.
7. Reward the donors owner! (eg Starbucks/Tim Horton’s card)

Fee to sell syringes of serum varies by clinic – cost of materials, labour
Surgical treatment for descemetoceles or lacerated corneas
1. Primary corneal sutures (laceration or very small descemetocele)
2. Conjunctival graft (pedicle graft, island graft, bridge graft) – descemetocele/laceration
3. Corneal-scleral transposition (descemetocele)
4. Corneal transplant – rare (descemetocele)

Further Reading:

A quick word about corneal lacerations

If partial thickness (long or short), treat as per any ulcer but monitor closely for uveitis and infection

REFER when
- full-thickness and not leaking (because it is being sealed with iris/fibrin*)
- full-thickness and leaking*
- laceration crosses the limbus
- if you are not sure of depth

Initial medical plan:
Topical antibiotic solution (not ointment) q2-4h
Mydriatic – only if no iris incarceration TID until dilated, then to effect
Oral anti-inflammatory
Oral antibiotic – if full-thickness
E-collar
Recheck in 1 day

Surgery is indicated if laceration is large or iris or fibrin is keeping the eye inflated

*Sidel’s Test – place a drop of fluorescein on the cornea. Do not rinse the fluorescein away. Watch the laceration site closely for several minutes with a blue light and magnification. Active leakage of aqueous will be seen (with magnification) as a small rivulet of brighter green fluid egressing from the laceration site and through the duller sheet of fluorescein stain.
COMPANION ANIMAL OPHTHALMOLOGY

FELINE KERATITIS

Marnie Ford PhD, DVM, DACVO

‘Kerato-’ = cornea (Gk), ‘-itis’ = inflammation (Gk)

The causes of keratitis in cats are numerous however of these, only a few are seen routinely in clinics and apart from ulcers, include herpesvirus, eosinophilic keratitis, and corneal sequestra. As these will likely be encountered in clinical practice, they will be discussed in the most detail today. Other less common causes of feline keratitis include corneal dystrophy, corneal degeneration, acute bullous keratopathy, neoplasia, and dermoids. These later conditions are typically not painful and not easily treated medically. Surgical therapy is often the only option for neoplasia, bullous keratopathy, and dermoids.

Clinical signs of keratitis may include squinting, mucoid or watery ocular discharge, and conjunctival hyperemia that may also be swollen or thickened. The cornea can be clear, or cloudy if there is an ulcer, erosion, or if scar tissue is present. The iris may be a “muddier” or duller color than normal if uveitis is present. Sometimes the cat shows signs of an upper respiratory tract infection (URTI) and is sneezing. The URT often precedes ocular disease. Severe keratitis can lead to abnormal blood vessel growth into the cornea.

Often, the medical history and the clinical signs present (including fluorescein stain) are sufficient to aid in diagnosis, but sometimes special tests are needed. These tests include (but are not limited to): collection of conjunctival and/or corneal cells for culture, cytology, or special DNA tests for FHV-1 infection, blood testing for FeLV, FIV, and FHV-1.

Microscopic evaluation of the cells collected from the ocular surface can help determine the health of the cornea and conjunctiva and help identify whether primary or secondary infections are involved. For suspected viral infections, samples can be sent for virus isolation (VI), fluorescent antibody (FA), and/or polymerase chain reaction (PCR) testing by a diagnostic laboratory. Although these tests are highly specific, they are not 100 percent sensitive; therefore, repeated samples may need to be submitted to the lab to achieve a positive result for a definitive diagnosis. Collection of cells for cytology is best achieved by lightly scraping the conjunctival surface with the blunt end of a scalpel blade or Kimura spatula. For conjunctival tissue specifically, a histologic sample can be easily obtaining via a snip biopsy following application of topical anesthesia.

**Herpes (FHV-1) (gk. herpein - “to creep”)**

“Herpes is the glitter of craft supplies” – Demetri Martin

**Etiology:** Feline Herpesvirus- 1 (FHV-1) is an alpha herpesvirinae, a common herpes strain found in many species. FHV-1 has a short replication cycle which leads to cell
lysis, induces lifelong neuronal latency (primarily in an episomal form in the nuclei of trigeminal ganglion), has a narrow host range (species specific to domestic and wild cats), causes ocular lesions, and causes feline viral rhinotracheitis in approximately 50% of all URT infections in cats. It has been suggested that 95% of cats are seropositive for FHV-1 via exposure to vaccine or transfer of wild-type virus between cats (macrodroplets/fomites). FHV-1 is labile in environment (lasts up to 18 hours) and susceptible to drying, disinfectants, fluorescein, rose Bengal, and proparacaine

While most cats have FHV-1 and are exposed to it when they are kittens, acute infections mainly affect susceptible kittens and juveniles. Kittens are normally protected by passive immunity until 2 months old. Kittens with residual passive immunity may not show clinical signs when exposed but can still become latently infected. As a permanent infection, the virus can be dormant in the cat’s body for the rest of the cat’s life, or flare up (recrudesce) at any time. Stress is the biggest factor in the frequency of recrudescence.

Primary FHV-1 infection generates humoral and cellular immune responses. Active immunity via natural FHV-1 infection or immunization protects cats from disease, but not from infection. Passive immunity persists for 2 to 10 weeks (depending upon colostrum concentration and intake). With low maternally derived antibodies kittens may develop subclinical infection and latency and may respond to early vaccination. With high maternally derived antibodies kittens may interfere with vaccination at 12–14 weeks of age. All current commercial systemic vaccines against FVR are trivalent (FVR, FCV, FPV) = FVRCP. Protection induced is lowest against FHV-1. A modified-live vaccine may have residual virulence and may induce clinical signs. Protection develops in 2-3 weeks. An inactivated vaccine is used in pregnant queens and cats infected with FeLV or FIV. Protection develops in 2-3 weeks. Intranasal vaccination results in protection in 1 week.

**Clinical Signs:** There are two mechanisms of pathogenesis that include: direct damage by the cytoplastic virus and indirect damage by immune-mediated effect. The cytoplastic virus causes ulceration of epithelial cells of mucosae and cornea. Immune effects mediated by inflammatory cells cause stromal keratitis or lymphoplasmacytic conjunctivitis (when chronic).

In the **acute phase**, fever, inappetence, and sneezing is followed by serous to mucopurulent nasal discharge, acute hyperemic conjunctivitis, ocular discharge, chemosis + URT signs, acute corneal ulceration (dendritic to geographic epithelial), and chronic corneal ulceration (a dense vascular response and immune-mediated stromal keratitis). Infection in neonatal kittens can result in ophthalmia neonatorum, symblepharon formation, neurologic signs, or high mortality. Pregnant queens infected with herpesvirus can rarely result in abortion. Primary disease in cats is usually self-limiting (10-20 days) however, it is during this phase that viral latency is established in the majority of cats.

The **latent phase** is the same as the acute phase whereby FHV-1 DNA persists in episomal form in the nuclei of trigeminal ganglia, but is less severe because FHV-1 RNA transcription is very limited and infectious virus is not produced. Lifelong latency
occurs in at least 80% of cats. At least half of these cats carry on with latent reactivation and shedding of virus.

**Reactivation** of a latent virus requires establishment and maintenance of the virus. While sensory nerves are damaged during reactivation (<0.5% of latently infected ganglia reactive and when they do, they die), the reserve of ganglia is large that repeated reactivation can take place throughout the life of the host. (eg 20 year old cat with 100 reactivations/year). Spontaneous reactivation is uncommon and often associated with a stress event (moving, lactation, corticosteroid administration).

**Recrudescence** occurs as intermittent and recurrent episodes of viral reactivation of virus from the latent stage back along the sensory axons to peripheral epithelial sites. When recrudescent, huge variation in severity of signs can be seen (ulcerative/non-ulcerative, unilateral, systemic/non-ocular signs). Generally conjunctivitis is less severe than with primary infection and secondary inflammatory cell infiltrate results in thickened conjunctiva and redness.

**Diagnosis:** The most common lab diagnostic methods to demonstrate presence of FHV-1 (tissue/swabs) are FA, VI, and PCR. VI (oronasal/conjunctival swabs) is considered to be the gold standard. Three aspects of laboratory diagnosis of FHV-1 frustrate the testing process: 1. The importance of confirming that chronic lesions are caused by FHV-1 can be complicated by the lack of shedding during the maintenance or latent phases, 2. FHV-1 or viral 1 DNA can be detected in samples from clinically normal cats therefore is a positive result coincidental, consequential, or causal?, and 3. Virus neutralizing antibodies can be low/slow to develop and as such, a low level of neutralizing antibodies does not imply the absence of protection against clinical disease. Serologic test results are predictably positive in most cats because of the widespread exposure to the vaccine or wild-type virus.

**Treatment** for herpes virus corneal lesions is typically supportive and includes broad-spectrum eye drops or ointments and antivirals (nucleoside analogues) and adjunct therapies (non-nucleoside analogues). Similarity between FHV-1 and other alpha herpesvirinae (specifically human Herpes Simplex Virus -1 (cold sores)) has led to the empirical use of HSV-1 therapies for FHV-1. Unfortunately, these medications have had less than desirable effects in cats. All antiviral medications are virustatic not virucidal because viruses are not truly “living” organisms and require host cells for replication.

Nucleoside analogues are structurally analogous to nucleosides used in viral and host DNA and RNA synthesis but modified to disrupt the virus replication cycle by preventing DNA chain elongation. Efficacy: Trifluridine>>>Ganicyclovir>Idoxuridine>Cidofovir>Pencyclovir>Vidarabine. Acyclovir / Trifluridine and INF-α have been reported to be synergistic in vitro.

In an effort to help minimize viral recrudescence, adjunct therapies are prescribed. These include 5% hypertonic saline, L-Lysine, lactoferrin, interferon, vitamins, antioxidants, serum, and lubrication. Arginine is an essential amino acid in the cat and is required for viral replication. Lysine antagonizes arginine. When treated with lysine, cats have a decreased incidence of recrudescence but not a decreased severity or shedding of the virus. The duration of an outbreak however is unchanged.
Anti-viral medications may fail because they cannot distinguish between infected and non-infected cells therefore causes host cell toxicity. (The ideal anti-viral would suppress viral replication without suppressing host cell function but this has yet to be developed), virostatic nature of the mediation requires a very high frequency of application for appropriate response, high cost of medications, pain upon application.

**Eosinophilic Keratitis (EK)**

**Etiology:** EK is an immune-mediated inflammatory disease of the cornea and or conjunctiva of young adult, mixed breed cats (or horses) that is characterized by progressive vascularization and cellular infiltration. The cause of eosinophilic keratitis is believed to be related to an underlying feline herpesvirus infection and/or an autoimmune reaction. This disease is progressive and can grow to involve the entire surface of the eye causing blindness and discomfort. Often it is initially detected in one eye; however, the disease may progress to involve both eyes.

**Clinical Signs:** EK often starts near the lateral or ventromedial limbus, and may affect one or both eyes. Typical clinical findings include vascularization and infiltration of the perilimbal cornea, presence of gritty, white corneal plaque(s) composed of inflammatory cells including eosinophils, thickening and hyperemia of the adjacent conjunctiva and third eyelid, and ocular discharge. These plaques are composed of. EK should be suspected in any cat with a relatively pain-free slowly progressive corneal vascularization with white infiltrative plaques in the cornea.

**Diagnosis:** The diagnosis can be confirmed by cytologic examination of a corneal scrape specimen using a Kimura spatula. Eosinophils and mast cells are present.

**Treatment:** Treatment consists of topical corticosteroids such as 1% prednisolone acetate or dexamethasone 0.1%. These medications are used initially 2-4 times a day until all clinical signs disappear and then slowly tapered to determine the lowest frequency of application that will maintain a clear cornea. Life-long treatment is often necessary. In the presence of corneal ulceration, treatment is restricted to oral prednisone and a topical antibiotic. A topical steroid-antibiotic combination solution may be used at a reduced frequency but only under STRICT observation for worsening of the ulcer. Recurrences are common, especially if medications are discontinued too quickly. Systemic megestrol acetate can be used if ulcerative keratitis is present or if the character of the cat prevents treatment with topical eye medications. While the dose recommended for use in cats in the treatment of EK is very low, the potential risks associated with this medication in cats may include profound adrenocortical suppression, adrenal atrophy, an iatrogenic “Addison’s” syndrome, transient diabetes mellitus, enlargement or (rarely) cancer of the mammary gland, and liver toxicity. These potential risks must be discussed with owners prior to use.

FHV-1 infection has been shown to be present in a large number of cats with eosinophilic keratitis. Its exact role in the pathogenesis of the disease is still unknown since the prevalence of FHV-1 in cats is almost ubiquitous. Topical use of corticosteroids in treatment of EK may reactivate latent FHV-1 virus resulting in FHV-1...
keratitis. Treatment with antiviral medications needs to be started and topical steroid use discontinued until the FHV-1 keratitis has resolved. It has been suggested to use both antiviral medications and corticosteroids in the treatment of eosinophilic keratitis, but this is still debated. To my mind, treatment of EK and FHV-1 recrudescence is analogous to balancing treatments in a patient with heart and renal failure.

**Corneal Sequestrum**

**Etiology:** A sequestrum is a localized necrosis of the epithelium and anterior stroma. The affected area becomes infiltrated with dark pigment that is present in the tear film resulting in the characteristic black lesion in the cornea. Numerous factors have been suggested in the etiology of corneal sequestration including FHV-1 infection and chronic irritation such as entropion, low tear production, decreased blink response, decreased corneal sensation, non-healing corneal ulcers, trichiasis and exposure in brachycephalic cats. Feline herpes virus infection is more likely to be a factor in the etiology of corneal sequestration in domestic long and short hair cats that in Persian and Himalayan cats. Sequestra can recur in the same eye or in the other eye in predisposed patients.

**Who:** Corneal sequestrum is a corneal disease unique to cats. Persian, Himalayan, Burmese and Siamese cats are predisposed. While corneal sequestra have been documented in horses, the disease is most common in cats.

**Clinical Signs:** Sequestra are often located in the (para) central cornea. A rim of loose epithelium is frequently present around the sequestrum. Other clinical signs include increased tearing, brown ocular discharge, and blepharospasm. The sequestrum is slowly extruded by the cornea through vascularization around and beneath the sequestrum lifting off the necrotic piece of tissue. The sequestrum may affect only the outer layers of the corneal stroma (tissue), but in some cases the sequestrum extends deeper into the cornea and may lead to deep ulceration, pain and possibly, corneal rupture. A corneal sequestrum can lead to corneal ulcers (superficial to deep), ocular infections, corneal scarring, corneal vascularization and corneal mineralization.

**Diagnosis:** Diagnosis is by the clinical appearance of a black corneal lesion in an intact cornea. It is easy to distinguish a corneal sequestrum from iris prolapse associated with corneal perforation. The iris can be seen to extend to the cornea in a perforation.

**Treatment:** Treatment options include surgical removal or medical management. If an underlying predisposing factor can be identified, such as entropion, it needs to be corrected. Medical management is aimed at preventing secondary bacterial infection and provide lubrication. A topical antibiotic ointment is used three to four times a day allowing the cornea to slough the sequestrum. This may take many months, especially in brachycephalic cats. Surgical removal by use of a superficial keratectomy can shorten recovery time. If the sequestrum involves the deeper layers of the cornea, it may be necessary to place a conjunctival pedicle graft. Other surgical options described include placement of a free island graft, lamellar keratoplasty, corneal scleral transposition, or placement of a biologic/collagen disc. Recurrences are not
uncommon. Placement of a conjunctival pedicle graft has been suggested to reduce the chance of recurrence.

Further Reading:
Golden Retriever Pigmentary Uveitis (GRPU), once seen sporadically in the northeastern United States has been spreading rapidly across North America since the early 1990’s. At that time, the impending and widespread magnitude of this disease was unknown. As such, like so many geographically isolated but locally recurrent conditions, various names for this condition were published over the following years and included: pigmentary uveitis, Golden retriever uveitis, ocular melanosis. With increased reporting and discussion among ophthalmologists, a consensus was made to rename this condition GRPU. With this consensus came reduced confusion with other similarly named but clinically different diseases in other breeds – eg Cairn Terrier Ocular Melanosis, brachycephalic pigmentary keratitis. Furthermore, by defining the constellation of clinical signs associated with GRPU, more accurate diagnosis for inclusion could be made.

The cause of GRPU is poorly understood but genetic factors have been proposed based on apparent breed predilection and an absence of demonstrable systemic diseases, and infectious or neoplastic causes. A rise in success and ease of mailing frozen sperm has made it possible for one affected /carrier male to sire several litters, thus magnifying the extent and rate of genetic spread. To make matters worse, affected dogs are not usually diagnosed until they are (on average) 8 to 9 years of age —usually after they are finished breeding. Unless dogs can be diagnosed before they are bred, GRPU will continue to spread from second or even third generation offspring from these affected older dogs. While the mode of inheritance is unknown, it is suspected to be either autosomal dominant with incomplete penetrance, or autosomal recessive. Investigation of pedigrees has revealed a common ancestry, as many Golden Retrievers in the Northeastern US have arisen from one common breeding stock. An immune-mediated mechanism is believed to be part of the pathogenesis of GRPU as evidenced by response to anti-inflammatory agents and occasional positive antinuclear antibody titers. There is no sex predilection, and only the eyes are affected. Any age of dog can be affected with GRPU but in a previous retrospective study involving 75 Golden Retriever dogs diagnosed with GRPU, dogs ranged in age from between 4.5 to 14.5 years with a mean of 8.6 ± 2.1 years. The clinical signs can be seen in one eye or both eyes, but typically GRPU affects both eyes in time.

This rapid spread of GRPU creates a moving target for which estimates of prevalence are difficult to assess. Results of genetic eye screening examinations (CERF / OFA data) of Golden Retrievers examined 2000-2009 yielded a diagnosis of pigmentary uveitis in 0.3% of 62695 dogs examined. This percentage has increased steadily since that time (2010-2013, 1.5% of 31174 dogs to 1.9% of 7539 dogs by 2014. Similarly, the reports of uveal cysts (to include iris, ciliary body, and free floating cysts) have increased in prevalence from 2.5% (1991-1999, 50489 dogs) to 5.0% (2000-2009, 62695 dogs) to 7.1% (2010-2013, 31174 dogs) and then 6.9% (2014, 7539 dogs). In 2013, a sampling of the general pet population of Golden Retrievers was examined by veterinary ophthalmologists in three Midwestern States and the prevalence of GRPU was found to be 5.5%. When data generated by the same group of dogs was selected for age as 8
years or more, this prevalence was found to be nearly 10%!\(^2\) The disparity between the clinical study in the Midwest and the data obtained by CERF/OFA is most likely attributed to the inherent bias of data generated by CERF/OFA data. Unlike family pets of all ages examined in the clinical setting, CERF/OFA data is generated by generally high-quality younger breeding and/or show animals, and does not reflect the general pet population. It is for this imbalance in data pools that the Golden Retriever Club of America now recommends lifetime annual OFA examinations for ALL Golden Retrievers.\(^3\)

In the early stages of GRPU, clinical signs are not unique and are typically seen to be associated with non-specific uveitis or confused with conjunctivitis. These signs may include intermittent conjunctival hyperemia and tearing, with or without iris cysts. With progression, increased pigmentation of the iris or pigment dusting on the anterior lens surface, persistent conjunctival and scleral hyperemia, photophobia, and low intraocular pressure are frequently observed.

To differentiate GRPU from any other uveitis, the pigmentary changes inside the eye must be identified. One hallmark sign of GRPU is the fine radial pigment dispersion (spoke-wheel pattern) on the anterior lens capsule.\(^1\) This is usually only seen with magnification. Pigment changes can also be seen in the irises. Both irises can become mottled in appearance with areas of hyperpigmentation against the normal light brown iris pigmentation. In some affected dogs the iris may become diffusely darkly pigmented. Iris discolouration does not always occur simultaneously or symmetrically between the two eyes. For this reason, it is important to monitor the iris color of both eyes for comparison over time.

Uveal cysts have long been reported to occur frequently in the Golden Retriever without much significance to the ocular health. Uveal cysts (specifically iridociliary cysts) may be seen in eyes diagnosed with GRPU.\(^4\) They can be one of the first clinical signs, develop later in the disease, or never form.\(^2\) The frequency of iris cysts in Golden Retrievers was reported in one study to be as high as 34.8%!\(^2\) The relationship between cysts and GRPU is not fully understood. Originating on the posterior side of the iris and/or ciliary body, iris cysts are small fluid-filled structures, which either remain attached to the iris or the ciliary body (most common) or uncommonly become free floating inside the eye. Iris cysts can range in colour from light to dark brown and rarely are they filled with blood. With magnification and pupil dilation, iridociliary cysts are seen laterally. Iris cysts can rupture to spatulated a small amount of pigment onto the anterior lens capsule, the corneal endothelium, or the iridocorneal angle.

When uncontrolled, GRPU can lead to the development of cataracts, intraocular scarring, synechia formation, iris bombé, severe pigment deposition, preiridal fibrovascular membranes, glaucoma, pain and blindness. In one study, as many as 46% of dogs with GRPU developed glaucoma and subsequently became blind.\(^1\) The development of glaucoma has been postulated to be due to cysts,\(^5\) intraocular scarring (pupillary block glaucoma),\(^1,4\) pigment deposition and subsequent obstruction of the iridocorneal drainage angle,\(^5\) or secondary to complex auto-immune mechanisms triggering or triggered by pigment dispersion inside the eye.\(^4\)
Sadly, many dogs present clinically with sore, red eyes, and acute blindness/vision deficits in one or both eyes. While seemingly acute, these changes were likely to have been mounting for many months-years. Upon further questioning, an intermittent history of ocular redness attributed to allergies is almost always identified. Unless painful (manifested with blepharospasm) or blind, many dogs are not seen by a veterinarian and as such, these early warning signals go undiagnosed and untreated. Upon closer examination, both eyes are almost always affected to some degree. The cause of impaired vision/blindness is usually glaucoma (increased intraocular pressure), but other conditions, such as cataracts and/or intraocular hemorrhage, can impair vision. If GRPU is too advanced and the eye(s) are blinded by scarring and glaucoma, vision cannot be recovered and enucleation is recommended. We now know that that early intervention with topical and sometimes oral anti-inflammatory therapy, that clinical scarring within the eyes can be prevented.

GRPU is a chronic disease that requires long-term treatment and management for the purpose of maintaining a comfortable, visual eye. While treatments of GRPU vary depending on the severity of clinical signs present, early treatment is recommended and is directed at reducing inflammation and subsequent scarring. Early detection is best accomplished by monitoring for pigmentation of the iris or pigment on the anterior lens capsule, conjunctival hyperemia, blepharospasm or photophobia, tearing, and low intraocular pressure (IOP). Intraocular pressure measurement is a valuable method for detecting inflammation in the eye that is not yet apparent clinically. Interestingly, histopathological examination of enucleated globes that had received medical therapy showed strikingly little evidence of inflammation. It is important to bear in mind that IOP decreases with age and that many older patients naturally have low intraocular pressures (6-10 mmHg). It is therefore imperative to base the diagnosis of GRPU not solely on IOP. Similarly, later in the disease process, serial intraocular pressure measurements can detect early trends towards glaucoma.

Following diagnosis, life-long topical therapy using anti-inflammatory medications is indicated and initiated in the very early stages of the disease. Treatment regimens vary by clinical experience and are driven largely by stage of the disease. Eyes with few clinical signs may at first be treated with topical NSAIDs while eyes with advances disease may receive topical corticosteroids like Prednisolone acetate 1% or Dexamethasone. The more advanced the disease, the more intense the therapy with more frequent applications of anti-inflammatory medication and glaucoma medications if indicated. Oral anti-inflammatory medication may be used to augment topical therapies. The response to therapy is assessed by performing repeated ocular examinations and measurements of the IOP. If the disease is well controlled the patients may be monitored every 3-6 months. If active disease is observed it is very important to watch for any changes including changes in IOP with greater frequency, because of the high risk of glaucoma in the more chronically affected eye. Blind eyes afflicted with painful glaucoma should be enucleated or eviscerated. Intravitreal injection of gentocin is not recommended as it will create additional uveitis and discomfort in these dogs.

The prognosis for GRPU is guarded. Many dogs are presented initially with advanced changes in one eye. With early detection, frequent monitoring, and continuous therapy, dogs with GRPU can be expected to have long-term vision in the second eye.
When not treated, dogs with advanced GRPU invariably lose their vision because of glaucoma. In one study, the average age at enucleation was 9.1 years (range 6.2 - 11.8 years). It is recommended to have the eyes examined regularly starting at the age of 3 years.

Further Reading:

Future work: Dr. Wendy Townsend is performing GRPU research at Purdue University College of Veterinary Medicine. This complex and time-consuming research is concentrated on identifying genetic markers which could then be used to develop a DNA test for GRPU.

Support the research efforts of Dr. Wendy Townsend (Purdue University College of Veterinary Medicine, Dept of Veterinary Clinical Sciences) to identify the genetic marker for GRPU. To this end, she is requesting:
- Blood samples from affected dogs, AND from normal-eyed elderly dogs 12 years of age and older (these animals must have been examined by an ophthalmologist).
- Tissues (enucleated eyes and also blood samples) be submitted from affected dogs of all ages,
- AND from normal-eyed dogs (that are euthanized) 12 years of age and older

Submission form and instructions are available at https://www.grca.org/about-the-breed/health-research/how-to-participate-in-pigmentary-uveitis-research/. If you have any questions, please contact Dr Townsend (townsenw@purdue.edu).
Ocular Neoplasia

Marnie Ford PhD, DVM, DACVO

The complexity and array of abnormal ocular growth is vast. Fortunately the frequency of neoplastic occurrence is very low (0.87% dogs, 0.34% cats). Primary ocular neoplasms are uncommon in dogs and cats relative to neoplasia affecting other organs. Unlike dogs, ocular neoplasms tend to be more malignant in cats. Owing to the space occupying effects of a mass (benign or malignant) within the closed and highly organized structure of the eye, changes in vision, appearance, comfort, and tissue destruction can be expected. Neoplasms may be primary (more common), arising from the tissue they affect, or secondary, arising from a distant tissue as part of a metastatic process. Secondary neoplasms are most likely to affect the intraocular structures of the eye.

Signs of ocular neoplasia are most commonly identified by the owner and as such, early intervention is largely dependent upon their observational skills. Clinical signs that are commonly reported include blepharospasm, tearing, redness, and/or elevation of the nictitans. Vision deficits and/or a cloudy ocular medium may follow. None of these signs are specific for tumor formation however they will be the incentive to request the initial examination. Exophthalmia, third eyelid elevation, reduced retropulsion, and/or strabismus are more commonly noticed as later signs and should raise concern for a retrobulbar mass. The importance of performing a good physical examination, including palpation of the periocular structures, face, and behind the last molar, and obtaining a thorough history cannot be overstated. Other early diagnostic tools include transillumination, slit-lamp biomicroscopy, ophthalmoscopy, orbital ultrasonography, gonioscopy, and CT / MRI are used to help localize and further characterize the nature of a lesion.

Armed with a certain amount of familiarity of the appearance and predictable growth sites of most ocular neoplasias, a degree of confidence regarding growth rates and prognosis for retained vision and ocular comfort can be provided. This relaxed approach to neoplastic diagnosis is supported by the low rate, if any, of metastatic spread of most disease away from the ocular environment of origin. Surgical and medical options are encouraged where the behaviour of a mass is uncertain or there is an opportunity to slow the growth of mass or improve comfort.

As with any unknown growth and where possible, tissue submission for histologic interpretation and conformation of tumor type is strongly recommended to establish a definitive diagnosis and better define treatment and prognostic plans. Where there is suspicion of metastatic spread, additional testing may include skull and thoracic radiographs, CT/MRI, abdominal ultrasound, CSF taps, and lymph node aspirates, and are warranted prior to embarking on surgical procedures involving general anesthesia. For some scleral or conjunctival masses, cytologic samples can be obtained very simply via scrape, fine-needle aspiration, or needle core biopsy. Histologic samples may be obtained by biopsy or wedge-resection of the eyelid, conjunctiva, or sclera, or by enucleation. More advanced techniques are required for keratectomy of corneal lesions, sclerotomy of scleral masses, or orbitotomy or exenteration of orbital masses. Often, biopsy of eyelid, conjunctival, and sometimes scleral masses results in the
complete removal of the tumor. The decision to biopsy or aspirate can be hampered by limited tissue availability and/or the limited potential to re-visit the surgical site following histopathologic diagnosis. Histologic interpretation of an ocular mass may impart a different prognosis than that of the same mass located elsewhere on the body.

Treatment of an ocular neoplasm is based on many factors that include: the type of neoplasia, the overall health of the globe and the dog, the presence of metastatic disease, and the financial constraints of the owner. Surgical treatment options may include local excision, enucleation, orbital exenteration, laser ablation (Nd:YAG, Diode), cryotherapy, radiation, or chemotherapy. Alternatively, given the seemingly benign biologic behaviour of many ocular tumors, some clinicians elect to simply observe the mass for progression (benign neglect) or pursue less radical options rather than to hastily remove a sighted and comfortable eye. While surgical debulking of a mass via iridectomy or iridocyclectomy may appear to be an obvious option for treatment and/or diagnosis of intraocular masses, the perfectly organized compartments of the eye are profoundly sensitive to surgeon proficiency and have a high potential for complications that include hemorrhage and/or retinal detachment. Clinical preference and financial constraints may warrant euthanasia to be more appropriate than enucleation (or exenteration) among select cases in which metastasis is present, the patent is debilitated, or owner has declined therapy.

Owing to the irritability and intolerance of the globe for typical cell/tissue collection methods, attempting to definitively diagnose a mass in or behind a globe is daunting. As such, we are sometimes forced to diagnose and prognosticate solely on clinical presentation and our own experiences. In an effort to provide some insight into the more common tumors likely to be encountered, this seminar will focus on the most common ocular tumors of the adnexa (periocular skin, eyelid, conjunctiva, third eyelid, cornea, sclera), uvea, and orbital space in dogs and cats where possible. There are a large number of ocular tumors; most of them are either underreported in the literature or varied in rate of occurrence by region, county, and breed types.

**Adnexa**

Neoplasia with a predisposition for the head and neck areas is more likely to affect the periocular skin. In dogs, this includes, but is not limited to, histiocytoma, melanocytoma, sebaceous hyperplasia, adenoma, and epithelioma, squamous and viral papillomas, and trichoblastoma. The vast majority of eyelid neoplasms in dogs are benign (74-88 %), local, minimally invasive, and respond to fairly conservative surgical procedures. For these reasons, benign neglect and careful monitoring is a reasonable therapeutic option. Distinct metastasis from eyelid neoplasms in dogs has not been reported. Eyelid tumors must be distinguished from conjunctival neoplasms, which tend to be locally invasive and often recur after attempt s of surgical excision. The epithelial tumors outnumber the mesenchymal tumors by a ratio of about 5 to 1.

**Eyelid margin** neoplasms, the most frequent ophthalmic neoplasms in dogs, include meibomian gland adenoma and adenocarcinoma, melanoma, histiocytoma, mastocytoma, and papilloma. Neoplastic growth associated with the meibomian glands of the eyelid margin are the most frequently reported neoplasia at 44-70% of eyelid neoplasms. Meibomian neoplasms (adenomas, epitheliomas, carcinomas)
appear clinical similar as tan, pink, grey, or black and with irregularly textured surfaces. With exposure, advanced meibomian adenomas or adenocarcinomas may ulcerate and even hemorrhage to cause local irritation resulting in blepharospasm, epiphora, conjunctival hyperemia, corneal vascularization, and pigmentation. When large or obstructive to the gland orifice, the gland can rupture and release glandular lipid secretions into the surrounding tissue to set up a marked inflammatory response. While locally invasive and histologically malignant, meibomian adenocarcinomas are not known to metastasize. Removal via complete excision or by debulking and freezing of both adenomas and adenocarcinomas is recommended to reduce local irritation to the cornea and surrounding tissues.

**Eyelid melanomas** are the second-largest group of tumors and appear as two distinct types that arise from the eyelid skin (single/multiple, easily excised) or from the pigmented eyelid margin (aggressive, expansile). As with most eyelid tumors, eyelid melanomas are considered to be benign but should be removed when small to avoid extensive blepharoplasty procedures.

**Conjunctival (including nictitans) neoplasia** occurs infrequently in the dog and the cat. While morphologically malignant, most tumors of the conjunctiva can grow to be quite locally aggressive and recurrent after removal but seldom metastasize. Hemangioma / hemangiosarcoma, melanoma, and mastocytoma appear to be the most commonly presenting tumors affecting the canine conjunctiva. Mast cell tumor, hemangiosarcoma, fibrosarcoma, and adenocarcinoma are most commonly seen primary conjunctival tumors seen in cats.

**Melanocytic neoplasia of the conjunctiva in dogs** appear clinically as pink-lightly pigmented-darkly pigmented masses on the palpebral, bulbar, and third eyelid conjunctiva. Differentiating a benign from a malignant mass has traditionally been determined by the mitotic index (MI), however, for conjunctival malignancies, a cut-off MI number has yet to be established. Benign melanocytomas (0-1 MI) appear clinically as well circumscribed and heavily pigmented. In contrast, most melanocytic neoplasms (high MI (>4?)) are malignant. These masses are poorly circumscribed, may be multifocal, mildly pigmented relative to melanocytomas or may be amelanotic, with ulcerated overlying epithelium. The metastatic rate of malignant conjunctival melanoma has been reported to be between 17-33% and as such, wide surgical excision followed by cryotherapy, enucleation, or exenteration are treatment options. Local recurrence may occur.

**Vascular neoplasms** are classified as hemangiomas or hemangiosarcomas but clinically are difficult to differentiate. Hemangiomas tend to be well-circumscribed masses whereas hemangiosarcomas are described as variably infiltrative. With complete surgical excision, the prognosis for life and for the globe is good. Surgical excision of hemangiomas is typically curative. With an increased risk of local recurrence, adjunctive cryotherapy has been recommended for after wide surgical excision.

**Conjunctival mast cell tumors (MCT) in dogs** are seen clinically as smooth, firm, and subconjunctival edematous masses with or without conjunctival hyperemia or hemorrhages and fluctuation in size. Prognosis for life and the globe is good and metastatic spread is rare after surgical excision.
Feline tumors of the eyelid and conjunctiva, while less common in cats than dogs, are usually malignant and more difficult to treat than those in dogs. **Squamous cell carcinoma** (SCC), while rare in general, occur more commonly in white cats with nonpigmented eyelid margins. SCC can involve the eyelids, conjunctivae, and the nictitating membrane, they are pink, roughened, irregular masses or are seen as thickened ulcerations. Adenocarcinomas, fibrosarcomas, neurofibrosarcomas, and basal cell carcinoma have also been uncommonly reported. As outline above, treatment varies with the tumor type, location, and size and includes surgical excision, radiation therapy, and cryotherapy. Adjunct therapy is often required as poor margins are often achieved. Prognosis for these malignant tumors is poor, with survival of only 1–2 mo. **Lymphoma**, is the second most common tumor of the feline eyelid conjunctiva with the hallmarks being local edema and a tan/white colour to the conjunctiva. Ocular lymphoma in cats is usually part of multicentric disease and may be a manifestation of cutaneous epitheliomatous lymphoma.

**Apocrine hidrocystadenomas** are rare benign cystic adenomas of the apocrine sweat gland. Persians and Himalayans, 7-11 years of age, are most commonly affected. These cysts can be solitary or multiple and range in size from pin point to greater than half a centimeter. Darkly pigmented, often black, concern is often raised regarding their potential malignancy. The cause of these cysts is not clear but retention or proliferative processes have been suggested.

**Third eyelid gland adenoma/adenocarcinoma** appear clinically similar as protrusion of the third eyelid with the body of the third eyelid expanded by a firm pink mass. This may result in exophthalmos, strabismus, or enophthalmos. Third eyelid gland adenocarcinomas have been described as expansile and variably infiltrative masses to well differentiated or solid. Most third eyelid gland neoplasms are histologically malignant however a good prognosis for life with third eyelid gland adenomas and adenocarcinomas is given with complete surgical excision/exenteration.

**Limbal (epibulbar) melanomas**, identified in dogs (3.5%) and cats (1%), are typically benign neoplasms without cellular atypia and with rare-absent mitosis. Female, German Shepherds, and Labrador or Golden Retrievers breed predilections have been inconsistently reported. Clinically, limbal melanomas appear as heavily pigmented masses that are presumed to arise from either the melanocytes that demarcate the limbus and expand into the adjacent cornea and sclera, or as masses arising posterior to the limbus and expanding into the adjacent sclera. Gonioscopy and B-scan ultrasound can help to detect possible penetration of the sclera. Unlike most neoplastic masses, limbal melanomas are identified in two age groups and with different behavioural characteristic. Tumors in the young group (2-4 years old) tend to be rapidly invasive into the globe whereas tumors in the older group (8-11 years old) tend to be stationary or found incidentally on physical exam. Primary limbal melanomas must be differentiated from external extension of intraocular melanomas via complete intraocular examination, gonioscopy, and high-resolution ultrasonography. Consequences of rapidly growing limbal melanoma with intraocular extension include glaucoma and uveitis. Benign neglect and careful monitoring may be warranted in the older group of dogs. Surgical excision with/without cryotherapy,
or laser photocoagulation may preserve the globe in cases with more rapid progression.

Considered to be a primary carcinoma in situ, corneal squamous cell carcinoma may develop secondary to chronic irritation (pigmentary keratitis, environmental exposure, KCS) or possibly to use of topical immunomodulators in dogs. SCC can present clinically as a raised, multilobulated or discrete, pink-white mass associated with significant corneal vascularization. The adjacent corneal epithelium may be hyperplastic and is often pigmented. Diagnosis and treatment is via keratectomy followed by cryotherapy to decrease recurrence.

**Intraocular Neoplasia**

Intraocular tumors are relatively uncommon in the dog. The tumors may be primary (91%) or secondary (9%) from metastatic disease or local invasion with their origin in the anterior uvea. Distant metastasis from primary intraocular tumors is rare, however local tissue destruction, uveitis, and secondary glaucoma occur commonly. While unilateral disease is more suggestive of a primary neoplasm, bilateral disease supports secondary or metastatic neoplasia. Presence of an intraocular mass is suggested by a visible IO or scleral mass, glaucoma, pupil distortion, hyphema, anterior uveitis, or extrabulbar spread. These changes must be differentiated from other intraocular masses, including iris cysts, granulomatous lesions, and staphylomas and must be considered in any eye with secondary glaucoma or opaque media. As outlined above, diagnostic techniques are abundant but, without risk to vision, restricted to external methods of imaging.

Canine primary IO tumors are generally carefully monitored with digital photographs being a valuable aid in assessing progression. Enucleation is advised if there is concern about malignancy, intractable uveitis, or secondary glaucoma. Isolated primary masses involving a portion of the iris/CB may be amenable to local resection, however this requires an accomplished ophthalmic surgeon and often have unsatisfactory long-term results. Transscleral and transcorneal Nd:YAG or diode laser therapy has induced remission in some small- to moderate-sized primary intraocular tumors. The three most commonly identified uveal neoplasias in dogs in two veterinary pathology databases (3496 neoplasias) were uveal melanocytoma (41.5%), iridociliary adenoma (21.1%), and uveal malignant melanomas (13.1%). Other neoplasms identified included lymphoma (primary or secondary), metastatic neoplasia (transmissible venereal tumor, hemangiosarcoma), iridociliary adenocarcinoma, optic nerve meningioma, histiocytic sarcoma, peripheral nerve sheath tumor, astrocytoma, and medulloepitheliomas.

**Uveal melanocytic neoplasia** can be divided into benign (melanocytoma) and malignant (melanoma). Clinically, it is difficult to distinguish between benign and malignant anterior uveal melanocytic neoplasia. 93% are located mainly in the iris and ciliary body, anterior uveal melanocytic tumors can vary in appearance from heavily pigmented to nonpigmented as well as in location; seen as iris expansion and extension into the anterior chamber, anterior displacement of the iris face, or pigment expansion into the adjacent sclera with two age-groups more commonly affected (2 mo – 7 years, >7 years). Mitotic index (MI) is the most reliable criterion to make the
distinction between a benign and malignant diagnosis (benign MI < 2, malignant MI > 4 (often >30)).

Benign uveal melanocytomas are most common (75-94%) in the iris and/or ciliary body. These anterior uveal melanocytomas (grow rapidly to extend beyond the iridocorneal angle to infiltrate the deep peripheral corneal stroma). Secondary glaucoma may develop without the presence of uveitis and frequently requires enucleation.

Approximately 10-25% of all anterior uveal melanocytic neoplasms are malignant with a metastatic rate of 4% (hematogenous is most common) estimated. Uveal malignant melanoma occurs more commonly in the anterior uvea (~95%) than in the choroid (~5%). The prognosis for life with malignant melanoma is guarded and removal of the globe is frequently required.

Choroidal melanocytomas are located most commonly in the peripapillary retina and optic nerve and have no age predilection but 6-7 year old, medium-large breed dogs predominate. Choroidal melanocytomas are uncommon and account for only 6% of all benign uveal melanocytomas. Unlike anterior uveal melanocytomas, choroidal melanocytic neoplasms are typically pigmented focal and subretinal masses with or without retinal detachment. Like anterior uveal melanocytomas, choroidal pigment may also infiltrate into the corneal stroma. 15% of choroidal melanocytic neoplasm are malignant however metastastic spread occurs infrequently (<5%).

**Iridociliary neoplasia** occurs infrequently in dogs and cats. Middle aged to older Labrador and Golden Retrievers may be predisposed. These tumors, whether benign (adenoma) or malignant (adenocarcinoma = invasion of the sclera), arise from the ciliary body epithelium and appear as non- to lightly pigmented pink masses that may be papillary (57%) or solid (43%). With growth these masses may be seen in the pupillary space and displace the iris face anteriorly to cause dyscoria, lens subluxation, cataract, retinal detachment, and secondary glaucoma may occur. Approximately 15% of iridociliary neoplasms can be classified as malignant and invasion of the sclera which occurs late in the disease, is reported to be a major criteria of malignancy. (As with many uveal tumors, the prognosis for life with an iridociliary neoplasia is good, but enucleation is required in most cases secondary to its expansive and destructive nature.

Approximately 90% of uveal neoplasias in older cats (>9 years) are diffuse iris melanomas (DIM) which presents as progressive hyperpigmentation of the iris with an expanding irregular surface, pupil abnormalities, and secondary glaucoma. In early feline DIM, diffuse or nodular tumor is usually confined to the iris with a long prodromal period of months to years. Typically benign, this tumor can undergo malignant transformation with metastasis (usually to lung/liver) occurring late in the disease. There is no single morphologic feature that is predictive of outcome. Metastatic rate in cats is much higher than in dogs (35-65%) and linked to MI, large tumors, and extension into iris and ciliary body stroma and scleral venous plexus. Metastatic spread may not become evident for 1.3 years following enucleation. Pre-surgical chest radiographs and abdominal ultrasound are recommended when fast-
growing tumors are to be removed with enucleation or when secondary glaucoma is already present.

**Feline post-traumatic intraocular sarcoma (PTS)** formation is the second most common primary intraocular tumor of cats with a frequency reported in one study to be 8% (!). Feline PTS occurs in older (7-15 years) cats with a history (2 mo – 15 years earlier) of chronic uveitis, previous ocular damage, or intraocular injections of gentamicin. 67% are male (intact/neutered). Clinically, cats present with glaucoma, phthisis bulbi, or chronic uveitis. Spindle cell and round cell variants are seen. Spindle cell variants are of lens epithelial origin (lens damage) and round cell variants (a variant of lymphoma). There are also a very rare subset that includes osteo- and chondrosarcomas of unknown cell of origin. Chronic uveitis and lens damage are known risk factors. These are aggressive tumors in cats and have a propensity for spread along the optic nerve and meninges to affect the central nervous system (and hence can be fatal). Cases that have tumor extension beyond the sclera have a poor prognosis with local recurrence and extension towards the brain. Eyes removed with tumor present within the sclera have a good prognosis. Prophylactic enucleation with removal of as much ON as possible is the treatment of choice.

**Orbital Neoplasia**

Orbital masses are rare and account for approximately 5% of ocular neoplasia in dogs however of these, approximately 90% are malignant with region infiltration (into CNS) or distant metastasis common. As such, prognosis for long-term survival is often poor. Most orbital neoplasms in dogs are primary 75% (most are secondary in cats) and identified in middle-aged to older animals, possibly large breed dogs (A67) and possibly female dogs and male cats. The most frequently diagnosed tumors include osteosarcomas, fibrosarcomas, and nasal adenocarcinomas. While early diagnosis and surgical therapy provides the best potential for globe retention or even survival, the typical slowly progressive and non-painful exophthalmia (sometimes enophthalmia) with decreased retropulsion, and third eyelid elevation with or without strabismus that is suggestive of orbital neoplasia, nearly always prevents early discovery. As such, a guarded prognosis is offered with 25%-40% of affected dogs euthanized on diagnosis. Surgery, sometimes combined with chemotherapy, can often prolong life for ≥6 mo. Painful exophthalmia is more commonly associated with non-neoplastic disease such as cellulitis or abscess formation.

When vision is compromised (via compression not infiltration of the optic nerve), especially when exophthalmia is considered to be minimal, concern for **orbital meningo**ma is raised. Orbital meningioma is the most commonly identified orbital neoplasia of the optic nerve in dogs, but accounts for only approximately 3% of all meningiomas in dogs. Generally slow growing and benign, intraocular invasion and extracranial metastases have been rarely reported. Ophthalmoscopy reveals the hallmarks of meningioma; exophthalmos, papilledema and blindness with abnormal optic disc appearance. Exenteration of the orbit is recommended and a fair rate of recurrence to this and the fellow eye is likely.

**Orbital lobular adenoma**, the second most commonly identified orbital neoplasia in dogs, is typically derived from the lacrimal or salivary glands. Patients often present with swollen lids, protrusion of the nictitans, enophthalmia or exophthalmia, and
subconjunctival masses. This latter sign is not common with most orbital masses. Owing to the large friable and expansile nature of this benign tumor, removal of all affected tissue is not possible and recurrence (within 1-2 years) is common despite exenteration.

**Feline lymphosarcoma-leukemia complex (FeLLC)** is an aggressive secondary orbital neoplasia in cats. Cats with ocular FeLLC have clinical signs ranging from isolated ocular lesions (affecting one or both eyes) to severe systemic illness. Corneal abnormalities may include keratitis, edema, neovascularization, corneal infiltrates, and hemorrhages within the stroma. Ulcerative keratitis may result. Masses can be found in the orbit, globe, conjunctivae, and eyelids. Pupillary abnormalities, including mydriasis, anisocoria, spastic pupil syndrome, "D" or reverse "D" pupil shape, and lack of light-induced pupillary reflexes, may develop months before other clinical signs. Anterior uveitis is the most common clinical finding in FeLLC and is manifested as ocular hypotension, changes in iridal pigmentation and color, keratic precipitates, hyphema, anterior and posterior synechiae, miosis, and aqueous flare. Posterior segment changes include retinal hemorrhages, tortuous dilated vessels, perivascular cuffing, and detachment and degeneration of the retina. Few therapy studies of cats with ophthalmic lymphoma exist, but cats with lymphoma and feline leukemia virus infection have lower overall survival times.

**References available upon request**
SAFE AND COST-EFFECTIVE ANESTHESIA FOR SPAY-NEUTER PROGRAMS

Paulo Steagall

Spay-neuter programs are becoming more popular as a mandatory component to decrease overpopulation and euthanasia of stray dogs and cats.\(^1\)\(^2\) Adequate anesthesia and pain management are crucial parts of a successful canine and feline spay-neuter programs.\(^3\) These environments present a challenge for the veterinarian in regards to geography and limited personnel and drug/equipment availability. Both anesthetic and analgesic protocols will have to be tailored to each specific situation.\(^3\)

The most ideal protocol should be reversible, economically viable, provide pain relief, avoid adverse-effects, species-specific, and have a small injection volume.\(^4\)\(^5\)\(^6\)\(^7\)\(^8\) This session will provide guidelines for: perioperative care (physical examination, patient housing, infectious control, minimal equipment), anesthetic-analgesic management (monitoring, drug protocols, fluidtherapy, emergency setting, recovery) and general considerations based on current evidence and expert opinion. Particular anesthetic and analgesic protocols will be presented. The ultimate goal is to provide humane methods for neutering large number of cats and dogs and an alternative to massive euthanasia due to overpopulation.

References:
THE USE OF KETAMINE IN CLINICAL PRACTICE: ANESTHESIA OR ANALGESIA?

Paulo Steagall

THE NMDA RECEPTOR

A noxious stimulus (i.e. surgical trauma) is translated into electrical impulses that are transmitted to the dorsal horn of the spinal cord. During this process, there is the release of glutamate which is an important excitatory neurotransmitter that activates N-Methyl D-Aspartate (NMDA) receptors. There is now evidence that long-term changes in the central nervous system following peripheral tissue or nerve injury are dependent on the activation of NMDA receptors. The role of the NMDA receptor in the processing of nociceptive input has led to renewed interest in NMDA receptor antagonists such as ketamine. These drugs may provide useful insight in the treatment of different painful conditions.

Ketamine is a popular dissociative anesthetic that acts as NMDA receptor antagonist. This drug may prevent central sensitization and cumulative depolarization (“wind-up”) from occurring. Doses of ketamine associated with NMDA antagonism are considered to be sub-anesthetic and lower than dosage regiments required for induction of anesthesia.

CLINICAL USE

Doses - Sub-anesthetic doses of ketamine have been used as an adjunctive analgesic agent in dogs undergoing surgery. In this species, dosage regimens usually consist of administering a loading dose (0.15-0.7 mg kg⁻¹) followed by a constant rate infusion (CRI) (2-10 µg kg⁻¹ minute⁻¹). As an alternative option for the loading dose, anesthesia can be induced with a combination of ketamine and diazepam.

Studies in dogs - Ketamine has improved feeding behavior when administered as a CRI in dogs after mastectomy but did not provide an opioid-sparing effect. In a recent study, pain scores after ketamine were not significantly different than a group receiving butorphanol, and the drug did not provide adequate analgesia in 37.5% of dogs undergoing ovariohysterectomy. Ketamine should be used as part of a multimodal analgesic approach and not as a sole method of providing pain relief in dogs after surgery.

Indeed, when combined with opioids and other analgesic techniques, a CRI after a loading dose has been associated with analgesia of longer duration in dogs undergoing limb amputation and with an anesthetic-sparing effect in dogs. It seems to be advantageous to combine opioids, loco-regional blocks, NSAID therapy and NMDA antagonists in the treatment of acute and chronic pain if one considers the concept of multimodal analgesia.

Studies in cats - the administration of ketamine has reduced the minimum alveolar concentration of isoflurane. At high doses or after prolonged administration, ketamine may impair anesthetic recovery in feline patients. In a recent case report, a combination of an NSAID and fentanyl-ketamine CRI was administered to cats, and ketamine was used as an adjunctive analgesic agent in order to minimize the risk of
central sensitization, potentially decreasing opioid requirements.\textsuperscript{10} In the clinical setting, ketamine may be used as an additional tool for decreasing inhalant anesthetic requirements and to provide analgesia in cats that are poorly responsive to opioid analgesics.

Practical tip: If one wants to administer a ketamine CRI intraoperatively at 10 \textmu g kg\textsuperscript{-1} minute\textsuperscript{-1}, one can add 60 mg of ketamine into a 1L bag of a crystalloid solution and set the fluid rate at 10 mL kg\textsuperscript{-1} hour\textsuperscript{-1}. A fluid pump is recommended for volume accuracy. A bolus of 0.5 mg kg\textsuperscript{-1} has been used by the author.

References:
A STEPWISE APPROACH TO THE TREATMENT OF HYPOTENSION IN SMALL ANIMAL PRACTICE

Paulo Steagall

By definition, blood pressure (BP) is the force that the flow of blood exerts on the walls of the vessels and what drives tissue perfusion. A mean arterial blood pressure (MAP) of 70 mmHg is required for adequate perfusion of vital organs such as the brain, heart and kidneys. Blood pressure is directly affected by cardiac output and systemic vascular resistance (BP = CO x SVR). Because cardiac output is dependent on the heart rate and stroke volume, the latter two components will affect blood pressure. In addition, stroke volume is affected by preload, afterload and cardiac contractility. Therefore, hypotension is normally a result of a combination of factors such as bradycardia, vasodilation (decreases in systemic vascular resistance, SVR), and decreases in cardiac contractility which in turn will reduce stroke volume and cardiac output.

Blood pressure is measured either indirectly (oscillometric sphygmonomanometry or Doppler ultrasonography) or directly when a catheter is placed in an artery and connected to a pressure transducer that allows constant and continuous evaluation of systolic, mean and diastolic blood pressure. In fact, invasive blood pressure is widely used for accurate BP monitoring of critical patients or during general anesthesia where arterial blood sampling is required. Cardiac output may be evaluated noninvasively in client-owned animals; however, such techniques are most commonly applied in research.

Within physiological conditions, neural, hormonal and local mediators control systemic blood pressure. These strict mechanisms might be affected during disease and anesthesia, and hypotension inevitably occurs. Common causes of hypotension include hypovolemia (hemorrhage, fluid deficits, relative hypovolemia due to vasodilation), vasodilation (anesthetic drug-induced, severe metabolic or respiratory acidosis, severe hypoxemia, endotoxemia, septicemia, anaphylactic reactions), myocardial depression (decreased contractility caused by anesthetics and sedatives, hypoxemia, acid-base disturbances, electrolyte imbalances, cardiomyopathy, catecholamine depletion), cardiac arrhythmias (bradycardia, bradyarrhythmias, atrial fibrillation, ventricular tachycardia), obstruction of venous return (mechanical ventilation, gastric-dilatation volvulus, pericardial effusion, tumors, surgery packing), vagal stimulation (drug-induced - opioids, excessive traction of abdominal organs, pressure on the eye) and sympathetic blockade (locoregional anesthesia), among others. It is crucial to understand the possible causes of hypotension for its adequate treatment.

A step-wise approach to the treatment of hypotension is recommended.

- Diagnose and identify the underlying cause(s) of hypotension based on physical examination (heart rate, mucous membrane colours, capillary refill time, pulse quality, evident blood loss, arrhythmias on auscultation, behavioral changes), blood work (PCV, TP, blood gas, CBC, electrolyte imbalance, lactate), drugs that were administered (anesthetics, opioids, epidural administration of local anesthetics), monitoring (ECG and arrhythmias) and diagnostic tools (radiology, ultrasonography and echocardiography for tumours, GDV, pericardial effusion, peritonitis, etc.). It is also important to identify technical errors such as cuff size and position, transducer quality, evident blood loss, arrhythmias on auscultation, behavioral changes), blood work (PCV, TP, blood gas, CBC, electrolyte imbalance, lactate), drugs that were administered (anesthetics, opioids, epidural administration of local anesthetics), monitoring (ECG and arrhythmias) and diagnostic tools (radiology, ultrasonography and echocardiography for tumours, GDV, pericardial effusion, peritonitis, etc.). It is also important to identify technical errors such as cuff size and position, transducer
calibration and height and inaccuracy of the monitor before triggering treatment for hypotension.

- Fluid therapy is administered only when primary cardiogenic disease has been excluded. In this case, positive inotropes (dobutamine 1-5 mcg/kg/min IV - an agonist of beta-adrenergic receptors) and diuretics are administered depending on the cardiomyopathy.

- Fluid therapy aims to correct fluid deficits and ameliorate preload, stroke volume, and cardiac output. Fluid therapy will correct ongoing losses, and balanced, isotonic crystalloids are the first option to increase intravascular volume (10-20 mL/kg over 15 min or so-called “fluid challenge”). This is usually not attempted in patients with oliguria or anuria, and heart disease. Colloids (3-5 mL/kg over 20 minutes) are administered in combination (or not) with crystalloids especially in hypoalbuminemia. Hypertonic saline (saline 7.5%) is administered (3-4 mL/kg in 5-10 minutes) if excessive hypotension is observed for rapid volume resuscitation. These therapies are better used in combination in high-risk cases such as gastric dilation volvulus. Blood pressure is closely monitored for observation of “end-points”. Fluid therapy may produce adverse-effects which are discussed in the course of the lecture. Current trends indicate pulse-pressure variation as means of monitoring the effectiveness of fluid therapy.

- Hypoxemia, hypercapnia and electrolyte disturbances should be corrected.

- Doses of anesthetics and other cardiovascular depressant drugs should be reduced or adjusted in cases where hypotension is hypothetically caused by bradycardia and excessive vasodilation. In these cases, fluid therapy will most likely have no effect unless if dehydration and hypovolemia are concomitantly present. For example, it is important to adjust depth of anesthesia during surgery according to the surgical stimulus and anesthetic monitoring. Inhalant anesthetics, propofol and barbiturates are well-known for decreasing myocardial contractility and systemic vascular resistance (vasodilation). Balanced anesthesia aims to administer additional analgesics or sedatives with anesthetic-sparing effects. Ventilation is adjusted to reduce peak airway pressure and positive end expiratory pressure without affecting gas alveolar change.

- Hypotension is commonly associated with bradycardia or bradyarrhythmias (2nd degree AV block or ventricular escape beats). The cause of bradycardia is identified and treatment with an anticholinergic (atropine or glycopyrrolate) is recommended especially in opioid-vagal induced bradycardia. Hypothermia is another common cause of bradycardia in which pharmacotherapy may not be effective. On the other hand, dexmedetomidine-induced bradycardia associated with hypertension should not treated.

- Tachycardia may affect diastolic filling and reduce coronary perfusion which will consequently affect stroke volume and cardiac output. It increases myocardial oxygen consumption and work. Tachycardia is treated with fluid bolus (in cases of hypovolemia and dehydration, and reflex tachycardia) or antagonists of beta-adrenergic receptors (“beta-blockers”) in case of excessive sympathetic stimulation.

- Sympathetic support is recommended when positive inotropism, chronotropism and vasoconstriction are desired, especially in normovolemia. Drugs such as dopamine
(5-20 mcg/kg/min; an agonist of D1, D2, beta and alpha receptors depending on the dose) and ephedrine (0.2 mg/kg, IV; an agonist of beta and alpha receptors) are administered during anesthesia. In cases of severe vasodilation during sepsis and endotoxemia, norepinephrine (0.05-2 µg/kg/min; an agonist of alpha receptors) that provides arterial vasoconstriction, and/or arginine vasopressin (0.5-2 mU/kg/min, IV) that causes arterial and venoconstriction and increase venous return, are administered.

It is important to understand the physiologic, humoral and tissue mechanisms that regulate blood pressure. Hypotension is only treated appropriately when the underlying causes are properly identified. Ultimately, the therapy will aim to correct fluid deficits while maximizing cardiac output (heart rate and stroke volume, myocardial contractility) and systemic vascular resistance.

This section discusses a clinical approach for treatment of hypotension in the clinical setting.

References:
1. Klein BG. Cunningham’s Textbook of Veterinary Physiology, 5e, Elsevier Saunders, 2012
Animal welfare organizations across North America are increasingly involved in relocation programs, which are typically defined as programs in which animals are transferred over some distance from organization or individual to another. In Canada, these programs may involve the interprovincial transport of animals as well as the importation of cats and dogs from the United States or other counties. Relocation programs have tremendous potential to save lives by transporting shelter animals from areas of oversupply, where the chance of adoption are low, to locations where there is a greater demand for that type of dog or cat. At the same time, relocation can be very stressful for the animals involved and carries with it the potential for the spread of a number of infectious diseases. These risks can, however, be minimized with meticulous planning, careful consideration of the risks and benefits, and veterinary involvement in all aspects of the program.

Published guidelines and best practices for animal transport and relocation programs do exist, including those created by the Association of Shelter Veterinarians, the American Veterinary Medical Association, and the National Federation of Humane Societies. The recommendations contained therein are largely, but not completely, consistent across these documents. It is important to recognize that there is still much to be learned about both minimum standards and best practices for relocation programs. Existing recommendations may be based on expert opinion or extrapolated evidence, as little scientific data exists specific to these programs. Veterinarians should be familiar with current requirements and recommendations, and should expect that these documents, as well as the applicable laws and regulations, will continue to be refined over time as the body of knowledge relevant to relocation programs expands. Furthermore, it is likely that there is no universally perfect system. Modifications may need to be made in different regions or even between different source and destination partners to create programs that effectively and efficiently ensure the health and safety of animals being transported as well as the communities they enter.

Source shelters should have a comprehensive preventive healthcare program in place. Administration of core vaccinations at or prior to intake is considered a minimum standard, as is the administration of a rabies vaccination prior to transport in accordance with applicable law (e.g. determined by the animal’s species, age, and destination). Core vaccines for cats in a shelter setting include a parenteral FVRCP. Core vaccines for dogs in a shelter setting include a parental DA2PP and intranasal Bordetella bronchiseptica and parainfluenza (with or without adenovirus). The use of modified live rather than killed products is strongly recommended because of the more rapid onset of immunity and greater efficacy resulting from generation of both humoral and cell-mediated immune responses, particularly in the face of maternal antibody interference. Core vaccines should typically be administered to all animals of sufficient age (e.g. 4 weeks or older), including those with mild fever, illnesses, or injuries as well as to nursing queens and dams. It is also recommended that animals be treated for internal and external parasites common to the region and for any obvious detrimental parasite infection they are harboring; minimally, product(s) effective against hookworms, roundworms, fleas, and ticks should be administered. Dogs 6
months of age and older should be tested for heartworm disease prior to transport. Any additional treatments or diagnostic testing required by law at the destination must be performed as well.

Accurate assessment and documentation of an animal’s identity and physical condition is a critical component of successful relocations programs. Furthermore, such examination and record-keeping is often legally required by local, provincial (or state), and/or federal regulations. It is recommended that all animals receive an examination within 24 hours of transport and then again at the time of arrival to the destination. These examinations are in addition to those performed at the time of entry to the source facility as well as by the veterinarian for completion of a health certificate, if that is performed separately. Transport of animals with communicable diseases is generally prohibited. If an animal has an infectious disease, transport should either be delayed or arranged such that the animal can travel alone and be isolated at the destination. Officials in the receiving jurisdiction should be contacted in advance of making any arrangements to ensure that such transport will be permitted.

Spay or neuter prior to adoption is considered a minimum standard for animals participating in relocation programs, but recommendations regarding the timing of such procedures are inconsistent. It is frequently recommended that animals be spayed or neutered and allowed sufficient time for recover prior to transport. Performing such surgeries in the source communities may allow for more timely adoption of the animals once they arrive at the destination shelter, but the resources are often inadequate in source communities to facilitate this arrangement. In these situations, both the animals and programs may be better served by delaying such surgery to be performed in the destination community. Open dialogue between the source and destination organizations is critical in determining which arrangement will be most appropriate in a particular circumstance.

Appropriate housing and stress reduction at the source shelter prior to relocation, during transport, and at the destination shelter prior to adoption are critical considerations in maintaining the health and welfare of animals participating in relocation programs. Extended holding at the source shelter is generally not recommended and the animals’ health is often best served by being transported to the destination shelter sooner. Few source organizations have an adequate ability to segregate the population of animals in their care and there is an on-going risk of infectious disease exposure. In many cases, a true quarantine is not possible and the risk of disease spread can be better managed with strong preventive medical protocols, careful examination of animals prior to transport, and (if needed) quarantine upon arrival at the destination facility.

Further Readings:
INTAKE PROTOCOLS FOR SHELTER DOGS AND CATS

Stephanie Janeczko, DVM, MS, DABVP, CAWA

The experiences leading up to and occurring at the time of intake can have a profound effect on the behavior, health, and well-being of animals arriving at shelters. Stressful and negative experiences may hinder or even prevent successful acclimation to the shelter environment and result in increased anxiety and mental suffering, which can ultimately affect the animal’s disposition. It is critical that the waiting area is appealing to both humans and animals and functions in such a way as to facilitate the various steps of the intake process that must occur. Ideally, shelters are designed with an area for the intake and handling of newly arrived animals that is separate from areas used to process adoptions and return pets to their owners. Regardless of the physical facility, standard operating procedures should be put in place that minimize the stress experienced by all animals from the moment they enter the sheltering facility.

A minimum database of information should be obtained for every animal entering a shelter and included as part of the animal's individual record. This includes information about the circumstances under which the animal arrived at the shelter, specific animal characteristics, medical and behavioral examinations, treatments and procedures, and information about the disposition of the animal. Information on the animal’s history, including medical and behavioral observations and concerns, should be obtained at the time of intake. It is ideal to obtain this information via personal interviews, but written questionnaires are acceptable as well. The process should be standardized to ensure that as much relevant information is captured as possible.

All animals must be restrained upon arrival to the shelter. Larger dogs should be on a leash while smaller dogs, cats, and other small mammals should be safely confined in a crate or transport carrier. The shelter should have ample leashes and carriers for those animals arriving at the shelter without appropriate restraint. The environment should be arranged to reduce auditory and visual contact between cats, dogs, and other animals that may also be present in the immediate vicinity. Every area where cats may wait in carriers should have sufficient chairs, shelving, or counter space to prevent carriers from being placed at or near the level of the floor. Towels, sheets, or other coverings should be kept behind the front counter or in an easily accessible location so that transport cages can be covered while awaiting completion of the intake process and for initial movement of the animal through the shelter. Pheromones (e.g. synthetic feline facial pheromones and dog appeasing pheromone) can be utilized in waiting areas and throughout the shelter as an additional step to help calm cats or dogs. Auditory input should be minimized to the extent possible from both animal and non-animal sources such as fans, phones, and loud-speakers.

Timely handling of arriving animals to reduce crowding in the waiting room and overstimulation is also important. Critically ill or injured animals must be given priority to allow for prompt veterinary care, administration of pain relief, and/or humane euthanasia to relieve suffering. Similarly, triage decisions should also consider the behavior and stress levels of animals awaiting intake. Aggressive, unruly, or otherwise disruptive animals should be quickly admitted to reduce the negative impacts of their...
behavior on other animals. Fearful, fractious, and otherwise highly stressed animals should also be given high priority for completion of the intake process.

Medical intake procedures are intended to identify and treat any medical issues in a timely fashion and to administer vaccinations as close to the time of admittance as possible. Examinations should be conducted in an area that is quiet, well-lit, and stocked with all necessary supplies to ensure the medical intake process is as efficient as possible. Tasty food treats should be available used readily unless a specific contraindication exists or the animal is unable to ingest foodstuffs (e.g. age, injury, etc). The room and all surfaces within it that may come into direct or indirect contact with the animals must be made of durable, non-porous materials that can be cleaned and disinfected between animals or that can be replaced in between animals and either laundered (e.g. bedding) or disposed of. Medical care must be delivered with consideration for humane handling and restraint strategies that are tailored to the individual animal. The restraint used should be least necessary, in terms of both intensity and duration, to accomplish the necessary procedures without injury to animal or person.

The examination should start with a thorough inspection of the animal for any forms of identification, including collars and tags, microchips, and tattoos. Scanning for a microchip multiple times using proper scanning technique and a scanner capable of reading all microchip frequencies is strongly recommended to increase the chances of detection. A complete and accurate physical description must be recorded for each animal and should contain as much detail as possible. Photographs, including both close-up and full body views, are strongly recommended to facilitate efforts to reunite lost pets with their owners as well as to promote pets for adoption.

A thorough physical examination should be performed on every animal as close to the time of entry to the shelter as possible. It is ideal if such an examination can be performed by a veterinarian, but staff can be trained to perform a basic assessment of the animal’s physical condition and to identify abnormalities that require further evaluation and/or treatment. Initial exam findings should be used to determine the most appropriate type of housing as well as any special handling precautions. If a complete physical exam cannot be performed at the time of intake, the animal should be held in a separate area of the shelter until at least a preliminary examination can be performed to triage the animal, identify any conditions requiring immediate treatment, and assist in the determination of appropriate housing. Both normal and abnormal findings should be recorded in the medical record, and a standardized form should be used to ensure the exam is thorough and documentation is complete.

If the examination is performed by trained staff, detailed protocols should exist regarding further assessment by a veterinarian, including what conditions require emergency care. If appropriate treatment cannot be provided at the shelter, arrangements must be made to transfer the animal to a private veterinary facility or for humane euthanasia and these should be specified in advance per a standard protocol. Similarly, protocols should exist regarding the management of animals with clinical signs that are consistent with common infectious diseases; generally, animals with these signs should be considered contagious until determined otherwise by a veterinarian and must be managed accordingly.
Timely vaccination is a significant component of shelter wellness plans because of the high likelihood of exposure to and great risk of infection with serious or even potentially fatal diseases. Vaccinations are an important tool but are insufficient to safeguard animal health and prevent disease transmission in a vacuum. Control of infectious diseases requires a holistic plan that minimizes exposure to pathogens (e.g. adequate sanitation procedures, isolation of contagious animals, etc.) and strengthens host defenses (e.g. appropriate husbandry, stress reduction, etc).

It is important to note that vaccination protocols commonly recommended for shelters differ from those recommended for most privately owned pets seen in general practice. Such distinctions are important, as animals entering shelters are (in general) at much greater risk of disease exposure compared to the average pet in a home. Most sheltering facilities house animals in high-density environments where they may have daily exposure to animals with unknown medical histories and disease risk. At the same time, many animals admitted to shelters may have had little or no preventive care prior to admission. As such, the likelihood of infection is much greater, and the risk of not vaccinating is elevated.

Published guidelines from the American Animal Hospital Association and the American Association of Feline Practitioners provide an excellent overview of shelter-specific recommendations that can be used as the basis of a protocol specific to the population in question. Core vaccines for cats housed in a shelter setting include a parenteral FVRCP. Core vaccines for dogs housed in a shelter setting include a parental DA2PP and intranasal Bordetella bronchiseptica and parainfluenza (with or without adenovirus). Vaccinations should be given at or before the time of intake to maximize efficacy. The use of modified live rather than killed products is strongly recommended because of the more rapid onset of immunity and greater efficacy resulting from generation of both humoral and cell-mediated immune responses, particularly in the face of maternal antibody interference. Core vaccines should typically be administered to all animals of sufficient age (e.g. 4 weeks or older), including those with mild fever, illnesses, or injuries as well as to nursing queens and dams. For pregnant animals, the risk of exposure must be weighed against the risk of vaccination. Rabies vaccination for all cats and dogs should be administered in accordance with local law, either at the time of release or the time intake if there is an expectation of a long-term stay.

Staff must be trained on the proper handling and administration of vaccines. Detailed records regarding vaccination must be maintained for each animal as part of the complete medical record and every shelter should have a written standard operating protocol for the recognition, treatment, and reporting of adverse vaccine reactions.

Failure to vaccinate at the time of intake, or at all, is likely to result in more outbreaks of preventable disease, leading to unnecessary morbidity and mortality as well as probable criticism for the shelter. Unless valid medical records are available documenting an adequate previous vaccination history, shelters should administer the core vaccinations at the time of intake. It is not prudent to assume that a particular population of animals or an individual is likely to have immunity, either from previous vaccination or natural infection. Previous studies have documented that as high as
60-80% of dogs and up to 70% of cats entering shelters do not have positive antibody titers for canine parvovirus, canine distemper virus, feline panleukopenia, respectively.

Parasite control is of particular importance to and a challenge for shelters given their ubiquitous nature, the range of clinical disease that may result, and the potential for zoonotic transmission in many instances. Furthermore, parasitism can contribute to and exacerbate clinical signs caused by other pathogens. Many parasite eggs, larvae, or oocysts are highly resilient in the environment, which may lead to significant contamination of the facility that poses an ongoing risk of infection to newly admitted animals.

At a minimum, all cats and dogs 2 weeks of age and older should be treated with a drug or product effective against hookworms and roundworms, such as pyrantel pamoate, prior to release from the shelter. Ideally, the oral anthelmintic should be given upon intake and repeated as needed during the shelter stay. Coccidiosis may be very common in certain populations, particularly young kittens and puppies and those animals previously living in poor conditions (e.g. hoarding situations). For this reason, routine administration of an anti-coccidial treatment (e.g. ponazuril) at the time of intake may be indicated to treat infected animals and limit environmental contamination and disease transmission that may otherwise occur. Consideration must also be given to treatment of common ectoparasites, particularly ear mites and fleas. Accurate identification and treatment of infested animals at the time of intake is critical. Treatment options for fleas include a variety of topical treatments such as fipronil, selamectin, and others, many of which also treat louse infestations. Routine screening for and/or treatment of other parasites should be determined by the prevalence in the population served; any diagnostic testing as well as any medications administered must be recorded in the medical record.

Special accommodations must be provided for individual animals based on their specific needs. The history, physical exam findings, and observed behavior of the animal will inform staff as to which housing options are most appropriate. Housing should be segregated based on the animal's species, age, health, behavior, and reproductive status. Aggressive, very shy or fearful, or otherwise distressed animals should have special accommodations made to meet their behavioral needs, prevent escape, and limit the risk of injury to both animal and staff and volunteers. They should be housed in a quiet area of the shelter and be assigned consistent, dedicated caretakers who are knowledgeable about animal behavior and comfortable with handling cats or dogs across the spectrum of stress, fear, and aggression. Routine quarantine procedures are not typically recommended for most animals entering shelters unless there is a specific concern with a subpopulation of animals or a disease outbreak. Animals with evidence of contagious disease must be isolated from the general population. Ideally, animals who are badly injured, suffering from severe illness, have chronic medical conditions that require intensive monitoring, or who are recovering from surgeries other than routine spay and neuter should not be routinely housed in shelters unless the facility has a veterinary clinic with sufficient staff to provide for their needs. If humane care cannot be provided in the shelter arrangements must be made for treatment at a private veterinary facility or for humane euthanasia. Similarly, placement in foster care is generally the best option for
care of underaged kittens or puppies and lactating queens or dams as it is very difficult to meet these animals’ physical and behavioral needs in most shelter facilities.
Management of infectious respiratory disease in dogs in shelter environments remains a particular challenge and health concern. The term canine infectious respiratory disease complex (CIRDC) is typically used to describe a range of clinical signs consistent with upper respiratory infection in dogs, including sneezing, coughing and nasal discharge. However, such a diagnosis does little to inform the practitioner as to the underlying causes, as nearly a dozen pathogens have been associated with the disease complex. Incubation periods range from as little as 48 hours (acute canine influenza virus infection) to as long as 4-5 weeks (canine distemper infection), as does the presence of a carrier state and duration of shedding following infection. Most of the abnormalities detected in infected dogs are similar and overlap from pathogen to pathogen, resulting in clinically indistinguishable disease that requires laboratory testing to determine an etiologic diagnosis. Coinfections are thought to be common and have been documented to result in increased morbidity and mortality.

The following organisms have been associated with CIRDC in dogs, either alone or in combination with one another or with opportunistic bacterial infections:

- Bordetella bronchiseptica
- Canine adenovirus type 2 (CAV-2)
- Canine distemper virus (CDV)
- Canine herpesvirus (CHV)
- Canine influenza virus (CIV), H3N8 and H3N2 strains
- Canine parainfluenza virus (CPiV)
- Canine pneumovirus
- Canine respiratory coronavirus (CRCoV)
- *Mycoplasma* spp.
- *StREPtococcus equi subsp. zooepidemicus*

Several of the etiologic agents associated with CIRDC are either emerging or reemerging pathogens. Many were first detected in dogs housed in high density environments, including CRCoV (2003), H3N8 CIV (2004), and *StREPtococcus equi subsp. zooepidemicus* (2007). Canine distemper is re-emerging as a significant cause of respiratory disease in some shelters and communities as well. An incomplete picture exists for many of these emerging pathogens with regards to their role as primary or secondary contributors to CIRDC and more study is needed.

Disease caused by various etiologic agents in the CIRDC is usually mild with low mortality rates, but severe and even fatal disease can occur with some pathogens and in certain situations. Dogs of all breeds and ages are susceptible to CIRDC, with increased risk of infection and severe disease for puppies and immunocompromised individuals. A higher incidence of disease may be noted in summer and fall months, but for many shelters the level of endemic disease remains constant year-round. Environmental conditions (e.g. high density housing, poor facility design, inadequate ventilation systems) and high stress levels found in many shelters are significant risk factor for the development of disease and more severe clinical signs.
Typical clinical signs include coughing, sneezing, nasal discharge, and mild pyrexia. Some infected dogs will remain asymptomatic, including up to 20% of those infected with canine influenza virus. Additionally, severe clinical signs of lower respiratory disease and/or systemic illness can be seen in dogs infected with various pathogens of the CIRDC. For example, canine influenza virus infection can produce pneumonia in a small number of infected dogs and *Streptococcus equi* subsp. *zooepidemicus* has been implicated as the causative agent in several outbreaks of hemorrhagic pneumonia, characterized by sudden death and high mortality rates.

Diagnostic testing must be performed in order to identify the specific cause(s) and can be particularly helping in targeting treatment and control measures. Clinical signs, patterns in the affected population, and severity of illness may provide an index of suspicion for one or more of the etiologic agent(s). For example, a very high incidence of upper respiratory signs in all the dogs in the shelter (e.g. puppies and adults, vaccinated and unvaccinated) can be expected when canine influenza virus is introduced into a naïve population. However, involved this index of suspicion cannot be used as a substitute for appropriate laboratory testing. It is important to remember that the same pathogen can have a mild presentation in one dog yet cause severe or even fatal disease in another dog.

Diagnostics are available for all of the pathogens implicated in CIRDC but the most sensitive and specific tests will vary from organism to organism. Similarly, the ideal sample(s) to be collected and time of collection will vary with organisms of concerns and clinical signs noted. Options include polymerase chain reaction (PCR) testing, virus isolation, bacterial culture and sensitivity, enzyme-linked immunosorbent assays (ELISA), serology, immunofluorescence and immunohistochemistry, and histopathology. When choosing tests it is important to confirm with the laboratory that the individual selections or panels are appropriate options to detect the pathogens of concern in the samples that are being collected. Diagnostic test selections should be made with consideration to the timing and location of sample collection, with careful attention paid to the onset and duration of shedding.

Deep nasal and oropharyngeal samples are likely to be the highest yield in dogs with signs limited to the upper respiratory tract, while tracheal wash samples may be more appropriate for dogs with evidence of lower airway involvement. Invaluable information can be obtained through necropsies when disease is severe enough that dogs die and/or are humanely euthanized. Post mortem examination can help identify the presence of various etiologic agents as well as provide insight as to the role they play in the clinical condition observed. Necropsy has been shown to be the most accurate method for collecting appropriate diagnostic samples and often allows for sample collection and testing methods that are not possible or practical ante-mortem. Depending on the available resources and services provided at the laboratories accessible to the practitioner the entire body be submitted for a complete necropsy or the gross evaluation can be performed on-site and samples submitted for histopathology, viral isolation, and bacterial culture. Lung and tracheal samples are particularly valuable and several pieces should be collected and stored appropriately (e.g. formalin-fixed, fresh or fresh/frozen) for these respective tests. If possible, samples collected during post mortem examination should be from dogs with clinical signs representative of that seen in other affected animals. Ideally, these dogs would
not have received prior therapy that would be expected to alter results (e.g. previous antimicrobial use) unless poor response to treatment is a concern also being investigated.

Testing all individual cases of CIRDC is seldom practical or feasible in the shelter setting. For this reason, most diagnostic testing for CIRDC is performed to determine which pathogens are present and causing disease in the population. Testing should be pursued when the severity or incidence of disease changes, when unusual clinical signs are noted or mortality has increased, and in the face of an outbreak. Acutely affected dogs should be sampled, prior to initiation of treatment and in sufficient numbers to provide data representative of the larger population (typically 10-30% of the population with a minimum of 3-5 animals). Larger sample sizes improve one's ability to detect uncommon diseases or those organisms for which a diagnosis may be challenging to obtain. In addition, positive results in several dogs for one pathogen or another provides much more convincing evidence of that organism’s involvement in the disease seen then when such results are confined to one or two cases as many of the pathogens can be isolated from clinically normal dogs.

Treatment is usually symptomatic and supportive in nature, and aimed at preventing secondary bacterial infections as well as providing definitive treatment, if appropriate, for any primary pathogens and other medical conditions. Antibiotic therapy is indicated for primary and secondary bacterial infections, which do occur commonly in shelter settings. Although it is ideal to make therapeutic choices based on sensitivity results, this is seldom possible on a consistent basis for all infected dogs due to financial and logistical restraints. Empirical therapy can be instituted based on likely pathogens and historical susceptibility data, but no one antibiotic will be appropriate for all cases. For example, doxycycline often has good efficacy against *Bordetella* and *Mycoplasma* but would be expected to be less successful when used to treat dogs infected with *Streptococcus equi* subsp. *zooepidemicus*. Culture and sensitivity should be performed when adequate response to empirical therapy is not seen and should be considered during outbreaks to identify the most efficient course of treatment.

The use of steroids, antitussives, and expectorants is generally contraindicated in the shelter environment. Because coughing is often exacerbated with excitement, exercise (particularly pulling on the leash) and barking, appropriate environmental modifications should be made to limit these triggers. More severely affected dogs require more aggressive treatment, which may include hospitalization, intravenous fluid therapy, oxygen support, nebulization and coupage. If it is not possible to provide such intensive treatment or if inadequate response to treatment is noted in individual cases these dogs must be considered for transfer to a private veterinary facility or for humane euthanasia.

Despite the relatively mild disease that is typically seen, CIRDC remains a particular concern for shelters (as well as other facilities where large numbers of dogs are housed in close proximity to one another, such as boarding facilities) because of the significant morbidity and ease of transmission. Prompt identification and diagnosis of cases, treatment of infected dogs, and implementation of control measures is critical.

Timely removal of clinically affected dogs to strict isolation must be undertaken to limit the spread of disease. Dogs should be moved as soon as possible once clinical signs are
noted, as the intensity of shedding does not necessarily correlate with the severity or duration of clinical signs observed. Most of the pathogens are efficiently transmitted by direct oronasal contact as well as aerosolization of respiratory secretions from infected dogs. Thus, wherever possible, isolation areas should have separate ventilation to reduce the risk of further airborne transmission of CIRDC. The need for isolation should be based on the presence of compatible clinical signs without regard to their severity because mild clinical signs can be the result of potentially deadly pathogens capable of causing much more severe symptoms, such as canine distemper virus. Depending on the nature of the disease and available resources for treatment, placement into foster care, release to rescue organizations, or hospitalization at a private veterinary clinic may be appropriate options for handling and treatment of infected dogs.

Appropriate precautions to prevent further spread of disease and to protect shelter, household, and/or community pets and people must be implemented. Special care should be taken to protect the health of other dogs in the facility as well as that of other species, including humans, given the potential for transmission. There has been at least one report of clinical disease in cats in a cattery setting caused by Streptococcus equi subsp. zooepidemicus as well as transmission from an infected dog to his handler, and Bordetella bronchiseptica is also known to infect both cats and humans (albeit most human infections occur infrequently and in immunocompromised individuals). Contact with infected dogs should be restricted to key staff and volunteers. Whenever possible, these individuals should not work with other dogs. If this is not feasible, appropriate personal protective equipment (PPE) must be worn and should handle dogs in isolation only after handling healthy animals in the general population.

Sanitation protocols, procedures for movement of animals through the facility, and preventive medicine protocols must be reviewed periodically and whenever an outbreak is identified. Although most of the various CIRDC pathogens are not particularly resilient in the environment, adequate cleaning and disinfection practices are necessary to limit transmission. Canine adenovirus is a notable exception and thus disinfectants with efficacy against non-enveloped viruses (e.g. sodium hypochlorite or similar products, potassium peroxymonosulfate, accelerated hydrogen peroxide) must be used. A thorough review of sanitation procedures should include an evaluation of the product(s) used as well the manner in which they are diluted and applied to various surfaces by staff (including allowed contact time); this evaluation should consider written protocols as well as a visual assessment of these procedures as they are actually performed. In addition, staff assignments should be made to limit the spread of disease and reduce fomite transmission, including restricting access to small subsets of the population, the use of appropriate personal protective equipment, and the provision of dedicated supplies used only in limited areas of the facility.

Humane conditions and medical care as well as adequate isolation space to prevent transmission to the rest of the population must be available. When it is not possible to meet these needs euthanasia must be considered. Depending on the size of the facility, number of staff, and number of infected dogs it may become necessary to temporarily halt intake to avoid jeopardizing the health of incoming animals and avoid the need for depopulation. While the depopulation of a group of animals in a shelter is
one option available for control of disease in the face of an outbreak, the need for such drastic actions is rare and it should be utilized only as a carefully considered last resort.

CIRDC is not a vaccine-preventable disease. While vaccines are commercially available for many of the more common and serious pathogens associated with the disease complex, others (including canine respiratory coronavirus, herpesvirus, and pneumonivirus as well as *Mycoplasma* spp. and *Streptococcus equi* subsp. *zooepidemicus*) do not exist or are not currently available for use in the United States. In addition, many of the products that are available simply limit severity without preventing infection – creating a situation where respiratory disease can still be frequently seen despite adherence to recommended vaccination protocols appropriate for dogs in animal shelter settings. Despite these limitations, vaccination remains an important tool for limiting both the incidence and severity of CIRDC in shelter-housed dogs by providing protection against specific pathogens as well as reducing morbidity associated with co-infections.

It is critical that animals entering the facility receive appropriate vaccinations at or before their entry to the shelter in order to maximize effectiveness and reduce the risk of disease outbreaks. Training should be periodically provided to ensure that vaccinations are being handled appropriately (e.g. kept refrigerated, reconstituted prior to use) and administered in compliance with shelter protocols. Core vaccinations for shelter dogs include canine distemper virus, canine parvovirus, and canine adenovirus-2 given parenterally as a modified live product as well as intranasal vaccination using a bivalent product that contains *Bordetella bronchiseptica* and canine parainfluenza virus. Vaccination against canine influenza may be appropriate in some facilities, but immunity should not be expected until approximately one week following the second dose (e.g. three weeks following initial vaccination). Perhaps the most critical of all control measures is the reduction of stress and overcrowding, which go hand and hand. Overcrowding is an extremely potent stressor of dogs and exacerbates many negative factors that shelters are already struggling with. High density housing, particularly when young and naïve dogs are intermixed with other dogs in the facility, is a significant risk factor for disease. Length of stay in a shelter environment has been shown to be a risk factor for seroconversion and development of clinical signs associated with CIRDC pathogens. Keeping fewer dogs in the facility at any given time, particularly by reducing the length of time they spend in the shelter, generally improves the level of animal care and sanitation that can be provided and reduces the risk of direct or indirect contact with infected animals. Direct physical benefits of stress reduction include reductions in circulating cortisol levels and decreased barking, which in and of itself can help to reduce respiratory irritation and aerosol spread of the causative agents.
INTRODUCTION

The ability of dairy cattle to adapt to the natural change of energy balance in early lactation is an important aspect of the transition period, as the demands for milk production cannot be met by feed intake alone. In order to maintain homeorhesis during this period of negative energy balance, cows break down adipose tissue to produce non-esterified fatty acids (NEFA), which are partially converted to ketone bodies (e.g., β-hydroxybutyrate; BHB) and can both act as alternate fuel sources. An elevation of blood NEFA and BHB is normal during this time in order for cows to adapt to the physiologic changes that occur from late gestation through early lactation, however cows that have a poor adaptive response to negative energy balance will produce excessive NEFA and/or BHB. This excessive production, particularly in cows diagnosed with hyperketonemia (a blood BHB concentration ≥1.2 mmol/L), has been shown to have detrimental effects on immune function, milk production, and overall health. Given that 85-95% of hyperketonemic cows do not show signs consistent with clinical ketosis\(^1,2\), the health and production consequences of a poor transition into lactation are often unseen.

DIAGNOSTICS

Evaluation and monitoring of individual and herd-level negative energy balance in early lactation has revolved around measurement of NEFA and BHB. Blood NEFA concentrations have been found to be a stronger indicator of disease, milk production, and reproductive performance than blood BHB concentrations\(^3,4\); however, current technology does not allow for easy collection and processing of blood samples for NEFA analysis. To obtain samples and accurate results, blood for NEFA analysis should be collected in an EDTA or non-anticoagulant tube, placed on ice immediately after collection, kept at 4°C until processing, and the plasma or serum separated within 24 hours\(^5\). Samples with a hemolytic index ≥ 300 should be interpreted with caution. Due to a natural rise of NEFA a few days before parturition, collection of samples between 14 and 3 days prior to expected calving is optimal\(^6\); some samples may need to be discarded if cows then calve outside of this window. There is currently no true “cowside” test for NEFA, and current costs for NEFA analysis range from $11-16 at the New York State Animal Health Diagnostic Center and the Michigan State Diagnostic Center. Hyperketonemia monitoring through blood BHB measurement is less expensive and more practical than determination of blood NEFA concentrations and will be discussed below.

As BHB is the most stable ketone body in blood, the gold standard for detection of hyperketonemia is laboratory analysis of blood BHB via a kinetic enzymatic assay. However, there are numerous cowside tests varying in accuracy that are available for practical on-farm diagnosis of hyperketonemia. The most common ketone tests use either urine, milk, or blood, although the sensitivity and specificity of each type of test varies tremendously (Table 1). To complicate matters when using a urine test, only 50%
of cows can typically be induced to urinate while sampling. Meters for blood BHB monitoring can be purchased for $20-50 with the largest complaint being that they don’t work well in cold temperatures.

Table 1. Cowside ketone test characteristics.

<table>
<thead>
<tr>
<th>Test</th>
<th>Reading</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Cost per test (USD)</th>
<th>Lab BHB Reference Conc.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine – acetoacetic acid (Ketostix)(^6)</td>
<td>≥15 mg/dL (small)</td>
<td>78%</td>
<td>96%</td>
<td>$0.20</td>
<td>1.4 mmol/L</td>
</tr>
<tr>
<td></td>
<td>≥40 mg/dL (moderate)</td>
<td>49%</td>
<td>99%</td>
<td>$0.20</td>
<td>1.4 mmol/L</td>
</tr>
<tr>
<td>Milk – acetoacetic acid (KetoCheck)(^6)</td>
<td>Purple color</td>
<td>41%</td>
<td>99%</td>
<td>$0.60</td>
<td>1.4 mmol/L</td>
</tr>
<tr>
<td>Milk – BHB (KetoTest)(^6,7)</td>
<td>100 µmol/L</td>
<td>73-80%</td>
<td>76-96%</td>
<td>$1.70</td>
<td>1.4 mmol/L</td>
</tr>
<tr>
<td></td>
<td>200 µmol/L</td>
<td>27-59%</td>
<td>90-99%</td>
<td>$1.70</td>
<td>1.4 mmol/L</td>
</tr>
<tr>
<td>Milk – BHB (PortaBHB)(^8)</td>
<td>100 µmol/L</td>
<td>89%</td>
<td>80%</td>
<td>$2.00</td>
<td>1.4 mmol/L</td>
</tr>
<tr>
<td></td>
<td>200 µmol/L</td>
<td>40%</td>
<td>100%</td>
<td>$2.00</td>
<td>1.4 mmol/L</td>
</tr>
<tr>
<td>Blood – BHB (Precision Xtra)(^9,10,11)</td>
<td>1.2 mmol/L</td>
<td>85-96%</td>
<td>94-98%</td>
<td>$1.50</td>
<td>1.2 mmol/L</td>
</tr>
<tr>
<td></td>
<td>1.4 mmol/L</td>
<td>90-100%</td>
<td>98-100%</td>
<td>$1.50</td>
<td>1.4 mmol/L</td>
</tr>
<tr>
<td>Blood – BHB (Novamax)(^12)</td>
<td>1.2 mmol/L</td>
<td>97%</td>
<td>82%</td>
<td>$2.75</td>
<td>1.2 mmol/L</td>
</tr>
</tbody>
</table>

Hyperketonemia, by definition, is the elevation of ketone bodies above a physiologically normal range. Although historically the terms “subclinical” and “clinical” ketosis have been used to describe this metabolic state of ketone elevation, classification into these two categories is quite subjective. It is important to remember that expression of signs consistent with ketosis has much individual-animal variation and that manifestation of these signs most likely suggests a more severe hyperketonemia compared with cows that do not show clinical signs. For this reason, rather than refer to different severities of the same disease as subclinical or clinical, I prefer to use the term “hyperketonemia” which encompasses both groups. Recent research has provided solid and repetitive evidence of quantitative blood BHB concentration cut-points that result in increased risks of subsequent disease development and production loss. This cut-point falls somewhere around 1.2 to 1.4 mmol/L depending on the desired sensitivity and specificity\(^3,4,13\); a lower cut-point will result in a higher test sensitivity and lower specificity and a higher cut-point will results in a lower sensitivity and higher specificity.
INDIVIDUAL ANIMAL HEALTH EVENTS AND PRODUCTION
The incidence of early lactation hyperketonemia (number new cases divided by the number of cows at risk), has been reported in only 2 studies\textsuperscript{14,15} as repetitive testing for incidence estimation is time and labor intensive. One of these studies\textsuperscript{2} followed 1,717 cows from 3-16 days in milk (DIM) in 4 free-stall, total mixed ration fed herds in New York and Wisconsin. Using a Precision Xtra meter, all cows were monitored for hyperketonemia (BHB \( \geq 1.2 \) mmol/L) on a Monday, Wednesday, Friday testing schedule, and each cow was tested 6 times between 3-16 DIM. Peak incidence occurred at 5 DIM (Figure 1) with 22% of cows testing hyperketonemia positive for the first time; 75% of cows that developed hyperketonemia between 3 and 16 DIM were first positive between 3 and 7 DIM.

Figure 1. Histogram of incidence of hyperketonemia (HYK) in 1,717 Holstein dairy cows undergoing repeated testing for HYK from 3 to 16 DIM. A positive test was defined as a blood BHB concentration \( \geq 1.2 \) mmol/L (Adapted from McArt et al., 2012).

Multiple studies have shown that the risk or odds of metritis, displaced abomasum (DA), and early lactation culling are increased for hyperketonemic animals\textsuperscript{2,4,13,14}. Measurements of reproductive performance have shown more inconstancies in their association with elevated BHB in early lactation. In addition, most studies report a decrease in milk production (in the range of 2-3 kg/cow/day) in hyperketonemic cows compared to non-hyperketonemic animals, although this effect appears to wane as lactation progresses\textsuperscript{1,4,14}. Many of the risks regarding subsequent disease development and milk production loss increase as blood BHB concentration increases\textsuperscript{2,13} (i.e. the more severely hyperketonemic a cow is, the higher her chances of incurring additional disease events as well as a more pronounced drop in milk yield). The time of onset of hyperketonemia also affects the risk of subsequent disease development, reproduction, and milk production. Cows that became hyperketonemic between 3 to 7 DIM were over 6 times more likely to develop a DA, 4.5 times more likely to be removed from the herd, 0.7 times as likely to conceive to first service, and made almost 2.5 kg less milk per cow per day for the first 30 DIM than cows first testing HYK positive between 8 and 16 DIM\textsuperscript{2}. Thus it is important to identify these hyperketonemic animals early in lactation in order to reduce the risk of subsequent negative events.

INDIVIDUAL ANIMAL TREATMENT
Once a hyperketonemic animal is identified, immediate treatment is warranted to prevent negative subsequent health events and production losses. An excellent systematic review of hyperketonemia treatment in lactating dairy cattle found only 10 publications that reported a clearly defined method of hyperketonemia diagnosis before initiation of treatment, included a control group, had defined outcomes of interest, and used appropriate statistical methodology. Interestingly, no studies have evaluated the effect of intravenous dextrose administration on hyperketonemia although this treatment is commonplace. Treatment of hyperketonemic cows with oral propylene glycol has been shown to improve resolution of hyperketonemia, decrease subsequent negative health events, and increase milk production. Propylene glycol acts by increasing blood glucose concentrations through its conversion to propionate as well as its actions in decreasing peripheral tissue glucose demand. A dose of 300 mL of propylene glycol orally once a day for 3-5 days is recommended given the median duration of a hyperketonemic event. Glucocorticoids have been used for years to treat hyperketonemia given their action in stimulating gluconeogenesis and providing glucose precursors, however few studies have scientifically supported the use of glucocorticoids in the treatment of hyperketonemia. Recent work suggests even one dose of dexamethasone at diagnosis of HYK may be detrimental to cows with BHB $> 2.0$ mmol/L. Similarly, few studies have described the benefits of B vitamin use in the treatment of hyperketonemia, although two studies have reported a decreasing trend of blood BHB concentration in hyperketonemic cows treated with a butaphosphan and cyanocobalamin combination product.

CONCLUSIONS
Excessive negative energy balance in early lactation is an issue in many dairy herds. As most cases of hyperketonemia are subclinical in nature, associated disease events, early removal from the herd, and production losses are often unrecognized. Cows that develop hyperketonemia within the first week of lactation are at a greater risk of subsequent disease development and milk loss; this risk increases with higher concentrations of blood BHB. Testing should be focused on cows that are in the first 2 weeks of lactation with even more emphasis placed on animals 3-9 DIM, as a cow that is hyperketonemic during this period has the highest risk for subsequent negative health and production effects. Additionally, test sick cows that are off feed, appear depressed, or have had a recent drop in milk production.

REFERENCES
INTRODUCTION
Given the ease and economics of cowside β-hydroxybutyrate (BHB) testing, knowledge of herd-level monitoring approaches is an important tool for bovine veterinarians from both a disease and economic standpoint. Recent research has estimated the financial impact of hyperketonemia on a component cost per case (i.e. the sum of the cost of diagnostics, therapeutics, labor, death loss, and future milk production, culling, and reproduction losses) and a total cost per case basis (i.e. the component cost plus cost related to hyperketonemia-attributable diseases). The average component cost per case was estimated to be $117; the average total cost per case when accounting for costs attributable to displaced abomasum and metritis was estimated to be $289\textsuperscript{1}. Given the high total cost, reported incidences of hyperketonemia at 40-60% highlight the importance of appropriate nutrition and management strategies as well as herd-level monitoring and individual animal treatment plans.

HERD-LEVEL MONITORING
Whereas previously discussed ketone tests have great use as tools for diagnosis of hyperketonemia in individual animals, they can also be incorporated into a program for herd-level monitoring in order to assist with management decisions. It is important to note that the sensitivity and specificity of different tests can have a large impact on individual test results and, and as a consequence, herd diagnosis and treatment\textsuperscript{2}. The probability of correctly identifying a herd as positive (i.e. having a prevalence of hyperketonemia above a certain threshold) decreases when a test with low sensitivity is used. For these reasons, use of handheld blood BHB meters are suggested for herd-level monitoring.

Once you have decided which tool you will use to monitor ketones in your herd, there are two important measurements which will help you evaluate the degree of ketosis on a farm: incidence and prevalence. Incidence is the number of cows that develop ketosis any time during early lactation (typically about the first 2-3 weeks in milk) divided by the total number of cows that have gone through this early lactation time period. In order to calculate incidence, choose a group of cows to follow through the early lactation period; determining the incidence of hyperketonemia requires repeated testing of cows throughout this risk period. Testing must occur twice or more per week in order to accurately assess the incidence of hyperketonemia. This is necessary because the median time for the resolution of hyperketonemia is approximately 5 days\textsuperscript{3}. If, for example, 15 cows within a group of 50 cows develop hyperketonemia sometime in early lactation, the incidence is 30% (15/50). Prevalence is a snapshot of the amount of hyperketonemia in the herd at one point in time. It is calculated by dividing the number of hyperketonemic cows by the number of cows sampled at that point in time. For example, if 3 cows were hyperketonemic out of 20 cows tested that day, the prevalence would be 15%. Prevalence is more current and a lot easier to determine than incidence. It is used for general herd monitoring and for gauging the
effectiveness of changes in dry or fresh cow management. Although the prevalence of hyperketonemia in a herd is much easier to determine than its incidence, the incidence of hyperketonemia must be known in order to estimate the overall negative impacts of hyperketonemia on herd performance. The repeated testing necessary to determine the incidence of hyperketonemia within a herd is daunting and requires testing a large number of cows twice weekly for about the first two weeks of lactation. Fortunately, the incidence of hyperketonemia can be estimated from its prevalence; the incidence of hyperketonemia has been reported to be approximately 2.2 times the prevalence. The number of animals tested in order to determine herd-level prevalence of hyperketonemia is also important, as an increase in sample size will increase the precision of the estimate.

**HERD-LEVEL HEALTH EVENTS AND PRODUCTION**

Herds with a high prevalence of hyperketonemia are more likely to have a higher incidence of postpartum disease, poorer reproduction, and lower milk production. Research completed in over 150 herds in the United States and Canada suggests that this association between herd hyperketonemia prevalence and disease and production outcomes occurs when ≥ 15-25% of animals have elevated postpartum blood BHB concentrations. Ospina et al. (2010) found that 40% of the 100 herds tested were above the herd alarm level. Unfortunately, hyperketonemia is an issue on many herds, and additional studies have found the incidence and prevalence of hyperketonemia to be quite high across herds in the United States, Canada, and Europe (Table 1).
**Table 1.** Summary of recent research on incidence and prevalence of elevated postpartum blood BHB concentrations in dairy cows. Cow-level incidence and prevalence refer to the number of positive animals across all herds combined, whereas herd minimum and maximum refer to individual herd-level incidence and prevalence (Adapted from McArt et al., 2013).

<table>
<thead>
<tr>
<th>Study</th>
<th>No. Herds</th>
<th>No. Cows</th>
<th>Cut-point (mmol/L)</th>
<th>Time</th>
<th>Freq.</th>
<th>Cow-level (%)</th>
<th>Herd Min (%)</th>
<th>Herd Max (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Post-partum incidence</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duffield et al., 1998</td>
<td>25</td>
<td>507</td>
<td>≥1.2</td>
<td>1 to 63 DIM</td>
<td>1/week</td>
<td>59</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>507</td>
<td>≥1.4</td>
<td>1 to 63 DIM</td>
<td>1/week</td>
<td>43</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>McArt et al., 2012b</td>
<td>4</td>
<td>1,717</td>
<td>≥1.2</td>
<td>3 to 16 DIM</td>
<td>3/week</td>
<td>44</td>
<td>27</td>
<td>57</td>
</tr>
<tr>
<td><strong>Post-partum prevalence</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LeBlanc et al., 2005</td>
<td>20</td>
<td>1,063</td>
<td>≥1.2</td>
<td>1 to 7 DIM</td>
<td>-</td>
<td>20</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Duffield et al., 2009</td>
<td>1,010</td>
<td>987</td>
<td>≥1.2</td>
<td>1 to 7 DIM</td>
<td>-</td>
<td>24</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>1,010</td>
<td>941</td>
<td>≥1.2</td>
<td>8 to 14 DIM</td>
<td>-</td>
<td>25</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Ospina et al., 2010a</td>
<td>104</td>
<td>1,318</td>
<td>≥1.2</td>
<td>3 to 14 DIM</td>
<td>-</td>
<td>18</td>
<td>0</td>
<td>71</td>
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<tr>
<td>Seifi et al., 2011</td>
<td>16</td>
<td>849</td>
<td>≥1.0</td>
<td>1 to 7 DIM</td>
<td>-</td>
<td>31</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Chapinal et al., 2012a</td>
<td>45</td>
<td>2,069</td>
<td>≥1.4</td>
<td>1 to 7 DIM</td>
<td>-</td>
<td>12</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>45</td>
<td>2,069</td>
<td>≥1.2</td>
<td>8 to 14 DIM</td>
<td>-</td>
<td>20</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Suthar et al. (2013)b</td>
<td>528</td>
<td>5,884</td>
<td>≥1.2</td>
<td>2-15 DIM</td>
<td>-</td>
<td>22</td>
<td>11</td>
<td>37</td>
</tr>
</tbody>
</table>

**DIM,** days in milk

*a* Cows were classified as positive if they had at least one test above the cut-point.

*b* Prevalence in 10 European countries.
ECONOMICS OF TESTING AND TREATMENT
Figure 1 outlines a suggested approach for monitoring of hyperketonemia and action recommendations in herds following prevalence sampling. This flow chart is based from work that evaluated 4 different testing and treatment strategies at varying herd incidences of hyperketonemia using stochastic and iterative modeling. Results indicate that at herd incidences above 50%, blanket treatment of all fresh cows with 5 days of oral propylene glycol starting at 5 DIM was the most cost-effective strategy. At incidences between 15 and 50%, testing cows that are 3 through 9 DIM two days per week (e.g. Mondays and Thursdays) and treating hyperketonemic cows with 5 days of oral propylene glycol was the most cost-effective strategy, although testing all cows 3 through 16 DIM one day per week (e.g. Mondays) also provided a positive return on investment. For a herd with a 40% incidence of hyperketonemia that freshens 1,000 cows per year, choosing to test cows two days per week and treating the positives will return a benefit of $10,000 to $25,000 per year.

![Flowchart of Monitoring and Action Recommendations](image)

**Figure 1.** Monitoring recommendations for BHBA testing based on herd-level prevalence of elevated BHBA (Adapted from Ospina et al., 2013).

PUTTING IT ALL TOGETHER
Choose a test with a high sensitivity and specificity. Conduct a hyperketonemia prevalence assessment (sample 15 to 20 cows) in a herd in order to approximate herd incidence and determine the best testing and treatment plan. For herds with an estimated incidence greater than 50%, where blanket treatment with PG is initiated, repeated prevalence testing is necessary after management changes to determine if treating all fresh cows remains the best option. For herds with an incidence of 15 to 50%, either a one or two day per week testing strategy will allow for repeated
monitoring of herd incidence and improve individual animal health. Repeated incidence or prevalence testing is recommended in order to evaluate changes in transition cow management and allow appropriate adjustment of farm hyperketonemia testing and treatment protocols. Remember, the goal is to not treat many, if any, cows with propylene glycol, but rather have transition cow management strategies in place such that the prevalence of hyperketonemia is lower than 10%.

CONCLUSIONS
Excessive negative energy balance in early lactation is an issue in many dairy herds. As most cases of hyperketonemia are subclinical in nature, associated disease events, early removal from the herd, and production losses are often unrecognized. Cows that develop hyperketonemia within the first week of lactation are at a greater risk of subsequent disease development and milk loss; this risk increases with higher concentrations of blood BHBA. Individual and herd-level testing should focus on the first week to two weeks of lactation in order to optimize individual animal treatment and herd management practices.

REFERENCES
HYPOCALCEMIA IN POSTPARTURIENT DAIRY CATTLE

Jessica McArt, DVM, PhD
Ithaca, NY

INTRODUCTION
One of the challenges in dairy cattle management is the maintenance of optimum blood calcium concentration during the early postpartum period. Postparturient hypocalcemia occurs due to the massive mobilization of calcium needed to initiate and sustain lactation. When there is increased demand for calcium, falling serum calcium concentration stimulates parathyroid hormone secretion, which increases serum calcium via effects in the kidney, gastrointestinal tract and bone. Hypocalcemia results when serum calcium concentration falls faster than homeostatic mechanisms can adapt to the demands of lactation.

Clinical hypocalcemia (milk fever) affects approximately 5% of periparturient dairy cattle\(^1\). This disease results in weakness that can progress to recumbency and death and is a widely accepted risk factor for subsequent displaced abomasum, ketosis, metritis, retained placenta, mastitis, culling, and decreased milk production\(^2-6\). Subclinical hypocalcemia, recently defined by Reinhardt and colleagues as a serum calcium concentration <2.0 mmol/L within 48 hours of calving, is much more prevalent, affecting up to 50% of postpartum multiparous cows\(^7\). Recent evidence suggests subclinical hypocalcemia is also a risk factor for future adverse health events\(^1,2,4-7\). It has been estimated that single-herd annual costs for hypocalcemia can exceed $50,000\(^7\).

MONITORING TOOLS
The majority of calcium in the blood is in a free or ionized form (50-60%), which is recognized to be the biologically active form responsible for calcium homeostasis. Approximately 30% is bound to proteins, primarily albumin, with another 10% in a complexed form bound with phosphate, bicarbonate, sulfate, citrate, or lactate. Although ionized calcium makes up the majority of total blood calcium concentration, measurement of ionized calcium is expensive and requires special handling of samples, thus most research has been conducted using total calcium measurements. Although some studies show that total calcium is reasonably associated with ionized calcium concentrations in bovine blood, this relationship has been found to change near calving and requires further investigation\(^8\).

Total calcium is more stable than ionized calcium and is currently the suggested form to analyze. Blood should be collected into heparin (green top) or non-anticoagulant (red top) tubes; collection into EDTA, citrate, or sodium fluoride tubes may reduce total calcium concentrations to an unmeasurable level due to binding agents found in these tubes. Whole blood can be stored in a green or red top tube for up to 6 hours at 4 or 22°C with minimal changes in total calcium measurements (Neves and Stokol,
Cornell University, unpublished data). For best results, however, plasma or serum should be separated as soon as possible.

Until recently, the only well-validated way to measure total calcium was via benchtop analyzer. Costs per sample range from USD$10-$20 at accredited veterinary diagnostic laboratories, and analyzers found in veterinary clinics range from USD$5-$7.50 per sample. These machines are not economical for individual cow monitoring due to their cost and slow turnaround time; however, herd-level monitoring, while expensive, is more realistic. Recent work by Sweeney and colleagues showed that the IDEXX VetTest is an option for on-farm calcium testing at both the individual and herd-level, however between-machine variation needs to be addressed\(^8\), and cost of the machine and slides is still significant. A few additional machines have recently come on the market advertising their use as “on-farm” total calcium measurement tools, but to date, none of these machines has been well validated although current trials are underway to this end.

Cut-points for hypocalcemia diagnosis taken within the first 24 hours postpartum range from 8.6 mg/dL (2.15 mmol/L) to 8.0 mg/dL (2.0 mmol/L)\(^1\). As with all tests, a use of a higher cut-point decreases the sensitivity and increases the specificity of the result. Clinical hypocalcemia is much easier to diagnose based on observation alone; total calcium concentrations are often < 6.0 mg/dL (1.5 mmol/L) in animals displaying signs of milk fever\(^1\).

Ionized calcium is extremely problematic to measure due to sample collection and handling difficulties. Exposure of a blood sample to air changes the pH and thus the amount of ionized calcium in the sample; stability over time is questionable, and the sample must be processed as soon as possible. Samples can be submitted to veterinary diagnostic labs or run in veterinary clinics, but results are often not accurate due to the aforementioned handling difficulties and the time it takes for a sample to arrive at a lab or clinic location. Machines targeted for on-farm use (e.g. iSTAT, VetStat, Nova Stat) cost approximately USD$5,000-$15,000 in addition to per sample costs. At USD$15-$20 per sample, ionized calcium is not currently a cost-effective method of individual animal or herd-level hypocalcemia monitoring on farms, in veterinary clinics, or by diagnostic laboratories.

Given the sample handling and cost issues with obtaining accurate ionized calcium measurements, not much work has been reported on cut-points for diagnosis of hypocalcemia with this analyte. Current cut-point suggestions or diagnosis of hypocalcemia range from 1.00 mmol/L to 1.10 mmol/L\(^9,10\).

**PREVENTION**

As hypocalcemia increases the risk that a cow will experience adverse health events during her lactation and also decreases milk production, prepartum management aimed at prevention of hypocalcemia represents a sizable opportunity for preventing other post-partum diseases. The nutritional strategy of reducing the dietary cation-anion difference (DCAD) is the mainstay of hypocalcemia prevention, although vitamin D supplementation or provision of a calcium-deficient diet are also used\(^1,11,12\). Cows
RUMINANT THE PERIPARTURIENT COW

fed low DCAD rations develop mild acidosis that is thought to increase tissue sensitivity to PTH, allowing faster mobilization of calcium when demand increases\(^3\). Some evidence suggests that feeding a low DCAD ration can reduce the incidence of periparturient hypocalcemia from 50% to 30%\(^{13}\). Current recommendations are to feed a DCAD diet of approximately -10 to -15 mEq/100 g of dry matter for 3 weeks before expected calving\(^{14}\). To ensure the diet is being fed appropriately, monitor urine pH in close-up dry cows; target urine pH is 5.5-6.5 (normal ~ 8.0).

POSTPARTUM INTERVENTION

Given the technological constraints of accurately measuring calcium and the negative consequences of hypocalcemia to dairy cattle health and farm economics, many cows are commonly supplemented with calcium immediately after calving, which can become an expensive and time-consuming task. Common on-farm interventions include subcutaneous or intravenous administration of calcium salts, oral administration of calcium pastes or boluses, or oral drenching with calcium propionate\(^{1,10,15}\). A lack of progression to clinical symptoms of milk fever is often used to measure the benefits of these interventions, which overlooks the importance of monitoring and treating subclinical hypocalcemia.

The standby therapy for clinical hypocalcemia is intravenous administration of 500 mL of 23% calcium gluconate, which can also be given subcutaneously as a treatment or preventative for subclinical hypocalcemia. While cows with milk fever need an immediate source of calcium, caution should be used given the potential “rebound effect” found after intravenous administration of calcium when blood calcium concentrations decrease approximately 4 hours after administration\(^1\). These animals benefit from an additional sustained calcium sources, with a further dose of calcium given subcutaneously or orally.

Oral calcium supplements containing calcium chloride, calcium propionate or calcium carbonate are also available. Calcium chloride is both highly bioavailable and acidifying (supporting mobilization of the cow’s own calcium stores) but is irritating to oral mucous membranes\(^{11}\). Calcium propionate is absorbed more slowly than calcium chloride, but has equivalent efficacy and longer duration of action, while calcium carbonate is ineffective due to its poor bioavailability and alkalinizing effect\(^{16,17}\). Several manufacturers have introduced oral calcium boluses containing rapidly- and slowly-absorbed calcium salts. Depending on the product, label instruction require administration of 1 to 3 boluses at calving, with some products requiring a second bolus administration 12 hours later. Oetzel and Miller\(^{10}\) demonstrated that use of Bovikalc® oral calcium boluses decreased adverse health events for lame cows and increased milk production in high-producing multiparous cows but could not recommend them as a blanket treatment.

Oral drenching of calcium propionate has the benefit of being less irritating to mucosal surfaces and not acidogenic. In addition, it supplies propionate, a glucogenic precursor, to postpartum cows that may be in a state of excessive negative energy balance. Common drench formulations include 0.45-0.68 kg calcium propionate and encourage
administration within a few hours postpartum and again 24 hours later. The hesitation of some herds to lock up and drench postpartum cows, coupled with the technical skills required to prevent the catastrophic side-effects associated with improper orogastric tubing, makes routine drenching of calcium propionate uncommon.

CONCLUSIONS
Although clinical hypocalcemia is becoming a rare occurrence on modern dairy farms employing good nutritional management strategies, subclinical hypocalcemia is an often underdiagnosed problem at both the individual animal and herd level. Testing and monitoring options remain expensive although current studies to improve on-farm, economically-viable testing methods are under way. Prevention of hypocalcemia through nutritional management is key, with multiple options available for postpartum prevention and treatment.

REFERENCES
In recent years there have been important changes in antimicrobial therapy. There are new antimicrobials available and there is a greater database of species-specific pharmacokinetic information available for antimicrobials used in veterinary medicine, which allows for more accurate drug dosing. Concerns over drug residues in food animals and the continued development of bacterial resistance to antibiotics has heightened the awareness of rational use of antimicrobials.

The following questions must be considered when developing an antimicrobial regimen:

**DOES THE DIAGNOSIS WARRANT ANTIMICROBIAL THERAPY?** Using antimicrobials to treat minor infections or purely viral or inflammatory diseases is irrational, expensive, can be hazardous to the patient and encourages antimicrobial resistance. Clients have come to expect antimicrobials for trivial infections or “just in case” an infection may develop. Equine practitioners must resist client pressure to use or prescribe unnecessary drugs.

**WHAT ORGANISM(S) IS/ARE LIKELY TO BE INVOLVED?** For many infections, the likely organism can be successfully predicted from the history and clinical signs (eg, urinary tract and skin infections in dogs, “strangles” in horses).

**WHAT IS THE IN VITRO ANTIMICROBIAL SUSCEPTIBILITY OF THE ORGANISM?** For many pathogens, the in vitro susceptibility can reliably predicted. For example, *Streptococcus* species are typically susceptible to penicillin. However, many Gram-negative bacteria have unpredictable susceptibilities and susceptibility testing is essential for determining appropriate drug therapy.

**IN WHAT PART OF THE BODY OR TISSUE IS THE INFECTION LOCATED? WILL THE ANTIMICROBIAL PENETRATE TO THE INFECTION?** Consideration of the pathophysiology of the infection will aid in selection of effective therapy. Treatment of sequestered infections such as mastitis or meningitis requires antimicrobials that readily cross membrane barriers. Antimicrobials characterized by low values for volume of distribution are unlikely to reach therapeutic concentrations in such sites.

**WILL THE ANTIMICROBIAL BE EFFECTIVE IN THE LOCAL ENVIRONMENT OF THE ORGANISM?** For some antimicrobials the local infection environment reduces their efficacy. Sulfonamides are ineffective in purulent debris, as para-amino benzoic acid (PABA) released from decaying neutrophils serves as a PABA source for bacteria and reduces the competitive effect of the sulfonamide. Aminoglycosides are ineffective in an abscess due to the acidic, anaerobic environment along with the presence of nucleic acid material from decaying cells which inactivates the aminoglycosides.
INTERPRETING MICS
By the Clinical Laboratory Standards Institute (CLSI) definition, the MIC values are derived as serially doubling concentrations (in μg/ml). Susceptible (“S”), intermediate (“I”), and resistant (“R”) designations are derived from “breakpoints” assigned by the laboratory based on safely achievable plasma concentrations and results of clinical trials. When a pathogen is reported as susceptible, it means that the recommended dosage of the antimicrobial will reach plasma or tissue concentrations that will inhibit bacterial growth in vivo. When a pathogen is reported as resistant, inhibitory antimicrobial concentrations are not safely attainable in the patient. If the pathogen is reported as intermediate, then administering the antimicrobial at higher than recommended doses may result in effective therapy. The relationship between drug concentration and microbial inhibition is not a linear predication. As antimicrobial concentration increases in vitro, eventually all bacteria will be inhibited or killed. The “S”, “I”, “R” designations are assigned by the laboratory based on safely achievable plasma concentrations. Antimicrobial susceptibility data also do not account for:

HOST DEFENSES: The interaction between the host and the pathogen are complex and not predicted by in vitro tests. Antimicrobial drug action takes place in concert with host defences such as humoral and cell mediated immunity, complement components, and nonspecific antibacterial factors such as lactoferrin, lactoperoxidase and lysozyme.

DRUG DISTRIBUTION IN THE BODY: The “S”, “I”, “R” designations assigned by the microbiology laboratory are based on safely achievable plasma concentrations. This does not take into account for extremely high concentrations of antimicrobials achieved in organs and fluids of excretion (kidney, urine, bile) or with local administration of high drug concentrations (e.g. ophthalmic ointments). Pathophysiology may alter drug distribution. For example, some antimicrobials, such as the tetracyclines, accumulate in pneumonic lung tissues for effective therapy that would not be predicted from studies in healthy cattle.

GROWTH RATES AND SIZE OF INOCULUM AT THE INFECTION SITE: The incubator of the microbiology laboratory is an ideal world for bacterial growth. Conditions are managed to promote rapid growth, and rapidly dividing bacteria are more susceptible to antimicrobial drugs. Replication rates may be much slower at the infection site and MIC’s are generally unreliable for slow growing bacteria. Standardized inoculums used in the laboratory may over- or under-represent pathogen numbers in infected tissues.

MIXED INFECTIONS: Separate susceptibility testing of pathogens in a mixed infection does not account for the pathological synergism between bacteria. The by-products of one bacterial species may facilitate the establishment and growth of another.

INFECTION ENVIRONMENT: Many antimicrobials are inactive in purulent exudate, which is typically anaerobic, acidic and hyperosmolar. Some antimicrobials will have different activity in body fluids (plasma, milk, bile) than in nutrient-rich laboratory media. Deposition of fibrin may alter tissue penetration of antimicrobials. Many bacteria are capable of producing a polysaccharide slime capsule to protect them from host factors. Mastitis pathogens typically increase their replication rate when incubated in mastitic milk.

IN VIVO SYNERGISM WITH ANTIMICROBIAL COMBINATIONS: Despite predictions of resistance from susceptibility testing, therapy may be successful because of synergistic combinations of antimicrobials. Synergism between penicillins and aminoglycosides
has been recognized for some streptococcal, enterococcal and staphylococcal infections. The synergism is attributed to increased cellular uptake of the aminoglycoside after cell wall damage from the penicillin.

**TOPICALLY ADMINISTERED ANTIMICROBIALS ARE NOT TESTED:** Veterinary microbiology laboratories may not routinely do susceptibility testing for antimicrobials that are only used topically. Polymyxin B is one of the most effective antimicrobials for superficial Pseudomonas infections, but it causes neurotoxicity and nephrotoxicity if administered systemically, so it is rarely included in susceptibility testing.

**CLSI BREAKPOINTS MAY BE INAPPROPRIATE:** Veterinary breakpoints for susceptibility testing are developed by the Veterinary Antimicrobial Susceptibility Testing (VAST) Subcommittee of the Clinical and Laboratory Standards Institute (CLSI), but only for a limited number of drugs/indications. Note the specificity of the CLSI breakpoints. Breakpoints used in diagnostic laboratories for these antimicrobials with other bacteria or other antimicrobial/bacteria combinations may be based on human derived breakpoints which may or may not be predictive for veterinary patients. So the true relevance of any in vitro MIC predicting the in vivo results of drug therapy is questionable. But by convention, drug dosage regimens use a target plasma drug concentration that is based on some multiple (2 to 10, most often 4) of the in vitro MIC.

<table>
<thead>
<tr>
<th>Veterinary Drugs with CLSI Breakpoints</th>
</tr>
</thead>
<tbody>
<tr>
<td>ampicillin</td>
</tr>
<tr>
<td>Eq: respiratory K9: dermal, soft tissue infections</td>
</tr>
<tr>
<td>cefpodoxime</td>
</tr>
<tr>
<td>K9: dermal</td>
</tr>
<tr>
<td>cefovecin</td>
</tr>
<tr>
<td>K9: dermal, UTI Fe: dermal</td>
</tr>
<tr>
<td>ceftiofur</td>
</tr>
<tr>
<td>Bo: respiratory disease, mastitis Eq &amp; Po: respiratory disease</td>
</tr>
<tr>
<td>clindamycin</td>
</tr>
<tr>
<td>K9: soft tissue infections</td>
</tr>
<tr>
<td>danofloxacin</td>
</tr>
<tr>
<td>Bo: respiratory disease</td>
</tr>
<tr>
<td>enrofloxacin</td>
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<tr>
<td>florfenicol</td>
</tr>
<tr>
<td>Bo &amp; Po: respiratory disease</td>
</tr>
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<td>gentamicin</td>
</tr>
<tr>
<td>penicillin/novobiocin</td>
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<tr>
<td>Bo: mastitis</td>
</tr>
<tr>
<td>orbi-, marbofloxacin</td>
</tr>
<tr>
<td>K9: dermal, UTI Fe: dermal</td>
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<td>Bo &amp; Po: respiratory disease</td>
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<tr>
<td>Bo: mastitis</td>
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<tr>
<td>spectinomycin</td>
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PHARMACOKINETICS/PHARMACODYNAMICS OF ANTIMICROBIAL DRUGS
Successful antimicrobial therapy depends on both a measure of drug exposure (pharmacokinetics) and a measure of the potency of the drug against the infecting organism (pharmacodynamics). These PK/PD parameters required for clinical antimicrobial efficacy are remarkably similar in different animal species and humans, for different dosage regimens, for different drugs within the same class, and at different sites of infection. The PK parameters most useful in drug dosage design are the area under the plasma concentration versus time curve (AUC) from 0 to 24 hours, the maximum plasma concentration (Cmax), and the time the antimicrobial concentration exceeds a defined PD threshold. The most commonly used PD parameter is the bacterial MIC. In relating the PK to PD parameters to clinical efficacy, antimicrobial drugs are currently being classified as either concentration-dependent or time-dependent. For antimicrobials whose efficacy is concentration-dependent, high plasma concentrations relative to the MIC of the pathogen (Cmax:MIC) and the area under the plasma concentration-time curve that is above the bacterial MIC during the dosage interval (area under the inhibitory curve, AUC0-24 hr:MIC) are the major determinants of clinical efficacy. For antimicrobials whose efficacy is concentration-dependent, high plasma concentrations relative to the MIC of the pathogen (Cmax:MIC) and the area under the plasma concentration–time curve that is above the bacterial MIC during the dosage interval (area under the inhibitory curve, AUC0-24 hr:MIC) are the major determinants of clinical efficacy. These drugs also have prolonged post administration effects, which allow once-daily dosing with maintenance of maximum clinical efficacy. For fluoroquinolones (enrofloxacin, orbifloxacin, marbofloxacin, pradofloxacin), clinical efficacy is associated with achieving either an AUC0-24 hr:MIC of more than 125 or a Cmax:MIC of more than 10. For the fluoroquinolones, the two PK/PD relationships are not independent, because as Cmax increases, so does AUC. For aminoglycosides (gentamicin, amikacin), achieving a Cmax:MIC of more than 10 is considered optimal for efficacy. These PK:PD targets are met when antimicrobials are administered at label dosages for the pathogens indicated on the label. For extralabel pathogens with high MIC values, such as Pseudomonas aeruginosa, achieving the optimum PK:PD ratios systemically may be impossible with label or even higher than label dosages. In such cases, under dosing is ineffective and merely contributes to antimicrobial resistance. For time-dependent bacterial killers, the time during which the antimicrobial concentration exceeds the MIC of the pathogen determines clinical efficacy (T>MIC). How much above the MIC and for what percentage of the dosing interval concentrations should be above the MIC is specific for individual bacteria-drug combinations. It is generally accepted that exceeding the MIC by 1-5 multiples for between 40-100% is appropriate for most time-dependent killers. T>MIC should be closer to 100% for bacteriostatic drugs and for patients that are immunosuppressed.
Consumers are increasingly concerned about drug residues and antimicrobial-resistant bacteria in the food they consume. A large part of consumers’ growing interest in humane animal practices was actually fueled by food safety concerns, which created awareness of how food animals were being raised and slaughtered. There is increasing concern over the adverse effects of antimicrobial drugs on human intestinal flora, including selection of resistant bacteria and disruption of the barrier effect of the normal resident intestinal flora.

To protect consumers from adverse health effects, federal programs are charged with the regulation of chemicals and drugs and the detection of chemical and drug residues in foods of animal origin. The United States Food and Drug Administration’s (FDA) Center for Veterinary Medicine (CVM) and the Health Canada’s Veterinary Drugs Directorate (VDD) approve veterinary drugs and establish the acceptable concentrations of drug residues in animal-origin food products. The United States Department of Agriculture's Food Safety and Inspection Service (FSIS) and the Canadian Food Inspection Agency (CFIA) monitor meat, poultry, eggs and honey for residues of drugs and chemicals. Monitoring of antimicrobial residues in milk and dairy products is mainly carried out on a state or provincial basis at the processor level. Before any drug can be approved in the United States or Canada for use in a food producing animal, an extensive toxicologic evaluation of the drug and its metabolites is undertaken. This ensures that any drug residues in animal-derived foods do not harm the consumer. Based on the results of toxicity tests, regulatory agencies establish an acceptable daily intake (ADI). The ADI represents a level of daily intake of a chemical which, during an entire lifetime, appears to be without appreciable risk to the health of the consumer. The ADI is used to determine the maximum concentration of a marker residue in edible tissues, honey, milk, or eggs that is legally permitted or recognized as acceptable. In the U.S., these acceptable concentrations are termed tolerances while in Canada and the European Union they are termed maximum residue limits (MRLs). The MRL is calculated such that daily intake of food with residues at the MRL will result in a total daily consumption of residues in quantities at or below the ADI. ADIs are based on the total residue of a chemical present in food (parent compound and all metabolites) whereas MRLs are based on a single, measurable marker residue, which may be the parent compound or any of its metabolites. In establishing MRLs, consumption estimates for the various foods are taken into account so that foods consumed infrequently or in small amounts are allowed greater MRL values than those foods likely to be consumed daily or which represent a major component of the diet. Because of differences in consumption factors, MRLs and label withdrawal times may differ between countries, even though ADIs are equivalent. International values for MRLs can be searched for on the International Maximum Residue Level Database.
CALCULATING THE WITHDRAWAL TIME
Drug manufacturers administer the proposed label drug dose to a number of animals and perform sequential euthanasia to measure tissue drug concentrations. The label withdrawal time (WDT) is determined by identifying the time that is required for marker residue concentrations in the target tissue (eg, muscle, liver, kidney) to fall below the tolerance level with a confidence level of 95%. Therefore, small errors in dosing or mild disease states are accounted for in determining the WDT.

RESIDUE MONITORING PROGRAMS
The Canadian Food Inspection Agency (CFIA) and U.S. Department of Agriculture’s Food Safety and Inspection Service (FSIS), test for chemical contaminants in meat, poultry, milk and egg products. They screen for chemical residues from approved and unapproved veterinary drugs, pesticides, and environmental compounds. When a violation is detected, the FSIS or the CFIA condemns the carcass or adulterated product. If the product has been distributed into commerce, it is subject to a voluntary recall. The federal agencies take appropriate action when a violation is detected. These actions include follow-up inspections, further directed sampling according to a surveillance plan, or even seizure and recall of products when the human health risk is considered unacceptable. Follow-up actions vary according to the magnitude of the health risk; regulatory emphasis is on preventing repeat violations and preventing distribution of contaminated products into the public food supply. With increasing public concern over the risks of chemical contamination, there has been greater focus on strengthening the identification, ranking, and testing for chemical hazards in meat, poultry, and egg products. The U.S. and Canada now use multi-analytic methods that analyze more compounds per sample while using fewer samples (>350 compounds can be screened for in a single sample).

When inspection program personnel detect evidence of disease or drug use in an animal carcass, they hold and test samples from those carcasses. An animal may be suspect because of historical information on a production class, or appearance on ante- and post-mortem inspections. Typical suspect animals include culled dairy cows, bob veal calves (calves <3 weeks of age and weighing <68 kg), any animal with visible evidence of an injection site, any animal showing evidence of an infectious disease, or animals of a given production class for which a high incidence of residue violations has been detected through the monitoring program.

CAUSES AND INCIDENCE OF RESIDUE VIOLATIONS IN THE UNITED STATES AND CANADA
Drugs, pesticides, environmental contaminants, and naturally occurring toxicants can leave residues in meat, milk, eggs and honey. Of these, drugs are the most frequently detected chemicals and the overwhelming majority of violations are from antimicrobials. When approved veterinary drugs are administered according to their label directions, the prevalence of violative drug residues in animal products should be less than 1%. Residue violation rates greater than 1% indicate that a drug has been used in a manner inconsistent with label directions. Analysis of the probable causes for violative residues reveal that failure to observe withdrawal times, drugs administered in error, treatment of animals with greater than labeled doses, failure to use the appropriate route of administration and improper maintenance of medication records are identifiable risk factors. Medicated feeds are a frequent cause of residue violations in market hogs and poultry. Adherence to medicated feed withdrawal times may be
burdensome, inconvenient, and expensive in that nonmedicated feed must be provided during the withdrawal period and this requires the changing of feed programs and containers for a short time at the end of the feeding period. Lack of treatment records or failure to adequately identify treated animals can lead to insufficient withdrawal periods. When drugs are administered to animals at higher than label dosages, or when drugs are used in species for which they are not approved, the prescribing veterinarian is responsible for withdrawal recommendations.

Most of the drugs that result in residues can be purchased by producers without a veterinary prescription. According to the FDA, 31% of the drugs that cause violations are obtained from feed stores or other drug suppliers. Veterinary supplied drugs account for 26% of violations. Veterinarians contribute to 3% of violations by extralabel drug use, while 9% of violations are from producer initiated extralabel drug use. Dairy farms are responsible for 33.5% of violations from all species.

In Canada, there is a legal loophole that allows producers to purchase drugs “for personal use” as active pharmaceutical ingredients. This is mostly raw drug in bulk barrels, imported from third world countries. The products do not have DIN (drug identification numbers) and there is little assurance of quality, purity and efficacy. Unless the use of such products results in a detected residue violation, producers can legally buy and use these drugs in their animals. Withdrawal recommendations made by veterinarians are often rough estimates and may be inadequate for depletion of drug residues from the carcass, milk, honey or eggs. Salvaging diseased animals for slaughter that have been treated with antimicrobial drugs is a common cause of violative drug residues, especially in cull dairy cows and veal calves. In the U.S., neomycin accounts for the most residue violations (25%), followed by penicillin (14%). The high rate of neomycin violations in veal calves is mainly due to neomycin-medicated milk replacers fed to calves with enteritis. In normal calves, the oral bioavailability of aminoglycosides such as neomycin is very poor. But with inflammation and damage to the mucosal barriers with enteritis, sufficient quantities of neomycin are absorbed systemically and result in violative kidney residues. Because of human health concerns, there are a number of banned drugs that cannot be used in food animals under any circumstances. The best documentation of adverse human health effects from consumption of residues from a veterinary drug is from clenbuterol-tainted food products. Diethylstilbesterol (DES) was once used for growth promotion in cattle but is banned in food animals as it is carcinogenic in humans. A number of antimicrobials are banned from veterinary use in many countries because of similar concerns. Idiosyncratic (non-dose dependent) aplastic anemia can occur in humans exposed to chloramphenicol. Nitroimidazoles (eg, metronidazole), nitrofurans (eg, nitrofurazone) and carbadox are banned for veterinary use in many jurisdictions due to carcinogenicity potential. Ironically, all of these banned antimicrobials are still used therapeutically in humans and there is there is no evidence that consuming food animal products with residues of these drugs impacts human health.

The veterinarian's role in extralabel drug use is essential to avoid violative residues and minimize antimicrobial resistance. An accurate diagnosis assures that proper treatment is rapidly instituted. This may halt progression of the disease before it became severe enough to alter the pharmacokinetic profile of the drug. The veterinarian's training in pathophysiology allows assessment of how the disease could
affect pharmacokinetic disposition. For example: Does the disease reduce clearance by altering renal or liver function? Is volume of distribution altered by fever, endotoxemia, dehydration, or pregnancy? Would concurrently administered drugs or pesticide use alter biotransformation or protein binding? For lactating dairy cows, the veterinarian can conduct on-site screening tests for antimicrobial residues. On-Farm Food Safety programs are now enforcing the involvement of veterinarians to insure adequate record keeping.

When using drugs in an extralabel manner in food animals, or in the event of animal exposure to pesticides, herbicides and other toxic chemicals, veterinarians request withdrawal recommendations from the U.S. FARAD or the Canadian gFARAD (www.cgfarad.usask.ca). When contacting a FARAD center, the veterinarian should be prepared to provide information regarding the brand name and generic name of the drug, the dose, the type and number of animals treated and the disease condition prompting treatment. The FARADs cannot provide withdrawal guidance for banned drugs or compounded drugs in food animals, or if there is no depletion data available. So it is always best to consult the service before considering extralabel drug use.
The dairy industry actively promotes milk and dairy products as safe and nutritious, and veterinarians play an important role ensuring residues of medications used on farm do not end up reaching the consumer. Milk contaminated with antimicrobials is considered a public health hazard because of possible adverse reactions and antimicrobial resistance. In Canada, the Food and Drug Act: Dairy Products section establishes the standard of dairy products and raw milk that are processed into dairy products. However, regulation of milk and dairy products is done on a provincial basis. Results of residue testing are not readily available, and testing protocols may vary with jurisdiction and individual processors. In the province of Saskatchewan alone, the value of milk discarded due to antimicrobial drug residues is estimated to be $200,000 per year. Surveys indicate that improper use of drugs in the control of mastitis is the major source of residues in milk. The odds that a violative antimicrobial residue will be found in bulk tank milk increases with increasing somatic cell count (SCC) status of the herd. The SCC is an indicator of the prevalence of mastitis within a herd and mastitic cows are routinely treated with antimicrobials in order to lower the SCC to acceptable levels. Drugs administered for dry cow therapy are unlikely to cause drug residues if milk is not shipped for the first four days after calving, if dry periods are longer than six weeks, and if dry cows are not accidentally run through the parlour and milked.

When a drug is approved for use in a food animal species, regulatory authorities establish an acceptable daily intake (ADI). The ADI represents a level of daily intake of a drug which, during an entire lifetime, is without appreciable risk to the health of the consumer. The ADI is used to determine the maximum concentration of a marker residue in edible tissues, honey, milk, or eggs that is legally permitted or recognized as acceptable. In Canada and the European Union these acceptable concentrations are termed maximum residue limits (MRLs), while in the United States they are called tolerances (TOLs). The MRL is calculated such that daily intake of food containing drug residues at the MRL results in total daily consumption of residues in quantities at or below the ADI. The ADI is based on the total residue of a drug present in food (parent compound and all metabolites), whereas MRLs are based on a single, measurable marker residue (which may be the parent compound or any of its metabolites). In establishing MRLs, consumption factors for the various foods are taken into account. Therefore, foods consumed infrequently or in small amounts are allowed greater MRL values than those foods likely to be consumed daily or which represent a major component of the diet. Because of differences in consumption factors, MRLs may differ between countries; even if ADIs are equivalent. An example is ampicillin which has a European MRL of 4 ppb in milk versus 10 ppb in the US and Canada. Canadian MRLs are set by the Veterinary Drugs Directorate.

The milk withdrawal time (WDT) for a drug approved in lactating dairy cows is based on the time required after treatment for milk residues to fall below the MRL in 99% of
animals, 95% of the time. The milk WDT is not the point at which residues can no longer be detected. Label milk WDTs for the same product may be different in Canada than the US, even if the MRLs are the same. In Canada there is no assumption regarding dilution of drug residues in the bulk tank, so milk from an individual cow must be below the legal MRL to establish the WDT. The Food and Drug Administration in the US assumes that no more than one-third of the milk in the bulk tank will come from treated cows. Therefore the label WDT is determined so that the milk from any treated cow will be less than 3 times the legal MRL. While the MRLs and WDTs are established federally, the milk supply is regulated provincially in Canada. The provincial milk regulations vary considerably and may contain wording that contravenes the federal requirements. For example, the Saskatchewan milk regulations state: “No persons shall sell, supply or offer for sale any milk that contains antibiotics, insecticides, herbicides, colouring matter, blood, preservatives, added water, ...” Therefore drug residue testing at the provincial level may result in drug “positives” called at concentrations below the MRLs, rendering the Canadian label WDTs essentially meaningless.

Antimicrobial drug residues in milk can be detected by several methods. In the US and Canada, most rapid, on-farm screening tests are microbial growth inhibition assays such as the Charm CowSide II (Charm Sciences Inc) and Delvotest SP-NT (DSM Food Specialties), or ELISA-based tests such as the IDEXX SNAP series (IDEXX Laboratories, Inc). BetaStar is receptor-based lateral flow assay, and the Penzyme Milk Test uses a DD-carboxypeptidase enzyme assay. More expensive and bulky equipment used at processing plants includes the Charm II system (an immuno- or microbial receptor assay) and the Charm ROSA immunoassay and very sensitive and quantitative assays using high performance liquid chromatography (HPLC).

Rapid antimicrobial screening tests are validated only for use with bulk tank or tanker-truck milk samples. Despite brand names that may include the term “cow side”, none of the tests are currently validated for testing milk from individual cows. To become certified by the Association of Official Analytical Chemists (AOAC), milk residue tests must pass rigorous evaluations for sensitivity, repeatability, and robustness. Each test has a ninety percent sensitivity level (90/95) for specific drugs. This is the estimated lowest concentration of that drug in milk that will give a positive result on 90% of truly positive samples, with 95% confidence (19 times out of 20). For a test kit to be AOAC certified, this 90/95 number must be below the MRL (Canada, Europe) or tolerance (US) of the jurisdiction where the test is sold. As well, drug residues at the MRL concentration must be detected with 100% sensitivity (all truly positive samples will test positive). Because the 90/95 levels must be below the MRL, screening tests can produce a positive result when the drug concentration is below the legal MRL. These “subviolate” test results are positive test results on a milk sample in which the actual drug concentration is at or above the detectable concentration of the test, but below the established MRL. With all of the tests, there is a characteristic response curve, which means that as the drug concentration increases in the milk, there is a corresponding increase in the percentage of positive tests until a plateau is reached and all samples test positive. Even if two different tests have the same 90/95 results at the MRL, the responses at less than MRL concentrations can differ.
If provincial law or the contract between the processor and the producer states that "there shall be no drugs in the milk", then the provincial authority or the processor is free to use any validated residue detection test, even if its 90/95 sensitivity level is far below what was determined to be safe for human consumption (the legal MRL). This is problematic for some drugs like ceftiofur and cepahirin (intrauterine formulations) that have zero milk WDTs on the label, but for which screening test sensitivity is far below the MRL that was used to establish the zero WDT. When evaluating the values for a screening test, the sensitivity is the concentration of the drug in the milk that the test will detect at the 90/95 level. For example, if the sensitivity of the SNAP Beta-lactam test is 5.4 ppb for ceftiofur, then it will correctly detect 90% of samples that actually contain 5.4 ppb ceftiofur, 95% of the time. But it is possible for the SNAP Beta-lactam test to detect as little as 1 ppb of ceftiofur in the milk on occasion – a subviola7ve positive at 1/100th of the legal MRL of 100 ppb. To determine the actual drug concentration of a sample, a truly quantitative method must be used such as high performance liquid chromatography (HPLC).

The issue with subviola7ve positives becomes more complicated when using multi-residue tests such as the β-lactam screening tests. Each multi-residue test detects one or more drugs at concentrations below their respective MRL, but is not ideal for detecting all drugs. In the case of the multi-residue β-lactam tests, they are all very accurate for detecting penicillin, but they are too sensitive for cepahirin and not sensitive enough for cloxacillin.

The rejection of subviola7ve but “safe” milk is an economic issue for dairy producers, who may not understand how they can use an approved drug according to label directions, follow the label WDT, and still have a residue violation. The regulatory authorities and processors know that these testing methods will result in a very small percentage of milk being dumped for testing positive, even though the drug residues are safe for human consumption (below the MRL). Identifying the specific drug and quantity present in a milk sample requires more specific chemical analysis, such as HPLC or mass spectrometry. This is not feasible for every milk sample testing positive with a rapid screening test, due to the time and expense of withholding a positive tanker of milk from the food supply until conclusive results are obtained. The authorities accept the imprecision of the screening tests for the sake of the public good and the efficient delivery of milk products to consumers. But dairy farmers and veterinarians often do not understand this risk mitigation strategy.

Because rapid milk residue tests will only detect certain antimicrobials or classes of antimicrobials, veterinarians should review farm drug use with their clients so an appropriate test can be recommended. If more than one drug is given (eg, intramammary erythromycin and intramuscular procaine penicillin G), a single test may not be adequate to ensure that the milk is free of detectable residues. Despite the fact that test kits are not validated for individual animals, milk from all sick or dehydrated cows that have been treated with antimicrobials should be tested, even if label instructions were followed. It is better to unnecessarily dump the milk from one treated cow than to hope that the “solution to pollution is dilution” and potentially have violative residues in the bulk tank. When testing for a known or suspected drug in
milk, it is best to use a test that is designed specifically for that drug. When testing milk from cows where the treatment history is unknown, it is better to use a multi-drug screening test. However, a positive result on a multi-drug test will not identify which specific drug is present. Unfortunately, most producers only invest in on-farm testing after they’ve had a residue violation. If there is no suitable on-farm test available, the producer should ask the processor to check a milk sample before adding milk from a treated cow to the bulk tank. Rapid screening tests do not detect drugs other than antimicrobials. However, provincial regulators may carry out quantitative testing for other drugs and chemicals, including flunixin, macrocyclic lactones (eg, ivermectin), phenylbutazone, and pesticides. Clients must be warned that screening tests are not meant to shorten the official milk WDT. As well, producers should not dilute “positive” milk by adding it to the bulk tank milk as the actual amount of drug present cannot be quantified with the screening test and the dilution may not be sufficient to prevent a positive test on the bulk tank. And never use a cow side screening tests in attempt to use a withdrawal period shorter than the label withdrawal time.

If using drugs in a legal but extralabel manner in lactating dairy cow, you may contact the Canadian gFARAD for advice on meat and milk withdrawal times. If data is available, the service will provide withdrawal recommendations. But for milk, the recommendations will always suggest a minimal discard time followed by either appropriate cow side testing or a suggestion to send a sample to the processor for testing prior to adding the milk to the bulk tank.

If your producer incurs an antimicrobial drug residue violation, especially if it involves drugs that you prescribed, our advice is to obtain some of the positive sample and send it to an analytical laboratory that can specify the offending drug and quantify the concentration. In Canada, Maxxam is a contract laboratory that can provide such services (for $300-$600 per sample). Especially in cases where the screening test is a multi-drug test, the offending drug may not be the one first thought. For example, you prescribed ceftiofur to a lactating cow and the β-lactam screening test on a bulk tank sample was positive. But when specified, the sample is found to contain penicillin and then the producer remembers treating a different cow with procaine penicillin G and administering more than 15 ml per injection site. Or you prescribed cephapirin (Metricure®) for intrauterine treatment of metritis, and the product label says that no milk withdrawal time is necessary. Quantification of the positive sample reveals cephapirin at 18 ppb, which is below the legal MRL, but above the sensitivity of the processor’s screening test.
Overview of the Issue:
Evaluation of lameness in the horse often includes suspicion of injury to the proximal suspensory ligament. This suspicion can be based on history, sensitivity to palpation, gait evaluation and/or diagnostic anesthesia. While diagnostic ultrasound is often utilized to evaluate the proximal suspensory ligament structure, it can remain a challenging entity to accurately assess and diagnose.

Objectives of the Presentation:
1. Anatomy
   a. Fore limb
      i. Origin attachments of proximal suspensory ligament:
         1. Proximal palmar MCIII
         2. Axial margin of proximal MCII and MCIV
         3. Palmar C3
      ii. Structure
         1. 0-8 cm distal to carpal metacarpal joint (DCMCJ): bilobed structure
            a. Medial lobe is wider and oval in shape
            b. Lateral lobe is more cuboidal in shape
            c. Both lobes have irregular nonlinear tissue that contains fat, muscle and other connective tissue
         2. 8-20 cm DCMCJ
            a. Lobes unify to become one oval structure
            b. Progressive increase in more uniform linear tissue
      iii. Unique characteristics compared to hind limb proximal suspensory ligament anatomy
         1. Definitive bilobular structure
         2. Palmar attachment is perpendicular to body axis
         3. Palmar attachment is less concave
   b. Hind limb
      i. Origin attachments of proximal suspensory ligament:
         1. Proximal plantar MTIII
         2. Axial margin of proximal MTII and MTIV
         3. Plantar T4
      ii. Structure
         1. 0-10 cm distal to tarsal metatarsal joint (DTMTJ):
            a. Highly irregular, ill-defined origin
            b. At 3-10 cm DTMTJ: a multicomponent structure becomes evident.
               i. Usually an irregular oval shape
               ii. Contains two bundles of non-linear tissue
EQUINE LAMENESS

1. One medial and one lateral
2. Contain fat, muscle tissue and other connective tissue
   iii. The bundles divide the ligament into medial, middle and lateral parts
2. 8-20 cm DCMCJ
   a. Structure become more uniformly linear
   b. Cross section becomes more uniformly oval
iii. Unique characteristics compared to fore limb proximal suspensory ligament anatomy
   1. Highly ill-defined proximal origin (0-3 cm)
   2. Unilobular structure
   3. Plantar attachment is angled to body axis
   4. Plantar attachment is more concave

2. Skin preparation
   a. Optimize acoustic energy transmission by clipping and scrubbing with soap
      i. Reduce dirt accumulation
      ii. Reduce air interference
      iii. Hydrate dermis
         1. Topical water/alcohol
         2. Internal dermal hydration by physical stimulation of scrubbing
      iv. #10 blade clipping will typically yield adequate results
   b. Reference markings
      i. No standard of reference
      ii. Use Wite-Out correction fluid to mark every 5 cm
      iii. Suggest use of carpal metacarpal/tarsal metatarsal joint as a point of reference-more accurate than measurements from accessory carpal bone.

3. Probe placement /selection
   a. Fore limb
      i. Transverse images
         1. Non-weight bearing with limb in minimal flexion
         2. Displace palmar tendon structures somewhat laterally as probe is applied on midline over the relaxed flexor tendons while maintaining a perpendicular orientation to the palmar bone surface.
      ii. Longitudinal images
         1. Limb weight bearing
         2. Apply probe along mid axial skin surface
         3. Important to fan medial to lateral to completely evaluate structure
   b. Hind limb
      i. Transverse images
         1. Non-weight bearing with limb in minimal flexion
2. Apply probe to medial surface just under the distal aspect of the chestnut
   ii. Longitudinal images
      1. Limb weight bearing
      2. Apply probe along medial surface under chestnut
      3. Fan medial to lateral to completely evaluate structure
      4. May need to direct probe slightly proximally to get beam perpendicular to plantar bone concavity

c. Probe frequency
   i. Most probes from 5-10 MHz yield adequate images
   ii. The best opportunities to improve suspensory imaging are associated with better skin preparation or improved technique

4. Image acquisition
   a. It is important from a legal and clinical perspective to systematically label all images
      i. Patient
      ii. Limb
      iii. Location of probe
         1. Distance from anatomic point of reference
         2. Zone measurement
   b. Image orientation is not currently standardized; therefore, it is important to clarify the orientation of the image. A convention used by many:
      i. Left of image is proximal or medial
      ii. Right of image is distal or lateral
   c. Comparison images of contralateral limb are essential
      i. Left limb on left half of image
      ii. Right limb on right half of image
      iii. Usually with both orientations similarly presented i.e. with medials both to the left
      iv. Some ultrasonographers prefer mirror images, especially of hind PSLs

5. Criteria for ultrasonographic evaluation of soft tissue structure. Note: severity of ultrasonographic change is not correlated to severity of lameness!
   a. Echogenicity
      i. Standard (perpendicular) probe orientation
         1. Hypoechoogenicity indicates
            a. Artifact
            b. Tissue edema
            c. Nonlinear tissue
         2. Echogenicity indicates
            a. Artifact
            b. Fibrosis
            c. Linear tissue
            d. Bone surface
      ii. Contrast enhancement (off-angle) probe orientation
         1. Hypoechoogenicity
EQUINE LAMENESS

1. Artifact
2. Tissue edema
3. Linear tissue

2. Echogenicity
   a. Artifact
   b. Nonlinear tissue
      i. Fibrosis
      ii. Connective tissue

b. Architecture
   i. Normal tissue has variation of ultra-architecture
   ii. Normal tissue has characteristic size, amount and shape of structure architecture
   iii. Injury to tissue almost always reduces architectural detail
   iv. Important to compare to contralateral limb and other horses

c. Bone surface
   i. Irregularities are typical at insertion points of ligaments
   ii. Bone remodeling is common with insertional desmopathies
      1. Most enthesiophytes do not resorb making it difficult to determine if they are the current source of discomfort
      2. It is important to sweep medial to lateral to assess the entire bone surface during the examination

d. Thickness
   i. Injury typically increases the thickness of the structure
      1. Allowing measurement of structure dimensions for comparison
      2. Causes deformation of skin surface which is visible with use of standoff pad.
   ii. Cross sectional measurements have been advocated to evaluate injury and healing
      1. Difficult to identify actual boarders of tissue
      2. Software measurement is subject to operator control

Summary:
While it is generally recognized that MRI serves as the gold standard for imaging of the proximal suspensory ligament, the reality is that ultrasound serves as the primary imaging modality that most veterinarians use. With knowledge of anatomy, proper skin preparation and advancement of technique, ultrasound images can be acquired that will provide more reliable information in the clinical assessment and management of proximal suspensory ligament injuries.
Lameness assessment is one of the most common reasons why clients request the services of a veterinarian. Lameness of the hind limb, in particular, seems to be more difficult to assess than those of the fore limb. This difficulty can be caused by unfamiliarity/risk of diagnostic anesthesia, variability of gait, and variability in observation. In addition, the owner/trainer often has a strong opinion as the source of the lameness. As a veterinarian we need to understand and minimize the variables of the assessment, develop a rationale for diagnostic approach and then validate the suspected pathology. Ultimately, our goal is to provide a therapeutic regime that will accommodate the owner and the horse.

To start, lameness is not an objective quality. As a biologic entity, horses are inherently asymmetrical in structure and gait. As an example, a close observation of the third phalanx bones of the same horse will reveal that they are similar in size and shape but that on closer inspection, they are quite different. Likewise, the gait of a horse may appear symmetrical, but with close measurement there is always some degree of asymmetry in impact, push off or swing characteristics. To repeat, every horse has some inherent degree of asymmetry of gait. However, when that gait causes the horse not to be able to perform at his intended level or if the owner is not enjoying the horse because of the amount of asymmetry; the asymmetry is then termed “a lameness”. This explains why several observers can look at the same horse and grade various levels of lameness.

Assessment of gait relies on the observation of the stride. Each stride is typically divided into stance and swing phases. During the stance phase, the foot rapidly decelerates and bears weight as weight is transferred to the limb and is termed “impact”. As weight is entirely transferred to the limb, the limb begins to accelerate to the end of the stance phase and is called “push off”. The swing phase occurs during non-weight bearing while limb is in retraction as it moves to maximum protraction. Primary gait asymmetry can occur because of discomfort or mechanical restriction during any portion of the stride. Additionally, hind limb gait asymmetry can be caused by compensation from a primary fore limb lameness.

Detection of hind limb asymmetry is typically based on pelvic movement and occasionally on head/neck movement. Most commonly, observation of movement of the tuber coxae, tuber sacrale and pelvic rotation are used as parameters for assessment. Other assessment criteria include assessment of the “drop” of each fetlock (MTPJ extension) during weight bearing, the protraction/retraction of the stride and characterization of the swing of the limb. It has been my experience that there are proponents of the significance of each of these criteria. Unfortunately, no single parameter yields absolutely reliable results. For example, the tuber coxae may “hike” (elevate) when the affected limb is weight bearing in one horse while another horse may “drop” (descend) when the affected limb is weight bearing. Additionally,
human cognition has difficulty assessing change when the time becomes less than 0.25 seconds in duration. Finally, human cognition has a tendency to validate observation to preconceived outcomes: “we see what we want to see”.

Research by K Kegean DVM has demonstrated that variation of tuber sacrale height is the most reliable indicator of hind limb gait asymmetry. In order to remain as objective as possible, I use inertial sensor based gait analysis (Equinosis Lameness Locator) in each gait evaluation. I do not solely rely on the objective data but rather use it to support my subjective observations that involve the other criteria. These systems are relatively inexpensive and quite reliable. Regardless of the criteria employed by the individual practitioner, it is paramount that a methodical repeatable process be employed to attempt reduce the variability of observation.

Variables during assessment need to be keep at a minimum. There are no standard parameters but rather we should be consistent and assess horses in similar circumstances. For example, some practitioners prefer to palpate prior to dynamic examination while others prefer to palpate after dynamic examination. Neither method is absolutely correct; rather, each has an advantage and disadvantage when compared to the other. Consistency is the key to reducing variability. Variables include:

1) Amount of warm up time before movement examination or clinical palpation
   a. Movement may reduce swelling and discomfort noted on palpation
   b. Movement may change gait asymmetry
2) Surface type
   a. Density of surface often changes amount of asymmetry because of altered loading of soft tissue structures
   b. Arena footing is seldom consistent; ends are usually deeper and dryer while long sides are worn in paths
3) Handler
   a. Variation in speed often changes amount of asymmetry; different handlers run at different speeds
   b. Handlers may unintentionally interfere with the horse's way of going
      i. By pulling on lead
      ii. By holding head tight
      iii. By allowing horse to play while running
4) Environment
   a. Distractions
      i. Feed time
      ii. Lesson time
      iii. Cell phones
      iv. Machinery
   b. Weather
      i. Cold
      ii. Wind
      iii. Insects

As an ambulatory practitioner, we all face these challenges every day but it is essential to keep as many of variables minimized as much as possible. My most significant
challenges are usually associated with a novice handler and a distracted horse. I try not to change the handler in the middle of an examination but rather prefer a light sedation to aid in reducing the distractions by the horse. A sedative cocktail of 5 mg acepromazine with 50 mg xylazine typically yields a relaxed assessable horse.

Once the horse has been assessed and lameness is assigned to a limb, the next step is a clinical examination with flexion tests. It is important to evaluate all limbs as often compensatory asymmetries are in effect making accurate assessment more difficult. Additionally, the axial spine should be assessed through static and motion palpation examination. If obvious heat, pain and swelling are evident and associated with onset of lameness, then the area should be imaged to determine pathology.

If no obvious heat, pain or swelling is evident then a decision needs to be made in consultation with the owner to consider the following options:

1) Conservative management: reduced work, NSAID therapy
2) Therapeutic/diagnostic treatment trial
3) Diagnostic anesthesia

The basis for this decision lies in the recognition of the resources of time, money and emotion. It is most difficult to determine the best treatment plan without having a specific diagnosis. Unfortunately, specific structure injury often does not yield a pathognomonic gait asymmetry. We can say that a horse moves like his stifles are sore but we cannot be sure until intra articular anesthesia is performed. Because of the horse’s vocation, it may reasonable to pursue a therapeutic trial because of probability but it will be trial and error process.

To limit the trial and error process, I typically use diagnostic anesthesia to validate the origin of lameness and consider diagnostic anesthesia to be the “gold standard” of lameness diagnostics. Usually gait asymmetry is associated with sensory discomfort and I prefer the use of anesthetic to evaluate the origin of the discomfort. Conversely, the use of imaging without validation will often lead to misdiagnosis. As examples, ultrasonography can demonstrate significant changes in the sound limb or radiography demonstrates remarkable bony remodeling of the distal tarsus in a sound horse.

Because of increased risk of diagnostic anesthesia it is important to develop a plan. If two limbs are asymmetric and they are contralateral (i.e. LF and RH), I will start anesthesia of the front limb because compensation of primary front limbs transfers to contralateral hind limbs. Often the hind limb asymmetry will decrease to an acceptable level. Conversely, if the two affected limbs are ipsilateral, I will start anesthesia of the hind limb because compensation of primary hind limb lameness is almost always transferred to the ipsilateral fore limb. It is not uncommon to have a relatively mild hind limb lameness cause a moderate fore limb asymmetry. The corollary of this situation is: when blocking an obvious forelimb asymmetry is unsuccessful, consider anesthesia of the ipsilateral hind limb. It is unusual to have a single primary lameness. Almost all horses have at least two asymmetries in their gait.

Once the limb of question is identified, I check to see if the horse has sensitivity to torque of the digit or sensitivity to palpation of the dorsal surface of P1. I sensitivity is noted then survey radiographs are acquired to check for sagittal fractures of the distal limb prior to anesthesia. I then perform intraarticular anesthesia of any joints that
appear to have effusion. If no joints have effusion, I typically start regional anesthesia at the distal metatarsus (low 6 point block). As a matter of note, horses are remarkably tolerant to diagnostic anesthesia of the hind limb as long as the limb is picked up during anesthetic placement. After five minutes, I validate skin desensitization. If improved, I image the distal limb. If partially improved I wait an additional five minutes and repeat the evaluation. If not improved, I continue to the proximal metatarsus. Evaluation is based on objective data from the inertial sensor system and subjective indicators.

At the proximal metatarsus, I anesthetize the lateral plantar nerve 1cm distal to the TMT joint. I evaluate after five minutes. Careful attention is paid to the inertial sensor information, because many horses have significant changes in compensatory patterns and asymmetry timing while appearing to have little subjective change. If no change is noted, I will then proceed to tibial and fibular nerve anesthesia.

Anesthesia of the tibia nerve should be performed under ultrasound guidance. This relatively easily accomplished by having the leg picked up and held in a farrier’s position. With a micro convex probe, the needle can be guided close to the tibial nerve with a minimum of discomfort. It is essential to get the needleed adjacent to the nerve as the nerve resides in somewhat of a sheathed anatomic structure. Under ultrasound, the veterinarian can easily visualize the nerve and see the anesthetic solution surround the structure. Likewise, both the deep and superficial branches of the fibular nerve are anesthetized although the distal limb is held in protraction during the procedure. If the horse shows improvement, I may opt to come back and then continue with intra synovial anesthesia of the tarsal joints or tendon sheath.

If no improvement is noted, I then proceed to intra articular anesthesia of the femoral tibial joint. I typically anesthetize the medial femoral tibial joint, patellar femoral joint and lateral femoral tibial joint by ultrasound guided injection. The MFTJ is injected at the medial dorsal recess. The FPJ is injected at the lateral pouch on the lateral distal femur. The LFTJ is injected by ultrasound guided technique at the subextensoris recess of the proximal lateral tibia.

Once a region is identified, then imaging is acquired. In an ambulatory setting, radiography and ultrasonography are typically available. Most pathology is identifiable with these modalities. If no pathology is evident, then referral for MRI or scintigraphy is indicated. I will often repeat the diagnostic anesthesia to revalidate the area of inquiry prior to more expensive diagnostic procedures.

In summary, successful hind limb gait assessment depends on a methodical approach to limit the effects of variables in the observation of gait asymmetry. Most horses have multiple origins of gait asymmetries and it is important to evaluate the entire horse in the assessment of the gait. Additionally, imaging without validation by diagnostic anesthesia or presence of heat, pain or swelling will often be misleading. Accuracy of diagnosis is best achieved by the consideration of clinical examination, diagnostic anesthesia and imaging. In most cases, hind limb asymmetry can be diagnosed in the field by ambulatory practitioners while reducing client expense and improving comfort/performance of the horse.
EQUINE LAMENESS

INJURY SPECIFIC SHOEING FOR THE EQUINE

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Outline:

1. Introduction: Best results are obtained when accurate and specific diagnoses are established
   a. Treatment program includes
      i. Reduction of inflammation
      ii. Regenerative medicine
      iii. Hoof biomechanics
      iv. Rehabilitation program
      v. Nutritional support
      vi. Progress assessment/evaluation
   b. Biomechanics of distal limb structures can be modified by variation of sagittal and medial-lateral balance of the hoof capsule
      i. Allows modification of structure loading during healing phase of tissue
      ii. Accomplished by:
          1. Trimming hoof capsule
          2. Application of appliances (wedges, swedged shoes, rolls, bevels)
             a. Muscles contract in response to reduced tension therefore effect is often temporary.
             b. Difficult to lengthen muscle after contraction
             c. Asymmetric shoes can give same load relief while maintaining muscle length.
             d. Wedges are still useful in some situations
   3. Application of asymmetric shoes
   c. Asymmetric shoes alter loading based on surface area of contact with ground
      i. Every action has an equal and opposite reaction>>GRF is the force exerted by the ground against the hoof
      ii. Increasing the surface area of contact reduces penetration of that area into a deformable surface (soft footing)
      iii. Therefore asymmetric shoes have minimal effect when horse is on a firm surface and only are effective when on deformable footing.
   d. Sagittal soft tissue structures (SDFT, DDFT, ALDDFT, SL etc.)
      i. Loaded at specific portions of the locomotion
         1. SDFT and SL are maximally loaded during the impact (first part) of the stance phase
         2. DDFT and ALDDFT are maximally loaded during the push-off (second part) of the stance phase.
         3. All of these structures are loaded in mid-stance to support the angle of the fetlock to prevent
hyperextension and yet retain energy to continue propulsion with minimal energy expenditure

4. A reduction in tension of one structure is accommodated by the other structures

e. Non-sagittal structures
   i. Collateral ligaments
   ii. Non-sagittal articular pathology
   iii. Are loaded during asymmetric movements like circling and unilateral weight bearing on soft surfaces
      1. Lateral collateral ligaments on the outside limb and medial collateral ligaments on the inside limb are loaded more during circling.
      2. Medial joint pathology on the outside limb and lateral joint pathologies on the inside limb are loaded more during circling.
      3. During unilateral weight bearing, the lateral aspect of the hoof penetrates more than the medial aspect.

f. Affecting biomechanics
   i. During the stance phase of locomotion in soft footing, a horse will typically:
      1. Push the toe of the hoof deeper than the heels
      2. Push the lateral side of the hoof deeper than the medial wall while in unilateral weight bearing.
   ii. Using a shoe to increase the surface area at the toe reduces penetration of the foot into the cushion.
      1. The palmar angle decreases slightly
      2. The effect of a reversed #2 wedge pad can be approximated by using a width of 35 mm at the toe with 17 mm branch width. The effect of both varies according to the softness of the cushion. Note that minimal effect was noted by only using beveling to reduce surface area.
      3. Maintains (increases) tension on the DDFT and ALDDFT therefore minimizes tension on the SDFT and SL
   iii. Using a shoe to increase the surface area at the heel decreases penetration of the heels into the cushion.
      1. The palmar angle increases markedly
      2. The effect of a #2 wedge pad can be approximated by using a width of 17 mm at the toe coupled with 35 mm branches or using an egg bar shoe. Again, the effect of both varies according to the softness of the cushion. Note that minimal effect was noted by only using beveling to reduce surface area.
      3. Reduces tension of the DDFT and ALDDFT therefore increases tension on the SDFT and the SL
   iv. Using a shoe to unilaterally increase the surface area on one bar reduces the penetration of the bar into the cushion.
      1. The medial-lateral loading of the joint surface is altered so that the area of joint above the wider bar will not penetrate into the cushion as much as the contralateral

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bar therefore increasing the load on structures above the wider bar
   a. The loading of joint surfaces above the wider bar have more loading than the adjoining articular hemisphere.
   b. The loading if of collateral structures (ligaments, tendon insertions) above the wider bar have less loading.

2. Narrowing the opposite bar increases the effect

3. The effect of a 35 mm branch coupled with a 17 mm branch yielded approximately half of the effect of a #2 wedge placed sideways. As in the other applications, beveling yielded minimal change.

2. Framework to approach injury specific shoeing
   a. Soft tissue structures (tendons and ligaments) are usually loaded in tension
      i. Goal is to reduce tension during healing
      ii. Gradually increase tension during rehabilitation
   b. Weight bearing structure (articular surface) injury are more painful in compression
      i. Goal is to reduce compression during healing
      ii. Gradually increase compression load during rehabilitation

3. Specific injury shoeing suggestions
   a. Wide medial branch shoe
      i. Medial collateral ligament desmitis
      ii. Lateral arthropathies
      iii. Medial SDFT insertion injuries
      iv. Medial SL branch injuries
   b. Wide lateral branch shoe
      i. Lateral collateral ligament desmitis
      ii. Medial arthropathies
      iii. Lateral SDFT insertion injuries
      iv. Lateral SL branch injuries
   c. Caudal heel support shoe (reverse, egg bar, onion shoe)
      i. DDFT tendonitis
      ii. ALDDFT desmitis
      iii. Navicular bone edema
      iv. Podotrochlear bursitis
   d. Wide toe/narrow branch shoe
      i. SDFT tendonitis
      ii. SL desmitis
      iii. ALSDFT desmitis

4. Rehabilitation
   a. Gradually increase loading of injured structures after proliferative stage of healing is completed; usually after 1-3 months
   b. Increases of loading may be obtained by:
      i. Trimming
      ii. Use of less severe appliances (wedges)
      iii. Use of less dramatic asymmetry of shoe structure
5. Summary
   a. Best results are obtained when accurate and specific diagnoses are established.
   b. Tendon and ligament injuries occur because of excessive load while being in tension. Shoeing should attempt to reduce loading to facilitate healing.
   c. Arthropathy associated discomfort is accentuated by compressive loading during weight bearing. Shoeing should attempt to reduce compressive forces during healing and comfort management.
   d. Loading forces of limb structures can be altered by
      i. Trimming hoof capsule
      ii. Application of appliances (wedges)
      iii. Use of asymmetric shoes to modify hoof penetration into soft surfaces
EMERGENCIES OF THE EQUINE DIGIT: ASSESSMENT AND CARE

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Wounds to the equine digit are a common and potentially grave injury. Most injuries are superficial in nature and respond well to conservative treatment. However, because of the risk of sepsis, deep injuries that involve synovial structures require prompt recognition and aggressive treatment. Unfortunately, the clinical signs of superficial and synovial injuries overlap, necessitating a logical and accurate assessment to formulate a successful treatment plan.

The goal of this presentation is to present a rational approach for the assessment of penetrating injuries of equine digit for the ambulatory practitioner. This assessment will be based on recognition of anatomic structures at risk, awareness of imaging options and treatment requirements. The owner and veterinarian can then have a meaningful dialogue as to the selection of the best treatment plan.

Emergencies of the digit involve injury to the sole of the hoof, wall of the hoof and soft tissue adjacent to the hoof (coronet). Trauma types include puncture, laceration and avulsion. Puncture injury is most often caused by foreign bodies. Nails are by far the most common source representing >80% of sole punctures. In addition, clips of shoes and other linear foreign bodies (wood, glass, wire) are also encountered as penetrating objects. Iatrogenic punctures from intra articular medication administration and diagnostic anesthesia should also be considered as possible origins. Lacerations occur typically above the coronet, while avulsions are usually associated with entrapment wounds and usually involve heel bulb structures and side walls of the hoof. Because of proximity to synovial structures and collateral cartilage structures, all wounds require assessment of synovial structures and collateral cartilage involvement.

Clinical assessment of sole punctures starts with evaluation of location. Penetrations of the sole are especially serious when they involve the central regions of the frog. This includes the frog tissue, central sulcus and collateral sulci. Punctures of the frog are reported to have a better than 50% return to soundness while sole punctures have a >95% return to soundness. Penetrations of the middle and caudal third of the frog are twice as likely to involve synovial structures. 12% of frog punctures involve synovial structures. It should be noted that hind foot involvement has a better prognosis than front foot injury. This may be because of less weight bearing or that the observer is less able to detect hind limb gait asymmetry. In addition, to the location, the direction of puncture should be assessed prior to removal of object if possible. The type of object will usually give indication as to the probability of completeness of foreign body removal.

If the foreign body is no longer present, careful paring of hoof as indicated by hoof testers needs to be performed to identify the wound. Sometimes this is quite challenging as the soft horn often collapses around the tract. As expected, increased digital pulses, heat and swelling (if chronic) are expected. Lameness (typically AAEP grade 3-5) is always severe and not reliable as an indicator of severity of injury.
It is essential to establish the length of time from injury prior to establishing a prognosis. If treatment is initiated within 48 hours, a success rate of 82% has been reported compared to a 7% success rate if the injury is greater than 7 days.

Radiography must be used to assess for presence of gas, foreign body, fracture and/or bone defect. A sterile probe, teat cannula or blunted needle can be carefully placed in the tract to aid to location/direction determination. Multiple projections including LM, DP, DPPP and PPPP are required for accurate assessment.

Ultrasoundography can be quite useful in assessment of punctures of the frog especially if the penetrating object has been removed. The frog needs to be paired so that it is soft. Because soaking is often not necessary, this modality can be very useful for detection of air or particulate matter along the tract of penetration.

Regional anesthesia may be necessary to facilitate radiography and will certainly help with centesis procedures. If clinical evaluation and radiography suggest the possibility of synovial involvement then synoviocentesis should be performed. After insertion of the needle, a fluid sample should be retained for cytology and culture. The structure should then be pressurized with a balanced electrolyte solution to evaluate compartment integrity. If frank leakage is not evident, then contrast radiography should be performed.

The first synovial structure to be assessed should be the podotrochlear bursa (PTB) as this structure must be penetrated by a foreign body before the object can come in contact with the distal interphalangeal joint or flexor tendon sheath. If the PTB is intact, then it is highly likely that the other synovial structures are also intact. A caveat is that retrieval of fluid is not always possible from the PTB. If no fluid is able to be collected, then a small amount of saline can be used to assist in retrieving a sample for cytology and culture.

Culture of septic synovial fluid yields negative results in 45-77% of cases. These results are improved with use of automated blood culture media enrichment systems. In addition, cultures take greater than 24 hours to yield the most preliminary results making them impractical for initial assessment. Visual appearance is often accurate in determining presence of contamination.

The verification of involvement of the PTB warrants further evaluation of other synovial structures. The ambulatory practitioner may opt to refer the case at this time or more fully assess by then performing the same procedures on the distal interphalangeal joint and digital flexor tendon sheath. A fistulogram is especially important to perform if the object has been removed. The veterinarian should note that fistulograms are prone to negative results and that the contrast agent may obscure the visibility of contrast from synovial cavities. Hence, arthrograms should be performed first, then, followed by fistulograms.

Advanced diagnostic options exist to aid in difficult situations. MRI is not required for first line assessment. It may be useful for after care assessment for prognostication and rehabilitation. Surgical exploration is indicated by presence of foreign material, synovial sepsis, osteochondral fragmentation, osteomyelitis or an unanticipated poor response to treatment.
Treatment goals include removal of foreign material, debridement of contaminated/devitalized tissue, elimination of microorganisms, removal of destructive enzymes and to promote tissue healing. Conservative treatment including debridement, standing surgical procedures has demonstrated significant success as reported by Kilcoyne et al, 2001.

Conservative treatment must be multifaceted and include tetanus toxoid if previous vaccination was greater than six months. Because the wounds are heavily contaminated by soil and feces, they typically are colonized by mixed infections of Gram (+), Gram (-) and anaerobic organisms. Consider the use of a broad spectrum antibiotic therapy like PPG and gentamycin with metronidazole or a cephalosporin and amikacin. The therapy should continue for at least 7 days or until the wound has a bed of granulation tissue. Regional limb perfusion with gentamycin or amikacin is effective to augment therapy response.

Soaking should only be used if the wound is superficial in nature. Soaking aids in the future pairing of sole material and the release of purulent material. The affected tissue should be assessed to determine if necrosis is present. The necrotic material can be debrided surgically after regional anesthesia in a standing manner or using a recumbent surgical procedure. Interestingly, 25% of P3 can be removed with minimal consequence. Additionally, medical maggots have been shown to be effective in the removal of necrotic material. If financial constraints are present, it is reasonable to use 18 gauge needle lavage to lavage synovial structures as long as expectations of success are tempered.

Surgical exploration and treatment is typically associated with a higher success rate in synovial contamination despite the results of Kilcoyne et al. 2001. Surgery termed “street nail surgery”, has been superseded by endoscopy because of the reduction of morbidity and mortality. Endoscopy allows better visualization of structures, improved identification of foreign material and devitalized tissue and access to larger area of synovial surfaces.

Wound care can use a variety of techniques as preferred by the attending veterinarian. This author prefers the application of metronidazole paste to the debrided wound. General guidelines restrict the use of sclerosing agents until the wound is granulated or epithelized. A hospital plate with extreme attention to stall cleanliness will optimize the effect of daily wound dressing changes. Topical astringents (2% iodine) can be applied to epithelized tissue to facilitate resistance to environmental trauma.

Wounds at the level of the coronet and pastern may intrude into the digital flexor tendon sheath and/or the distal interphalangeal joint. Wounds of the dorsal aspect of the coronet are an indication for careful evaluation of possible intrusion into the DIP synovial capsule. Whereas, wounds of the lateral, medial and palmar pastern are indication for careful evaluation of possible intrusion into the DIP joint and the digital flexor tendon sheath. Ultrasound is particularity helpful in assessment of synovial involvement of the pastern. If unclear, then synovial assessment should be performed starting with the synovial structures of greatest risk and if warranted followed by a fistulogram.
The treatment of these wounds is guided by the same protocols as outlined above. If synovial structures are not involved, the wound should be stabilized with sutures, casting or conservative management.

A methodic rational approach to emergencies of the foot can yield an accurate and timely assessment of the severity of injury. This information coupled with prognostic data will help the owner and veterinarian select the best treatment decision for the given presentation which, ultimately, will improve therapy success and reduce horse suffering.

**Further Reading:**
- Kilcoyne, I, et al; Penetrating injuries to the frog and collateral sulci of the foot in equids: 63 cases (1998-2008); *JAVMA*. 2011; 239 (8): 1104-9
- O’Neill, H, O’Meara, B. Diagnosis and treatment of penetrating injuries of the hoof in horses; *In Practice*. 2010; 32: 484-90
- Parks, A. Equine Foot Wounds: General Principles of Healing and Treatment: In proceedings, 55th Annual Convention of the AAEP 2009, pp 180-7
- Redding, W. An In-Depth Look at Puncture Wounds to the Foot; In proceedings, 56th Annual Convention of the AAEP 2010, pp 512-21
- Smith, M. Penetrating injuries of the foot; *Equine vet Educ*. 2013;25 (8): 422-31
INTEGRATION OF ACUPUNCTURE, MUSCULOSKELETAL ALIGNMENT TECHNIQUES & TCVM HERBALS SUPPLEMENTS FOR EQUINE BACK ISSUES

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The diagnosis and treatment of equine back issues has greatly improved with the advent of improved diagnostic imaging. A comprehensive review of equine back conditions, their diagnosis and treatment is beyond the scope of this treatise. The focus is on my practical integrative approach to the diagnosis and treatment of equine back issues based on 35 years of clinical practice, teaching and research. A brief review of the scientific basis and practical applications of acupuncture, chiropractic and other approaches to musculoskeletal alignment are presented.

My philosophy is that no form of medicine has all the answers to all problems and that an open minded integrative approach to both diagnosis and treatment of equine back issues is essential to offer the best outcome for horses and their caretakers.

"Whoever understands to set himself up as a judge of truth and knowledge is shipwrecked by the laughter of the G-ds" …Albert Einstein

This presentation is based on my 30 years of integrating equine acupuncture & musculoskeletal alignment

- Integrative Veterinary Medicine:
  Dr. Schoen's Philosophy
  - INTEGRATE the best diagnostic & therapeutic modalities into a new comprehensive approach creating a new paradigm of veterinary medicine
  - Integration of
    - Acupuncture
    - Musculoskeletal alignment therapies
  - Essential to equine mobility, joint health & overall performance
- Dr. Schoen's AP Approach
  - Integrative Diagnostic & Therapeutic approach combining
    - Western medical, neurophysiologic approach
    - TCM
    - Japanese, European & other perspectives
  - Based on The Liquid Crystalline Collagen Continuum Theory
    - Encompassing neurophysiology, TCM, etc.
- Dr. Schoen's Integrative Approach
- This is not the only way to integrate these approaches
- Sharing 30 years of practical holistic, integrative approaches of what is working to be of benefit to the horses under our care
- All complementary to conventional medicine & surgery
- Dr. Schoen's Integrative Approach
- Conventional Medicine & Surgery
- Acupuncture
- Manual Therapies
- Botanical Medicine
- Self Help Techniques for Clients
- Conclusions
- Reductionistic vs. Holistic Approach
LAMENESS AND INTEGRATIVE TREATMENTS FOR EQUINE BACK PROBLEMS

- Reductionistic Approach
  Try to find one specific cause of a problem i.e. localize a lameness to a particular joint – hock- or a tendon or ligament)
  Assume that that will resolve all secondary compensatory issues
  - Reductionistic Vs. Holistic Integrative Approach
  - Holistic, Integrative Approach
  Primary lower leg lamenesses? Essential & a key to successful lameness diagnosis & treatment

Acupuncture & Manual Therapies
Help identify that location
Treat compensatory problems, secondary restrictive mobility issues

- Clinical Applications of Equine Acupuncture
- Diagnostic value: localize lameness, secondary problems, internal medicine
- Musculoskeletal problems: tx primary lameness, back problems, subclinical problems, compensatory problems
- Laminitis, navicular disease
- Hock, stifle, hip, foot, knee, shoulder problems
- Myositis, myofascial syndromes
- Acupuncture:
  Physiologic Effects: Osteoarthritis
  - Endorphin release: analgesia
  - ACTH and cortisone release: anti-inflammatory
  - Decreased muscle spasms, trigger points
  - Increased local circulation
  - Release myofascial restrictions
- AP & Manual Therapies Integration
  - Many different approaches to Acupuncture & to Manual Therapies
  - I term my manual therapy approach “musculoskeletal alignment” (MSA) ©
  - Integration of osteopathy, chiropractic, craniosacral techniques, rolling, stretching,
  - Physical therapy, laser, magnetic, etc.
  - Prevent & correct primary etiologies
  - Diagnostic AP Palpation Examination
  - Palpation of diagnostic acupuncture points in cervical, shoulder, thoracolumbar &lumbosacral regions.
  - Association points: Bladder meridian
  - Paravertebral: between longissimus dorsi & iliocostalis muscles.
  - Hindleg points along BL &GB channels between quadriceps, semimemb.&semitend.
- AP Points for Neck &Back Problems
  - Begin w/ BL-25, reevaluate points,
  - BL-23, BL28, then reevaluate...
  - Neck: begin w/ LI-16, BL-10, LI17,
  - Check local intervertebral & paravertebral points
  - check ting points
  - BL-25: Large Intestine Association Pt
  - BL-25: medial to cranial edge of wings of ileum; 3 cun lateral to midline between 5th & 6th lumbar vertebrae
  - Sensitive w/ hindleg issues, chronic back issues, S-I issues, back pain, as well as g.i. issues:
  - Evaluate based on which other points are sensitive.
- Types of Trigger Points
- Myofascial
- Cutaneous
- Fascial

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LAMENESS AND INTEGRATIVE TREATMENTS FOR EQUINE BACK PROBLEMS

• LIGAMENTOUS
• PERIOSTEAL
• AP VS. TP POINTS
• TP’S ARE NOT SPECIFIC LOCATIONS
• EVERY MUSCLE CAN DEVELOP TP’S
• MUSCLES MAY HAVE MULTIPLE TPS
• ACTIVATION OF LATENT TRIGGER POINTS
• MUSCLE REMAINS IN SHORTENED POSITION FOR PROLONGED PERIODS
• REPETITIVE STRAINS
• CHILLED BY COLD DRAFT
• POST EXERCISE STIFFNESS
• VIRAL or INFECTIOUS ILLNESS
• MYOFASCIAL CHAINS
• IN CHRONIC PAIN CASES THERE APPEARS TO BE CHAINS OF MYOFASCIAL TRIGGER POINTS IN PATTERNS ANALOGOUS TP AP MERIDIANS.
• THREE MAJOR ZONES: DORSAL, LATERAL & VENTRAL
• Equine Myofascial Chains
• These correlate w/ diagnostic acupoint palpation patterns associated w/
  • various primary lamenesses or
  • secondary lamenesses or
  • multiple leg lamenesses or
  • internal medical problems
• DAPE: Individual Points Vs. Myofascial Chains
• More likely to be individual ap point sensitivity if somatovisceral
  • i.e. BL-22: Triple Heater Association Pt: Ovarian issues: if just this point is sensitive on the back in a mare and the complaint is back problems, stiffness, resistance, consider ovarian issues
  • i.e.: in heat, ovarian cysts, “marish behavior”
• Integrative Prevention
• Proper shoeing
• proper training techniques: avoid unnecessary training devices
• Foot issues may reverberate into the back creating ap point reactivity; i.e. Bl13-15 & Li-16 sensitivity with front foot issues
• AP Points for Frontleg Conditions
• Laminitis: PC-9, med. to lat. cartilages, ting points,
• Navicular: same, & SI9,
• Shoulder: LI15, TH-14, LU-1,
• Association (Shu) Points & Somatovisceral Effects
• Back Shu points may be reactive due to primary back issues
• Saddle issues
• Rider issues
• Lower leg issues
• Somatovisceral issues & somatovisceral effects
• May cause somatovisceral reflexes
• Somatoautonomic Reflexes (SARS)
• Autonomic responses initiated by the transmission of signals from somatic afferent fibers located in dermatomes
• Spinal fixations (SF) or muscle spasms, trigger points, can cause somatic afferent bombardment of dorsal horn cells that may alter normal autonomic reflexes or may evoke abnormal autonomic reflexes
• Somatoautonomic Reflexes
• Noxious or nociceptive (painful) impulses evoked from TP’s predictably may have some influence on normal somatoautonomic pathways
• Saddle, rider issues may possibly create or exacerbate somatovisceral issues.
• BL-22: Triple Heater Association Pt
• In a mare, if only BL-22 is sensitive, consider ovarian issues as a possible cause of back problems, behavioral problems, stiff back, resistance,
• When palpated, mare may get very defensive, ear back, try to kick you, even rear, severe muscle spasm there
• Calms down with AP; aquapuncture or dry needle
• Equine Association (Shu) Points (transpositional)
• BL-13: Lung Shu Pt: Cough, COPD, allergic bronchitis
• BL-15: Heart Association Pt: cardiac pain, cough
• BL-18: Liver Association Pt: reactive with liver issues, myofascial syndromes, back soreness
• BL-21: G.I. Issues
• BL-21: Stomach Association Pt: cdl to last rib; colic, constipation, gastroenteritis.
• ST-10: between sternomandibular m. & ventral aspect of brachiocephalicus, one hand cranial to shoulder inn.: supraclavicular n.: stifle issues, stomach meridian issues
• ACUPUNCTURE FOR EQUINE BACK PAIN: Approaches:
  • Trigger points and local points
  • Distal points: command points, ting pts.
  • Ting points: around coronary band
  • TCM differentiation techniques
  • Primary and secondary points
  • Equine Musculoskeletal Alignment: Therapeutics
    • Integration of conventional veterinary lameness therapeutics along w/
    • Acupuncture therapy from a western & eastern approach along w/
    • Physical manipulative therapy (chiropractic) & osteopathic techniques, physical therapy along w/
    • Correcting shoeing, saddle fit, riding etc.

Truly integrative approach
• Veterinary Chiropractic Research
• Research is limited
• Research by Dr. Kevin Haussler at Cornell Veterinary School & Colorado Veterinary School
• Demonstrated mobility of equine spine
• Demonstrated movement of equine spine w/ chiropractic adjustment
• Chiropractic Motor Unit
  MOTOR UNIT: Two adjacent vertebrae & their associated structures
  • Tendons & Ligaments
  • Nerves
  • Blood vessels & Lymphatics
  • Joints
  • Muscles
  • Contents of intervertebral foramen

• Chiropractic Definitions: Subluxations
• Major controversy b/w veterinarians & chiropractors
• A disrelationship
  Between a vertebral segment
  In association w/ contiguous vertebrae
  Resulting in disturbance of normal biomechanical & neurologic function
  • Vertebral subluxation complex or segmental dysfunction
• Vertebral Subluxation Complex
LAMENESS AND INTEGRATIVE TREATMENTS FOR EQUINE BACK PROBLEMS

- Clinical entity of disrelationship of two vertebrae in a motor unit resulting in disturbance of normal function.
- Kinesiopathy: Pathology of movement of the motor unit (hyper or hypo mobile)
- Neuropathy: Pathology of neural component (facilitation or inhibition of neural function)

WOLF’S LAW
- Structure Follows Function.
- First there is a functional change, shift from normal balanced mobility to decreased or increased mobility.
- This then leads to structural changes and degeneration.
- Osteopathic Research:
  - Studies on vertebral lesions & their effects on the spines of cats, dogs, rats, rabbits, guinea pigs demonstrated changes in blood chemistry, urine, gastric secretions, glandular tissues, vascular reflexes, tissue fluids, g.i. motility, and on cytology & histology & matched with unlesioned control animals
  - Somatic dysfunction & TP’s may result in pathophysiologic changes in various systems
  - Osteopathic Research:
    - Circulation affected by sympathetic nerves that control vasoconstrictive tone, especially to the viscera. These somatosympathetic pathways become routes for aberrant neural transmission when they are affected adversely by TP’s
    - May lead to muscle atrophy of lumbar musculature as well
  - DORSAL ZONE DISTURBANCE
    - RELIEVE STRESSES FROM SPONDYLITIC RADICULOPATHIES BY RELEASING MYOFASCIAL & PARASPINAL CONSTRICCTIONS.
    - RESTORES EFFERENT FLOW OF MOTOR IMPULSES RELEASES MUSCLE SHORTENING

DORSAL ZONE DISTURBANCES
- SIGNS OF NERVOUS SYS. DYSFUNCTION ACCORDING TO GUNN
- PARASPINAL T.P.’S AT LEVEL OF SEGMENT OF PAIN.
- M.S. PAIN SYNDROMES POINT TO N.S. DYSFUNCTION. I.E. NEUROPATHIES
- SENSORIMOTOR & AUTONOMIC PERIPHERAL N. DISTURBANCE: RADICULOPATHIES.
  - Cervicogenic Headache Origin
    - Articulations: Atlanto-occipital, C1-C2, C2-C3, C3-C4
    - Muscles: upper cervical muscles
    - Suboccipital musculature
    - Semispinalis capitus, longissimus capitus, splenius capitus
    - Vertebral Artery
    - Posterior Cranial Fossa Dura Mater
  - Clinical Implications of TP’s
    - In addition to a comprehensive conventional physical examination...
    - Diagnostic Acupuncture Point Palpation Exam
    - Evaluate spinal fixations, triggers points, muscle spasms, myofascial restrictions
    - Evaluate any correlation with presenting signs
    - Consider acupuncture & manipulations/adjustments as part of an integrative approach
  - Phases of Subluxation
    - Vertebral misalignment
    - Neuropathy
    - Kinesiopathy
    - Dysfunction
    - Symptoms
    - Degeneration
LAMENESS AND INTEGRATIVE TREATMENTS FOR EQUINE BACK PROBLEMS

• Goal: correct at earliest phase possible

**Pathophysiology of Subluxations**
• May compress spinal nerve roots or spinal cord
• May cause vertebrobasilar arterial insufficiency
• May cause somatovisceral dysfunction
• May decrease mobility, cause fixations

**Etiology of Subluxation**
• Traumatic injury
• Birth: abnormal force at delivery
• Excess weight: rider, saddle, obesity
• Drugs & stress
• Hereditary/Congenital

**Chiropractic Definitions: Adjustment vs. Manipulation**
• Adjustment
  A short-lever, high velocity, quick thrust
  Specific force applied in a specific direction to a specific vertebra
  Designed to deliver maximal force w/ minimal tissue damage
• Manipulation
  Distribute force to multiple segments
  Long-lever, slow velocity, non-thrust techniques

**Musculoskeletal Alignment Therapy: Terminology**

Terms I prefer to use:
• FIXATIONS
  Decreased mobility: dorsal, ventral, right, left, cranial, caudal,
  Sometimes in 3 different dimensions
• MISALIGNMENTS: asymmetry
• ADJUSTMENTS & MANIPULATIONS: improve mobility, correct misalignments
• Resulting in correct musculoskeletal alignment

**Why Do Horses Benefit from MSA Therapy?**
• Confinement
• Riding, rider, training
• Saddle fit, shoeing
• Training, conditioning
• Transportation
• Accidents, traumatic injuries

**Why Do Horses Benefit from MSA Therapy?**
• Any traumatic injury
  Trailer accidents, falls over jumps, etc.
• Birth
• Illness
• Postoperatively
• Conformation

**Signs of Equine Fixations**

Abnormal standing posture
• Discomfort when saddling
• Discomfort when riding
• Evasions
  (e.g. extending head & neck, hollowing back)

**Signs of Equine Fixations**
• Wringing tail & pinning ears
• Refusal or unwillingness over jumps
• Refusal or resistance in any performance (e.g. lateral or collected movements)
• Facial expressions (apprehension, pain)
• Sensitivity
• Stiffness, resistance to move
• **Signs of Equine Fixations**
  • Resistance to gait changes
  • Gait abnormalities
  • Stiffness when coming out of the stall
  • Stiffness in head & neck lateral movements
  • Muscle atrophy
  • Shortened strides
• **Signs of Equine Fixations**
  • Inability to engage rear quarters
  • Difficulty flexing at poll; head shy, shaking
  • Lameness
  • Rider unable to sit centered on horse
  • Asymmetrical sweating
• **Integrative Prevention**
  • Proper shoeing/ Barefoot trimming
  • Proper training techniques: avoid unnecessary training devices
  • Proper warm-up & warm-down periods
  • Proper fitting blankets, saddles
• **Prevention: Saddle Fit**

Beware of Saddle-induced injuries:
• Obvious sores
• White hairs under saddle
• Swellings, hair rubs
• Deep muscle spasms
• Spasms in the withers
• Atrophy of muscles on side of withers
• **Prevention: Saddle Fit**
  • Check for: enough space, but not too much in withers
  • Even weight distribution of panels throughout the saddle w/ contact w/ horse
  • Not too hard a saddle, good cushion
  • Structure of saddle: broken tree
  • Position too far forward or back
  • Check for pressure points
• **Diagnostic & Therapeutic Options**
  • Conventional medical lameness approach
  • Flexion tests, gait analysis, Appropriate blocks
  • Ultrasounds, Radiographs, Scintigraphy
  • Consider appropriate medical & surgical options
  • Weigh risk/benefit of diagnostic tests & therapies
• **Therapeutic Options**
  • Acupuncture
  • Chiropractic/ MSA
  • Riding & training techniques
  • Massage, TEAM,
  • Physical therapies: ultrasound, laser, magnetic therapy
  • Rest, heat, cold therapy
  • Botanical medicine, homeopathic remedies
• MSA Therapy Examination Techniques

Initial observation of patient:
  • Posture: standing in stall
  • Gait coming out of stall, watching movement
  • Behavior
  • Conformation
• **Manual Therapy Examination Techniques: Static Palpation**
  • Palpation of symmetry of spinous processes: dorsal, transverse
  • Symmetry
    – left to right
    – cranial to caudal
    – dorsal/ventral
  • Symmetry of musculature; muscle atrophy
  • Exam for kyphosis (roach back), scoliosis, swaybacked
  • Heat, pain, muscle tone, spasms
• **MSA Examination:**
  • **Motion Palpation**
  • Evaluation of active & passive range of motion in vertebral motor units
  • Lateral flexion, axial rotation, flexion, extension
  • Movement of equine spine documented by Dr. Hausler of Cornell Veterinary School
  • Requires experience to know what is normal vs. abnormal
• **MSA Techniques:**
  • Adjustments
    • Short-lever, specific high velocity controlled forceful thrust by hand
    • Directed at specific articulations
    • Designed to restore normal biomechanical & neurologic function.
    • \( F = M \times A \):
      force is created by the interplay of mass & acceleration
• **MSA Techniques**
  • Short lever
  Specific vertebrae
  • Long lever
  Used on an extremity occasionally
  Risky & potentially harmful if done w/o knowledge of anatomy & experience
• **MSA Technique**
• **Diagnostic Acupuncture Point Evaluation (DAPE) & Musculoskeletal alignment evaluation** pain, mobility, spasms, asymmetry & alignment issues
• **Chiropractic Evaluation**
• Adjustments to correct fixations and improve mobility
• AP to resolve unresolved trigger points & trigger point patterns
• **Companion Animal**
  • **Transition Regions**
    • Atlanto-occipital
    • Cervical-thoracic
    • Thoracolumbar
    • Lumbosacral
    • Most common locations for biomechanical mobility issues to begin
    • And then spread from here throughout the entire cervical and thoracolumbar and sacroiliac musculature
• **Clinical Examples:**

Atlanto-occipital and sacro-iliac fixations
LAMENESS AND INTEGRATIVE TREATMENTS FOR EQUINE BACK PROBLEMS

- One of the most common examples
- Musculoskeletal alignment of those regions corrects alignment and improve mobility
- Acupuncture releases any trigger points
- Combination improves overall performance
- Clinical Application Examples

AP & musculoskeletal alignment to correct these issues:
- Jumper with coffin joint issues and hock issues and saddle issues
- IA injections to appropriate joints
- Saddle fit correction
- Decreased mobility in cervical & thoracolumbar & Sacro-iliac regions
- Clinical Examples:
  - Which came first, chicken or egg?
  - Primary hindleg issues (e.g. hock, stifle problems)
  - Secondary decreased mobility and fixations in the sacro-iliac region and pelvis
  - Correction of sacro-iliac mobility after treating the primary lower leg issues improves performance
  - Diagnostic challenge sometimes: varies by horse
  - Kindergarten song: hip bone connected to knee bone
  - Pelvic Rotations
  - One of the most common issues: 3-D pelvic rotation: right, dorsal, oblique
  - Adjustment: On right, right hand on right ventral ileal wing & left hand on left ischeal tuberosity
  - AQP to release associated TP’s: i.e. BL23, BL-25, BL 28, sometimes: BL54
  - Clinical Examples
  - Incorrect shoeing or foot trimming, poor saddle fit and incorrect riding may result in neck & back pain, decreased mobility and fixations throughout the neck, back and pelvic regions
  - TX: Correct shoeing, saddle fit and riding
  - Correct misalignments, fixations with manual therapies
  - Correct trigger point patterns with AP
  - Improve overall performance
  - **Manual Therapy Techniques**
  - Correct primary fixations, misalignments
  - Correct secondary compensatory problems
  - Ancillary care may include:

  - Acupuncture, muscle relaxants, rest
  - Stretching exercises, massage, laser therapy
  - Therapeutic ultrasound, magnet therapy
  - Long & low riding techniques

Acupuncture Approach
- Most commonly used acupoints: LI-16, BL-21, BL-23, BL-25, BL-28
- Aquapuncture in appropriate points, dry needle appropriate points, electroap etc.
- Revaluation of DAPE and treat appropriate remaining points
- Other approaches:
  - Ting point therapy, TCM approaches

**Integration of AP & Manual Therapies into Equine Practice**
- Preventive health care
- Sports medicine
- Treatment of back & neck pain
- Treatment to correct primary back problems or secondary compensation
- Along w/ acupuncture for muscle spasms
LAMENESS AND INTEGRATIVE TREATMENTS FOR EQUINE BACK PROBLEMS

- Follow-up w/ stretching, massage
- Additional Physical Therapeutic Options
- Low Level Light Laser Therapy:
  - Low Level (Cold) Laser Therapy (LLLT)
  - Class II-III Laser Therapy vs.
  - Class IV Laser Therapy
  - Pulsed vs. nonpulsed

Great research available on pro's & con's of each
- Additional Physical Therapeutic Options
- Magnetic Therapy:
- Electromagnetic Blankets (EMB):
  - I do not use these the same day that I do acupuncture
  - EMB prior to and after heavy work
- Magnetic Blankets with static magnets
- Additional Physical Therapeutic Options
- Back on Trac Carbon fabric blankets, saddle pads, foot and ankle wraps
  - Also for humans
  - Reflects body head back into the muscles
  - Stretching Techniques
  - Equine Yoga: neck stretches: 3-D
    - frontleg stretches,
    - Back stretches, tail traction,
    - Hindleg stretches
    - Before and after work
  - Soft tissue bodywork
  - Massage
  - Craniosacral
  - Rolfing
  - Myofascial release
- TCVM Herbs For Bi Syndrome
  - Bi syndrome refers to pain & stiffness in muscles, tendons, bones & joints.
  - TCM: Invasion of Wind, Cold, Damp & Heat
  - Includes osteoarthritis, rheumatoid arthritis, spondylitis etc.
  - Kidney Qi Def., Bony Bi, KidneyYin & Qi/Yang Def.Bony Bi, Painful Bi, & Fixed Bi
- TCVM Herbs For Bi Syndrome
  - Painful (Cold) Bi: joint pain, difficult movement, heat relieves pain, dislikes massage,
    - Tongue purple or slightly pale
    - Pulse: tight & wiry
  - JT Formula: EquineDu Huo or Dok’s formula
- TCVM Herbs For Bi Syndrome
  - Fixed (Damp) Bi: Difficult movement, more stiff than pain, heat relieves pain
  - Tongue: greasy & pale coating
  - Pulse: Soft & Slow
- Coix formula (JT)
- TCVM Herbs For Bi Syndrome
  - Kidney Qi/Yang Deficiency
  - Difficulty getting Up or walking
  - Cold back
  - Lameness worse in cold/ damp or winter
  - Likes being massaged
  - Tongue: pale, wet; Pulse: deep & weak
  - Loranthus Formula (JT)
LAMENESS AND INTEGRATIVE TREATMENTS FOR EQUINE BACK PROBLEMS

- TCVM Herbs For Bi Syndrome
- Kidney Yin & Qi/Yang Deficiency
- Dry skin, thirsty,
- Lameness worse in hot summer
- Lethargy
- Tongue: pale or red
- Pulse: thready & weak
- Di Gu Pi (JT formula)

- TCVM Herbal Supplements
- Body Sore (Jin Tang): generalized, nonspecific body muscle soreness of undiagnosed etiology: pain in soft tissues, muscles, ligaments
- JT Tendon Ligament Formula: assists in healing tendons and ligaments;
- I also use it as a preventive when bringing horses back from suspensory injuries

- TCVM Herbal Supplements
- Hot Hoof 1 Formula (JT): Laminitis, Navicular syndrome, Sore heels
- Correct etiology & conventional medical approaches as well

- TCVM Herbal Supplements
- Infectious diseases & musculoskeletal/ neurologic issues:
- Lyme formula and Wei Qi formula (JT)
- Nerve Restore (Noahs Ark)
- Medications
- Methocarbamol (Robaxin)
- NSAIDS
- Topical liniments

Veterinary Chiropractic: Future Implications
- More than “animal crackers”
- Beware of “animal crackers”
- Check for appropriate training
- Can harm your horse if not done properly: fractured femurs, necks
- Integral part of sports medicine practice

EQUINE THERAPEUTIC OPTIONS
- Our primary goal is to help our patients heal
- We should look at all options for diagnosis and treatment from conventional medicine and surgery to complementary therapeutics
- Acupuncture, manual therapies & physical therapy
- The most important consideration is

We treat all beings with kindness, compassion and love!

Equine Integrative Approach: Conclusions
- Communicate, listen to your horse
- Respect them as a living, loving, sensing, feeling, intelligent being
- Treat them as you would like to be treated, or even better!
- The greatest healing force is Love & Compassion!
- Go forth & do good!
THE VETERINARIAN’S ROLE IN ANIMAL CRUELTY INVESTIGATIONS

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Veterinarians encounter animals that appear to be suffering from non-accidental injury in the course of regular practice. Veterinarians may also be called upon to document animal cruelty cases presented by law enforcement. Understanding what information investigators and prosecutors seek, case packaging and presentation can help strengthen an investigation. This presentation will give an overview of veterinary forensics, useful techniques as well as pitfalls to avoid.

Forensics and Veterinary Forensics
Cases of serial killers getting their starts as animal abusers are dramatic and startling examples of the link between violence to animals and people. While not all animal abusers go on to abuse people, most heinously violent abusers of human beings have also abused animals. More commonly, in domestic situations, family members may abuse or threaten to abuse family pets to exercise power and control over their human victims.

Not only is the investigation of animal cruelty the right animal welfare intervention for the animal victim, it is a social imperative to mitigate the damage done by animal abusers. Veterinarians’ contributions to the investigation of animal cruelty and neglect can help create a more successful and just prosecution.

Reporting of Animal Cruelty by Veterinarians
Veterinarians have been and continue to be important reporters of animal abuse and neglect when blatant cases have been presented by clients. Unfortunately, many veterinarians are still reluctant to report animal cruelty for a number of reasons: a lack of recognized, published standards defining neglect vs. abuse, concerns about confidentiality clauses in the practice acts regarding medical records, and civil liability attached to reporting, concerns of personal safety or retaliation when dealing with potentially violent clients, loss of personal income, and concern about time lost testifying in court.

The CVMA states “Most cases of animal abuse and neglect can be handled through client education. When education fails, a report should be filed with the appropriate authorities for investigation.”

Veterinarians should remember that a report of a suspicion of animal cruelty does not convict a perpetrator, it only opens the investigation. Many provinces provide immunity for the veterinarian reporting suspected animal abuse in good faith and some mandate that a veterinarian must report suspicions. When reporting of suspected animal abuse is required by law, it removes hesitation, ethical dilemma, or struggle on the part of the reporting veterinarian.

How veterinarians can contribute to the forensic process
Beyond identification and reporting of animal cruelty within their own practice, veterinarians have skills and tools to help build the case against a suspected animal
SUSPECTED TO IDENTIFY FRACTURES IN VARIOUS STAGES OF HEALING.

RADIOPHGRAPHS

RADIOPHGRAPHS MUST BE TAKEN IN CASES WHERE SERIAL ABUSE (BATTERED PET SYNDROME) IS SUSPECTED TO IDENTIFY FRACTURES IN VARIOUS STAGES OF HEALING.

WHOLE BODY

WHOLE BODY RADIOPHGRAPHS SHOULD BE TAKEN OF AN EXPOSURE THAT ALLOWS VISUALIZATION OF THE SOFT TISSUE.

The animal should be photographed on presentation (or on arrival at the scene). Photographs of lesions should be taken in series of three, an overall of the animal with the affected area in the photograph for orientation, a second zooming in at the level of the lesion with anatomical markers apparent, and a third, a close up of the lesion itself with a photographer’s scale in place. The date and animal identification or case number should be legible within the overall view of the animal. The physical exam should be done in a meticulous systematic fashion. Signs of injury can be subtle such as scleral injection (redness of the whites of the eye), hyphema (blood pooling in front of the iris) or subtle collapse of the frontal sinus. Puppies that have been dragged will have scabbing on the perineum and foot pads.

RADIOPHGRAPHS

RADIOPHGRAPHS SHOULD BE TAKEN OF AN AREA OF BLUNT TRAUMA, ASYMMETRY, HEAT OR SWELLING AND BE OF AN EXPOSURE THAT ALLOWS VISUALIZATION OF THE SOFT TISSUE. WHOLE BODY RADIOPHGRAPHS SHOULD BE TAKEN IN CASES WHERE SERIAL ABUSE (BATTERED PET SYNDROME) IS SUSPECTED TO IDENTIFY FRACTURES IN VARIOUS STAGES OF HEALING. RADIOPHGRAPHS MUST BE TAKEN IN SERIES OF THREE, AN OVERALL OF THE ANIMAL WITH THE AFFECTED AREA IN THE PHOTOGRAPH FOR ORIENTATION, A SECOND ZOOMING IN AT THE LEVEL OF THE LESION WITH ANATOMICAL MARKERS APPARENT, AND A THIRD, A CLOSE UP OF THE LESION ITSELF WITH A PHOTOGRAPHER’S SCALE IN PLACE. THE DATE AND ANIMAL IDENTIFICATION OR CASE NUMBER SHOULD BE LEGIBLE WITHIN THE OVERALL VIEW OF THE ANIMAL. THE PHYSICAL EXAM SHOULD BE DONE IN A Meticulous SYSTEMATIC FASHION. SIGNS OF INJURY CAN BE SUBLT SUCH AS SCLERAL INJECTION (REDNESS OF THE WHITES OF THE EYE), HYPERHMA (BLOOD POOLING IN FRONT OF THE IRIS) OR SUBLT COLLAPSE OF THE FRONTAL SINUS. PUPPIES THAT HAVE BEEN DRAGGED WILL HAVE SCABBING ON THE PERINEUM AND FOOT PADS.

DETAILED DOCUMENTATION

DETAILED DOCUMENTATION OF A CASE FROM THE OUTSET IS ESSENTIAL, AND MUST BEGIN BEFORE ANY INTERVENTIONS. AS CAREGIVERS, IT IS OUR IMPULSE TO FEED, CLEAN, HYDRATE. HOWEVER, THERE IS ONLY ONE OPPORTUNITY TO DOCUMENT THE ANIMAL THROUGH THE COLLECTION OF LABORATORY WORK AND PHOTOGRAPHY BEFORE CHANGES ARE MADE.

At the beginning of the examination process, blood should be drawn for baseline references; blood chemistry, complete blood count, and urinalysis are a minimum database. Feces should be collected for parasite analysis. If cachexia (physical wasting with loss of muscle mass) is suspected to be due to starvation, blood work should be drawn to rule out malabsorptive and catabolic conditions (such as hyperthyroidism). However, if finances do not allow advanced blood chemistry analysis, careful tracking of weight gain with feeding as the only therapeutic intervention may be performed.

Initial suspicions and thoughts should be conveyed verbally to the investigative team as any written materials may be subject to discovery. Detailed exam reports (medical records) must be kept. Once the results of initial exams and testing are available, a preliminary summary report may be written. A final summary report should only be written when all medical issues are resolved and all test results are returned. Conclusions should not be presented as absolute but rather with terms such as “consistent with,”“compatible with,”“similar in all respects as,”“indicative of” in order to avoid inevitable cross examination questioning traps. In large scale cases, summary charts (Excel or Access) should be provided to show numbers of animals suffering from various conditions (eg emaciated, thin, anemic, fleas, dental disease, dehydrated etc). Each individual animal must have a medical record reflecting its particular conditions. In addition to recording weights, each animal should be given a body condition score using an established body condition scoring system (Purina scale or TACC scale). The ASPCA maintains a library of sample forms on their ASPCApro website. (www.aspcapro.org/sample-forms.php)

The animal should be photographed on presentation (or on arrival at the scene). Photographs of lesions should be taken in series of three, an overall of the animal with the affected area in the photograph for orientation, a second zooming in at the level of the lesion with anatomical markers apparent, and a third, a close up of the lesion itself with a photographer’s scale in place. The date and animal identification or case number should be legible within the overall view of the animal. The physical exam should be done in a meticulous systematic fashion. Signs of injury can be subtle such as scleral injection (redness of the whites of the eye), hyphema (blood pooling in front of the iris) or subtle collapse of the frontal sinus. Puppies that have been dragged will have scabbing on the perineum and foot pads. Radiographs should be taken of any area of blunt trauma, asymmetry, heat or swelling and be of an exposure that allows visualization of the soft tissue. Whole body radiographs should be taken in cases where serial abuse (battered pet syndrome) is suspected to identify fractures in various stages of healing. Radiographs must be
permanently identified in the radiograph with the date and the animal identifying information. Whenever possible, and when necessary for subtle findings, radiographs should be interpreted by a board certified radiologist.

PAIN AND SUFFERING
The documentation of pain and suffering is one of the most important aspects of the veterinary forensic examination. In daily clinical practice, we acknowledge when pain exists, but the focus is on the alleviation of pain through pharmaceutical and other interventions. Of course the pain of a cruelty victim must be alleviated as swiftly as possible, but it must also be characterized to the best of our ability. The perception of pain is an activity that takes place in the cerebral cortex. As veterinarians, we do not have access to that inner process but we can infer from behaviors that an animal is painful (gait analysis, posture, vocalization, trembling, level of or lack of activity, response to handling) or in a state of comfort (good appetite, drinking, grooming, normal sleeping, social interaction). These behaviors should be recorded and inferences drawn. Also, the change in behavior after pain medication is administered should be recorded.

The University of Glasgow, Pain and Welfare Research Group have developed a short form for the scoring of post-surgical animal pain. Colorado State University has dog and cat specific acute pain scoring systems. While not intended to be used as scales for acute or chronic pain resulting from non-accidental injury, these references may be helpful to demonstrate that animal pain can be recorded and scaled using university based resources. This helps provide credibility to documentation.

Biologists recognize that those stimuli which cause pain (noxious stimuli) are liable to damage tissue. In these cases, biopsy samples should be taken to help in the classification and characterization of the injury. For example, for most animals, including dogs and cats, skin does not blister, so the human classification system using 1st, 2nd and 3rd degree burns does not apply (even for humans, this system is changing). Instead, burns should be categorized by their depth: superficial, partial thickness-superficial, partial thickness-deep and deep.

Other types of discomfort must be recorded and scaled when possible (overgrown nails, hair coat matting, morbid obesity). Mats are uncomfortable, painful and in some cases debilitating. The duration of these conditions should be characterized in the summary reports (days, weeks or months). The Tufts Animal Care and Condition scoring system allows for the objective classification of physical care in dogs as well as exposure to environmental conditions.

Suffering is defined as a deviation from a state of comfort. In order to suffer, an individual must be able to cognize. Fortunately, the pet food companies are enhancing our ability to argue that our patients do cognize by marketing foods specifically developed and produced to enhance cognition! Suffering has also been defined as “different unpleasant states that one would avoid or remove themselves from if they could.” (Dawkins, 2005) Or “an unpleasant state of mind that disrupts quality of life - this mental state is associated with unpleasant experiences including pain, malaise, distress, injury and emotional numbness.” (Gregg 2004) The medical record should reflect the extent of matting and dermatitis for example, and the
summary report should suggest the duration and degree of pain, discomfort and suffering.

EVIDENCE COLLECTION
During the course of initial examination, forensic materials (evidence) may be observed requiring specific preservation. For example, in gunshot wounds, the trajectory through tissue should be established (via radiography, CT if possible, careful probing of tissue) and recovered projectile fragments (bullets) should be handled only by the gloved hand or plastic forceps in order to avoid scarring ballistic information.

The age of a wound or duration of suffering may be estimated based on granulation bed formation. Granulation takes 5 to 7 days post injury to start becoming apparent, and then progresses at about 1 mm a day for the initial week or two, and then slows to one cm a month. This measure is important in refuting a perspective that the animal was only injured “yesterday.” Hair mats clipped from animals should be saved as evidence as well as any material entrapped in the hair mats. Embedded collars should be preserved in a zip lock plastic bag and frozen until trial. The odor will be well preserved and provides powerful testimony of its own. Additionally, the circumference of the neck (where not swollen or cut into by the collar) should be taken and recorded in comparison to the length of the embedded collar around the dog’s neck. When animals are tethered by chains or burdened with chains to “strengthen” them, the chains should be weighed and expressed in percentages of body weight and human equivalents.

A list of drugs and veterinary materials (antiseptics, bandage materials, suture material, etc) found at the scene of an animal crime should be documented (especially animal fighting and animal hoarding cases). This in combination with the totality of evidence can build an argument of intent and awareness as well as building a case of practicing veterinary medicine without a license.

In the deceased patient, necropsies should be systematic and complete. During the course of necropsy, skin should be reflected (peeled back) to demonstrate areas of bruising on the underside of the skin from blunt force injury. Animals have thicker skin and reduced capillary circulation as compared to people which often does not allow bruising to be seen on the outer surface of the skin.

STARVATION
Starvation in the adult animal causes loss of tissue (fat stores and muscle mass). Starvation in the young will cause the loss of body tissue and the stunting of growth. Very underweight or emaciated animals will adapt a posture typical of weakness, this should be described. Fat stores will be used from external body stores first, then internal stores (around the heart and the kidney) and finally from the bone marrow. Starved animals will be anemic, hypoproteinemic (low levels of protein in the blood), may have pica (eating foreign objects - document via radiographs, collecting 1st feces passed in custody or gastric contents at necropsy). End stage starved animals may have melena (black tarry stool from digested blood) from gastric ulceration and decubital ulcers (“bed sores”). Starved cats may develop reddish coats from decreased melanin deposition. It can be difficult to determine how long an animal has been...
starved depending on whether they were absolutely starved or if they had access to inadequate amounts or quality of nutrition.

Studies done with total calorie restriction in obese dogs showed that they would lose 8% of their body weight in the first week, 5% in the second week and 3 – 4 % in subsequent weeks. Calculations can be performed to estimate how long it would have taken a dog to go from ideal body condition to the presenting weight at absolute starvation. The timeline to that body condition would be delayed by intermittent or inadequate nutrition. Blood urea nitrogen, creatinine and urine specific gravity are low during starvation and will rise after refeeding, taking weeks to reach normal levels. Refeeding syndrome is an electrolyte disturbance that is well documented in starved humans causing diarrhea which can be seriously compromising. This phenomenon has not been demonstrated in dogs. Refeeding dogs can be done by calculating the dog’s energy requirement at just above the current weight, feeding amounts should be adjusted up weekly until ideal body condition is reached. The dog that was starved as a juvenile will have a smaller stature than expected for their age (age can be estimated from dentition charts and radiographic ossification charts). Compare the growth of the starved dog as it is fed to a standard growth chart (Current Veterinary Therapy VI, p 1366). Compensatory growth is evidence of prior starvation. On average, dogs gain 80% of their body weight from the 2nd month of life to the 3rd month, 45% from 3 months to 4 months, 20 – 25% from 4 to 5 months of age, and about 15% from 5 months to 6 months of age.

DNA EVIDENCE
DNA evidence can be helpful to prove that a particular object was used to harm an animal. DNA collection techniques are clearly described at the University of California Veterinary Genetics Diagnostic Lab (VGL) website. Gloves should be worn when handling materials to be tested for DNA. Dry samples can be collected on a cotton tipped swab moistened with sterile water. The swab should be allowed to air dry and sent in paper (not plastic) for analysis. Genetic testing is very expensive, but there is occasionally grant money available to provide for discounted or free testing. Suspected human genetic material from animal sexual abuse cases will not be processed by veterinary genetic laboratories. Work with local law enforcement to access the state police laboratory for these samples.

PREDATION
Cases of eviscerated cat bodies or cat parts left behind can be reported in the media as “a cat killer on the loose.” A careful veterinary examination may help determine that the suspect is a coyote, fisher cat, fox, bird of prey or other animal. Post-mortem predation may also explain missing eyes, tongue, genitalia and perineum. Histological examination of tissues will help to elucidate pre vs post-mortem injury, and etymologic evidence may help establish a time of death.

Where the field is being developed
Maples Center for Forensic Medicine at the University of Florida has an online certificate and master’s degree course in veterinary forensics. See the University of Florida website for information. Shelter medicine courses at many veterinary schools incorporate components of forensic medicine; consulting with your local veterinary school’s shelter medicine faculty may help you find a veterinarian with the right
training or interest. Veterinarians at animal welfare agencies, which have been performing law enforcement functions likely have been performing veterinary forensics for years. These folks have a wealth of knowledge, seek them out. The International Veterinary Forensic Science Association was formed in 2008 at the first annual Veterinary Forensic Sciences Conference. This conference is held annually in May in Florida.

Calculations and statements:
BLOOD LOSS In the patient who has had sudden blood loss due to hemorrhage with a normal total protein, the blood volume loss can be calculated.

\[
\text{(Normal standard hematocrit – current hematocrit)} \\
\text{Normal standard hematocrit} = \text{Fractional blood loss}
\]

Example (using the low end of normal for “normal” hematocrit) - for a 10 kg dog with a hematocrit of 13: \((37 -13)/37 = 0.64\) or 64% loss of blood volume. Dogs have in general 66 cc of blood per kg of weight. Fractional blood loss X animal’s normal blood volume = blood volume loss.\(64 \times 60 = 38.4\) ccs. Therefore, a previously healthy 10 kg dog with a post-hemorrhage PCV of 13 can be estimated to have lost at least 38 ccs of blood (or as much as 44 ccs using 50 as the upper limit of normal).

WEIGHT GAIN
Weight changes should be calculated and expressed in percentage changes, ie: A starved dog comes in at 23.6 pounds and is determined to be at ideal body condition after refueling for several weeks. She weighs 19.9 pounds more at the ideal condition than she did at intake. \((43.5 - 23.6) = 19.9\) (Recent weight - incoming weight = weight gain) \(19.9 / 23.6 = 84\%\) weight gain in 2 months (or it may be expressed as \(19.9 / 43.5 = 46\%\) weight loss from Ideal weight) (Weight gain divided by incoming weight = percentage gain) “At presentation, the dog’s weight was 43.5 pounds. As of the writing of this statement, the dog’s weight has increased to 43.5 pounds. This is a 19.9 pound weight gain in 19 days. This is a 84% increase in weight in less than 3 weeks. There has been no treatment other than feeding a balanced diet of commercial dog food.”

CHAIN WEIGHT
“This 34 pound dog was tethered by a 7 pound chain, representing 20% of his body weight. This is equivalent to a 150 pound man being tethered by a 30 pound chain.”

Further Reading:
- Melinda Merck’s Veterinary Forensics (forms, references) [www.veterinaryforensics.com](http://www.veterinaryforensics.com)
- Glasgow Composite Pain Scale (short form) [www.gla.ac.uk/media/media_61908_en.pdf](http://www.gla.ac.uk/media/media_61908_en.pdf)
- Hoarding of Animals Research Consortium [www.tufts.edu/vet/hoarding](http://www.tufts.edu/vet/hoarding)
- International Veterinary Forensic Sciences Association [www.ivfsa.org](http://www.ivfsa.org)
- UF distance learning forensic programs [http://www.forensicscience.ufl.edu/veterinary/programs/ALDF](http://www.forensicscience.ufl.edu/veterinary/programs/ALDF)
ANIMAL WELFARE COMPANION ANIMAL

- Tufts Animal Care and Condition Scales [www.tufts.edu/vet/hoarding/pubs/tacc.pdf](http://www.tufts.edu/vet/hoarding/pubs/tacc.pdf)
- Introduction to Veterinary and Comparative Forensic Medicine, Cooper & Cooper, Blackwell Publishing, 2007.
- Special thanks to the ASPCA, Dr. Lila Miller, Dr. Robert Riesman and Dr. Melinda Merck for your leadership in the areas of animal cruelty prevention and veterinary forensics.
ANIMAL WELFARE COMPANION ANIMAL

ANIMAL SEXUAL ABUSE

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The sexual abuse of animals includes a wide range of sexual behaviors. The elements of a crime of animal sexual abuse depends on the jurisdiction and applicable laws. This presentation will give an overview of animal sexual abuse, the behavior of offenders and tips for physical exam and necropsy of the animal sexual abuse victim.

WHY SHOULD THE VETERINARIAN BE INFORMED ABOUT ANIMAL SEX ABUSE?

The simple answer is: Sooner or later, most of us will encounter a case of animal sexual abuse. With training, a veterinarian is more likely to recognize non-accidental injuries (NAI) or death as having been sexual assault in origin. Signs that should arouse suspicions of non-accidental injury include inconsistent history, untreated injuries, recurring injuries, unusual meekness of an animal, suspicious behavior of the owner and injuries consistent with abuse. Additionally, animal sex abusers may seek hormone therapy for their female victims or sedatives for any target in order to manipulate their sexual receptivity.

A study of NAI in small animals in the UK by Munro and Thrusfield identified 6% of the 448 reported cases as being sexual in nature. Proper identification of a case of animal sexual abuse increases the likelihood of appropriate intervention for both the animal victim and animal abuser. As with any forensic veterinary work, it is not the veterinarian’s duty to draw any conclusion of innocence or guilt, but rather to perform a meticulous exam and document it appropriately. Inferences may be drawn based on veterinary expertise and clinical experience to the likelihood of injury being accidental or not.

Unfortunately, many veterinarians continue to be reluctant to report animal cruelty for a number of reasons: a lack of recognized, published standard definitions, concerns about confidentiality clauses in the practice acts regarding medical records, and fears of civil liability attached to reporting, concerns of personal safety or retaliation when dealing with potentially violent clients, loss of personal income, and concern about time lost testifying in court. The reporting of animal sexual abuse carries the increased burden of being a taboo subject.

HUMAN SEXUAL CONTACT WITH ANIMALS & TERMINOLOGY

Human sexual contact with animals is a form of animal abuse that has impacts for the animal victim, the abuser and society. There is disagreement within the sociology, criminology, legal and medical fields as to the most appropriate term to describe human sexual contact with animals. The veterinary field is only just beginning to explore this phenomenon, and it is rarely brought to the public forum for discussion. The term “animal sexual abuse” is preferred by some in the animal welfare field over “zoophilia” and “bestiality”, as both of those terms are perpetrator-centric and thus may fail to convey a sense of the harm that occurs to the animal.
Other terms that may be used are “zooerasty”, “animal sexual assault”, or “sex with animals”. Some argue that the term “bestiality” brings to mind rape, which may be preferred for conveying the brutality of the abuse when trying a case in court. Regardless of what terminology is used, as veterinarians, our primary concern is with the animal welfare implications of human sexual contact with animals, the recognition of injuries and meticulous documentation of evidence.

SHADES OF GREY – A SPECTRUM OF BEHAVIORS

Human sexual contact with animals may occur because of opportunity, not necessarily reflecting a sexual desire for animals, or it may occur because the abuser has a preference for animals for arousal. The range of human sexual contact with animals is a spectrum from role playing (which may be a gateway to animal sexual assault), to zoophilia and bestiality.

Role Playing “Furry Fandom” is a subculture interested in fictional anthropomorphic animal characters with human personalities and characteristics; it is also used to refer to the community of people who gather on the Internet and at conventions. Some ‘Furs” build elaborate costumes called “fursuits” to gatherings. “ Anthrocon” held in Philadelphia each year, attracted 5,000 attendees in 2012. Most “furries” are only interested in the anthropomorphic life, it is a community in which animal abusers circulate. In a survey of Anthrocon attendees in 2008, 17% of respondents self-reported zoophilia.

Zoophilia Zoophiles (“zoos”) claim to have a deep and mutual emotional attachment to their animals, often regarding these animals as their lovers or mates. Zoos believe they are "conscientious" in their behavior and feel a shared romantic attraction with an animal. Zoos will groom animals to accept sexual contact in a very similar manner to the way that pedophiles groom children. The method is to use positive reinforcement to gain increasing physical access. Another parallel between pedophiles and animal sexual abusers is that they will share photos and videos on the internet (referred to as “aniporn”), and even offer their animals to share or for meet-ups.

Many “zoos” assert an ethical superiority to or disdain for bestialists who they liken to rapists – people who use animals for sexual gratification with no regard for the animals’ feelings. In the most recent edition of the Diagnostic and Statistical Manual of Mental Disorders, DSM-V (2013) zoophilia is not considered a diagnosable mental health problem unless it causes distress to the person who does it. This may be because zoophiles rarely seek treatment, and not all people who engage in sex with animals are zoophiles. Most regrettably, the DSM doesn’t consider animals as victims (a requirement for diagnosis of a paraphilic disorder typically requires that a human be harmed in some way).

Bestiality is the physically forceful sexual molestation of animals by humans and includes a wide range of behaviors, including fondling; vaginal, anal, or oral penetration; oral-genital contact; penetration with an object; and injuring or killing an animal for sexual gratification. Bestiality may be a crime of opportunity, such as the choice of an animal victim by a child molester to avoid the risk of being caught or re-offending.
The historic cliché of a farm boy with farm animals arises from data gathered in the Kinsey report in the late 1940s. Some of the currently available literature pointing to a decline in bestial activity is somewhat misleading given that we are no longer a rural, agricultural society. It is not that bestial activity is no longer taking place, but rather that it has moved from the farm to the house or apartment, and the prevalent species affected are no longer farm species.

The Moral Position In cases where an animal is not physically injured, some question where the harm lies in animal sex abuse behavior. Similar to pedophilia, in animal sexual abuse, there is an imbalance of power that makes sexual contact with an animal coercive – here the imbalance of power is based on species rather than age difference. Animals are neither able to consent nor report such an episode.

THE ABUSERS
Animal sexual abusers are difficult to profile because they come from every walk of life, span age groups from the teenaged to the elderly and have many different types of human relationships. Like some pedophiles, many “zoos” are actively engaged or employed in high tech jobs; they may run complicated, encrypted websites, and frequently work in computer-related fields. They may demonstrate a failure to relate to members of their own species. Bestiality in the sexual history of a juvenile offender may be an indication of risk for sexually abusing a person in the future. Sexual abusers of animals are more likely to have been victims of emotional neglect and abuse as children.

Bestiality is most commonly found among violent offenders, sex offenders and the sexually abused. This is thought to be because sexuality and aggression become developmentally fused when people or animals are treated as objects: manipulated, exploited and controlled. Various studies do point to a link between bestiality and other forms of animal or human cruelty. There unfortunately is not enough research to support any particular hypothesis. For bestialists, generalized deviation and sexual polymorphism as descriptors fit better than a graduation hypothesis. Some people do graduate from viewing “aniporn” to having sexual contact with an animal, but more often, they started with physical contact. Much is still unknown about the sexual abusers of animals and their propensity to violence against people because what we do know comes from a small handful of limited studies.

Animal sexual abusers, like pedophiles, may associate with one another in chat rooms or in get-togethers where they may bring animals to swap and share. They are very difficult to track or identify, and many do not bring their animals to the veterinarian for care for fear of being identified.

THE LAW
Canadian Federal law states “Bestiality is an indictable offence and punishable by up to a term of ten years or, alternatively, is a summary conviction offence.” And “Committing bestiality in the presence of a person under the age of sixteen years or inciting a person under the age of sixteen years to commit bestiality is an indictable offence and punishable by up to a term of ten years or, alternatively, is a summary conviction offence.”
Crush Videos A crush video graphically depicts the torture, beating, burning, or mutilation, and ultimate death of an animal, often being crushed underfoot. The prototypical crush video depicts a scantily clad woman in stiletto heels. The “woman” may be an underage girl, hired for as little as a few dollars, or as much as a thousand dollars or more. The torturous acts are videotaped and then sold on porn websites where users may pay anywhere from $35 to $4,000 to view or download them. The films are intended to provide sexual gratification for those who can only be aroused by the torturing of animals. The most common depiction is that of a female from the legs down in high heels physically dominating and brutally killing small creatures with high-heeled shoes. The animal may be pinned by one stiletto and slashed with the other, in the eyes or stomach. After “teasing” the animal, its life is taken by crushing it with a shod foot or between the breasts or buttocks.

Crush films were outlawed by the Animal Crush Video Prohibition Act of 2010 which states that it is illegal to create, sell, distribute, advertise, market or exchange crush films. Unfortunately, U.S. District Court Judge Simeon Lake, in the Southern District of Texas recently ruled that the conduct of the defendants in recent videos was not obscene and the Act infringed upon the First Amendment rights of the defendants. Federal prosecutors have filed an appeal with the Fifth Circuit.

SPECIAL CONSIDERATIONS FOR THE EXAMINATION OF THE ANIMAL SEX ABUSE VICTIM

Injuries to the victim may include blunt force trauma, used to stun the animal, tears to the vagina, vaginal vault, anus or colon, strangulation injuries or ligature injuries to the limbs to prevent the animal from avoiding contact. Injuries to the anus, nipples or genitalia should immediately bring to mind the possibility of animal sexual abuse. Careful otic and ophthalmic exams are important to detect head injury, and evidence of prior strangulation. The ear canals can become thickened from chronic abuse, using the ears to hold the head and prevent the animal from turning and biting. For some animals, the only presenting condition may be an unexplained peritonitis. Others may have recurrent vaginitis or prostatitis. Foreign bodies may be inserted into a spayed dog, and exit the uterine stump to be free in the abdominal cavity, or foreign bodies may be retained in the uterus of the intact dog causing an open pyometra. The particularly sadistic animal sexual abuser may torture the animal as part of their abusive behavior, so any variety of injuries may be found (burns, cuts, contusions etc).

Trace Evidence It is helpful to know Locards’ principle: “Wherever he steps, whatever he touches, whatever he leaves, even unconsciously, will serve as a silent witness against him. Not only his fingerprints or his footprints, but his hair, the fibers from his clothes, the glass he breaks, the tool mark he leaves, the paint he scratches, the blood or semen he deposits or collects. All of these and more bear mute witness against him. This is evidence that does not forget. It is not confused by the excitement of the moment. It is not absent because human witnesses are. It is factual evidence. Physical evidence cannot be wrong, it cannot perjure itself, it cannot be wholly absent. Only human failure to find it, study and understand it can diminish its value ”

Evidence collection During the course of initial examination, forensic materials (evidence) may be observed requiring specific preservation. Sexual assault evidence collection kits or crime scene collection kits for evidence collection can be ordered
from commercial sources, or in an urgent situation may be available from the local human emergency room. Most hospital emergency rooms have a sexual assault nurse examiners [SANE]. This person may be a valuable resource to contact and consult before ever suspecting a sexual assault case. It is important to note that if swabs are being taken to examine for trace evidence (sperm, DNA, condom lubricants etc), they must be fully air dried before packaging for preservation in paper.

Semen may be found anywhere on the animal, not just at the perineal region. Semen will cause the fur to clump together and this fur can be collected by snipping the clumps of fur with scissors. Of course, it goes without saying that gloves must be worn for these exams, and they should be powder free gloves in order to avoid contaminating the evidence.

The vaginal vault can be washed with saline solution, and this solution preserved for further analysis. The patient should be combed out over butcher paper to collect trace evidence if appropriate to the case. When possible, an investigator or officer should be present during evidence collection to take control of the evidence so that it is properly secured and stored. The evidence lab can identify semen and sperm from different species by a variety of tests. The morphology of sperm varies in size and shape. Bull and human sperm, for example, have paddle-shaped heads, rodent sperm have hook-shaped heads, and the heads of chicken sperm are spindle-shaped and almost difficult to distinguish from the midpiece.

It may be tempting to look at the evidence microscopically yourself to confirm your suspicions, but know that your identification of semen as human in origin will probably not stand up in court as your veterinary practice is not a certified forensics lab. The first priority for any evidence is to make it available to law enforcement. With smaller samples, testing will likely be fully consumptive, and they will need every little bit of evidence available.

**Animal DNA** DNA evidence can be helpful to prove that a particular object was used to harm an animal. DNA collection techniques are clearly described at the University of California Veterinary Genetics Diagnostic Lab (VGL) website. Once again, gloves must be worn when handling materials to be tested for DNA. Dry samples can be collected on a cotton tipped swab moistened with sterile water. The swab should be allowed to air dry and sent in paper (not plastic) for analysis. If damp materials are packaged in plastic, mold can develop and denature the DNA evidence.

Genetic testing is very expensive. Samples should be collected and archived appropriately even in the absence of resources for in the case. Should the case develop, resources may be found, and there is only one opportunity to perform any initial evidence gathering.

**Human DNA** Suspected human genetic material from animal sexual abuse cases will not be processed by veterinary genetic laboratories. Law enforcement officers or prosecutors will submit materials to state or county police laboratory for these samples. Although there are a number of bench tests available for purchase, it is not appropriate for veterinarians to perform testing on collected materials. Materials should be collected, logged and made available to law enforcement officials.
**Condom Lubricants** Condom lubricants may be collected on swab samples that will corroborate witness statements that sexual contact occurred in the absence of semen and in fact will account for the absence of seminal fluid. The presence of condom lubricants in a body cavity is evidence of penetration. Laboratory testing of the lubricant may be able to match brand of condom. The most common lubricant PDMS (polydimethylsiloxane – a silicone) is not water soluble, so it doesn’t absorb but it can wash away with urination. Some have particulates, like cornstarch or amorphous silica, so it is important wear powder-free gloves during evidence collection. Nonoxynol-9 spermicide, which is a strong detergent, may also be detectable from swabs.

**Evidence preservation** When moist materials are collected, direct impressions may be made on glass slides. Care must be taken not to contaminate slides or swabs while drying. When dried materials are to be sampled, fur may be cut and collected directly into a paper bundle (a piece of paper folded to prevent small traces from escaping). Alternatively, dried matter may be scraped with a scalpel blade and collected, or swabbed with a swab moistened with water and air dried before preserving in a paper. Each sample should be identified indicating when and where collected and logged on an evidence form. If swabs are used, an unused swab should be submitted in a similar manner as a negative control.

**Evidence management** Evidence management requires that it be collected in a fashion which does not compromise the nature of the evidence, kept in a fashion which maintains its integrity and handled in a fashion which allows no doubt that the evidence could not have been accidentally or deliberately altered or substituted. These procedures will avoid later allegations of tampering or misconduct which can compromise the case. The chain of custody requires that from the moment the evidence is collected, every transfer of evidence from person to person be documented and that it be provable that nobody else could have accessed that evidence. It is best to keep the number of transfers as low as possible, and a signed and dated log must accompany the evidence when it is transferred.

**Zoonotic Diseases, STDs, Injuries and Allergies**

People who engage in sexual contact with animals risk exposing themselves to infectious diseases such as (but not limited to) transmissible venereal tumor, brucellosis, leptospirosis, Q fever, rabies, echinococcosis, campylobacter, cryptosporidium, salmonella, toxocariasis, UTI’s and peritonitis.

Human sexually transmitted diseases ("STDs") are not carried or transmitted by animals. Many human pathogens can survive in animal fluids for a limited time, and therefore STDs may theoretically be transmitted via an animal that has consecutive human sexual contacts in a short enough time frame to allow pathogen survival. The veterinarian must take precautions to protect against exposure to such diseases while conducting a physical exam or necropsy.

Just as animals can be seriously injured by sexual contact by humans, humans may also be seriously harmed. Larger animals may have the strength and defensive attributes (e.g. teeth, hooves, horns, claws) to injure a human through bites or kicks, either in rejecting physical or sexual contact, or during sexual arousal. The penis of a sexually
aroused dog’s bulbous gland can cause injury if forcibly pulled from a human orifice, and equines can thrust suddenly and "flare" inside a human orifice causing significant damage or death. Allergies can also cause problems animal sexual abusers. Repeated exposure to secretions after sensitization has already occurred may subsequently provoke an anaphylactic reaction, which can be life-threatening.

Further Reading:

• The Chandler Edwards Group is a research and training law enforcement assistance organization focused on the issue of animal sexual abuse http://www.chandleredwards.org/
• UC Davis Veterinary Genetics Laboratory Forensics Unit www.vgl.ucdavis.edu/forensics/index.php
People with animal hoarding disorder can be bewildering to deal with, exhibiting frustrating behaviors that are difficult to understand. This presentation will describe three general classifications of animal hoarders, their typical behaviors and motivations in order to provide context for veterinary teams encountering animal hoarders.

Not a benign eccentricity
A variety of studies have supported the origin of the myth of typical animal hoarder as the solitary neighborhood “cat lady.” The majority of animal hoarders are single, older women accumulating cats. However, despite the stereotypes, animal hoarders come from the full range of demographic and economic backgrounds. Animal victims of this condition range across species, including dogs, cats, birds, reptiles, small mammals, horses, livestock and even captured wildlife. It is not uncommon for an animal hoarder to be living with dependent children or elderly family members. Some are reclusive and secretive while others live a “double lifestyle” with successful professional careers. Hoarding behavior has been documented in doctors, nurses, public officials, college professors and veterinarians.

Rather than being a benign eccentricity, the unconventional ownership of large numbers of animals kept in poor condition is thought to be a psychological disorder (or a symptom of a variety of disorders). People with animal hoarding disorder are also oftentimes vexing personalities - with labile mood changes, an adept ability to manipulate others, ranging from cunning to belligerent, hostile, paranoid, obstreperous and onerous to deal with.

There is a great disconnect between what the hoarder thinks, and the reality of the situation. They believe they’re providing good care, when in fact the animals are usually starving, diseased, and dying in severely overcrowded conditions and overwhelmingly filthy environments. Veterinarians and animal welfare workers undoubtedly come across clients that could be considered to be hoarders at least once in their careers. Understanding the variety of psychological origins thought to contribute to animal hoarding disorder and the broad categories of personality types expressed by animal hoarders can help equip veterinarians and staff to deal with these irrational and challenging individuals.

Animal hoarders are not in touch with reality
Without apparent intent or recognition, hoarders cause an enormous amount of suffering and death. They may keep animals for months or years in conditions of horrendous deprivation, in filth and poor air quality. They ignore the most basic animal care; their victims can become severely emaciated, and the animal population often has rampant compound parasitic, bacterial, viral and fungal infections. Animals may display behavioral patterns suggestive of extreme psychological suffering.
hoarders lack insight into the problematic nature of their behavior. Most believe they are providing adequate animal care, or even better care than anyone else could possibly provide.

Some animal hoarders are delusional and may be especially paranoid of government officials, and will go to great lengths to obscure how many animals they have or the extent of degeneration of living conditions within the house. Many animal hoarders, if left to their own devices, will end up in a home without electricity, working plumbing or sewage service. Frequently, the animal hoarder’s building will be condemned and will have to be torn down after the people and animals are evacuated. Animal hoarding scenes are truly human built disasters.

Animal hoarding is a disorder that begins at some point with adequate animal care. As the numbers of animals creep up, the quality of life and care for the animals declines. An incipient hoarder may meet the minimum standards of care for the animals as defined by law, and they may be very aware of the problem that is developing, but they are ill equipped to correct their ways. Recognizing that the animal hoarder may not be able to ask for help or admit being in over their heads means that social services should be notified sooner rather than later. The average animal hoarder is not going to be able to correct his or her ways based on the counseling of a veterinarian or staff.

Most jurisdictions have statutes that mandate that caretakers provide animals with sufficient food and water, a sanitary environment, and necessary veterinary care in case of illness or injury. Therefore the conditions implicit in animal hoarding violate animal cruelty statutes, making animal hoarders criminals under the law. However, in addition to being criminals they are usually individuals in desperate need of psychological intervention for their own well-being, for the welfare of the people around them and in order to prevent recidivism. Prosecution is sometimes the only way to provide lasting intervention, mandate psychological therapy and deliver relief for the suffering animals.

**Unstable childhoods can lead to axis II traits**
Attachment disorders arise from chaotic or traumatic childhoods characterized by loss of important parental or caregiving individuals, inconsistent parenting and emotional abuse. Axis II is classification dimension used with Diagnostic and Statistical Manual of Mental Disorders (DSM), which includes personality disorders—paranoid, schizoid, schizotypal, antisocial, borderline, histrionic, narcissistic, dependent, and obsessive-compulsive personality. Axis II traits are suspiciousness, mistrust, fear of abandonment, unstable and intense interpersonal relationships, feelings of emptiness, difficulty with anger and paranoia. Individuals expressing these traits often come from families with a history of unresolved grief due to tragic, untimely death or losses and emotional or physical abuse.

An absence of nurturing in childhood can cause people to have a deep sense of aloneness in adulthood that can never be remedied. This leads them to seek “perfect love” to repair their wounded selves and make them feel worthy. These unrealistic expectations cause them repeated disappointment, and can contribute to a downward
spiral. It has been conjectured that animals may be perceived to provide this “perfect, unconditional love.”

In a pattern of history repeating itself, the individual who was neglected as a child becomes the adult who neglect the needs of the animals dependent on them. And as animals die (due to a lack of proper care), the hoarder’s sense of unworthiness is reinforced, as is their fear of abandonment. In this case the abandonment is by that of the dying animal. It has been suggested that rather than supplying perfect love, the animal companions provide conflict free companionship. The control that the hoarder has over the animal and its destiny is more of a feature of the relationship than true love.

Pathological altruism
The compulsion to devote as much time as possible giving care rather than receiving care has been called “pathological altruism.” Adult compulsive caregiving has its roots in traumatic losses that leave children feeling like they need care and help which absent or distant parents failed to provide. As these children reach adulthood, they learn to reverse parenting roles but have lost the ability to express needs or ask for care. They will try to help others when deep inside, they have a desire to be cared for. Ironically, animal hoarders are known for failing to provide care, but they adopt a caregiving identity by claiming to save many animals from certain death.

Of course, it would be unreasonable to suggest all animal hoarders have psychological disorders. There may be a complex web of compromised social situations with contributing factors from familial, neighborhood or community social problems that lead to an animal hoarding situation. Once an animal hoarder has developed a reputation as an animal lover, people in the community may drop unwanted pets off at their home. This allows the individual abandoning the animal to assuage their guilt, believing the animal will be cared for, and not be euthanized as they assume will happen at the local animal shelter. Hoarders may have a circle of enablers who contribute to the amassing of animals, and who do nothing to intervene for fear of alienating personal relationships with the hoarder.

The 3 main personality typologies of the animal hoarder
These broad personality types are not meant to be diagnosis, and each hoarder may not fit exactly into any one category, and in fact may have overlapping traits from two or three categories. The classification within these designations is liquid; a hoarder may start as an overwhelmed caregiver and become a rescue hoarder or exploiter hoarder. For the purposes of the veterinarian and staff, these designations help to understand the motivations and provide context to otherwise bewildering behavior.

The overwhelmed caregiver hoarder often acquires animals passively, like the neighborhood cat lady who finds kittens dropped off at her home. Or they may have started as a hobby breeder, but then can’t bear to part with the offspring. He or she is often aware that things have gotten out of hand, perhaps made worse by a departure, illness or death of a partner who used to share some of the responsibility of caring for the animals. While these hoarders are often strongly attached to the animals, they don’t usually (or vigorously) object to people coming in to help.
The rescue hoarder’s once benevolent mission to save lives becomes a compulsion. This type of animal hoarder tends to be articulate, mission driven (often to save animals from “certain death” at animal shelters), media savvy, good at accumulating animals—and very difficult to stop. A “rescuer” identity means that the person can experience feelings of superiority for saving a life that would otherwise be lost; this feeling can become a high as addictive as any drug. In fact animal these individuals share many characteristics with substance abusers, including: a preoccupation with animals, denial of a problem, excuses for their behavior, claims of persecution, and neglect of personal and environmental conditions. They may have a squadron of enabling volunteers (or a rotating cast of characters as it is difficult for them to maintain long term relationships), and they often find reasons why each animal cannot be re-homed.

The exploiter hoarder acquires animals to fulfill their own psychological needs and is completely indifferent to the animals’ condition. This is most difficult hoarder to deal with. They tend to have serious personality disorders that border on or are sociopathic behavior. Manipulative and cunning, type of hoarder is possessive of the animals, and they will express violent opposition to letting even one animal go. Exploiter hoarders believe that their knowledge is superior to all others and they can be litigious to the point of being legal abusers (filing repeated frivolous and vexatious lawsuits). Once evicted from properties or convicted of animal cruelty, they will often move communities to start hoarding all over again. They may keep animals in an RV or bus in order to keep moving from community to community and avoid detection or prosecution. The megalomaniac or narcissistic personality disorder exploiter hoarder uses their charm and charisma to present themselves as competent and credible experts to the public, the media, and animal welfare authorities.

Exploiter hoarders lack guilt, remorse, social conscience, and empathy for animals or people. They demonstrate extreme denial regarding their hoarding situation and may lie, cheat or steal without remorse to achieve their own ends. They reject attempt by authorities to intervene and will manipulate situations to skirt the system e.g. asking enablers to hide animals and evade authorities.

<table>
<thead>
<tr>
<th>Overwhelmed caregiver</th>
<th>Rescuer hoarder</th>
<th>Exploiter hoarder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Some awareness, more reality-based</td>
<td>Mission leading to unavoidable compulsion</td>
<td>Tends to have sociopathic characteristics</td>
</tr>
<tr>
<td>More passive acquisition</td>
<td>Fear of death</td>
<td>Lacks empathy for people or animals</td>
</tr>
<tr>
<td>Problems triggered by change in circumstance</td>
<td>More active vs. passive acquisition</td>
<td>Indifferent to harm caused</td>
</tr>
<tr>
<td>Unable to problem-solve effectively</td>
<td>S/he is the only one who can provide care</td>
<td>Rejects outsiders concerns</td>
</tr>
<tr>
<td>Animals are family members. Dementia may play a role in some cases</td>
<td>Rescue-followed-by-adoption becomes rescue-only care</td>
<td>Superficial charm and charisma</td>
</tr>
<tr>
<td>Likely to be socially isolated</td>
<td>May have extensive network of enablers</td>
<td>Lacks guilt/remorse</td>
</tr>
<tr>
<td>Self-esteem linked to role of caregiver</td>
<td>Not as likely to live with the animals</td>
<td>Manipulative and cunning</td>
</tr>
</tbody>
</table>
Understanding the human aspects of animal hoarding will help the veterinarian and staff to comprehend the unusual presentation of the client who is an animal hoarder. A key understanding to dealing with these cases is recognizing that the animal hoarding is a symptom of a larger maladaptive psychological and social situation too complicated to be addressed by animal professionals alone.

Helpful resource:
- Reinisch, A. Understanding the human aspects of animal hoarding/Comprendre les aspects humains de l'accumulation d'animaux; The Canadian veterinary journal. La revue veterinaire canadienne 49(12):1211-4 · December 2008.

Special thanks to my friends and colleagues Dr. Gary Patronek, Jane Nathanson, LCSW and Dr. Arnie Arluke.
ANIMAL WELFARE LARGE ANIMAL

POULTRY WELFARE: HANDLING AND TRANSPORT OF BROILERS/
HOUSING OF LAYING HENS

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Broiler transport

Mortality/DOA
Mortality can occur at any stage (loading in the barn, during the journey and in the
holding barn/lairage) and some birds would have died, even if they had remained in
the barn. If death is quick and without suffering this is not a welfare issue. However, if
prolonged and associated with suffering, then it is a welfare concern. High mortality
rates indicate that the birds that survived will likely have suffered during the journey.

Main factors affecting mortality risk
Their relative importance will depend on a number of factors, but the thermal
conditions experienced by the birds is critical.

1. Health status of the flock
Health status of flocks is variable, but some broilers experience chronic illness, such as
congestive heart failure and ascites that cause mortality during and particularly
towards the end of the growing period. Some of the flock will continue to die from
these conditions during transportation and lairage, and their reduced physiological
fitness make them more susceptible to exposure to environmental extremes during
transportation. Some birds will die from acute conditions, such as sudden death
syndrome as they are more susceptible to stressors.

2. Physical injury during catching and loading
These injuries include fractures, dislocations, bruising, internal haemorrhage and crush
injuries. The number of birds injured and the severity of their injuries depend on the
manner in which the birds are handled and the type of handling system.
Types of handling systems.
   Loose crates: crates are brought into the barn or the birds carried out to the
   trailer and the birds are placed in the crate through a flap door.
   Modules: are brought into and out of barn using a fork-lift truck that loads the
   module onto the trailer. The module is placed close to the birds, reducing the
   carrying distance. The birds are placed into drawers or compartments in the
   module through larger openings resulting in less injury.

3. Thermal stress
Hyperthermia: occurs if the temperature is too hot and air humidity is too high (birds
have difficulty losing heat via evaporation of water from respiration when the humidity
is high). Death is likely to be preceded by a period of respiratory distress and metabolic
changes.
Hypothermia: occurs if the temperature is too cold and especially if the birds are wet
(birds lose heat via evaporation of water from their body and have reduced insulation). Death is likely to be preceded by huddling, a compact body posture, shivering, irregular breathing and cyanosis.

**Loading:** potential for exposure to:
- heat (temperature, sun, humidity and minimal ventilation)
- cold (temperature, wind, precipitation and reduced benefit from metabolic heat until sufficient birds have been loaded).

**Transport:** trailers do not provide a controlled environment
- manual adjustments to ventilation by adjusting external covers
- difficult to balance external protection from cold and wet with the need to remove build up of heat and humidity within the trailer
- ventilation mostly when the vehicle is moving (some trailers now have some fans)
- selection of stocking density during loading according to thermal conditions
- selection of time of day, consideration of weather forecast, adjustments to frequency and duration of stops during the journey.

**Lairage/holding barn:** difficult to provide ventilation to birds in crates/modules at high stocking densities.

**Stocking density**
- more birds per unit area/volume generate more metabolic heat and humidity this is a risk factor for hyperthermia
- fewer birds per unit area/volume generate less metabolic heat and this is beneficial in warm environments, but in cold conditions it is a risk factor for hypothermia.

**Trailer ventilation**
- Ventilation is required to remove heat and moisture produced by the birds.
- In warm conditions, the screens/side protection needs to be kept open to allow sufficient air movement to remove excessive heat and moisture.
- In cold conditions when screens/side protection are used to protect birds on the outside of the vehicle from cold external temperatures, precipitation and road spray, there may be insufficient ventilation in the ‘thermal core’ areas of the trailer and this has the potential to cause heat stress even though the external conditions are cold.

**Duration of handling, transport and lairage increase the risk of mortality**
- The longer the duration of each stage, the longer a bird has to die due to problems during the current stage or due to effects that started during a previous stage.
- Duration of feed and water restriction can affect risk of mortality on long journeys
  - Water should be available until loading. At high temperatures dehydration would increase the risk of heat stress.
  - Feed withdrawn 8-9 h before loading to reduce the risk of faecal contamination, might offer some advantage to reduce the risk of heat stress.
Long periods without feed can increase the risk of mortality due to reduced energy to cope with cold environments.

**Reductions in DOAs**
- More research, development, knowledge transfer, financial investment, quality control and legal enforcement
- Improve on-farm health and broiler genetics
- Improve catching/handling: supervision, modular systems and not loading wet birds
- Modify stocking density according to environmental temperature
- Improve trailer and lairage ventilation and temperature control
- Reduce journey and lairage durations.

**Further Reading:**
PAIN CONTROL FOR CASTRATION AND TAIL DOCKING IN LAMBS

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The sheep industry faces challenges in adopting optimal methods for minimising pain associated with castration and tail docking. There is considerable research on the effects of castration and tail docking on the behavioural and physiological signs of pain in lambs and on potential methods for controlling pain.

*Tail docking methods:* rubber ring, Burdizzo clamp and ring, surgery, and hot iron
*Castration methods:* rubber ring, Burdizzo clamp, Burdizzo clamp and ring, and surgery

**Pain control:** local anaesthetics (LA) and non-steroidal anti-inflammatory drugs (NSAIDs).

**Pain**

"An aversive sensory and emotional experience representing an awareness by the animal of damage or threat to the integrity of its tissues; it changes the animal’s physiology and behaviour to reduce or avoid damage, to reduce the likelihood of recurrence and to promote recovery" (Molony and Kent, 1997).

"Because direct measurement of subjective experiences is not possible, assessment of animal pain is of necessity a value judgement relying on physiological and behavioural indices to provide indirect evidence of this particular mental state” (Molony and Kent, 1997).

Measurements of the hypothalamic-pituitary-adrenocortical system and the sympathetic adrenomedullary system are used to assess distress. “They do not measure pain, but provide an indication of how unpleasant the experience is emotionally and physically” (Mellor and Stafford, 1999).

**Behavioural responses to pain from castration and tail docking**

Increased behavioural activity in an apparent attempt to reduce the pain, including restlessness, kicking, stamping, rolling, jumping, easing quarters and licking/biting at the site of damage.

Followed by a passive response to reduce the pain such as the adoption of an immobile stance or other abnormal posture to avoid or reduce stimulation of hyperalgesic tissues. The plasma cortisol response after castration and tail docking often follows the time course of changes in activity and posture. There are quantitative reductions in the behavioural and physiological responses to castration and tail docking if effective LA has been used to reduce nerve impulses from the site of the procedure. Castration and tail docking cause immediate tissue damage that initiates nerve impulses in pain pathways. Subsequent inflammation that develops after injury causes additional pain (Mellor and Stafford, 2000). Some methods e.g. rubber rings and Burdizzo clamp, cause an initial nerve response due to pressure on receptors. Once rubber rings constrict the blood vessels, this causes ischemic tissue damage and a second painful response occurs. A Burdizzo clamp crushes tissues including underlying nerves and this interrupts the transmission of sensory nerve impulses distal
to the crush. Surgical methods cause an initial acute pain response from the incision followed by the development of inflammation and associated pain. Thermal cautery causes an initial pain response, but if the pain receptors are destroyed by burning, this reduces the pain. Chronic pain can follow castration and tail docking.

**Methods for reducing pain associated with castration and tail docking**

- Improve existing methods
- Develop new methods that cause reduced pain
- Reduce pain by using LA and pre- and post-treatment analgesics
- If possible, perform the procedure on a lamb at an age that causes the least distress and tissue damage
- Use a method that involves minimal handling and restraint and returns lambs as quickly as possible to their dam
- Find an alternative management solution that avoids the painful procedure.

**Pharmacological methods to reduce pain responses**

- Multimodal analgesia i.e. inhibition of all the mechanisms responsible for pain
- Pre-emptive analgesia i.e. analgesia established before pain initiates
- Analgesia that lasts during the surgery
- Post-operative analgesia.

**Local anaesthetic (LA)** injections block nerve impulse transmission along affected nerves for the duration of action of the anaesthetic. They effectively prevent, reduce or delay acute pain resulting from procedures.

- Lidocaine (lignocaine) 2%: anaesthesia within 5-20 minutes and lasts for 45-90 minutes
- Bupivacaine 0.5%: slow onset and provides anaesthesia for 4-8 h.

**Analgesic (pain control) drugs**

- Xylazine: Sedative with analgesic and muscle relaxant effects
  50 µg/kg IM in 4-6 wk-old lambs, analgesia effective for at least 1 h.

**NSAIDs** reduce pain associated with inflammation

- Acts on the brain and spinal cord and damaged tissues
- Can provide both pre-emptive and prolonged post-operative analgesia
- Different types of NSAIDs have different effects
- Potential for toxicity: abomasal ulceration, renal toxicity. Administer for ≤ 3 days and keep within recommended dose.

**Tail docking**

**Rubber ring method**

- Physiological and behavioural evidence suggests acute pain for about 1 h. Some pathological evidence suggests that some pain might persist for several months.
- A rubber ring alone appears to more painful than using either rubber ring combined with a clamp or a hot iron alone. It might be more painful than using a surgical method.
- Use of LA at the site before the rubber ring is applied can reduce the signs of acute pain.
- The NSAID diclofenac reduces the plasma cortisol response, but does not
reduce behavioural signs of pain and is not as effective as LA.

**Combined ring and clamp method**
- Physiological and behavioural evidence suggests that a combined rubber ring and clamp method is acutely painful, but is less painful than using only a rubber ring.

**Surgical method**
- Physiological evidence suggests that the surgical method is acutely painful.

**Hot iron method**
- Physiological and behavioural responses are not as apparent after docking using a hot iron. LA has some benefit.

**Castration**
All methods of castration result in a response indicative of pain.

**Rubber ring method**
- Marked physiological and behavioural response indicative of acute pain.
- LA injected into the scrotal neck or the testis can reduce this response.
- NSAID: SC scrotal flunixin can reduce this response. SC scrotal meloxicam and SC carprofen (90 mins before) have some benefit, but they are not as effective and are not as effective as LA.
- Until the scrotum falls off after about 4 weeks, it remains swollen and appears to cause behavioural signs of discomfort.

**Clamp method**
- Physiological and behavioural response to clamp castration that is indicative of acute pain
- LA injected into the scrotal neck and cords can reduce the cortisol response, but not some of the behavioural responses.
- NSAID: IM diclofenac 20 mins before can effectively reduce behavioural responses.

**Clamp and ring method**
- The response to the combined clamp and ring method appears to be less than castration by rubber ring alone, but greater than castration by clamp alone.
- The clamp crushes each spermatic nerve and prevents the afferent transmission of nociceptor impulses from the testes as they become increasingly hypoxic and then anoxic after ring application.
- LA injected into the scrotal neck can reduce the cortisol and behavioural responses.

**Surgical method**
- There is a marked physiological and behavioural response indicative of acute pain
- LA does not always reduce the cortisol and behavioural responses. However, a spray-on topical anaesthetic (lignocaine and bupivacaine) can reduce pain responses.
Factors limiting use of pain control

• Drugs effective for pain mitigation in food animals are available, but their use in lambs constitutes an extra-label drug use.
• Analgesic drugs must be prescribed and dispensed by a veterinarian.
• Reluctance to dispense large volumes of drugs to producers for extra-label use.
• Producers see a practical difficulty in safely and effectively administering LA to lambs.
• Lack of knowledge by some producers and attitude of some producers to pain control.
• Low economic value of lambs and costs of pain control.
• Not required by law or assurance schemes.

Further Reading:

Health and welfare
Although the relationship between health/disease and welfare is self-evident to most veterinarians, it is not simply a matter of adding the word welfare to health. Although some definitions of health are so broad that they include welfare, the terms are not the same. If animals are healthy, it does not mean that other aspects of their welfare are always satisfactory, and it does not necessarily mean that all animals with ill-health are suffering. There can be confusion between and within disciplines on the exact nature of this relationship and on approaches to the meaning of animal welfare.

Approaches to animal welfare: Feelings, Physical fitness and Naturalness
Where does health fit?
• Firmly and primarily in the mental status/feelings approach: pain, feeling ill etc.
• Secondly within the physical fitness approach: physical and biological fitness: ability to undertake basic functions, opportunity to express positive emotions, growth and reproduction. However, increased productivity does not necessarily equate with good welfare.
• As a topic that urges caution in the adoption of a ‘natural’ approach to systems of production: e.g. increased exposure to some risk factors for disease and lack of optimal therapeutic and preventive treatments.

Welfare aspects of disease
• Welfare concerns related to animal health are not focused on the disease itself, but on how the animal experiences the consequences of disease.
• Inferences about how sick animals feel are based on behavioural, clinical or other observations of the animal, knowledge of its biology and our own experiences of pleasant and unpleasant feelings (Kirkwood, 2007).
• “to understand the suffering in disease one has to appreciate its pathophysiology and the feelings experienced by humans in comparable situations” (Gregory, 2004).

Effects of disease on welfare
• Diseases that are likely to cause pain and suffering are of particular concern.
• Diseased animals are likely to feel ill:
  • Inappetence
  • Thirst
  • Fever
  • Nausea
• Diseased animals might experience other negative emotional states
  • Fear because of disorientation or reduced ability to respond to perceived danger
  • Distress e.g. hypoxia from impaired oxygen supply.
• Some diseases can cause discomfort and reduce rest and sleep.
• A disease might also cause weakness reducing ability to compete for limited resources.
• Fever or prolonged immobility can increase heat loss and reduce ability to respond to the thermal environment.
• Reduced mobility, lameness or injury can prevent an animal from moving easily to gain access to resources such as food, water or a comfortable lying area.
• Some diseases can cause reduced fitness: emaciation, reduced function or loss of function, thereby reducing ability to perform normal biological functions and opportunity to experience pleasurable activities.
• However, if the disease reduces awareness e.g. hypoxaemia, hypothermia and drowsiness this can reduce the ability of the animal to be sufficiently aware or conscious and be able to experience sensory inputs and interpret them as noxious.

Example: Welfare implications of a cow with toxic mastitis

<table>
<thead>
<tr>
<th>Pain</th>
<th>Illness/sickness</th>
<th>Discomfort from treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>• signs of inflammation, heat, swelling, pain</td>
<td>• Fever</td>
<td>• ↑ risk of pain/distress</td>
</tr>
<tr>
<td>• hyperalgesia in infected quarter</td>
<td>• Anorexia</td>
<td>• ↑ risk of pain/distress/discomfort</td>
</tr>
<tr>
<td>↑ response to pressure on udder</td>
<td>• Weakness</td>
<td>• Handling and transport</td>
</tr>
<tr>
<td>secondary hyperalgesia in hind-legs</td>
<td>↓ maintenance behaviour</td>
<td>• ↑ risk of injury from other animals</td>
</tr>
<tr>
<td>↓ thermal threshold</td>
<td>• ↓ grooming</td>
<td>• ↑ fear/distress from social interactions</td>
</tr>
<tr>
<td>↑ response to pressure</td>
<td>• ↓ ruminating</td>
<td>• ↑ displacement at feeder</td>
</tr>
<tr>
<td>↑ sensitivity to touch during milking</td>
<td>• ↓ lying</td>
<td></td>
</tr>
<tr>
<td>↑ leg lifting, ↑ steps</td>
<td></td>
<td></td>
</tr>
<tr>
<td>restlessness/discomfort</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↑ no. of lying bouts</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↓ hind-leg weight shifting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↑ respiration rate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↑ heart rate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↑ serum cortisol concentration</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

References for mastitis example


Animal welfare assessments
Measurements and records of morbidity and mortality should form part of a welfare assessment of a livestock unit or system. It is essential that the veterinary profession is
involved in welfare assessment/assurance schemes.

**Treatment**
The main aim of treatment is to achieve rapid and permanent recovery and this helps to reduce suffering. However, attention must also be given to the alleviation of suffering and feelings associated with sickness during disease states.

**Factors affecting treatment decisions and type of action taken**
- *Severity of suffering*: should affect the promptness of any action, use of analgesia and whether euthanasia is considered.
- *Prognosis*: the likelihood of the animal recovering and having a good quality or productive life.
- *Attitude of the owner or carer*: influenced by many factors e.g. relationship with the animal and ethical views e.g. organic farming can restrict treatment options.
- *Cost of treatment*: can be expensive, some owners can be reluctant to pay for the services of a veterinarian or may delay too long before requesting a veterinarian.
- *Economics*: treatment options can be affected by the cost of
  - treatment in relation to the animal’s replacement value
  - loss of productivity associated with the disease
  - losses due to drug withdrawal times before products are safe to eat
  - slaughter value for human consumption
  - cost of euthanasia and carcase disposal
  - potential loss of premium associated with lack of conformity with any quality assurance scheme
  - biosecurity implications for the control of disease
  - controlling certain livestock production diseases as the costs may be higher than the economic gain and a certain amount of disease is tolerated.
- *Ability to provide appropriate care and treatment*: affected by factors such as
  - environmental conditions and facilities for handling, treatment and recovery
  - skill and dedication of the carer
  - availability of a suitable treatment, drug availability: e.g. not available, prescription only, extra label and potential for misuse.
- *Unpleasant side effects associated with the treatment*
- *Potential impact of treatment options on human health and environment*
- *Legal requirements:*
  - animal welfare legislation to prevent suffering caused by disease is present in many countries.
  - In Canada, no legal requirements other than in The Criminal Code, provincial regulations and ‘requirements’ in codes of practice.

**Examples of Health Requirements in NFACC Sheep Code**
- All producers must have a valid veterinary-client-patient relationship (VCPR) with a licensed veterinarian.
- Producers must have a flock health and welfare plan.
- Keep accurate and detailed animal health records.
• Producers must have the resources for and knowledge of the basics of care as stated in this Code and ensure such care is provided.
• Stockpeople must be familiar with and provide the basics of care as stated in this Code.
• The stockperson responsible for the monitoring and care of the sheep must be knowledgeable of basic sheep behaviour and common signs of illness and injury.
• Stockpeople must take responsibility to become competent across a range of health and welfare skills, including body condition scoring.
• All stockpeople must be knowledgeable of normal sheep behaviour and signs of illness, injury and disease; or work in conjunction with an experienced stockperson.
• Sheep must be monitored at intervals sufficient to ensure well-being in accordance with all sections of this Code.
• The frequency of inspection will depend on factors that affect sheep welfare at any particular time, ... and must be at least daily.
• Stockpeople must not cause, nor allow, unnecessary pain or unnecessary distress by leaving a sheep to suffer.
• Sick, injured, or diseased sheep must receive prompt treatment and nursing care, or be euthanized immediately. The treatment must be appropriate for the condition. If in doubt about the sheep’s health or the most effective treatment, consult a veterinarian without delay.
• For sick, injured, or diseased sheep that are not responding to treatment producers must, without delay, obtain veterinary advice on appropriate care and treatment or euthanize the sheep.
• Monitoring of sick, injured or diseased sheep must be appropriate for the condition and at least daily.

Further Reading:
Housing of Laying Hens

Several housing systems for laying hens
- Battery Cage
- Furnished/Enriched/Colony Cage
- Free run/Barn/Aviary/Litter
- Free range ± organic.

Campaign against battery cages and for cage-free systems.
Focus has been on behavioural restriction in cages. However, cage production systems offer benefits in terms of control of disease and injury.

“All housing systems have both costs and benefits for hen welfare” (Poultry (Layer) Code of Practice Scientific Committee, 2013). EU Research project, LayWel considered the welfare implications of changes in production systems for laying hens.

Conventional battery cages do not provide opportunities for hens to perch, dust bathe, use a nest box and do not provide sufficient space. Lack of exercise is associated with osteoporosis making the birds more susceptible to fractures during depopulation.

Furnished/enriched cages retain all of the benefits of a caged system i.e. low risks of disease, parasitism and predation, the small group size leads to a stable social hierarchy and lower risk of feather pecking and cannibalism. They provide hens with perches, nest box and a scratch mat with some litter. More space is provided and bone strength is improved. However, hens cannot undertake their full range of normal behaviour and opportunity to dust bathe is not fully provided.

Free run systems provide hens with a greater opportunity to express their full behavioural repertoire, especially foraging. Increased exercise leads to improved bone strength, but there is a greater risk of collisions leading to fractures. Large group sizes can be associated with increased risk of injury and mortality from feather pecking and cannibalism. There is a risk of internal parasites and disease from contact with droppings. In litter systems, air quality can be poor.

Free range systems cannot be used year-round in most parts of Canada as the winter is too cold for hens to be outside. There is also a major risk of predation.

Further Reading:


2016 Annual Convention Proceedings
NIAGARA FALLS 2016 CVMA CONVENTION

Sunday July 10, 2016

Welcome to the 2016 CVMA Convention in Niagara Falls. 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.

The Canadian Veterinary Medical Association has chosen The Personal as your group insurer. Start saving today with exclusive group rates and customized coverage!

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thePersonal
The veterinary visit can be thought of as a number of discrete chapters in a short story, each one key to the conclusion. In a well written short story, the development of the characters, the trajectory of the story and the place with which the story is told all combine to achieve a single purpose, the satisfactory conclusion. So it is particularly with the feline visit.

The opening chapter of this story begins at least 30-45 minutes before arriving at the veterinary establishment. In the home environment, the companion cat has a set of expectations and routine that he expects to be unchanging in the home range he has established whether entirely inside or inside and out. Suddenly and often with great drama, an object, a box, appears that has not been seen in the home range since the last, well-remembered crisis six months ago. What follows is a dramatic spike in the story as the owner chases the cat or finds the hiding place and snatches the resistant cat, shoving him into the box. No matter what happens next, a great change has occurred in this character, our cat’s physical state. The human has changed, too. Both are now highly aroused, but not in a good way. The human is by turns, anxious, angry, frustrated. The cat is anxious, frightened and having flashbacks about the last time a similar event occurred. One could imagine a form of post-traumatic stress.

The rest of the story will not go well for either character introduced so far or for the ones to come unless this opening is rewritten. The details that need to be revised are not complex, but are not known to the human character. The veterinary team who will not be introduced until later in this version of the story must be introduced before the initial crisis occurs. The character is instructed by the veterinary team to purchase the “right” (easily disassembled, sturdy, top loading) sort of carrier, and to leave the carrier out in the house not stowed in the garage where it accumulates debris and dead insect carcasses. This clean carrier is placed in an open area with bedding made of familiar soft material or a shirt from the favorite person and an open door. Highly valued treats are placed occasionally in the carrier and often it becomes a safe place to take a nap.

Occasional rides in the carrier are taken to the ATM, the coffee shop or post office when weather permits. Our human character becomes adept at carrying it without banging it around, good strength training and muscle control. The carrier and car are sprayed with feline facial pheromone to add to a sense of calm and familiarity. Our cat character is not fed before any of these trips to prevent car sickness and increase the likelihood that a high value treat will be a good reinforcement for any activity that comes later. Having learned from the veterinary team how to make the carrier and car ride a pleasant experience, the remainder of the story can develop more peaceably.

In the next chapter of this new version of the feline veterinary visit story, the cat is brought to this highly necessary check up for preservation of good health and well being because the human character understands how important this is. As they arrive, they are ushered immediately into an exam room. The team member who escorts them may put the carrier on the floor and open the door to it. By leaving the room to
the owner and cat for a few moments, the odds increase that the cat will come willingly out of the carrier and explore and become familiar with the room.

The next character, the veterinarian, enters the room quietly and introduces him or herself to the owner. They begin a conversation that involves the history of the patient and the context within which this cat’s story unfolds. As they talk, the veterinarian begins the examination from a distance, commenting upon what she observes regarding gait, mentation, coat quality, respiration and emotional state. If the requisite exit from the carrier has not occurred by the time the veterinarian has entered the room, the top half of the carrier should be removed if possible. In addition to all the other “tools” of the physical exam, a number of zip ties should be stocked in each room for safe reassembly of the often precariously assembled carrier.

The assessment of the previously mentioned characteristics can be made as the carrier is taken apart. The next chapter of the examination begins as the cat chooses the most comfortable place for this stranger to touch him. It may be in the bottom half of the carrier, on the exam table covered with a clean white fluffy towel or some other venue that seems appropriate to the cat. This chapter requires the veterinarian to be highly aware of changes to the emotional state of the patient and to adjust the tempo and the system examined accordingly. For example, many cats do not like to be touched on the ventrum, so abdominal palpation takes place later on. Most cats do not like to be stared at, therefore, the ophthalmic part of the exam is last or late in the visit. Each system is slowly and quietly examined. Talking to the client through each step as the systems are described.

Normal findings are also described as you work your way through the process in whatever order the cat is willing to allow. Watching for clues to arousal that indicate what is acceptable and not. Video and still photos of these steps will be used to exemplify good ideas for keeping the cat from a state of arousal that may end your ability to interact calmly and fully with the client. The goals here are twofold, to do a complete physical exam and to solidify the relationship you have with the client. A calm environment allows for conscientious completion of both of these goals.

When a cat becomes more aroused than can be examined without assistance, many techniques are available to make this part of the exam successful and as calm as possible. As few people as possible should be in the exam room at any given moment. When a new person is required, they are introduced to the client and their role explained. The goal is to prevent struggling. Towel techniques and other forms of gentle restraint will be shown in video and still photos. These concepts are hard to learn without visual examples.

The examination room should be the only part of the establishment an outpatient cat experiences. The “back” of the hospital is a place the owners are wary of and will often ask when their cat returns if it was their cat that was crying out. The patient has also become acclimated to the room and is at least somewhat familiar with that environment. To introduce the commotion of more people, animals, equipment, sounds and smells is likely to alter the patient from mildly to highly aroused. Therefore, the exam room should be fully equipped to perform most common procedures without moving in and out of the room. The client should be offered the
opportunity to leave the exam room if there is blood or urine to be collected or any other act that might cause the owner to be nervous or phobic.

Finally, despite the rewriting of this story to have a different trajectory than the usual feline visit to the veterinary practice, the level of arousal of the patient may be too high to fully examine. The client should receive a full explanation of why this is so, what is recommended and why sedation is more desirable than the struggle to restrain. Examples of language to use and appropriate drugs and doses will be discussed.

There are, again, three goals of the feline veterinary visit:
- To achieve a high quality physical examination and other appropriate diagnostic steps;
- To create and maintain a trusting relationship with the client; and
- To insure that the visit is successful enough that the client will be willing to return

Throughout this presentation as many video and still photo examples of techniques, activities and flow will be included.

Pharma Options for occasional use with highly aroused cats:

**Carry Out:**
- 100 mg Gabapentin 2 hours before visit
- 0.5-1.0 mg Alprazolam 1 hour before visit. Can be started 36 hours before visit given every 12 hours for 3 doses in very, very tough cats
- not a replacement for Feliway, disassembling carrier, dark, quiet room and soft voices
- additional sedation may be necessary for procedures

**Eat-In:**
- 0.02 mg/Kg Buprenorphine transmucosally
- Butorphanol 0.2 mg/# and 0.005 mg/# Dexdomitor +/- 1 mg/# Ketamine
- Hydromorphone/midazolam
- Again, no substitute for environmental consideration

Further Reading:
- Sharon L. Crowell-Davis, Terry M. Curtis, and Rebecca J. Knowles
- Social organization in the cat: A modern understanding
- Vicki Thayer, Deciphering the Cat: The medical History and Physical Examination
- 2011 AAFP/ISFM Feline-Friendly Handling Guidelines
- 2012 AAFP/ISFM Feline-Friendly Nursing Care Guidelines
In both people and companion animals, cachexia and sarcopenia are 2 important syndromes that occur in a variety of chronic diseases and aging, respectively. Although cachexia has been recognized in people for over 2,000 years, only recently has it become acknowledged as a common and detrimental finding that is associated with increased morbidity and mortality, and with this observation has come rapidly expanding interest and research. Both of these syndromes are becoming increasingly important in human and veterinary medicine because of their high prevalence and adverse clinical effects, and a better understanding of the mechanisms underlying these syndromes is critical for optimal patient care, whether human or veterinary.

Cachexia is defined as loss of weight and muscle mass secondary to chronic inflammation or disease. Sarcopenia, “poverty of flesh”, is an age-related loss of lean body mass. Sarcopenia is not caused by disease, is a gradual process and progresses with age. Loss of muscle can occur without fat loss or a decrease in Body Condition Score (BCS). Individual cats, particularly those with long coats or a history of obesity may appear to have a high BCS and yet be under muscled.

One of the keys to the management of cachexia and sarcopenia in dogs and cats is recognizing it in its earliest stages. To achieve this, BCS and Muscle Condition Score (MCS) must be consistently assessed. The goal for BCS in a healthy cat is 4–5 on a 9-point BCS scale. However, in certain diseases (eg, CHF, CKD), a slightly higher BCS may be desirable (ie, a BCS of 6–7/9), although further research is required to make specific recommendations. Even in animals with these diseases, obesity (BCS > 7/9) should be avoided.

The MCS differs from the BCS in that it specifically evaluates muscle mass. Evaluation of muscle mass includes visual examination and palpation of the head, scapulae, epaxial muscles over the thoracic and lumbar vertebrae, and pelvic bones.

In people, the loss of LBM has direct and deleterious effects on strength, immune function, wound healing, and survival. In fact, cachexia is an independent predictor of survival in people. The specific deleterious effects of muscle loss have not been as well studied in dogs and cats although there are studies associating thin body condition with decreased survival.

The weight loss that occurs in cachexia is unlike that seen in a healthy animal that loses weight. In a healthy animal that is receiving insufficient calories to meet requirements, metabolic adaptations allow fat to be used as the primary fuel source, thus preserving LBM. Conversely, acute and chronic diseases alter concentrations of a variety of mediators (eg, inflammatory cytokines, catecholamines, cortisol, insulin, glucagon), which then decrease the ability to make metabolic adaptations required to switch to fat utilization, and amino acids continue to be used as a primary source of energy. Therefore, muscle and LBM quickly are catabolized.

Numerous other factors can contribute to muscle and weight loss. Maintenance energy requirements vary with age, genetics, health status and gender (intact or
In presence of some disease states, maintenance energy requirements increase significantly. Decreased nutrient absorption is another possible mechanism for muscle loss in cachexia and sarcopenia. Studies in cats have shown decreased digestive ability. One investigator showed a reduced ability to digest protein in 20% of geriatric cats with about 33% having a significant reduction in ability to digest dietary fat. Micronutrient absorption, potassium, phosphorus, sodium, choline, B vitamins and Vitamin E, is also decreased.

Cats derive most of their energy requirements from protein and are metabolically less able to handle decreased amounts of protein and increased amounts of carbohydrates to maintain their energy requirements. Omnivores adapt to lower dietary protein by down regulation of their protein metabolism (protein sparing) but cats have been proven to be unable to make this physiologic adaptation. This preferential use of protein for energy can have clinical effects when cats are ill or anorectic as protein malnourishment can occur.

An important problem in cardiac and other forms of cachexia is a decreased calorie intake. The anorexia may be secondary to fatigue, dyspnea, or may be because of medication toxicity or alterations in appetite that often accompany CHF, cancer, and CKD in cats. Absolute food intake may decrease in animals with these diseases, but there also may be altered food preferences, cyclical appetite, and other issues that negatively affect overall food intake. Anorexia, for example, is present in 34–84% of dogs and cats with heart disease.

Increased energy requirements, alterations in nutrient absorption, and decreased energy intake all likely play important roles in the pathogenesis of cachexia by causing a net calorie deficit. However, a healthy animal that has a calorie deficit, either as a consequence of decreased food intake or increased energy requirements, would primarily lose fat. Therefore, these factors are not sufficient to explain the muscle and LBM loss and relative sparing of fat that are the hallmarks of cachexia and sarcopenia. This discrepancy suggests that metabolic alterations also are present.

Because of the important implications of cachexia and sarcopenia on morbidity and mortality in people, there is now extensive research into the prevention, diagnosis, and treatment of these syndromes. There are exciting opportunities for new and effective targets to decrease energy requirements, enhance energy intake, improve nutrient absorption, and modify metabolic alterations to prevent and even reverse the effects of both cachexia and sarcopenia.

A 2008 study on longevity in aging cats studied in a controlled environment for 5 years showed that all cats lost weight over time. However, cats supplemented with dietary antioxidants, prebiotic chicory root and a blend of Omega 3 and 6 fatty acids had a beneficial effect over a commercially fed diet alone or one supplemented only with antioxidants (Vitamin E and beta carotene). Cats in the fully supplemented group lost less weight, lived longer, had better LBM scores, improved fecal flora and fewer diseases.
In many cases, practical methods to help owners manage their animal’s appetite are critical to success. This is particularly important because anorexia is one of the most common contributing causes to an owner’s decision to euthanize his or her pet. Any issues that potentially can affect food intake should be addressed, whether physical or environmental. Dental disease, for example, can substantially impair food intake in an otherwise healthy or sick animal. Pain (eg, back or joint) can decrease an animal’s mobility and make it more difficult to secure adequate food intake. Environmental issues also can negatively impact food intake. Multi-pet households may impede the ability of an individual animal to gain access to food (eg, a more frail or timid animal may be crowded out from the food bowl). Stress often can increase for animals after diagnosis of any illness because of lifestyle changes (eg, medication administration, new foods), as well as increased stress on the part of the owner, which may be detected by the animal.

Once environmental issues are ruled out as a cause of weight loss, a nutritional screening is crucial. Older cats may need 5-6 g of protein/kg to prevent protein catabolism. Reduced digestive ability indicate that a high energy, highly digestible diet may be needed. Some kitten formulas may be more appropriate. Folate and cobalamin supplementation may be useful. Commercial cat foods vary quite widely in caloric density. Specific formulas should be investigated for adequacy.

Cachexia should be anticipated in animals with chronic diseases such as CHF, CKD, cancer, and others. Consistently evaluating MCS in all patients will help identify muscle loss at an early, mild stage in aging or ill animals, rather than waiting until muscle loss is moderate or severe, when it may be more difficult to successfully manage. Similarly, as animals age, muscle loss is likely to occur, even in healthy individuals. Therefore, muscle mass should be thoroughly evaluated in geriatric cats and dogs.

**Further Reading:**
- V. Paul Doria-Rose, DVM, and Janet M. Scarlett, DVM, PhD. Mortality rates and causes of death among emaciated cats. JAVMA Feb 2000, Vol. 216, No. 3, Pages 347-351
- [http://www.wsava.org/nutrition-toolkit](http://www.wsava.org/nutrition-toolkit)
Muscle Condition Score

Muscle condition score is assessed by visualization and palpation of the spine, scapulae, skull, and limbs of the legs. Muscle loss is typically first noted in the epaxial muscles on each side of the spine; muscle loss at other sites can be more variable. Muscle condition score is graded as normal, mild loss, moderate loss, or severe loss. Note that if animals can have significant muscle loss even if they are overweight (body condition score > 5/9). Conversely, animals can have a low body condition score (< 4/9) but have minimal muscle loss. Therefore, assessing both body condition score and muscle condition score on every animal at every visit is important. Palpation is especially important with mild muscle loss and in animals that are overweight. An example of each score is shown below.

Normal muscle mass

Mild muscle loss

Moderate muscle loss

Severe muscle loss

wsava.org

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The single biggest opportunity to grow small animal practices lies in the chronically underserved feline patient. With nearly 1500 participating practices there is emerging a body of knowledge that is critical for prospective practices to understand. Overcoming resistance on the part of unconvinced staff members and building a team to accomplish Cat Friendly designation are critical to accomplishing the establishment-wide changes that improve the experience that cats and their owners have. 83% of adopted cats are seen within the first year of adoption. Fewer than 50% of those return for regular veterinary care. Based upon HSUS adoption data, approximately 1.7 million cats are seen once and do not return. If 3000 practices became Cat Friendly Practices and the population was divided equally among them, there would be over 550 new patients per practice per year.

The Bayer Veterinary Care Usage Study 3 – Feline Findings focused on the population of cats and their owners that do not seek regular veterinary care and the views veterinarians and practice owners have of this underserved population. More than half of these 401 practice owners reported less than 70% of appointment times were filled. This represents a significant opportunity to better utilize veterinarians, professional staff and improve the work flow of the practice. When asked what could impact growth, the top two choices were increasing cat and dog visits. However, more than 50% had no method in place to monitor or evaluate the efficacy of their reminder systems. They did not, then, know whether their existing clients were being effectively encouraged to return to the practice.

While increasing cat visits was the second most cited way to grow and practices did not believe they would need to make many changes in the practice to increase visits, less than 1 in 5 had actually taken any steps to do so. More than 1 in 3 practice owners had no intention of implementing changes that would reduce stress for cats. Almost as many had made no attempt to train staff to make feline visits less stressful.

As research has shown, there are more companion cats than dogs. This should mean that veterinary practices see more cats than dogs, but the opposite is true. Many cat owners avoid veterinary visits for a variety of reasons. One major reason is that they are convinced that their cat hates the experience. Another is a lack of understanding of the need for preventive health care for creatures who seem to be independent and healthy. Clients also dislike the experience of the 30+ minutes that precede the visit during which conflict arises around the carrier, the traumatic experiences in the automobile and the disruption of routine that is so important to cats.

Cats seem to experience forceful handling by their otherwise predictable and beloved human as a betrayal of their trust. The car, carrier and veterinary establishment are unfamiliar to a creature who values a sense of control and familiar routine. As a veterinary team, we may not understand cats, their behavior cues, or normal behaviors. We may feel as if cats are more of a nuisance, take too much time or will
potentially cause injury. Our attitude is conveyed through approach, body language and other forms of communication apparent to both cats and their owners.

When a cat visit becomes disruptive we lose the fundamental opportunity to form the trusting relationship we need to have with our clients so that we can practice the best medicine. We lose the chance to calmly build rapport, establish trust and educate clients that is so crucial to our future with them and their cat.

The solution to declining cat visits, to resulting welfare issues, and to our ability to serve this patient population is to become cat friendly. We must create a practice culture in which the entire staff is committed to improving the experience of the feline patient and their owner. We must incorporate this into staff training and education, into the practice physical environment and into our plans for the future.

We must begin by educating our clients. By sharing with them our knowledge of the characteristics of the feline, we can teach them to have reasonable expectations, to understand the subtle signs of illness, and to prevent unacceptable behavior before it starts. By understanding the social groups in multiple cat households and how the social structure of cats has evolved, we can decrease the stress experienced by companion cats and their owners. We can teach breeders and “accidental” breeders to raise well-adjusted flexible, social kittens who will become wonderful cats for the people who adopt them. We can teach them how to lower the household stress by giving them a better understanding of their cats’ needs, sensory awareness, and perception of safety.

Our outreach has to be where our clients are, i.e., on the internet. We need lively web sites with important educational links. We need Facebook pages that are constantly updating and providing tips and entertaining topics that engage the clients before we meet them in the practice. Our educational efforts can result in happier households and healthier cats. Clients need to understand how cats prefer being alone when eating, why play is important and how cats interact with each other and humans.

The Bayer Brakke study showed that the recession did not cause the decline in visits but rather, unmasked a phenomenon that has been going on since the late 1990’s. This investigation made several recommendations regarding the goals that would improve cat visits including understanding the client household, addressing handling, communication, and safe transport.

Becoming cat friendly is not a construction project; it is seizing this opportunity to harness the talent and intellect of the staff to change behavior and attitudes. Cat friendly practices nurture relationships with clients by employing open communication and active listening. The staff becomes deeply committed to achieving skills in gentle handing, understanding behavior, and the unique medical and surgical needs of cat patients.

Change in the busy veterinary practice is difficult. One of the most important roles in affecting the practice culture is to assign a Cat Advocate to the project. That person is not responsible for doing all the work to become cat friendly but to make sure the work is done. Cat friendly is not a project, it is a cultural shift within the practice that
must be continually monitored and assessed. Education plans, physical changes, communication training are ongoing. By evaluating the cat and client’s experience from before the visit to the time they leave, we can establish a plan for improving that experience.

The first experience of the practice environment is often the first phone call. Using that contact to educate clients or potential clients about resources available to help make the pre-visit experience less stressful are key. Questions about carriers, automobile transport and other cats in the household can be satisfactorily answered. Resources can be sent in a variety of ways from web links, pdfs or written brochures.

The physical presence of other animals in the reception area is a key consideration for reduction of stress. Many strategies for reducing the negative effects can be implemented including, separate entrances, separate waiting areas, or “cat only” days. Voices should be kept low, sounds kept to a minimum, unnecessary odors like perfume or cologne avoided. Visual barriers can be employed to keep cats from seeing dogs or other cats. Staff members must be counseled not to look directly in the face/stare at cats.

In the exam room, the cat should be allowed to walk out of the carrier while the doctor is speaking calmly with the client. If the cat leaves the carrier, remove it from sight as it has become the most familiar thing in the room and the cat will be inclined to return to the carrier. If, after an appropriate time, the cat remains in the carrier unwilling to exit voluntarily, remove the lid of the carrier. This is far less stressful than other ways of removing the cat. Towels can be employed to help fearful cats remain calmer.

One of the most critical skills required for becoming cat friendly is to learn to read how cats communicate their emotional state through their body posture, facial expression and movement. Fear is the #1 cause of “bad behavior” in the veterinary environment. By learning to assess emotional states, we can avoid a fully aroused state that takes a cat 30-40 minutes to recover from. Cats leave behind a scent from their pads that indicates stress. Careful cleaning between appointments is not only important for disinfection but also to remove this form of communication between cats.

A cat examination room should contain all of the equipment and supplies needed to perform most outpatient services. By approaching in a calm manner, keeping the people in the room to a minimum, using quiet voices, towels for restraint if needed, and being flexible about the order the exam is performed in, there will be more successful experiences than usual. Scruffing or stretching should never be necessary and is counter-productive. In a calm environment the doctor can talk through the exam, making sure clients understand what is being done and the value and importance of the physical exam.

Many gentle techniques are described in the photos in the Cat Friendly Practice (CFP) program that offer ideas regarding restraint. The examination table may be the least necessary piece of equipment in the room. Cats may prefer the bottom of a carrier, a lap, a chair or the floor and should be accommodated. Moving cats by picking them up adds a level of stress to an already fearful cat. The reflex response to fear is to flee thus
maintaining all four feet on the floor is very important to a sense of control and reassurance. Every effort should be made to avoid taking the cat to the “back” of the hospital. The exam room is now somewhat familiar. To move to a foreign space offers new stressors, different smells, bright lights, more animals, people, and noises.

Cats who must be admitted to the hospital have an increased need for a sense of familiar comforts. This can be provided by asking the client to bring known items from home; bedding, brushes, food, bowls or toys. Soft bedding, a place to hide and gentle nursing techniques are critical. For cats who enjoy social interaction, petting, brushing and other forms of interaction can be employed.

The cat ward should be separate from dogs and other animals, big enough so that cats cannot see one another. Cages should not face each other. Cats passing each other for treatment or discharge should be shielded from view. When removing a cat from a hospital enclosure, allow the cat to come forward or use bedding, towels or the bottom of the carrier to slide the patient forward. Do not loom about the cat or block the light.

The entire inventory of equipment, instrumentation, physical facility should be examined to make sure they are appropriately sized for the feline patient.

The Cat Friendly Practice program provides veterinary practices with ALL of the information, tools and techniques for becoming cat friendly. There are ten areas to evaluate with resources to achieve compliance with all of them. This program will continue to evolve and grow as new phases are implemented. The next of these will be Preventative Health Care. To participate the practice must have one AAFP member, identify the Cat Advocate for the practice and use the website, manual and checklist to achieve either gold or silver CFP status.

In recognition of this effort, the program provides you with a toolkit to market your practice as one that has made this significant effort and to distinguish yours from other practices that have not. A searchable website will allow clients to look for Cat Friendly Practices in their region. Beginning in the fourth quarter of 2012, the AAFP began a national consumer awareness campaign to encourage cat owners to seek a Cat Friendly Practice. Refinements and additions to this campaign will continue.

As we discuss each aspect of the program, specific examples of creative and innovative methods CFP practices used to overcome barriers to certification, to market themselves and to significantly benefit by the effort made to implement the program will be discussed. Almost every CFP practice currently certified plans to renew their certification when the two -year membership period expires. Recertification is intended to reinforce the CFP concepts and to introduce new tools and resources made available since the program began.

The CFP task force and internal team are continually analyzing the feedback from member practices, both designated and working on becoming so. Based upon that feedback there are videos directed at both the veterinary team and clients to demonstrate techniques important to improving the experience. New tools are being developed through out the year to meet their needs for social media, staff meetings,
owner education and staff development. In 2015, the task force and AAFP board created a strategic plan for the future of Cat Friendly Practice. It is our intention to keep evolving the program to add value to participating practices, to create tools and resources for practices to attract cat owners and to drive cat owners to practices that participate.
Lower urinary tract signs (LUTS) – dysuria, periumuria, pollakiuria and stranguria – are a common reason pet cats are brought to veterinary practices. When presented with a cat with these signs clinicians need to know whether this is the first episode or whether it is a chronic, recurrent disease as well as what other health problems the cat may have. Armed with this information an appropriate diagnostic plan can be made.

Cats may have multiple reasons for their clinical signs as well as other medical conditions and environmental requirements that need to be addressed. For example, Buffington et al. have presented evidence that some cats with severe, chronic LUTS seem to have a functional rather than a structural lower urinary tract disorder and that periumuria can occur in apparently healthy cats exposed to stressful circumstances. There is significant overlap at the present time among treatment recommendations for some LUT disorders particularly with regard to ensuring that the patient’s environmental needs are met.

Severe chronic idiopathic LUTS has been described as a naturally occurring model of interstitial cystitis in women. Interstitial cystitis (IC) has been defined as a disease of chronic irritative voiding signs, sterile and cytologically negative urine and cystoscopic observation of submucosal petechial hemorrhages. The same description in which cystoscopy was not performed in cats but in which other appropriate diagnostic procedures did not identify a cause became defined as Feline Interstitial Cystitis (FIC).

In addition to epithelial abnormalities identified in the bladder of cats with FIC, investigators found significant alterations in components of acetylcholine synthesis and release in the esophageal mucosa from cats with FIC. This suggested that changes in the nonneuronal cholinergic system may contribute to alterations in cell-to-cell contacts and possibly communication with underlying cells that may, in turn, contribute to changes in sensory function and visceral hyperalgesia. Differences in sensory neuron anatomy and physiology also are present in cats with FIC suggesting a more widespread abnormality of sensory neuron function. The acoustic startle response is a reflex motor protective response to a perceived threat. It is a brainstem reflex response to unexpected auditory stimuli and is increased in cats with FIC.

Differences in sympathetic nervous system function have also been identified in cats with FIC. Among them are changes in the brain stem in the region associated with the most important source of norepinephrine in cats and humans. It is involved in such brain functions as vigilance, arousal and analgesia and mediates the visceral response to stress. Other changes in brainstem help to explain the waxing and waning course of symptoms and the aggravation of signs by environment stressors. Some cats with FIC appear to have abnormalities in the hypothalamic-pituitary-adrenal axis such that there is a decrease in serum cortisol secretion compared with healthy cats. Adrenal glands in these cats were grossly smaller in cats with FIC when compared to healthy cats.
Cats with FIC often have variable combinations of comorbid disorders such as behavioral, endocrine, cardiovascular and GI problems. External stressors appear to exacerbate clinical signs of these disorders. Many human beings with IC suffer from variable combinations of comorbid disorders as well. These appear to have no consistent pattern of onset and so cannot be attributed to LUTS but rather may be some common disorder affecting more than one organ which then responds in its own way.

Ongoing research in both humans and cats with chronic LUTS has begun to include a more comprehensive evaluation of the entire patient. Nosology is defined as the classification of diseases. Until a better understanding of the larger picture of cats presenting with LUTS, naming this constellation of symptoms and organs systems involved should remain vague and not reflect only LUTS. Dr. Buffington has suggested “Pandora’s Syndrome” He and his colleagues, Drs. Westropp and Chew propose tentative criteria for diagnosis of Pandora syndrome:

1. Presence of clinical signs referable to other organ systems in addition to chronic idiopathic signs for which the patient is being evaluated
2. Evidence of early adverse experience (e.g abandonment, orphaning) and which may differ by individual
3. Waxing and waning of severity of clinical signs with events that (presumably) activate the central stress response system
4. Resolution of signs with effective multimodal environmental modification

Whatever the eventual name, restricting the description of these patients to their LUTS does not capture all of the currently recognized features of the syndrome. A more comprehensive evaluation of cats with these and other chronic idiopathic signs may result in a more complete diagnosis and lead to additional treatment approaches that may improve outcomes. For example, the relationship between the environment and health is quadratic rather than linear, with both deficient and threatening environment increasing the risk of poor health outcomes.

Individual patients presenting with chronic LUTS benefit by a more comprehensive evaluation to elucidate the effect on risk for Pandora syndrome. Included in this history should be:

- Where the cat was obtained
- Any other health or behavior problems that may be present
- Structure of the cat’s environment – amount of time indoors, activity level, availability and management of resources, other cats in the home, people living with the cat.
- Presence of signs referable to other organ systems
- Perceived allergic responses to skin, lung or GI tract
- Any unusual or problematic behaviors

The physical exam should be performed with evaluation of the lower urinary tract last to avoid being distracted and missing other abnormalities such as over-grooming, obesity, acne, cardiac abnormalities or GI tract issues.

For an initial episode in an apparently healthy, young unobstructed patient, the most likely explanation is either a sickness behavior in an otherwise healthy cat or acute idiopathic LUTS. After ruling out other causes of LUTS, the client should be counseled
regarding individually tailored multimodal environmental modification (MEMO) to make sure the cat’s environmental needs are being met. The client can also be taught to look for other signs of sickness behaviors and to evaluate response to MEMO for adequacy of accommodation.
Table 1. Forms used as part of the evaluation of cats presented the Ohio State University Veterinary Medical Center for evaluation of chronic lower urinary tract signs. These forms have not been formally validated beyond their face validity for cases in the authors’ practice area. They are offered as an example of an instrument that could be developed and validated for broader use.

**Cat and Client History Form**

Cat’s name__________________ Owner name__________________________
Date_______________________

Contact information: Telephone: ☐ ☐ E-mail: ☐ ☐ ☐ ☐ Please check preferred method of contact

Cat Information: Breed ________ Color ________ Date of Birth ________ Weight ________ ☐ lb ☐ kg

Owned for? ______ years ______ months; ☐ M ☐ F ☐ Neutered? If yes, date: ________

( month/ year)

Declawed? ☐ N ☐ Y If yes, Front only ☐ All four paws ☐

Body Condition (please check box that looks most like your cat):

<table>
<thead>
<tr>
<th>☐ Skinny</th>
<th>☐ Lean</th>
<th>☐ Moderate</th>
<th>☐ Stout</th>
<th>☐ Obese</th>
</tr>
</thead>
<tbody>
<tr>
<td>![Skinny Cat]</td>
<td>![Lean Cat]</td>
<td>![Moderate Cat]</td>
<td>![Stout Cat]</td>
<td>![Obese Cat]</td>
</tr>
</tbody>
</table>

Please check the boxes that best apply to your cat:

Diet: (please be as specific as you can, eg, Buckeye Best (company) Adult Chicken and Rice (flavor)

Wet food: name_________________________

| ☐ None | ☐ 25% | ☐ 50% | ☐ 75% | ☐ 100% |

Dry food: name_________________________

| ☐ None | ☐ 25% | ☐ 50% | ☐ 75% | ☐ 100% |

How many hours each day, on average: does your cat spend indoors?

| ☐ Indoor only | ☐ 12-24 | ☐ 6-12 |

| ☐ 0-6 | Is time outside supervised? ☐ Yes ☐ No |

Page 360
If you have more than one cat, what is their relationship? □ Not related
□ Littermate □ Sibling □ Parent-Offspring □ Other
(____________________)

Where did you obtain your cat (source)?
□ Shelter □ Offspring from a pet I already own(ed)
□ Purchased from a friend □ Gift
□ Purchased from a breeder □ Purchased from a pet shop
□ Stray/orphan □ Other __________________________

Does your cat frequently (please check all that apply):
□ Try to escape
□ Pace at outside doors
□ Cry at outside doors
□ Hide
□ Act fearful
□ Act friendly
□ Follow owners around the home
□ Destroy things when left alone
□ Act ‘depressed’ (little interest in feeding, grooming, environment, etc.)

Housing (______): Apartment: □ studio □ 1-2 bedrooms □ 3 or more bedrooms,
Zip Code
House: □ attached/twin duplex □ attached, 3 or more units, □ single
□ other__________________

Total Cats_____ Total Dogs_____ Other Pets______________

Other People_____________
Please help us understand what your cat does around the house by placing a check (✓) in the box next to each behavior that best describes how commonly your cat does each of the behaviors described below.

<table>
<thead>
<tr>
<th>Does your cat:</th>
<th>All of the time</th>
<th>Most of the time</th>
<th>A good Bit of the Time</th>
<th>Some of the time</th>
<th>A little bit of the time</th>
<th>None of the Time</th>
<th>Does Not Apply</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leave household articles (furniture, drapes, clothing, plants, etc) alone</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Eat small amounts calmly at intervals throughout the day</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Drink small amounts calmly at intervals throughout the day</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Use the litterbox</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Get along with people in the home</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Get along with other pets in the home</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Remain calm when left alone</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Stay relaxed during normal, everyday handling (grooming, petting)</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Calm down quickly if startled or excited</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>React calmly to everyday events (telephone or doorbell ringing)</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Play well with people</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Play well with other family cats</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Show affection without acting clingy or annoying</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Tolerate confinement in a carrier (including travel)</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>
Groom entire body calmly

Use scratching posts

Play with toys

Comments; anything else your cat regularly does or does not do that you think might be helpful for us to know about?

________________________________________________________________________

________________________________________________________________________

________________________________________________________________________

2. Health History

The cat’s condition today is ...........................................

.............................................................................................

Previous illnesses or surgeries ...........................................

.............................................................................................

Current medications .........................................................

.............................................................................................

*Directions*: For items below, please use the following choices to describe how many times you have seen your pet experience the symptom, adding comments/explanation as appropriate.

*Score =

0 = I have NEVER seen it
1 = I have seen it at least ONCE
2 = I see it at least ONCE per YEAR
3 = I see it at least ONCE per MONTH
4 = I see it at least ONCE per WEEK
5 = I see it DAILY
Please check any of the following diseases your cat has been diagnosed with:

- □ Periodontal (dental) disease
- □ Asthma
- □ Inflammatory bowel disease
- □ Skin disease
- □ Allergies
- □ Diabetes mellitus
- □ Cardiomyopathy (heart problems)
- □ Obesity
- □ Other

<table>
<thead>
<tr>
<th>Scoring</th>
<th>How often does your cat:</th>
<th>Comments/Explanations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cough</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sneeze</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have difficulty breathing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stop eating</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vomit □ food □ hair □ bile □ other</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have hairballs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have diarrhea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have constipation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Defecate outside the litter box</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strain to urinate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have frequent attempts to urinate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urinate outside the litter box</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have blood in the urine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spray urine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Groom more than cats usually do</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shed more than cats usually do</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scratch him/herself more than cats usually do</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have discharge from eyes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seem fearful</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seem to need a great deal of contact or attention</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Destroy things when left alone</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

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Household Resource Checklist

The following questions ask about your cat's resources so we can learn more about the environment your cat(s) live in. Please ✓ DK if you don’t know, NA if it does not apply, or Yes or No after each question. If you have more than one cat, please answer for all cats. Resources (food, water, litter and resting areas) for each cat are assumed to be out of (cat) sight of each other, such as around a corner or in another room. If they are in sight of each other, please answer No.

<table>
<thead>
<tr>
<th>Space</th>
<th>DK</th>
<th>NA</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Each cat has its own resting area in a convenient location that provides some privacy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Resting areas are located such that another animal cannot sneak up on the cat while it rests</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 Resting areas are located away from appliances or air ducts that could come on unexpectedly (machinery) while the cat rests</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 Perches are provided so each cat can look down on its surroundings</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 Each cat can move about freely, explore, climb, stretch, and play if it chooses to</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 Each cat has the opportunity to move to a warmer or cooler area if it chooses to</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 A radio or TV is left playing when the cat is home alone</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Food and water</th>
<th>DK</th>
<th>NA</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 Each cat has its own food bowl</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 Each cat has its own water bowl</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 Bowls are located in a convenient location to provide privacy while the cat eats or drinks</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11 Bowls are located such that other animals cannot sneak up on the cat while it eats or drinks</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12 Bowls are washed regularly (at least weekly) with a mild detergent</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13 Bowls are located away from machinery</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Litter boxes
### Litter boxes (continued)

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th>D K</th>
<th>N A</th>
<th>Ye s</th>
<th>N o</th>
</tr>
</thead>
<tbody>
<tr>
<td>22</td>
<td>Unscented clumping litter is used</td>
<td></td>
<td></td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>23</td>
<td>A different brand or type of litter is purchased infrequently (less than monthly)</td>
<td></td>
<td></td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>24</td>
<td>If a different type of litter is provided, it is put in a separate box so the cat can choose to use it (or not) if it wants to</td>
<td></td>
<td></td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
</tbody>
</table>

### Social contact

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th>D K</th>
<th>N A</th>
<th>Ye s</th>
<th>N o</th>
</tr>
</thead>
<tbody>
<tr>
<td>25</td>
<td>Each cat has the opportunity to play with other animals or the owner if it chooses to on a daily basis</td>
<td></td>
<td></td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>26</td>
<td>Each cat has the option to disengage from other animals or people in the household at all times</td>
<td></td>
<td></td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>27</td>
<td>Do any cats interact with outdoor cats through windows?</td>
<td></td>
<td></td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
</tbody>
</table>

### Body care and activity

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th>D K</th>
<th>N A</th>
<th>Ye s</th>
<th>N o</th>
</tr>
</thead>
<tbody>
<tr>
<td>28</td>
<td>Horizontal scratching posts are provided</td>
<td></td>
<td></td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>29</td>
<td>Vertical scratching posts are provided</td>
<td></td>
<td></td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td></td>
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<td>---</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>Chew items (eg, cat-safe grasses) are provided</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>2</td>
<td>Toys to chase that mimic quickly moving prey are provided</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>3</td>
<td>Toys that can be picked up, carried, and tossed in the air are provided</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>Toys are rotated on a regular basis (at least weekly) to provide novelty</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

If you have additional comments on any of the questions, please write them below, including the question #.

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

By submitting this form, you agree that anonymous information from it may be used for cat health-related research.
ACUTE AND CHRONIC PANCREATITIS

Elizabeth J. Colleran DVM, MS Diplomate ABVP feline
American Association of Feline Practitioners

Pancreatitis is an inflammatory disease of the exocrine pancreas. It can be divided into acute and chronic types based upon histological findings. Two main forms have been described. Neutrophilic inflammation and varying amounts of pancreatic acinar cell and peripancreatic fat necrosis characterize acute pancreatitis. Chronic pancreatitis is characterized by lymphocytic inflammation, fibrosis and acinar atrophy. While the cell types involved differ in this description, they appear to represent, in some studies, different points on a continuum of disease.

Diagnosis of both forms is difficult. There may be comorbidities that complicate signs. Clinical signs may be vague or mild. The diagnostic tools like imaging or clinical tests can lack sensitivity and specificity. Biopsy samples may be difficult to interpret or unavailable for many reasons. There appears to be a strong association in several studies between pancreatitis, inflammatory bowel disease (IBD) and cholangitis, giving rise to the term “triaditis”. This may be partially explained by the proximity of the common bile duct and major pancreatic duct in the duodenal papilla. Enteric bacteria were found in >1/3 of cases supporting the suspicion of a relationship between pancreatitis and the translocation of bacteria from the gut. Vomiting, a common sign in cats with IBD or cholangitis may also raise intraluminal pressure and further increase the risk of pancreaticobiliary reflux. The relationship between cholangitis and pancreatitis has recently been challenged however, though its relationship with IBD has not.

Ischemia is another recognized cause of acute pancreatitis. Inadvertent compression or ligature and hypotension during surgery can cause ischemia of the pancreas. Careful surgical technique and anesthetic monitoring prevent these events from occurring. The pancreas can be the cause of ischemia if fibrosis, edema or inflammation compromise pancreatic blood flow. Other causes like infectious agents, hypercalcemia, drug reactions and nutritional imbalances have been reported but are rare. Most commonly pancreatitis is considered idiopathic as no obvious cause can be found.

Serum feline pancreatic lipase immunoreactivity (fPLI) is the most recent addition to laboratory tests seeking a useful diagnostic ante-mortem test for feline pancreatitis. There are two tests, developed by the same laboratory. SpecfPLI is a quantitative test for which concentrations > 5.3 are consistent with pancreatitis. A grey zone is found from 3.5-5.3 ug/l and is notable on the Spec test and on the Snap fPLI test, which is a semi-quantitative test. A positive Snap fPLI includes the grey zone when it is positive so results should be confirmed by Spec fPLI. The sensitivity of the Spec fPLI is still without adequate data. Moderate to severe pancreatitis was 100% sensitive in one study but much lower, 54%, for mild pancreatitis based upon histopathology. However, the number of patients was small and there was some bias evident on patient selection for histopathology. More studies are needed to properly evaluate sensitivity and specificity of SpecfPLI. Snap fPLI has not been independently validated. Importantly, fibrosis or atrophy from long-standing chronic pancreatitis would not be expected to increase fPLI.
Other abnormal laboratory findings have been observed but are not diagnostic as well. From 26-55% of cats have a normocytic, normochromic nonregenerative or regenerative anemia. Less than half have a leukocytosis. Leukopenia may be present and has a poorer prognosis. Other hematological findings are non-specific and cannot distinguish between acute, chronic or suppurative pancreatitis. Biochemistry abnormal values are often present but are not specific for pancreatitis and may represent comorbid conditions with pancreatitis.

Abdominal radiographs are may be suggestive of cranial loss of serosal detail or a mass effect but are largely useful to rule out concomitant conditions like intestinal obstruction. Ultrasound is relatively specific in differentiating pancreatitis from other GI disease but cannot differentiate between acute and chronic forms. Hyperechoic pancreas, hyperechoic peripancreatic adipose or abdominal effusion is relatively specific for pancreatitis in cats. Mild forms of pancreatitis are more difficult to discern than moderate to severe forms on ultrasound. In some cats, the pancreas is more difficult to detect and is dependent on operator experience.

The use of endosonography may improve the general visualization but did not alter the diagnosis of pancreatitis in one study. Ultrasound is still recommended for diagnosis of pancreatitis and will reveal other abnormal findings such as pancreatic masses, cysts or stones. Computed tomography has not been helpful and is not recommended for diagnosis. Magnetic resonance imaging is the modality of choice in humans and may be helpful in cats.

Histopathology remains the gold standard for ante-mortem diagnosis though there are limitations to this as well. Cats with severe pancreatitis are poor candidates for anesthesia. Even for those patients stable enough, the results may not alter treatment planning and patient management. Patients undergoing laparotomy or laparoscopy for other reasons should have the pancreas biopsied. Focal lesions may be visible as well as more generalized changes that will guide sample collection. Multiple samples are recommended as lesions can be geographically distributed or very mild and difficult to discern. Mild changes may not explain the patient’s clinical signs as well.

Despite the challenges of diagnosis, pancreatitis is an important condition. Anorexia and weight loss found with pancreatitis can cause concurrent hepatic lipidosis. Several studies have shown the relationship between Diabetes Mellitus (DM) and pancreatitis. Other concurrent diseases can be complicated by the presence of pancreatic inflammation most notably IBD. End-stage CP can result in exocrine pancreatic insufficiency.

Management of pancreatitis is comprised of three main aspects: nutrition and antiemetic therapy, fluid and electrolyte correction and analgesia. A high protein, low carbohydrate, moderate fat diet is the recommended formulation. While fasting is not recommended, gradual reintroduction of food should be instituted to avoid the electrolyte and other disturbances that occur with refeeding syndrome. Though nausea may be difficult to discern, it should be treated to insure adequate intake of food. NK-1 receptor antagonist maropitant and 5HT3 antagonists are beneficial. Maropitant may also relieve some of the pain associated with pancreatitis.
Cobalamin deficiency is common in cats and should be addressed with B12 injections weekly for 6 weeks and every 1-2 months thereafter. Appetite improvements with the use of cobalamin supplementation have been reported.

If voluntary food intake is not rapidly restored a nasoesophageal tube for short-term use or an esophagostomy or gastrostomy tube may be required. The goal of a nasoesophageal tube is for stabilization until anesthetic risk is lowered adequately to permit a more lasting tube to be placed. In the case of severe malnutrition and persistent anorexia, partial parental nutrition along with some enteral nutrition has been shown to maintain gut wall barrier function in humans.

Vomiting, anorexia and diarrhea can lead to severe dehydration and electrolyte disturbances. Hypokalemia and hypocalcemia are no uncommon. Aggressive fluid therapy is required to correct pancreatic hypoperfusion.

Pain is a common feature of pancreatitis though difficult to evaluate in cats. Buprenorphine, oxymorphone or fentanyl may be good choices.

Comorbidities must be treated at the same time, insulin for DM, therapy for diabetic ketoacidosis, cholangitis or inflammatory bowel disease. Plasma (20ml/kg i.v.) or colloid (10-20ml/kg/day i.v.). may be indicated in the presence of hypoproteinemia or shock. Colloids such as dextran 70 and hetastarch may also have antithrombotic effects that help maintain the microcirculation.

Prophylactic broad-spectrum antibiotics (e.g. amoxicillin ± enrofloxacin depending on severity) may be warranted in patients with shock, fever, diabetes mellitus or evidence of breakdown of the GI barrier. Bacterial translocation has been demonstrated in experimental feline pancreatitis using distinct E.coli placed in the colon, and other sites e.g. bile, and colonization was prevented with cefotaxime (50mg/kg TID). A recent study revealed that bacterial infection is present in the pancreas of 35% (11/31) of cats with moderate to severe pancreatitis. The high frequency of infection (71%, 5/7) in acute necrotizing and supplicative pancreatitis may be linked to the poor prognosis associated with this form of pancreatitis. These localization and type of intrapancreatic bacteria suggests translocation of enteric bacteria is a likely source of infection Coagulation abnormalities should be pursued and treatment with parenteral vitamin K can be assessed. Where a coagulopathy e.g. DIC, or hypoproteinemia are present, or the patient with pancreatitis is deteriorating, fresh frozen plasma (10-20 ml/kg) may be beneficial in alleviating the coagulopathy, hypoproteinemia and restoring a more normal protease-antiprotease balance. The administration of heparin (75-150 IU/kg TID) may be potentially useful in ameliorating DIC, promoting adequate microcirculation in the pancreas and clearing lipemic serum. In experimental pancreatitis isovolemic rehydration with dextran has also been shown to promote pancreatic microcirculation in dogs. A dopamine infusion (5μg/kg/min) had a protective effect when administered to cats within 12 hrs. of induction of experimental pancreatitis. H1 and H2- antagonists blocked the progression of edematous to hemorrhagic pancreatitis in experimental cats and may be beneficial in patients.
Oral pancreatic enzyme extracts have been reported to reduce pain in humans with chronic pancreatitis, though this is controversial. The presence of a protease mediated negative feedback system has not been described in cats.
The interrelationship between calcium, phosphorus, parathyroid hormone, activated vitamin D and fibroblast growth factor has a profound impact on the progression of chronic kidney disease (CKD) in dogs and cats. Beneficial effects of calcitriol treatment during CKD have traditionally been attributed to regulation of parathyroid hormone (PTH). New analysis of information emphasize direct renoprotective actions independent of PTH and calcium. It is now apparent that calcitriol exerts an important effect on renal tubular Vitamin D which may be important in maintaining adequate circulating Vitamin D. This in turn may be vital for important actions of Vitamin D on peripheral tissue. Limited information is available reporting the benefit of calcitriol treatment in dogs and cats with CKD. However, a survival benefit has been shown in dogs with CKD treated with calcitriol compared to placebo. The concentrations of circulating Vitamin D have recently been shown to be low in people and dogs with CKD and are related to survival in people. In 2015, there will be compelling data regarding the benefit of calcitriol use in cats with CKD.

Rather than focus on the dearth of evidence for several forms of intervention, this talk will focus on a historic review of the use of dietary therapy, phosphorus binding agents and calcitriol over a ten year period. These are all client-owner cats. Therefore, these are not randomized, blinded, controlled studies. Rather, these cases are a demonstration of practical interventions that have prolonged good quality of life in cats who may not have agreed to all of the recommendations made in the literature.

In assessing renal disease in cats, the most sensitive indicator is the loss of urine concentrating ability. The use of an early morning urine sample to assess urine specific gravity (USG) may help to counter effects of diet or drugs on a tested sample. Using the International Renal Interest Society (IRIS) values for classification of renal disease can be helpful in planning therapy. In some classifications IRIS 2 is divided in to 2a (Cr. 1.6-2.4 mg/dl) and 2b (2.5-2.8 mg/dl). In our practices the classification of 2a with USG less than 1.030 eating a mostly dry diet formula, for example, are started on treatment for chronic progressive renal disease (CPRD). Early intervention prolongs quality of life, good body condition score and wellbeing in a number of key ways. We use ultrasound guided cystocentesis in every cat from whom urine is obtained. This allows a quick early morning visit by the owner, a sterile sample for culture if indicated, a full assessment of the appearance of the urinary bladder and observation of complications such as uroliths.

One of the most frustrating aspects of treating this and any condition requiring lifelong therapy in cats is the difficulty clients have complying with our recommendations. Cats resist contact or intervention they haven’t agreed to and clients want to preserve the relationship they have with their cat, often at the expense of appropriate therapy. It is essential then to choose the most effective forms of therapy, to provide options when resistance is experienced and to communicate a willingness to the client to assist in preserving the relationship they have with their beloved cat.
While it has been shown that dietary modification has the most positive long-term effect on outcome, the relationship between survival and protein restriction or the attendant restriction of phosphorus has yet to be illuminated fully. Strong evidence, however, supports dietary phosphorus restriction in animals with kidney disease. Serum phosphorus is an independent predictor of disease in cats with chronic kidney disease. Cats with induced renal disease fed phosphorus-restricted diets had less severe histological renal changes than cats fed normal diets.

Phosphate retention and hyperphosphatemia are primarily due to impaired renal phosphate excretion. If renal function is normal, clinically significant hyperphosphatemia seldom develops. In the early stages of CPRD increased levels of PTH can keep serum phosphorus within the reference range by decreasing expression of the sodium-phosphate transport system in the proximal tubule resulting in increased urine phosphate excretion. This allows for normalization of serum phosphorus at the expense of hyperparathyroidism.

As cats are quite specific about preferences in taste, texture and flavor, the use of renal formulated diets may not always be possible. Alternatives may not have been thoroughly tested to the extent that prescription diets are but the truth of the statement “It is more important THAT he eats than WHAT he eats” is undeniable. Treatment goals of dietary modification start with maintaining body weight and a normal body condition score. If renal diets are not tolerated, warm canned diets diluted with some form of flavored moisture are a good choice. Other alternatives include adding other forms of moisture to food to increase fluid intake, providing flavored waters to encourage moisture consumption, water fountains and multiple drinking places throughout the house.

If a renal diet is not fed, most cats will tolerate low doses of aluminum hydroxide in food to act as a phosphorus binder, before serum phosphorus levels leave the normal range. Serum phosphorus should remain in the 4-5 mg/dl range, especially if calcitriol is considered. Low body condition scores and malnutrition are negative prognostic indicators in dogs and the same is likely to be true in cats. If adequate caloric intake and preservation of lean body mass does not occur, quality of life will decline.

Studies done to confirm preservation of lean body mass in cats fed a low protein diet, about 28% on an as-fed basis, were, as one would anticipate, time restricted to around 4 months. With the advent of a better plan for managing renal patients, they are living for years with stable renal values and hematocrits within the normal range. The effects of protein restriction on the body condition scores of cats with CPRD should be evaluated. Until then, we all have observed the protein cachexia of our renal patients. It is crucial to preserve adequate caloric intake and adequate protein for these patients.

The effects of uremia on appetite are well known, particularly in human renal patients. The use of H2 blockers for uremic gastritis can be helpful in encouraging consumption of adequate calories. The use of mirtazapine as an appetite stimulant is helpful in those cats who can tolerate it. We use 1/8 of a 15 mg tablet every day to every third day depending upon response to therapy. Many cats with CPRD are underweight and dosing of ¼ of a tablet as has been recommended is often followed by restlessness,
anxiety and vocalizing in cats who are sensitive to it. Clients can be quite upset by this and may be less inclined to follow other treatment recommendations. Both of these forms of therapy imply being able to accomplish giving fragments of a pill to a cat on a regular basis and over a prolonged period of time. Strategies for this should be included in client education including the use of “sticky” high value food like cheese in a can, cream cheese or pill pockets and other soft treats.

Calcitriol has long been reported to provide benefits to the human uremic patient by lowering parathyroid hormone concentration. This has also been reported in dogs and cats. Oral calcitriol has been shown to increase survival in human patients with CPRD including those treated prior to dialysis. The antiproteinuria effects of Vitamin D analogs are of crucial significance because proteinuria is a major risk factor for the progressive decline of renal function in both dogs and cats. Podocytes are critically important in overall glomerular function and structure. Injury to podocytes commonly leads to proteinuria and glomerulosclerosis. A marker for podocyte injury, desmin, was lowered by calcitriol in one model of CPRD in rats. Fibrosis as either glomerulosclerosis or tubulointerstitial fibrosis is a common sequela in CPRD. Calcitriol in physiologic doses interfered with glomerular proliferation and growth, lessening glomerulosclerosis in a rat model. Calcitriol treatment of an experimental glomerulonephritis model in rats inhibited medangial cell proliferation, glomerulosclerosis and albuminuria.

The renin-angiotensin-aldosterone system (RAAS) is a major mediator of progressive renal injury in CPRD. The RAAS system is present entirely within the kidney and is present in most renal cells including tubular epithelia.

Calcitriol is a negative endocrine regulator of RAAS. Calcitriol suppresses renin biosynthesis and has a protective role against hyperglycemia-induced renal injury in diabetic human patients. Through its effect to inhibit RAAS, calcitriol decreases production of Angiotensin II and thus lessens these fibrogenic consequences as well as other harmful renal effects.

A glomerular mesangial or interstitial inflammatory reaction with marked involvement of macrophages and lymphocytes attends all forms of renal disease. Together with control of RAAS, the ability of calcitriol to control inflammation are hallmarks of renoprotective actions.

In our practices, early diagnosis of CPRD at the IRIS 1 or 2a level is the key to successful management. A cat with or without proteinuria, with or without hypertension with a USG less than 1.030 and normal Calcium and Phosphorus will be started on Calcitriol at a dose of 2.5-3.5 ng/Kg per day. This is compounded into a chicken or fish flavored oil base by a compounding pharmacy licensed to produce compounded pharmaceuticals for the human market. Calcium, Phosphorus and their product will be measured in 2 weeks.

While the literature is clear that iCA is a far more accurate measure of total body calcium, it is an expensive test. Our protocol calls for frequent testing of renal values including calcium and phosphorus. We would be treating a fraction of the cats we can help if this costly test were included. Instead we use a protocol advocated by Larry
Nagode and Dennis Chew, Pathology and Urology professors respectively at the Ohio State University Veterinary College.

One of the benefits of the preservation of renal tissue using this protocol is the preservation of erythropoietin production and the consequent preservation of normal hematocrits. Cats with IRIS Stage 3-4 CPRD are still feeling better, more active and eating better with adequate circulating red cells. Anemia is a quality of life issue.

Hepcidin excess prevents iron absorption from the diet and blocks iron release from body stores by binding to and inducing the degradation of the iron export protein ferroportin. A mechanism for the EPO sparing effects of vitamin D is suggested by recent data demonstrating a hepcidin lowering effect of vitamin D. In vitro treatment with vitamin D of monocytes isolated from hemodialysis patients downregulated hepcidin transcription. Furthermore, oral administration of vitamin D in healthy volunteers lowered serum levels of hepcidin by 50% compared to baseline levels within 24 hr and persisted for 72 hr. Supplementation with vitamin D has also been reported to have beneficial effects on increasing erythropoiesis and decreasing inflammation. These initial results are promising, and a randomized controlled study is warranted to determine whether correction of vitamin D deficiency can ameliorate ACD.

Further Reading:
- Journal of Veterinary Emergency and Critical Care, *Calcitrol, Calcidiol, Parathyroid hormone and fibroblast growth factor-23 interactions in chronic kidney disease*. Volume 23 (2) 2013, pp 134-162.
A brief presentation will be made on the advantages and disadvantages of offering behavioural services in the general practice. There are 4 levels of services that can be offered: 1) a behaviour-centered practice; 2) prevention of behaviour problems; 3) detection of behaviour problems; 4) diagnosis and treatment of behavioural problems. Examples will be given to highlight each of those levels. Additional examples of a behaviour-centered practice and low stress handling will be provided.

**SO WHY SHOULD I INTEGRATE BEHAVIOURAL SERVICES IN MY PRACTICES?**

**Who is knowledgeable about behaviour?**
According to clients, the veterinary team is knowledgeable about behaviour (Hawn, 1998 in JAAHA). However veterinarians do not think that they are (Patronek, 1999 in JAVMA). In fact, the veterinary team knows more than they think. As examples, just think of behavioural changes that occur prior to the appearance of neurological signs, behavioural signs of pain, and signs of fear or imminent aggression.

**What are the client expectations?**
The pet is a family member and must behave almost “perfectly”. Information (not always valid) is widely available mostly through the internet. Pet owners are asking more questions than previously. Think of first-time pet owners in your practice and consider the ratio of behaviour questions as opposed to medical questions? Clients expect veterinarians to answer their questions about behaviour.

**Can you avoid talking and addressing behaviour questions?**
Behaviour is part of global health. The veterinary team is the most qualified and best suited to educate clients about their animal's health. When information is given from the veterinary clinic, it is done in a more standardized and appropriate way, based on scientific data (when available) as opposed to opinions.

**What happens if we ignore our client’s questions and concerns about behaviour?**
There may be decreased general satisfaction with the service offered at the veterinary clinic. This dissatisfaction can lead to decreased credibility of the entire veterinary team. Let’s consider the potential costs of not offering any behavioural advice: There is loss of income from behaviour consultations, as well as loss of income from low client referrals to your practice. Additionally, there may be decreased income secondary to fewer visits to the practice and decreased compliance with recommendations given.
PROFESSIONAL ADVANTAGES OF OFFERING BEHAVIOURAL ADVICE

Quality control of the information given to your clients
When you refer to non veterinarians, were do you refer your clients? Where are your clients going? Have you ever listened or seen how these trainers teach/train? Which techniques are they using? Are they based on learning principles (science-based)? Are clients enjoying themselves? Are they only offering training or are they also giving advice on nutrition (selling food) and other products (to treat animals)?

Mission of veterinarians
The quality of life of the animal is important. Distress can easily be under estimated. It therefore becomes an ethical question for cases in which animals with anxiety disorders are referred to non veterinarians... Who is best suited to evaluate the emotional state of the animal?
If appropriate advice is given within your practice and the animal behaves well, there will be a stronger bond of owners with the animal and the practice. This will lead to a better quality of life for owners and pets as well as a decreased risk of relinquishment and euthanasia.
Educating the dog requires a few minutes of training and activity per day. As a result the owner will no longer see his dog/cat the same way. The veterinary team can give a few simple tips that will enable owners to easily train their animals.

Protects our patients and improves the image of the profession
What is the credibility of non veterinary “behaviourists”? Where did they acquire their knowledge? Will they answer your client’s questions? Of course they will! And they will give advice on nutrition, vaccination, surgery, medicine, products, grooming, dentistry, etc.
Behavioural advice is expected from our clients. Integrating behaviour in our practices will change the approach and handling of animals, by taking into consideration the dog’s or cat’s emotional state. Owners will feel empathy. They will be reassured and the bond between the client, the patient and the professional will be strengthened.

ADVANTAGES FOR THE BUSINESS

Increase in number of referrals by clients
A satisfied client will talk and recommend your practice to others. More referrals will increase the number of new clients. Behavioural advice may require additional visits to the practice which allows for more advice and recommendations given to the client. Credibility of the practice and confidence in the veterinary team will lead to a stronger bond of the client to the practice and better compliance. Better compliance in turn will result in more satisfaction for the veterinary team.

PERSONNAL ADVANTAGES
The team will be working in an environment in which positive reinforcement and positive comments are emphasized. The environment ideally will become less stressful (better) for the animals, and the clients. Clients will enjoy their experience and the team will have pleasure working. Animals will become easier to handle, restraining will be reduced (without reducing safety) and there will be fewer injuries.

LOW STRESS HANDLING
This presentation will be based on video clips showing alternative ways for handling our patients in order to increase the bond between the patient and the client but also between the “patient/client” and the clinic staff members.
Assessing risk of injury from a dog
Dangerousness does not necessarily equate with aggressiveness. A 50 kg enthusiastic excited dog running right into an owner and knocking down or injuring that person is dangerous. On the other hand, a growling dog that has never bitten is aggressive but not necessarily dangerous. Aggression has been defined as “behaviour that leads to the damage or destruction of a target entity”. Some definitions of aggression also include the display of threats in the absence of injury. Thus aggression encompasses a wide variety of behaviours from subtle body postures and facial expressions to explosive attacks. Aggression can be an expression of normal or abnormal behaviour. Description of the behaviour sequence, context, frequency and severity of aggressive events as well as health status of the dog allows us to tease apart appropriate normal from inappropriate abnormal behaviours.

Behaviour sequence
Behaviour is always a sequence. Observing the entire sequence is essential to determine if an animal is behaving normally or not. In the case of canine aggression we could illustrate the sequence as initial warning such as a growl (initiation), then a pause, then in some cases a bite (action), and finally immediate volitional release (end of sequence). Behaviour becomes “abnormal” or illness-related if some of the steps from the sequence are omitted or altered. A dog growling and biting simultaneously without any other form of warning has an altered sequence because there is no clear initiation phase. Aggression can be the result of fear or anxiety. Anxiety is defined as anticipation of a future threat or danger (real or imaginary). Anxious dogs occasionally are unable to tell the difference between real threat and absence of threat. It is therefore important to realize that some aggressive dogs may in fact be ill and suffering from an anxiety disorder.

Appropriateness of the aggressive behaviour given the context
Context is also important to determine if behaviour is normal or illness-related. Behaviour can be inappropriate given the context. If I decide to randomly kick a person on the street given that this person did not even interact with me, kicking is inappropriate behaviour on my part in the described context. If on the other hand I am being mugged on the street and I am kicking my assailant, the same behaviour becomes appropriate. The context makes all the difference. A dog with an otitis bites the veterinarian (or owner). Context will be considered (painful condition, defensive aggression) when interpreting this aggressive event and this dog will likely not be labelled as a high-risk dangerous dog. A dog will bark or growl briefly at the approach of a stranger and then he will wait and watch for the response. Based on the response of the receiver (the stranger in this example), the dog will decide on its subsequent action. The behaviours will depend on the receiver’s response and the dog’s interpretation of the response. If a dog growls snarls and lunges systematically at everyone approaching without first warning (barking and/or growling) then pausing for
a response, that aggressive behaviour becomes inappropriate and out of context. That
dog is ill and is unable to make the distinction between threat and non-threat.

**Severity and frequency of the aggression given the context**
A dog can bark, growl, lift its lips, snarl, snap, bite, and then latch on or release. Bites
can be single or multiple, inhibited or uninhibited. Clients are questioned on the
severity and frequency of the aggressive events. Severity of bite can in some cases be
exacerbated by fear or pain. The veterinarian must determine whether the severity
and/or frequency of the aggressive behaviours are appropriate for that given context.

**Predictability of the aggressive events**
The context (triggers and specific situations) and the dog’s body language are used in
determining predictability of the aggressive events. If the dog exhibits only defensive
aggression, the events are more predictable. Defensive aggression for the purpose of
this presentation is defined as one individual “approaching or entering the animal’s
space” and interacting with the animal (touching, handling); the animal reacts
aggressively to the approach or physical contact. Offensive aggression for the purpose
of this presentation is defined as aggression occurring without interaction (touching,
handling or even looking at the animal). The aggressive animal is the one approaching
the individual (victim of aggression). The trigger for the aggressive behaviour is often
difficult to identify.

**Size of the patient**
A larger dog can potentially produce more damage.

**Health status of the patient**
A painful condition may exacerbate anxiety and aggression. Dogs that are behaving
abnormally can be more unpredictable and therefore more dangerous.

**Social environment (humans and other animals)**
Relative risk for a young child unable to read and interpret canine body language can
be increased as the child will not understand a warning growl.

**Conclusion**
Any dog can be aggressive and bite. A zero risk level of bite does not exist for a live
animal. So the only way to guarantee that a dog will never bite again is to kill the dog.
Assessing level of risk for a specific case requires a complete analysis: the animal, its
behaviours and health status, and all interactions in its social environment. Clinical
cases will be presented to illustrate various points.

**Further Reading:**
- Overall KL, (2013). Clinical Behavioral Medicine for Small Animals. Mosby, St Louis,
  MO.
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 Quebec 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 pure-breds. 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 min), 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).

Selecting patients for pharmacological treatment
Which patients should we medicate? Drugs in behavioural medicine are generally prescribed to decrease anxiety or reactivity, thus facilitating implementation of behaviour modification techniques and in some cases accelerating rate of progress. Antidepressants can help achieve these goals if patients are carefully selected.

Anxiety
Anxiety in humans is defined as the anticipation of a future danger or threat, real or imaginary. Anxiety can be normal or a sign of an illness. This definition can also be used for animals. Most dogs and cats presented to the veterinarian are fearful or anxious. Some will remain anxious as long as they are on the table but are fine when they are back on the floor or out the door from the veterinary hospital. These patients are normal. On the other hand, separation anxiety, panic disorder, generalized anxiety, phobias and obsessive-compulsive disorders are sub-groups of anxiety disorders.

Behaviours and body language during the appointment
During a behavioural consultation, the animal is usually free to move around the room. Physical examination is done at the end of the appointment. Dogs can express anxiety (and/or fear) by panting, puffing their cheeks, crinkling their brow, yawning repeatedly, licking their lips constantly, pulling their ears back, trembling, tucking their tail, trying to back up or escape, hiding, whining, or even seeking owner attention excessively. Many anxious dogs will have dilated pupils, will pace, and are unable to settle down and relax. Each sign is non-specific. Anxiety (or fear) can result in urination, defecation or excessive salivation. Finally aggression may also be a sign of anxiety. Similarly cats can express anxiety (and/or fear) by panting, licking their lips frequently, pulling their ears down and back, arching their back, tucking their tail, trying to escape, vocalizing, pacing, freezing or being aggressive. Each sign is non-specific.

Some animals will have increased motor activity whereas others will have decreased motor activity. Vigilance may be increased. Reactivity during the appointment may be exaggerated and may even increase over time. Exploratory behaviour of the consultation room may be absent and should be distinguished from increased motor activity. These behaviours (also compatible with anxiety) serve as baseline and can be compared with behaviours expressed during follow-up visits.

Anxiety during the behavioural consultation is not sufficient to conclude that a given animal suffers from an anxiety disorder. But clients during the appointment are educated on how to recognize subtle signs of their animal’s anxiety or fear. Following the appointment they will be much more attentive to the animal’s body language and
behaviours. They may realize that their animal is exhibiting signs of anxiety on a daily basis in the home environment in the absence of an identifiable cause. In the latter case, their animal is perhaps suffering from an anxiety-related disorder. The veterinarian should make it a point to ask if signs of anxiety (or fear) occur when the dog or cat is in its familiar environment and determine if that anxiety/fear is appropriate for the context.

**Reactivity**

Excessive reactivity can also be an indication of illness. An animal becoming more and more aggressive during the appointment in the absence of any threat may be “over-reactive”. An animal becoming disobedient may in fact be “over-reactive” in that context. This animal is unable to hear (“emergency mode”) any commands. Ask a person if following a near miss car accident, he/she would be able to tell what song had just played on the radio at the time of that close call… The ears may have heard the song but the brain did not register the information, as it was not essential for survival… Dogs and cats in “emergency mode” will require medication to decrease the level of reactivity.

**Videotapes**

Objective baseline data are obtained from tapes. Videotapes of the animal at home may reveal signs compatible with anxiety and in some cases may even be indicative of generalized anxiety. Videotapes are essential to confirm diagnosis of separation anxiety as well as assess response to pharmacological treatment. Tapes are also very useful to identify occurrence of silent threats occurring between household pets (inter-cat or inter-dog aggression) that are often unrecognized or missed by clients.

**Indications for antidepressant medication**

1. Signs compatible with generalized anxiety in familiar environments in the absence of danger or threat
2. Reactivity during the appointment for behavioural evaluation increases over time without any threat to the animal
3. Excessive reactivity to benign stimuli
4. Behaviour sequence is altered (other medical conditions ruled out)
5. Behaviour is inappropriate for the context
6. Frequency, severity or duration of the behaviour is excessive for the context
7. Recovery time after an undesirable behaviour is excessive
8. Animal is in “emergency mode” during episodes of undesirable behaviour

Examples of the following criteria will be given with clinical cases and video presentations.
Megaesophagus is one of the most common causes of regurgitation in dogs. Megaesophagus refers to a specific syndrome characterized by a dilated hypoperistaltic esophagus and should be differentiated from other causes of esophageal dilation (e.g., foreign body, vascular ring anomaly, mucosal stricture, neoplasia) which may or may not be characterized by abnormal peristalsis. Significant complications of regurgitation include aspiration pneumonia and chronic wasting disease.

Esophagitis occurs more commonly than is usually recognized in clinical practice. This seminar will emphasize diagnosis and management of esophageal hypomotility and esophagitis in dogs and cats.

Regurgitation refers to a passive, retrograde movement of ingested material to a level proximal to the upper esophageal sphincter. Usually this occurs before ingested material reaches the stomach. The term reflux refers to movement of gastric or duodenal contents into the esophagus without associated eructation or vomiting. This process may or may not produce symptoms.

Regurgitation is usually a clinical sign of an esophageal disorder. The esophagus is a tremendously dilatable muscular tube that acts via a series of well-coordinated peristaltic contractions to move ingesta from the mouth to the stomach. Regurgitation in most cases results from abnormal esophageal peristalsis, esophageal obstruction, or asynchronous function of the gastroesophageal junction.

Diagnostic Procedures
It is essential that the clinician make a clear differentiation between regurgitation and vomiting at the outset. Failure to recognize the difference between regurgitation and vomiting often leads to inappropriate testing (i.e., tests most useful for diagnosis of abdominal disorders are generally performed), misdiagnosis, and the use of ineffective treatment protocols. Therefore, the first diagnostic step is to obtain an accurate history. This is best accomplished by a clinician who maintains a high index of suspicion regarding the possible occurrence of regurgitation and who subsequently asks clear questions of the client about their pet’s clinical signs.

Thoracic radiography for survey evaluation of the esophagus is the most important screening procedure in the diagnosis of a regurgitation disorder. Radiographs are evaluated for evidence of esophageal dilation and the presence of a foreign body or thoracic mass. Remember that transient dilation may occasionally occur and can be related to aerophagia, anxiety, dyspnea, anesthesia, and vomiting. Knowledge of the history is important in differentiation of potentially transient causes from those that are more long-standing. If survey radiographs fail to provide a definitive diagnosis, a barium esophagram (with fluoroscopy if available) should be performed. A liquid barium suspension (10 to 20 ml prior to each exposure) is best for evaluating for esophageal dilation. A mixture of food and barium is superior for evaluating
esophageal motility because in some patients with slightly to moderately decreased contractility peristalsis may be adequate for liquid but clearly unable to propel solids aborally in a normal manner.

A baseline CBC and biochemical profile should be run in all patients with megaesophagus to look for evidence of underlying problems. Specific tests to evaluate for systemic disorders such as hypoadrenocorticism (ACTH stimulation), CK (polymyositis), and serum lead levels are done if the history and/or physical examination indicate that these primary disorders may exist. Myasthenia gravis should be considered in any patient with megaesophagus. The test of choice is an acetylcholine receptor antibody titer. Acetylcholine receptor antibody titers are run at the Comparative Neuromuscular Laboratory in La Jolla, CA (Dr. Diane Shelton). Contact the laboratory for forms and sample submission instructions.

The address is:
**Comparative Neuromuscular Laboratory**
9500 Gilman Drive
Basic Science Building, Rm. 2095
University of California, San Diego
La Jolla, CA 92093-0709
**Phone:** (858)534-1537 **Fax:** (858)534-0391
**Web:** [http://vetneuromuscular.ucsd.edu/](http://vetneuromuscular.ucsd.edu/)
**Email:** musclelab@ucsd.edu

If radiographs reveal any suggestion of esophageal stricture, foreign body, mass, or diverticulum, endoscopy provides the most rapid and cost effective method of making a definitive diagnosis.

**General Management Principles for Megaesophagus**
The main objectives of treatment for regurgitation disorders are to remove the initiating cause as early as possible, minimize chances for aspiration of esophageal content, and maximize nutrient intake to the GI tract. In most cases, idiopathic megaesophagus is incurable, and treatment involves an individually tailored feeding regimen with the patient eating in an elevated position. Esophageal foreign bodies and intraluminal strictures can often be managed successfully with endoscopic techniques and bougienage or balloon dilation respectively. Medical management is indicated for such secondary causes of esophageal dilation as myasthenia gravis, hypoadrenocorticism, systemic lupus erythematosis, polymyositis, and esophagitis.

Megaesophagus patients are best fed with the upper body in a fully upright position. This is best accomplished by positioning the dog in a special high chair (see information below about Bailey chairs). It is important that proper positioning be clearly demonstrated to the client so that there is no misunderstanding. Whenever possible the elevated position should be maintained for a full 10 minutes after ingestion of food is completed. Various props to aid in the elevation process have been used successfully, including ladders, stairs, ramps, tables, and chairs. Since the esophagus is virtually never completely empty in a megaesophagus patient it is often helpful to hold the animal in an elevated position for 5 to 10 minutes at a time sometime between meals and at bedtime (I ask all of my clients to at least do the
bedtime elevation in an effort to empty the esophagus as much as possible prior to an expected period of prolonged recumbency).

Megaesophagus patients are ideally fed 2 to 4 times daily. This depends, of course, on the caregiver’s time constraints. I have had the best success feeding soft moist to solid (chopped) canned food consistency. I only recommend trying gruels if the semi-moist consistency is not well tolerated. Some patients do well when fed a series of “meatballs” fashioned from canned food. Others can tolerate dry food fairly well. A key point is that each patient is an individual and clients should be instructed to experiment with various food consistencies in order to determine the best approach for their own pet.

Many patients with idiopathic megaesophagus can be managed successfully for months to years. I have known many dedicated owners who have managed to find the time required to care for their pets. As a result of this experience I try to offer as much encouragement as possible at the time of diagnosis. The most worrisome complications that can occur are aspiration pneumonia and significant weight loss. The prognosis is guarded to poor in patients that suffer recurrent episodes of pneumonia.

An option in cases where frequent regurgitation remains an ongoing problem with or without aspiration events is to place a gastric feeding tube (e.g., percutaneous endoscopy-guided gastrostomy tube [PEG]). All food and water can then be administered through the feeding tube (some patients have been maintained for as long as 3 or more years in this way). Periodic tube replacement will be necessary. Low profile feeding tubes often work best for long term tube feeding. This method of management has been highly successful for some dogs that continue to regurgitate frequently despite excellent efforts to manage them with an elevated feeding program.

Special Feeding Chairs

**Bailey Chair:** There is a lot of information available on the internet about a special feeding chair that was designed by Donna and Joe Koch, the owners of a dog named “Bailey.” “Bailey” had been diagnosed with megaesophagus. The dog sits in a totally upright (“begging”) position to eat, drink, or take medication and gravity aids transit of anything ingested to the stomach. Use key words “Bailey Chair” for an internet search and information on how to acquire or build a Bailey chair (also see support group information below).

**Custom Bailey chairs:** [www.BaileyChairs4Dogs.com](http://www.BaileyChairs4Dogs.com)

**Megaesophagus Client Information and Support Group**
[www.caninemegaesophagus.org](http://www.caninemegaesophagus.org)
Provides information on the causes of congenital and idiopathic canine *megaesophagus*, the clinical signs, risk factors and accompanying disorders, with lots of feeding tips.

A Support Group for owners of dogs that have, had or may have megaesophagus, was established at Yahoo Groups in 2002 by Dave Kay and Katy Weeks, in memory of their
Golden Retriever, Rusty. Members of the group can provide suggestions and ideas for feeding and care of dogs with megaesophagus. A veterinarian monitors the group as an advisor and offers suggestions for members to discuss with their veterinarians. The group is at: http://groups.yahoo.com/group/megaesophagus/ and requires membership.

Esophagitis

Inflammatory diseases of the esophagus occur more commonly than they are recognized. Inflammatory changes can range from mild mucosal inflammation that may or may not be grossly evident, to moderate to severe ulceration and transmural involvement. Any disorder that causes acute or chronic frequent vomiting has the potential for causing esophagitis. This especially includes causes of severe vomiting, such as intestinal foreign bodies, gastric foreign bodies, acute pancreatitis, parovirus enteritis, and gastrinoma. Dogs with parovirus enteritis that are debilitated and recumbent are especially at risk. Vomited fluid that is retained in the esophagus is not cleared adequately in weak and recumbent patients. As a result the esophageal mucosa is bathed with gastric acid and activated enzymes that will cause mucosal injury.

Other causes of esophagitis include esophageal foreign bodies, chemical and thermal injuries, injury from lodged medication (doxycycline capsules in cats can become lodged and cause esophagitis and even stricture formation), gastroesophageal reflux, and anesthesia related reflux.

Diagnosis of Esophagitis

The clinical signs of esophagitis vary considerably, depending on the degree of inflammation present. The clinician must maintain a high index of suspicion because in many cases only subtle clinical signs may be evident. With mild esophagitis there may be increased swallowing motions, salivation, and inappetence. In more severe cases there may be gulping, regurgitation, dysphagia due to pain, total anorexia, and signs that suggest esophageal pain, such as reluctance to move, standing with the head extended, reluctance to lie down, and trembling. Heartburn pain in humans can be quite intense, and it is suspected that a similar situation exists in animals. Esophageal hemorrhage may occur in severe cases. Signs such as increased attempts at swallowing, salivation, and regurgitation, and inappetence that occur within 1 to 4 days of an anesthetic procedure strongly suggest reflux esophagitis. Chronic reflux esophagitis occurs most commonly in patients with hiatal hernia disorders.

Radiographic survey and contrast studies are often normal in patients with mild to moderate esophagitis. Survey films may show increased esophageal density in moderate to severe esophagitis. There may also be various degrees of esophageal dilation, since esophageal inflammation may inhibit motility. Persistent contrast in the thoracic esophagus or esophageal dilation, or both, suggest the possibility of gastroesophageal reflux.

A definitive diagnosis of esophagitis is most often made by endoscopic visualization of the esophageal mucosa. Variable degrees of mucosal erythema or isolated patches of eroded mucosa may be seen. However, as also occurs in humans, some animals with esophagitis do not have gross esophageal abnormalities, and in these cases symptom
patterns in conjunction with positive response to therapy are the key components to a presumptive diagnosis.

Treatment of Esophagitis
It is important to note that, although the esophagus is physically a very tough and resilient structure, once it is injured it does not always heal very quickly. For inflammatory disorders fairly aggressive combination drug therapy is often required. Treatment may include dietary modification, proton pump inhibitors (PPIs), H2-receptor antagonists, GI promotility agents, anti-inflammatory drugs, and mucosal protectant therapy. Single or combination drug therapy may be required, depending on factors that include whether treatment is designed mostly for prevention, duration or severity of mucosal injury, and clinical signs. Most affected dogs and cats are managed with either an H2-receptor antagonist or a PPI (e.g., omeprazole). Additionally, high-protein and low-fat diets, a prokinetic drug, and cytoprotective medication are indicated in some cases.

Mild reflux esophagitis is often asymptomatic and generally resolves without therapy. If clinical signs suggestive of reflux esophagitis occur within several days of an anesthetic procedure, treatment should be instituted, regardless of whether endoscopy is available for definitive diagnosis. Treatment in this situation usually includes an H2-receptor antagonist or a PPI, and a prokinetic drug (metoclopramide or cisapride). The duration of therapy will typically be 7 to 14 days. A longer duration will be required if clinical signs persist.

H2-receptor antagonists are used to decrease gastric acid production, thereby decreasing acid volume available for reflux. H2-receptor antagonists also reduce the volume of gastric acid that is produced. There is no adverse effect on resting or stimulated LES pressure levels. Ranitidine (2.2 mg/kg [dog], 3 mg/kg [cat] orally every 12 hours), or famotidine (Pepcid, 1 to 1.2 mg/kg orally every 24 hours, or every 12 hours if there is severe esophagitis) is generally used for 2 to 3 weeks in dogs and cats with acute reflux esophagitis. I have strongly preferred to use famotidine (Pepcid) because of its long dosage interval and the fact that it is associated with fewer side effects. Further, studies have shown that famotidine is the most effective H2-blocker drug for dogs and cats. It should also be understood that none of the H2-receptor antagonists are highly effective in dogs and cats (i.e., okay to use in mild cases of esophagitis, but patients should be observed carefully for signs of adequate response). Another H2-receptor antagonist that can be tried is nizatidine (Axid). The dosage is 2.5 to 5 mg/kg orally every 24 hours. Ranitidine and nizatidine also have a mild gastric prokinetic effect. Long-term therapy should be used in hiatal hernia patients with chronic reflux esophagitis if corrective surgery either is not performed or is unsuccessful.

PPIs are drugs that much more significantly inhibit gastric acid secretion in response to all modes of stimulation. This class of drug is used when esophagitis is moderate to severe, as H2-receptor antagonists are not as effective in reducing acid levels. PPIs include omeprazole (Prilosec), lansoprazole (Prevacid), esomeprazole (Nexium), pantoprazole (Protonix), and rabeprazole (Aciphex). Omeprazole is the PPI that has been used most frequently in animal patients. PPIs decrease acid secretion by inhibiting H+, K+ ATPase (commonly called the proton pump), thereby blocking the
final, common step in the secretion of gastric acid. PPIs control both basal and meal-stimulated acid secretion. Therefore, the acid suppression achieved by a PPI is more complete and longer lasting than can be attained with an H2-receptor antagonist. The currently recommended dosage for omeprazole is 1.5-2.5 mg/kg orally (this dose is based on studies published in 2012), once to twice daily, administered 30-45 minutes prior to feeding. It may be best to administer twice daily as a matter of routine for the first 7 to 10 days, and BID for longer periods in more severe cases. Maximal acid reduction is not achieved until 2 to 5 days after oral administration is begun; therefore, famotidine is often administered concurrently during the first 5 days to ensure some level of acid reduction as early as possible.

**Moderate to Severe Esophagitis – IV PPI Protocol**
The PPI drug pantoprazole is currently available in an injectable preparation. Lansoprazole was available in an IV formulation at one time but this is not currently available. In situations where the patient is NPO or where more rapid effective blood levels are needed, pantoprazole is administered IV.

**Pantoprazole (Protonix) IV, 40 mg/vial** Marketed by: Wyeth(R) Pharmaceuticals Inc.

**Dose:** 0.7-0.8 mg/kg q24 hours (but it can be dosed at q22 hours to get 2 doses out of 1 bottle as it's $\$$)

**Administration:** The Protonix should be reconstituted with 10mL of 0.9% NaCl and then further diluted with 100mL of 0.9%NaCl, LRS or 5%Dextrose.

**Final concentration** = 0.4 mg/mL

Give over 15-20 minutes

**Esophagitis Associated with Frequent Vomiting**
Clinicians are especially cautioned to be more attentive to patients that might have esophagitis secondary to frequent or severe vomiting (e.g., caused by GI foreign bodies, parvoviral enteritis, acute pancreatitis, or renal failure). Esophagitis can easily develop in these situations, and it no doubt adds significantly to the discomfort that the patient is already experiencing. In these cases, both sucralfate and an H2-receptor antagonist are used to treat esophagitis. I use famotidine (Pepcid) injectable at 0.5 mg/kg IV BID. An antiemetic drug such as maropitant (Cerenia) is injected to help decrease the frequency of vomiting. We are also using Cerenia much more frequently now PRE-OP to help decrease the frequency of vomiting post-op. Studies have shown that Cerenia is very effective in reducing the incidence of perioperative vomiting and animals often return to feeding earlier and eat a greater volume of food if they received Cerenia before surgery.

Sucralfate is given orally, in suspension form so as to better coat the esophagus, usually 30 to 60 minutes after antiemetic therapy has been administered. The duration of therapy in patients with reflux esophagitis depends on the cause and degree of inflammation. For moderate to severe esophagitis, 4 to 8 weeks of therapy or more may be required to achieve full healing of the esophagus. For esophagitis related to
frequent or severe vomiting, treatment is usually administered 5 to 7 days, and only longer if clinical signs or endoscopic findings warrant.
Vomiting is among the most common reasons that dogs and cats are presented for evaluation. Because there are a multitude of causes of vomiting, ranging from simple to complex, this can be a challenging problem for clinicians to accurately diagnose and manage. The problem also causes significant concern for pet owners, especially when there is an onset of frequent severe vomiting or when the occurrence becomes more chronic and intermittent without adequate control. However, by following a systematic approach beginning with an accurate history, a thorough physical exam, and appropriate baseline testing (Stage 1), then performing tests more specific for certain conditions or organ systems (e.g., bile acids assay, leptospirosis serology, baseline cortisol or ACTH stimulation, ultrasonography) (Stage 2), and finally where indicated performing advanced procedures for more thorough examination and biopsy or definitive therapy (endoscopy, exploratory laparotomy), most cases can be diagnosed successfully and managed judiciously. Vomiting does not constitute a diagnosis in itself. It is emphasized that vomiting is simply a clinical sign of any of a number of disorders that can involve any organ system in the body. In fact, one diagnostic registry service listed over 400 potential causes of vomiting in dogs! These notes summarize diagnostic approach and various treatment options for managing dogs and cats with vomiting.

Vomiting refers to a forceful ejection of gastric and occasionally proximal small intestinal contents through the mouth. The vomiting act involves three stages: nausea, retching, and vomiting. Serious consequences of vomiting include volume and electrolyte depletion, acid-base imbalance, and aspiration pneumonia.

It is essential that the clinician make a clear differentiation between regurgitation and vomiting at the outset. Regurgitation is defined as passive, retrograde movement of ingested material, usually before it has reached the stomach. Failure to recognize the difference between regurgitation and vomiting often leads to misdiagnosis. Regurgitation may occur immediately after uptake of food or fluids or may be delayed for several hours or more.

A Detailed, Accurate History is ESSENTIAL
One of the most important early considerations is to determine if any toxins or foreign objects may have been ingested. Some compounds can cause life threatening sequelae. The earlier a toxicity is identified, the greater the chance for successful management. Currently, xylitol toxicity is being recognized more frequently, and sago palm plants, which can cause severe hepatotoxicity in dogs and cats, are found in more homes and yards than in previous years. Cocoa mulch toxicity (theobromine) is also occasionally seen. Many animals that have ingested toxins are presented with vomiting as a prominent sign.

History and Clinical Assessment: Clinical Features of Vomiting
Because of the wide variety of disorders and stimuli that can cause it, vomiting may present the clinician with a major diagnostic challenge. A complete historical review with emphasis on all body systems is essential for determining a realistic and effective initial work-up plan and treatment protocol. All too often concentration on only the gastrointestinal tract leads to an incorrect diagnosis and inappropriate treatment. Consideration of the following features is useful in assessing and diagnosing a patient with vomiting:

(1) duration of signs
(2) signalment and past pertinent history
(3) environment and diet
(4) systems review (e.g., history of PU/PD, coughing and sneezing, dysuria or dyschezia, etc.)
(5) time relation to eating (vomiting of undigested or partially digested food more than 8-10 hours after eating often indicates a gastric motility disorder [more common] or gastric outlet obstruction [less common])
(6) content of the vomitus (food, clear fluid, bile, blood, material with fecal odor), and
(7) type and frequency of vomiting (projectile?, chronic intermittent?, cyclic?, morning vomiting only?).

Most Common Causes of Acute or Chronic Vomiting in Dogs
First need to Rule-Out:

Dietary/ingestive problem (always investigate for any potential environmental materials that the patient may have been chewing on (plants [toxins], debris carpet, etc)
- Indiscretion (e.g., table scraps, sudden diet change, garbage ingestion; toxins, foreign body, ingesting plants in home or yard)
- Food adverse reaction (dietary sensitivity)
- True food allergy

Parasites
- Intestinal (including Giardia)
- Gastric (Physaloptera)

Drug related problems
- NSAIDS must always be considered
- Other drugs (e.g., cardiac glycosides, antibiotics, chemotherapeutic agents)
- Any drug can potentially cause vomiting, always ask about any supplements that are being given to a pet

Metabolic disorders
- Renal disease
- Liver disease
- Electrolyte abnormalities
- Addison’s disease (some are glucocorticoid and mineralocorticoid deficient and will demonstrate typical electrolyte abnormalities; others are only glucocorticoid deficient and require ACTH stim for diagnosis (JAVMA April 15, 2007, p. 1190-1194)
Table 1

Don’t Forget About Leptospirosis!
Clinical signs may include:

<table>
<thead>
<tr>
<th>Fever</th>
<th>PU/PD</th>
<th>Icterus</th>
<th>Vomiting</th>
</tr>
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<tbody>
<tr>
<td>Anorexia</td>
<td>Dehydration</td>
<td>Bleeding</td>
<td>Diarrhea</td>
</tr>
<tr>
<td>Myalgia/Arthralgia</td>
<td>Hypovolemic shock</td>
<td>Renomegaly Renal pain</td>
<td>Weight loss</td>
</tr>
<tr>
<td>Lethargy</td>
<td>Peripheral edema</td>
<td>Abdominal pain</td>
<td>Uveitis</td>
</tr>
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**Rule-Outs for Chronic Vomiting, Once the Causes Listed Above are Ruled Out:**

**Main Categories:**

- **Motility Disorders**
  - Gastric hypomotility (an underappreciated disorder)

- **Inflammatory Disorders**
  - Chronic gastritis (with or without *Helicobacter*)
  - Inflammatory bowel disease

- **Obstructive Disorders**
  - Foreign body not already diagnosed (including cases with a partial small bowel obstruction that has eluded early diagnosis)
  - Hypertrophic gastropathy (uncommon)

- **Neoplasia**

**Most Common Causes of Chronic Vomiting in Cats**

- Dietary problem
  - Food adverse reaction (dietary sensitivity), up to 25% of cases

- IBD
- Hyperthyroidism
- Liver disease
- Renal disease
- GI lymphoma (intestinal is more common)
- Chronic pancreatitis
- Heartworm disease

**Intermittent Chronic Vomiting**

Chronic intermittent vomiting is a common presenting complaint in veterinary medicine. Often there is no specific time relation to eating, the content of the vomitus varies, and the occurrence of vomiting may be very cyclic in nature. Depending on the
disorder, other signs such as diarrhea, lethargy, inappetence, and salivation (nausea) may occur as well. When presented with this pattern of clinical signs, the clinician should strongly consider chronic gastritis, inflammatory bowel disease, irritable bowel syndrome, and gastric motility disorders as leading differential diagnoses. A detailed work-up including gastric and intestinal biopsies is often required for definitive diagnosis in these cases. It is important to note that chronic intermittent vomiting is a common clinical sign of inflammatory bowel disease in both dogs and cats.

Vomiting from systemic or metabolic causes may be an acute or chronic sign and generally there is no direct correlation with eating and no predictable vomitus content.

Diagnostic Plan
If reasonable concern is established based on the history (e.g., patient is inappetent, ingested a toxin, is vomiting frequently) or physical assessment (e.g., patient is listless, dehydrated, in pain), then a minimum data base of CBC, complete biochemical profile (or specific tests for evaluation of liver, kidney, pancreas, electrolytes), complete urinalysis (pre-treatment urine specific gravity extremely important for diagnosis of renal failure), and fecal examination is essential. The best way to screen for GI parasites on a single fecal sample is to run both a centrifugal flotation test and a Giardia antigen test. If only a single zinc sulfate centrifugal flotation is run, 25-30% of Giardia cases will be missed. T4 and both a heartworm antibody test and heartworm antigen test are considered routine baseline tests for vomiting cats (approximately 40% of cats with adult heartworms will have vomiting as a clinical manifestation of the disease). Survey abdominal radiographs are indicated if thorough abdominal palpation is not possible or suggests an abnormality (e.g., foreign body, pancreatitis, pyometra). Some institutions now routinely order 3 view abdomen films on patients presented for vomiting (both laterals and a VD). Unfortunately these tests are often not done early enough. Even if baseline results are unremarkable they are more than justified because they help to rule out serious problems at the outset (e.g., vomiting due to renal failure, diabetes mellitus, liver disease). Alternatively, any abnormalities provide direction for initial treatment and further diagnostics.

The decision for performing more in-depth diagnostic tests is based on ongoing clinical signs, response to therapy, and initial test results. These tests include baseline cortisol or ACTH stimulation to confirm hypoadrenocorticism in a patient with an abnormal Na:K ratio or to investigate for this disorder if electrolytes are normal, complete barium series or BIPS study (for gastric or intestinal foreign body, gastric hypomotility, gastric outflow obstruction, partial or complete intestinal obstruction), cPLI* or fPLI*(canine and feline lipase immunoreactivity, respectively, for diagnosis of pancreatitis in dogs and cats), and serum bile acids assay (to assess for significant hepatic disease). Barium swallow with fluoroscopy is often necessary for diagnosis of hiatal hernia disorders and gastroesophageal reflux disease. Serum gastrin levels are run if a gastrinoma (Zollinger-Ellison Syndrome) is suspected.

Pancreatitis: Pancreatitis continues to be a challenging disorder to accurately diagnose, short of thorough direct examination and biopsy. Assays for amylase and lipase are of very limited value, especially in cats. In general, the following can be stated regarding the various diagnostic tests for pancreatitis:
Value of the Various Diagnostic Tests for Pancreatitis

Amylase/Lipase (sensitivity on lipase depends on which specific test is being done)
- of value as a screening test in dogs only
- need to be 3x or > above normal reference range in order to suggest pancreatitis
- normal does not rule-out pancreatitis
- **new lipase assay from Antech (2 DGGR) approximates sensitivity of PLI for diagnosis of pancreatitis
- Antech has discontinued the somewhat less sensitive 1,2-diglyceride assay as of October 4, 2015. The new assay is 2 DGGR and is on every biochemical profile for dogs and cats (where lipase is normally included)

Abdominal Ultrasound
- highly specific, but not very sensitive, especially in cats

Serum PLI
- highly sensitive for pancreatitis

Pancreatic Lipase Immunoreactivity (cPLI and fPLI)
- Exocrine Pancreatic Insufficiency (EPI)
  - cPLI is reliably significantly decreased
  - cPLI is specific for EPI
- Chronic Renal Failure
  - Increased, but usually still within reference range
- Dogs with Biopsy Proven Pancreatitis
  - cPLI sensitivity is > 80%
  - currently recommended cutoff value for dogs is >200 ug/L
  - results are also promising for cats

Negative contrast gastrography.
An excellent technique to quickly evaluate the stomach for presence of a nonradiopaque foreign body.

Technique:
Gastric tube, tranquilize as needed
(definitely tranq cats)
Dogs: 8-10 ml/lb air or stop if the animal shows discomfort
Cats: 5 ml/lb air
Remove tube, take rads immediately
(left lateral, VD first)
Can also use 60 ml carbonated beverage (e.g., Mountain Dew)

BIPS are barium impregnated polyethylene spheres. Traditionally, veterinarians have relied on barium liquid as the contrast agent of choice for gastrointestinal studies. However, recognized limitations of barium liquid have led to the development of barium-impregnated solid radiopaque markers for the diagnosis of motility disorders and bowel obstructions. Barium liquid contrast studies are of limited value in detecting hypomotility. Radiopaque markers can be used to investigate a number of common gastroenteric problems. These spheres have been specifically validated for use in dogs and cats and are the only radiopaque markers with which there is extensive clinical experience in veterinary medicine. BIPS are manufactured in New Zealand and are now available in many countries. Information on availability of this product,
Ultrasonography can be useful in the diagnostic work-up of a number of disorders that can cause vomiting. Among the problems that may be detected with ultrasonography are certain disorders of the liver (e.g., inflammatory disease, abscessation, cirrhosis, neoplasia, vascular problems), gall bladder (cholecystitis, choleliths, gallbladder mucocele), GI foreign bodies, intestinal and gastric wall thickening, intestinal masses, intussusception, kidney disorders, and others. Needle aspirations and/or biopsies can be done at many sites under ultrasound guidance.

One of the most reliable and cost efficient diagnostic tools currently available for evaluation of vomiting is flexible GI endoscopy. Endoscopy allows for direct gastric and duodenal examination, mucosal biopsy from these areas, and in many cases gastric foreign body retrieval. Endoscopy is considerably more reliable than barium series for diagnosis of gastric erosions, chronic gastritis, gastric neoplasia, and inflammatory bowel disease (a common cause of chronic intermittent vomiting in dogs and cats). It is stressed that biopsy samples should always be obtained from stomach and whenever possible small intestine regardless of gross mucosal appearance. Normal gastric biopsies may support gastric motility abnormalities, psychogenic vomiting, irritable bowel syndrome, or may be noncontributory (i.e., look elsewhere for diagnosis). Many dogs with vomiting due to inflammatory bowel disease have no abnormalities on gastric examination or biopsy. If only gastric biopsies are obtained, the diagnosis may be missed.

Abdominal exploratory is indicated for a variety of problems including foreign body removal, intussusception, gastric mucosal hypertrophy syndromes, procurement of biopsies, and for resection of neoplasia.

*FPLI* is available at Texas A&M University. Serum samples can either be sent directly to the GI Laboratory at Texas A&M University, or they can be forwarded to Texas A&M by a commercial laboratory.

The address is:
GI Lab at Texas A&M University
College of Veterinary Medicine
TAMU 4474
College Station, TX 77843-4474
979-862-2861
[www.cvm.tamu.edu/gilab](http://www.cvm.tamu.edu/gilab)

Diagnosis of Vomiting

Stage 1—Baseline Assessment

- History and physical examination
- Conservative vs. more aggressive diagnostic plan based on patient’s condition and clinician’s concern
**Conservative Approach**
- Fecal examination
- Selected diagnostics
- Specific/symptomatic therapy

**Serious or Systemic Clinical Signs**
- Complete blood count
- Complete biochemical profile
- Urinalysis
- Fecal examination
- Parvovirus test if indicated
- Survey abdominal radiographs (3 views)

- T4 (cats)
- Heartworm antibody and antigen

- Appropriate specific/supportive therapy

**Stage 2—Further assessment** (if vomiting persists or initial tests indicate further investigation should be performed promptly):

- **Special Blood Tests**
  - Corticotropin baseline or ACTH stimulation
  - cPLI or fPLI (pancreatitis)
  - Leptospirosis serology and/or lepto PCR
  - Bile acids assay (to assess liver function)
  - Coagulation tests (consider in patients with hematemesis/melena)

- **Contrast Radiography**
  - Barium contrast
  - Air contrast gastrogram (to further assess for gastric foreign body)
  - BIPS (barium-impregnated polyethylene spheres; with food to assess GI motility)

- **Ultrasonography**
  - Evidence of GI or non-GI disease
  - Aspirates or biopsy
  - Abdominocentesis

- **Nuclear Scintigraphy**
  - Transcolonic portal angiography for detection of portosystemic anomaly
  - GI motility study

**Stage 3—Invasive Procedures**

- **Flexible GI endoscopy** (minimally invasive)
  - Examination, biopsy, foreign body retrieval

- **Laparoscopy**
  - Biopsies (e.g., liver, pancreas)
  - Aspirates (e.g., gall bladder, lymph nodes, mass lesion)
  - Intestinal biopsy

- **Surgical intervention**
  - Therapeutic or exploratory with multiple biopsies
aGI parasites, including *Giardia*, should always be considered in dogs with acute or intermittent vomiting. Best baseline testing on a single fecal sample includes centrifugal flotation and *Giardia* antigen test.

bEndoscopy is a diagnostic or therapeutic tool that can be used in Stage 1, Stage 2, or Stage 3, depending on the clinical situation.

**Further Reading:**
Pharmacologic Control of Acute Vomiting
Initial nonspecific management of vomiting includes NPO (in minor cases a 4-12 hour period of nothing per os may be all that is required), fluid support, and antiemetics. Initial feeding includes small portions of a low fat, single source protein diet starting 6-12 hours after vomiting has ceased. Drugs used to control vomiting will be discussed here.

The most effective antiemetics are those that act at both the vomiting center and the chemoreceptor trigger zone. Vomiting is a protective reflex and when it occurs only occasionally treatment is not generally required. However, patients that continue to vomit should be given antiemetics to help reduce fluid loss, pain and discomfort.

For many years I strongly favored chlorpromazine (Thorazine), a phenothiazine drug, as the first choice for pharmacologic control of vomiting in most cases. The HT-3 receptor antagonists ondansetron (Zofran) and dolasetron (Anzemet) have also been effective antiemetic drugs for a variety of causes of vomiting. Metoclopramide (Reglan) is a reasonably good central antiemetic drug for dogs but not for cats. Maropitant (Cerenia) is a superior broad spectrum antiemetic drug and is now recognized as an excellent first choice for control of vomiting in dogs and cats. In addition to antiemetic effect, maropitant also provides visceral analgesic effect. Maropitant is also the first choice for prevention of motion sickness vomiting in both dogs and cats.

Metoclopramide (Reglan) is a gastric prokinetic drug that also has central antiemetic effect. Metoclopramide increases gastric and proximal small intestinal motility and emptying without causing acid secretion, decreases enterogastric reflux, and provides inhibition of the chemoreceptor trigger zone. The central antiemetic effect is mediated through antagonism of dopaminergic D2 receptors in the chemoreceptor trigger zone of the medulla to inhibit vomiting induced by drugs, toxins, metabolic disease, and acid-base imbalances. Metoclopramide is a less effective central antiemetic drug in cats than in dogs because serotonin receptors, rather than dopaminergic receptors, predominate in the CTZ of cats. For vomiting in cats, I generally usually use metoclopramide only if a prokinetic effect is desired. Chlorpromazine, dolasetron, ondansetron, or maropitant should be used as a first or second choice to control acute frequent vomiting in cats. Parvovirus can cause gastric hypomotility and therefore the promotility effects of metoclopramide may prove beneficial. However, maropitant, dolasetron, or ondansetron are more effective drugs than metoclopramide for managing vomiting caused by parvovirus. Further, maropitant also helps provide visceral analgesia and is the best single drug choice in parvo cases.

The recommended injectable dose of metoclopramide is 0.2 to 0.5 mg/kg IM or SC given TID to QID as needed. Metoclopramide can also be given IV as a constant rate
infusion (1 - 2 mg/kg over 24 hours). Metoclopramide should not be used if gastric outlet obstruction or GI perforation is suspected, or in patients with a seizure disorder.

**Metoclopramide - Clinical Applications for Chronic Vomiting**

Several clinical applications for use of metoclopramide in dogs with chronic vomiting have been identified. These include gastric motility disorders, gastroesophageal reflux disease (GERD), primary or adjunctive therapy for antral and pyloric mucosal hypertrophy, and as treatment for nausea and vomiting caused by various other disorders. While cisapride is a superior prokinetic drug, metoclopramide is an effective drug and is often the first choice for prokinetic effect, with cisapride used as a second choice if metoclopramide is not effective. Other drugs that are sometimes used for prokinesis are low dose erythromycin and the H2-receptor blocker ranitidine (Zantac).

Gastric motility disorders have been recognized with increased frequency in veterinary medicine, but are still overlooked. Gastric stasis, characterized by abdominal discomfort, periodic bloating, borborhygmus, nausea and vomiting may be associated with a number of clinical states that include inflammatory disorders (e.g., chronic gastritis, IBD), gastric ulcers, gastroesophageal reflux, infiltrative lesions (e.g., neoplasia), and chronic gastric dilatation. Metabolic disturbances that may cause gastric stasis include hypokalemia, hypercalcemia, acidosis, anemia, and hepatic encephalopathy. Short-term continued vomiting that is observed in some cases after apparent recovery from viral enteritis may be due to abnormal gastric motility. Transient (3 to 14 days) gastric hypomotility may also occur after gastric or abdominal surgery. Motility disorders with no organic cause may be best classified as idiopathic. For any of the disorders listed, the primary cause should be treated, and metoclopramide may be a valuable short-term adjunct to therapy in these cases, along with feeding low fat foods in divided amounts. Metoclopramide alternatively may be used as the primary treatment on a long-term basis for idiopathic hypomotility disorders. Metoclopramide has also been useful in treatment of dogs that have chronic vomiting characterized by episodes occurring routinely in the early morning and containing bilious fluid.

In general, patients less than 4.5 kg (10 lb) receive 2.5 mg per dose, 4.5 to 18 kg (11-40 lb) 5 mg per dose, and greater than 18 kg (40 lb) 10 mg per dose. Metoclopramide is given 30 to 45 minutes before meals and again at bedtime. Animals that require chronic medication may need only 1 to 2 doses daily. Because of its short half-life, the drug is not effective when given by intravenous or intramuscular bolus injection for purposes other than when only one treatment would be administered (i.e., to aid in evacuating the stomach if an anesthetic procedure in a non-fasted patient becomes necessary, pre-radiologic contrast study). Subcutaneous administration into fat may be of benefit when oral therapy is contraindicated and an intravenous line is not available.

Metoclopramide is less effective as a promotility drug than cisapride (see later discussion). While many animals with gastric hypomotility respond well to metoclopramide, some have a less than desired response. If a patient with a suspected gastric hypomotility disorder has an inadequate response to metoclopramide, cisapride should be tried next.
Side Effects
Some adverse effects may occur if metoclopramide is given in the usual therapeutic doses. Clients should be apprised of these before the medication is prescribed. These effects are uncommon in animals, and somewhat more common in humans.

Motor restlessness and hyperactivity may occur; and when observed, these signs usually begin 20 to 30 minutes after a dose and last 4 to 5 hours. The reaction can range from mild to quite dramatic. Alternatively, drowsiness and depression occasionally occur. Side effects are infrequent in cats, but clients have reported disorientation, frenzied behavior, and hiding tendencies associated with the medication. Hospitalized animals may chew excessively at catheter sites or be more aggressive toward hospital staff. Sometimes these effects are subtle and nursing staff need to be observant. These side effects are reversible (diphenhydramine [Benadryl 2.2 mg/kg IV] or discontinuing the drug) but generally do not subside when lower doses are given. Unless side effects are infrequent, the use of metoclopramide should be discontinued if adverse reactions are seen. Cisapride does NOT cause these same type of adverse reactions. Metoclopramide crosses the blood brain barrier, cisapride does not.

In general, metoclopramide should not be given to epileptic patients. Other contraindications include evidence of significant mechanical obstruction, simultaneous use of anticholinergic agents (antagonism of metoclopramide’s effects), and pheochromocytoma.

Ondansetron - Clinical Applications for Acute Vomiting
Ondansetron (Zofran) is a potent antiemetic drug that has proven to be effective in both humans and animals for control of severe vomiting. It has been used in human cancer patients undergoing cisplatin therapy, a drug that frequently causes nausea and severe vomiting, with very good results. Ondansetron acts as a selective antagonist of serotonin S3 receptors (a principal mediator of the emetic reflex). S3 receptors are found primarily in the CTZ, on vagal nerve terminals, and in the gut in enteric neurons. The principal site of action of ondansetron is in the area postrema, but it also has some peripheral gastric prokinetic activity.

In my experience, ondansetron has produced very good results in either controlling or at least significantly decreasing the frequency of vomiting in dogs and cats with frequent or severe vomiting, including in dogs with severe parvovirus enteritis, in pancreatitis patients, and cats with hepatic lipidosis. The recommended dose is 0.5 to 1 mg/kg IV given as a slow push every 6 to 12 hours (based on patient response). Frequently dogs that appear quite distressed due to nausea and vomiting look much more relaxed and comfortable within 15 minutes of receiving ondansetron. There are no reports of any significant side effects such as diarrhea, sedation, or extrapyramidal signs in human and animal trials. While Zofran was quite expensive for many years, it came off patent in 2007 and is now more affordable for use at any small animal hospital. Currently, however, my top antiemetic drug of choice is maropitant (Cerenia), because it is a highly effective antiemetic drug but also because it provides visceral analgesic effects as well. Animals with significant liver disease may be best managed with ondansetron or dolasetron, as maropitant should be used with caution in animals.
with significant hepatic dysfunction (although it is not contraindicated – some clinicians have used maropitant successfully and safely in animals with liver disease).

**Dolasetron**

Dolasetron (Anzemet) is also a 5-HT3 receptor antagonist antiemetic drug, with action similar to ondansetron. It is a slightly less expensive alternative to ondansetron and only needs to be administered once daily. Indications are the same as for ondansetron, namely, for control of frequent vomiting that is poorly responsive to lesser expensive front-line antiemetic drugs. The dose is 0.5-1 mg/kg IV once daily. Dolasetron is generally well tolerated in animals.

**A newer antiemetic drug for dogs**

Most drugs used to control vomiting in animals have been developed for use in humans. There has been a need for a broad-spectrum antiemetic drug for use in animals that is effective in a variety of situations, has a rapid onset of action, is safe and affordable, and is available in both injectable and oral preparations. **Maropitant citrate (Cerenia)** is a newer broad-spectrum antiemetic drug that is indicated for the treatment of acute vomiting in dogs. Maropitant is a neurokinin receptor antagonist that blocks the pharmacologic action of the neuropeptide substance P in the central nervous system. Substance P is found in significant concentrations in the nuclei comprising the emetic center and is considered a key neurotransmitter involved in emesis. By inhibiting the binding of substance P within the emetic center, maropitant provides broad-spectrum effectiveness against both neural and humoral causes of vomiting.

Clinical trials and recent clinical experience, since August 2007 when the drug was released for use in the U.S., have shown maropitant to be very effective for control of a variety of causes of acute vomiting in dogs. It is administered as a once-daily injection (0.45 mg/lb [1 mg/kg] SC for dogs), which is a significant advantage over many other antiemetic drugs, and has a rapid onset of action. Maropitant is also available in tablet form for outpatient use, which makes it a very attractive choice for use in small animal practice. It is the drug of choice for dogs with motion sickness.

**CAUTION:** We generally advise that Cerenia be used at a reduced dose (50%) for animals with significant hepatic dysfunction, OR select an alternative antiemetic for animals with liver disease – e.g., ondansetron or dolasetron.

**The issue of stinging on injection:** Information from clinical experience and studies indicates that there is less likelihood for stinging to occur with maropitant injections when the product is kept refrigerated. The current guidance is that the solution should be kept refrigerated and drawn up and injected right away at refrigerated temp. In practice a sting can still be expected in some patients even when the product is kept refrigerated.

**CATS:** Studies have now been done using maropitant in cats and some clinicians in general practice have been using it since 2008. In May 2012 Cerenia was approved for use in cats and also in puppies as young as 8 weeks of age.

**Recommended dose of maropitant for cats:**
Injectable: 0.5-1 mg/kg SC or IV (give SLOWLY over 60-90 seconds if administering IV) Oral: (1 to 2 mg/kg). This is the starting dose recommended for prevention of motion sickness in cats as well; i.e., somewhat lower than the canine dose for motion sickness.

**Note: On January 14, 2016, Zoetis announced a new label claim for IV use of Cerenia.** In two separate bioequivalence studies conducted in 2015 by Zoetis in dogs and cats, when delivered intravenously, CERENIA reached concentration and absorption levels as quickly as with subcutaneous injection. Additionally, two separate safety studies in dogs and cats indicated no related effects on survival or clinical findings, and there were no reports of pain on intravenous injection.

**Consider Using Cerenia More Routinely Administered PRE-Operatively**
Some practices have now instituted the practice of including an injection of Cerenia administered routinely in the pre-operative period. I am a strong proponent. Reasons for doing this include:
- Help prevent post-op vomiting and nausea and decrease chances of aspiration
- Adjunctive visceral analgesia
- Improved patient comfort in the post-op period
- Earlier return to eating, with improved appetite and volume of food consumption

In this setting, Cerenia can be administered anytime in the pre-op period. If morphine or hydromorphone are going to be given as part of the pre-anesthesia sedation and preemptive analgesia plan, and the clinician desires to prevent vomiting secondary to these emetogenic drugs, Cerenia is administered 45 minutes prior to the emetogenic drugs. In one study when Cerenia was administered 45 minutes prior to morphine at 0.5 mg/kg, 0/15 dogs vomited, while 15/16 dogs who received saline instead of Cerenia vomited at least once (and 4 of the dogs vomited 4 times). We have seen excellent post anesthesia recovery periods in dogs that have undergone a variety of procedures, including OVH/neuter as well as prolonged anesthesia for dental procedures, major abdominal procedures, etc. We are also using Cerenia more routinely prior to performing endoscopic procedures.

The uniform response is that most patients recover more smoothly, more quietly and are presumably more comfortable overall. Clients of course are very happy when their pet eats earlier than would be otherwise expected. This has represented a gratifying advance in patient care in many ways — helping our patients be more comfortable is always good.

**How long can Cerenia be used on a consecutive days schedule?**
The original label guidance stated that Cerenia should not be given for more than 5 consecutive days (injectable or oral at the anti-emesis dose) and for 2 days at the motion sickness prevention dose. However, experience has shown that in some patients Cerenia has been used safely and effectively on a longer term basis (anecdotal reports, e.g., patients with neoplasia or renal disease that were experiencing ongoing nausea, vomiting, and inappetence). Many of these patients have a much better quality of life while on Cerenia, as they have less nausea and vomiting and a much better appetite. There are cats that have been treated with a daily oral dose for months to several years. Use of Cerenia in this fashion is being investigated further.
Further, in 2015 the label was changed, based on studies that evaluated the effect of maropitant when given at various doses for longer periods of time. Cerenia has a high safety profile and a longer duration of use, based on each patient’s individual needs, is now well accepted.

A study was presented at the Veterinary Cancer Society (VCS) meeting in San Diego Oct. 29-November 1, 2010, and then subsequently at the ACVIM Forum in Denver in June 2011:

Pharmacokinetics of maropitant citrate dosed orally to dogs at 2 mg/kg and 8 mg/kg once daily for 14 consecutive days. Two groups of eight healthy beagle dogs were administered maropitant citrate at 2 or 8 mg/kg orally once daily for 14 days. Concentrations of maropitant and its metabolite were measured in plasma using a LC-MS/MS assay. Pharmacokinetic parameters were estimated using non-compartmental pharmacokinetic techniques and a modeling approach was used to estimate steady-state.

Results: The model estimate for the number of doses required to reach 90% of steady-state was 4.30 for 2 mg/kg and 8.09 for 8 mg/kg. Four dogs experienced a single dose of vomiting.

Conclusions: Dosing maropitant citrate beyond the original label duration of 5 days was well tolerated by healthy dogs. During the 14 days of dosing there was accumulation, however, steady-state was reached after approximately 4 doses for daily 2 mg/kg dosing and 8 doses for daily 8 mg/kg oral dosing.

Use of Oral Maropitant (Cerenia)

- Confident there is no GI foreign body (i.e., do not use ongoing antiemetic therapy if there could be a foreign body ledge in the GI tract)
- Prevent vomiting during cyclosporine, azithromycin, or other drug induction period (use for 3-5 days in conjunction with the start of a drug that might cause vomiting)
- Vomiting flare-ups in IBD patients (or other chronic disorders)
- Pancreatitis, parvovirus, etc for a few days after vomiting is fairly well controlled with injectable maropitant. Excellent control of nausea may help improve appetite and earlier food intake
- Prevention of vomiting in chemotherapy patients
- Prevention of motion (“car”) sickness
- Renal disease patients – and perhaps chronic use (these patients may benefit tremendously and we have observed many patients that eat better, do not vomit or exhibit nausea, and feel better overall. Studies are ongoing).
Cisapride
Cisapride is a potent GI prokinetic drug and is superior in action to metoclopramide. It is no longer on the market for use in humans, as of 2000, because of an association with fatal arrhythmias. There are no reports of similar complications existing in dogs and cats, however, and cisapride continues to be readily available to veterinarians through compounding pharmacies.

Cisapride has broader promotility effects than metoclopramide (e.g., cisapride has demonstrated excellent efficacy in management of colonic inertia and small intestinal ileus). In contrast to metoclopramide, which has central effect at the CRTZ in addition to its peripheral effects, cisapride has no known direct antiemetic properties. Another contrast is that metoclopramide's prokinetic effect is most significantly on the stomach. It is NOT a reasonable choice for treatment of small intestinal ileus.

The most relevant uses of cisapride in animal patients include treatment of gastroparesis, especially in patients that experience significant side effects from metoclopramide (e.g., hyperactivity and other dystonic reactions) or where metoclopramide is not sufficiently effective, idiopathic constipation, gastroesophageal reflux disease (if H2-receptor antagonists or proton pump inhibitors and dietary management alone are not effective), and postoperative ileus.

Cisapride is extremely well tolerated by animal patients. I have used cisapride in dogs and cats that have experienced neurologic side effects from metoclopramide. I have observed no adverse reactions to cisapride in any of these patients, even in those whose side effects to metoclopramide included very bizarre behavior changes. The suggested dose of cisapride is similar to what has been recommended for metoclopramide (see earlier discussion).

Table 1
Comparison of Actions of the Prokinetic Drugs Metoclopramide and Cisapride

<table>
<thead>
<tr>
<th>Metoclopramide</th>
<th>Cisapride</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Prokinetic Spectrum:</strong></td>
<td><strong>Prokinetic Spectrum:</strong></td>
</tr>
<tr>
<td>Stomach only, not effective for small intestinal ileus</td>
<td>Stomach, small intestine, large intestine</td>
</tr>
<tr>
<td><strong>Prokinetic “strength”</strong></td>
<td><strong>Prokinetic “strength”</strong></td>
</tr>
<tr>
<td>Moderate</td>
<td>Superior</td>
</tr>
<tr>
<td><strong>Central antiemetic effect</strong></td>
<td><strong>Central antiemetic effect</strong></td>
</tr>
<tr>
<td>Moderate (maropitant, ondansetron, dolasetron are superior)</td>
<td>None</td>
</tr>
<tr>
<td>Side effects:</td>
<td>Side effects:</td>
</tr>
<tr>
<td>--------------</td>
<td>--------------</td>
</tr>
<tr>
<td>Tardive dyskinesia, jittery behavior, somnolence</td>
<td>No significant adverse effects in animals, does not cross blood brain barrier</td>
</tr>
</tbody>
</table>
Introduction
Pancreatitis is a common disorder of dogs but due to challenges in establishing a definitive diagnosis the true incidence of pancreatitis seen in clinical practice is not known. Mild cases of pancreatitis that do not show “classical” signs such as acute vomiting and abdominal pain can be very difficult to diagnose. Conversely, pancreatitis is just one of many causes of vomiting and clinicians are challenged daily to try to determine a specific cause in patients presented with vomiting and other clinical signs. The incidence of histologically confirmed pancreatitis in cats and dogs ranges from 1.3-1.5%, respectively. Most dogs have acute pancreatitis and most cats have chronic pancreatitis. Clinical presentation and diagnostic criteria differ between species and type of pancreatitis. Acute pancreatitis is potentially reversible but can also be fatal while chronic pancreatitis generally has irreversible changes but is rarely fatal.

Acute Pancreatitis in the Dog
There is confusion and controversy regarding pathogenesis, diagnosis, and treatment of acute pancreatitis in the dog. The spectrum of clinical disease can range from mild signs to those which are fulminant and frequently fatal. It is the development of multisystemic abnormalities that separates mild from severe, potentially fatal pancreatitis. Pancreatitis can be broadly categorized as acute, recurrent acute, or chronic. Medical management is the main form of treatment in most cases. Surgical intervention in diagnosis and management of pancreatitis is not often undertaken and is generally reserved for complex cases of pancreatitis (i.e., acute necrotizing pancreatitis, pancreatic abscess, pancreatic pseudocyst, and generalized peritonitis secondary to pancreatitis).

Etiology
The etiology of pancreatitis is generally unknown. Factors that influence development of pancreatitis include high fat diet, obesity, hyperlipoproteinemia, drugs (e.g., thiazides, furosemide, tetracycline, L-Asparaginase, azathioprine, corticosteroids), duodenal reflux into pancreatic ducts, pancreatic duct obstruction (e.g., parasites, calculi, neoplasia, inflammation, surgery), hypercalcemia, trauma, and pancreatic ischemia (e.g., shock, GDV).

Risk Factors
Risk factors identified in patients with acute pancreatitis include breed (Yorkshire and Silky terrier, toy poodle, miniature Schnauzer, nonsporting breeds), overweight body condition, small breed size, prior gastrointestinal disease, diabetes mellitus, hyperadrenocorticism, hypothyroidism and a history of surgery within two weeks prior to development of pancreatitis.

Diagnosis
Acute pancreatitis is one of the most difficult diseases to diagnose. There is no one definitive diagnostic test for pancreatitis, except for histopathology. Biopsy procedures are not commonly done in dogs with suspected acute pancreatitis. Clinical diagnosis is based on a combination of history, physical examination, and compatible clinicopathologic and imaging findings. The clinician’s index of suspicion is important and keeping an open mind to various possibilities is essential (i.e., avoid “tunnel vision”), since multiple organs can be involved concurrently. When vomiting is associated with systemic signs or is persistent the clinician must differentiate metabolic, polysystemic, toxic, and infectious causes from abdominal causes. Pancreatitis can be “diagnosed” when it is not actually present and easily missed when it is present. Most patients with suspected pancreatitis are managed based on the patient’s condition (mild vs. severe signs) and the various test results and closely monitoring response to therapy.

Clinical factors that help support a diagnosis of pancreatitis include high index of suspicion, acute or chronic vomiting, weakness, abdominal pain, dehydration, diarrhea, fever and shock. There may be a history of recent dietary indiscretion or drug administration. There is no apparent sex predilection. Middle-age to older dogs (greater than 5 years) that are overweight are at higher risk.

**Laboratory Findings**

Laboratory findings in dogs with pancreatitis are quite variable and to some extent parallel the severity of the clinical disease and may be representative of multiple organ system involvement in patients with severe pancreatitis. Laboratory results might include leukocytosis, azotemia (prerenal and renal), increases in ALT, AST and ALP, icterus, hyperglycemia, hypokalemia, acid base changes, DIC, and increases in amylase, lipase, concentration of trypsin-like immunoreactivity (cTLI), and pancreatic lipase immunoreactivity (cPLI).

Hematologic findings are quite variable, ranging from mild neutrophilia and slightly increased hematocrit (dehydration) to marked leukocytosis with a left shift, leukopenia with a degenerative left shift, and anemia. However, some dogs with even moderate to severe pancreatitis can have a normal leukogram. Platelet numbers should be assessed. If there is thrombocytopenia, tests for DIC should be performed (one stage prothrombin time (OSPT), activated partial thromboplastin time (APTT), fibrin degradation products (FDP or D-Dimer), and antithrombin III.

Urinalysis allows azotemia to be characterized as prerenal or renal and provides key information for ruling-in or out other disorders (e.g., diabetes mellitus [glucosuria or ketonuria], pyelonephritis which can cause vomiting and abdominal pain [absence of white cell casts or bacteria helps rule-out]).

Serum amylase and lipase activities are insensitive and nonspecific for pancreatitis. Specificity for both tests is only around 50%. Amylase and lipase are found in the oral cavity, GI tract, pancreas, and liver. Dogs with gastritis or intestinal foreign bodies can have a significantly elevated lipase but no gross evidence of pancreatitis. Lipase is produced by the gastric mucosa which explains why it can be increased in disorders of gastric inflammation. Increases in amylase and lipase can be seen in renal disease as well (decreased clearance). Significantly increased amylase or lipase can certainly
suggest the possibility of pancreatitis, but, some dogs with even severe pancreatitis have demonstrated normal levels. All things considered, amylase and lipase assessment are not very useful in the diagnosis of pancreatitis.

The immunoreactive canine pancreatic lipase assay (cPLI) is the most sensitive assay currently available. Use of the immunoassay allows for the specific measurement of lipase originating from the exocrine pancreas. cTLI is still the test of choice for exocrine pancreatic insufficiency, but it is not very sensitive for pancreatitis (less than 35% sensitivity in dogs with proven pancreatitis). The sensitivity for serum cPLI concentration is over 80%. cPLI can be used for diagnosing pancreatitis in dogs with renal failure. A study has shown that, while cPLI is elevated in dogs with renal failure compared with healthy dog levels, it is usually still within the reference range (in the higher end of the reference range), but not above the currently recommended cutoff value for pancreatitis. The long term oral administration of prednisone does not affect cPLI values. Currently, the combination of a quantitated cPLI assay and ultrasound are considered to provide the most meaningful noninvasive diagnostic information in the clinical setting for determination of whether or not pancreatitis is present.

**Imaging of the Pancreas**
(This section on imaging is authored by David S. Biller, DVM, DACVR, Kansas State University)

**Radiology**
The pancreas is shaped like a boomerang. There are three parts: the right lobe, which lies adjacent to the descending duodenum; the body, which lies at the junction between the pylorus and duodenum; and the left lobe, which lies adjacent to the greater curvature of the stomach. The pancreas is not normally seen.

With a diseased pancreas, the duodenum may be displaced ventrally. On the ventrodorsal view, there is usually lateral (right) displacement of the descending duodenum and the pylorus is displaced to the left (widening of the duodenal pyloric angle). The transverse colon as well as ascending colon may be displaced centrally and caudally. Fluid and gas distention of the stomach, duodenum and colon may be present. Localized loss of abdominal detail (ground-glass appearance) may occur with inflammation of the pancreas. The most common change radiographically on survey films is actually no change at all.

Changes that may occur and be visualized relative to pancreatic disease and an upper GI series include fixed position and shape to the duodenum; widened proximal duodenal flexure (the angle between the pylorus and duodenum); and thickening and rigidity of the duodenum, pylorus, and greater curvature of the stomach. Gastric outflow obstruction and duodenal distention (ileus) may also be visualized.

**Ultrasonography**
Ultrasonography is the imaging method of choice for evaluation of the pancreas in small animals. It can provide information about the size, shape, and contour of the pancreas and may suggest the presence of inflammation, abscess formation, or neoplasia. Ideally, patients should be fasted before abdominal ultrasonography to minimize interference from GI gas.
There are several limitations to pancreatic ultrasonography:

- The normal pancreas is not always seen as a discrete structure; thus, it is actually the pancreatic area, and not the organ itself, that must be examined.
- Ultrasonography lacks specificity. For the most part, ultrasonographic findings do not allow differentiation between inflammatory and neoplastic processes.
- The proximity of the pancreas to gas in the stomach, colon, and duodenum may prevent complete and accurate evaluation of the pancreatic region.

Despite these disadvantages, ultrasonography can provide valuable diagnostic information in most animals with inflammatory or neoplastic diseases of the pancreas if it is done properly and with patience. The healthy pancreas is difficult to image as a distinct organ. Therefore, familiarity with the anatomy of adjacent structures is crucial to successful evaluation of the pancreatic area.

The left lobe of the pancreas is dorsocaudal to the stomach and dorsocranial to the transverse colon. Its distal aspect can be visualized cranial to the left kidney and medial to the spleen. The pancreatic body lies caudal to the pylorus. It is ventral to the portal vein and the caudate process of the liver and craniomedial to the right kidney. The right lobe of the pancreas is found dorsomedial to the descending duodenum, ventral to the right kidney, and ventrolateral to the portal vein. Both the cranial and caudal pancreaticoduodenal veins are located in the parenchyma of the right lobe and run parallel to the descending duodenum.

Normally, pancreatic parenchyma has a homogeneous echotexture. The pancreaticoduodenal vein may be apparent in the right lobe. The left lobe of the healthy pancreas occasionally is seen in the triangular region defined by the spleen, stomach, and left kidney. Gas in the adjacent stomach and transverse colon makes imaging of all but the most distal portion of the left lobe difficult.

**Ultrasonography Findings in Pancreatitis**

Ultrasonography has proven to be a reliable tool for identifying changes associated with pancreatic inflammation and is currently considered the imaging method of choice for evaluating pancreatitis and pancreatic neoplasia.

Ultrasonography provides several distinct advantages in the diagnostic evaluation of pancreatitis:

- It can identify abnormalities in animals with pancreatitis. In many cases, it also provides information about the severity of inflammation.
- It is noninvasive and can be repeated frequently, providing a means of following disease progression and/or resolution.
- It allows evaluation of peripancreatic structures, such as the biliary system, duodenum, and stomach, which are often secondarily involved in acute pancreatitis.
- It is an important tool for identifying complications of pancreatitis, such as biliary obstruction, abscess formation, and pseudocyst formation.
The ultrasonographic findings that characterize pancreatitis represent changes in either the pancreas itself or in peripancreatic structures. The most common ultrasonographic abnormality noted in animals with pancreatitis is a hypoechoic mass dorsomedial to the descending duodenum and caudal to the stomach. This mass represents the inflamed pancreas, and although its overall echogenicity is usually decreased, it may sometimes appear inhomogeneous.

The peripancreatic mesentery and associated fat are often hyperechoic but may have variable echogenicity. The edges of the pancreas are distinct if the inflammation is mild but become poorly defined when severe pancreatitis is present, probably as a result of the edema, necrosis, and hemorrhage that accompany severe pancreatic inflammation. The overall pancreatic image tends to become better defined with more distinct edges as inflammation subsides. Improved resolution of the pancreatic margins may be partially related to saponification of surrounding fat. The ultrasonographic changes associated with chronic pancreatitis are often less severe than those associated with acute pancreatitis, although it is difficult to differentiate one condition from another based on ultrasonography alone.

Changes in peripancreatic structures also contribute to the ultrasonographic diagnosis of pancreatitis. Free peritoneal fluid secondary to focal peritonitis may be apparent in the pancreatic region. The descending duodenum typically becomes dilated and fluid-filled, with thickened walls and no apparent peristalsis. With severe duodenitis, the duodenal wall may have a corrugated appearance.

Potential complications of pancreatitis include pseudocyst or phlegmon formation, abscess formation, and biliary obstruction. Pancreatic phlegmons are edematous masses of indurated pancreas and adjacent tissue with varying degrees of necrosis that develop within several days of the onset of acute pancreatitis. They may resolve spontaneously or may cause persistent fever and abdominal pain. Pancreatic abscesses result from secondary infection of necrotic pancreatic tissue or of a phlegmon. Ultrasonographically, pancreatic phlegmons and abscesses appear as pancreatic masses of mixed echogenicity and variable size. Gas in a pancreatic mass, identified as an echogenic interface with reverberation, suggests abscess formation.

Pancreatic pseudocysts appear ultrasonographically as primarily anechoic masses but they may contain some internal echoes. They cause mild acoustic enhancement of distal structures. Unfortunately, ultrasonography cannot usually differentiate between pancreatic phlegmons, abscesses, or pseudocysts. Extrahepatic biliary obstruction is another complication of acute pancreatitis that may necessitate surgery.

**Cytology**

Fine needle aspiration of suspected areas of pancreatitis and suppurative inflammation may help support the diagnosis.

Abdominocentesis may be helpful if effusion is present. Suppurative inflammation is the typical finding, but it is rarely septic. In addition, abdominal fluid analysis combined with measurement of abdominal fluid lipase concentration is helpful. Abdominal fluid lipase concentration higher than serum lipase concentration supports the diagnosis of pancreatitis in many cases.
Histopathology
Biopsy provides the definitive diagnosis. Surgery or laparoscopy is generally required to obtain a diagnostic biopsy. Pancreatic biopsy techniques are generally safe, given careful tissue handling. The primary consideration in obtaining biopsy samples is whether or not a patient with suspected acute pancreatitis is a safe anesthetic risk and whether or not the step of obtaining tissue samples from a patient will add significantly contributory information to patient management to justify any risks and also costs related to performing a biopsy procedure. Most patients that undergo pancreatic biopsy are examined during the course of a laparoscopic or surgical exploratory procedure in which various areas of the abdomen are being examined and biopsied (e.g., liver, intestines).

Although acute pancreatitis is not generally considered a surgical condition patients progressing to a more severe necrotizing pancreatitis with pancreatic abscess and peritonitis may be candidates for exploratory laparotomy.

Medical Management
Initial treatment of pancreatitis should be supportive and should be tailored for the individual patient, taking into consideration any abnormalities detected on physical examination and testing. Basic therapy involves correction of fluid and electrolyte imbalance, control of nausea and vomiting, pain management, nutritional considerations (early feeding is the goal), and the control of secondary complications.

Management considerations for severe and often life-threatening acute pancreatitis include pancreatic rest in the form of fasting for two to four days for vomiting patients (try to minimize the fasting period), fluid and electrolyte therapy and in some cases colloid administration, and antibiotics for severe cases or whenever there is evidence of sepsis or concurrent liver disease (antibiotics are not indicated for all cases of pancreatitis). Plasma or whole blood may be indicated in some cases. Antiemetics are given to control vomiting and decrease fluid loss and enhance patient comfort (i.e., control nausea and hopefully allow the patient to rest more and eat earlier). Maropitant (Cerenia) given at 1 mg/kg once daily SC is a highly effective antiemetic drug that also provides visceral analgesic effect, making it a very attractive antiemetic drug to use in patients that have visceral pain. Use of analgesics should be strongly considered in the patient with pancreatitis even if there is no outward evidence of abdominal pain (e.g., buprenorphine for mild pain; and morphine, hydromorphone, or fentanyl for moderate to severe pain). Constant rate infusion of pure opioid drugs is an ideal way to more effectively control pain consistently hour by hour. Lidocaine infusions can also be administered when additional pain control is needed. Careful attention to detail on pain control is absolutely essential and too often inadequate therapy is administered in this area.

Why is Earlier Feeding Important?
Nutritional support of critically ill patients has long been considered a supportive measure of low priority. Recent advances in both human and veterinary medicine have demonstrated that nutritional support is an important therapeutic modality and can aid in the management of many diseases. In diseased states, the inflammatory response triggers alterations in cytokines and hormone concentrations and shifts
metabolism toward a catabolic state. With a lack of food intake, the predominant energy source is derived from accelerated proteolysis, which in itself is an energy-consuming process. Thus, critically ill animals may actually preserve fat deposits in the face of lean muscle tissue loss. The goal of nutritional support in these catabolic patients is to feed the catabolism with exogenous sources of protein and fat thereby sparing endogenous protein which is critical to recovery.

Malnutrition in veterinary patients is thought to increase morbidity and mortality, but this has not been statistically quantified. However, nearly every body system is affected by negative energy balance. In the GI tract transit times increase, absorptive capabilities decrease, and there is an increased risk of bacterial translocation. In the kidneys, excretion of urinary calcium and phosphorus increases, ability to excrete acid decreases, gluconeogenesis increases and glomerular filtration rate decreases. Malnutrition has been documented to decrease humoral immunity and barrier function (skin and mucosal surfaces), inflammatory response, leukocyte motility and bactericidal activity. Patients are at risk for pulmonary complications as a result of decreased response to hypoxia, decreased lung elasticity, and secretin production, altered permeability and decreased tidal volume. Cardiovascular complications include increased incidence of arrhythmias and decreased weight of the heart muscle. Protein calorie malnutrition may also alter the normal or expected metabolism of certain drugs, which may increase or decrease their therapeutic effect even when given at recommended dosages.

**Feeding Dogs with Pancreatitis**

Critical illnesses associated with gut barrier dysfunction include severe acute pancreatitis, inflammatory and non-inflammatory bowel disease, severe burn injury, multisystem trauma and high risk surgery. Gut barrier dysfunction can exacerbate critical illnesses by leading to bacteremia, endotoxemia, systemic inflammatory response syndrome and multiple organ dysfunctions. The nutritional management of these disorders has traditionally included an initial period of starvation, ranging from 3 to 7 days. However, the most important stimulus for intestinal mucosal growth, repair, and integrity is the presence of nutrients within the gut lumen. The absence of luminal nutrients leads to marked small intestinal mucosal atrophy and suppressed crypt cell proliferation, marked reductions in gut-associated lymphoid tissue cell mass and function, increased intestinal permeability to bacteria and toxins, and enhanced pro-inflammatory cytokine generation and acute-phase responses. For pancreatitis the recommendation for starvation is based on the belief that “strict pancreatic rest” prevents stimulation of exocrine pancreatic secretion, thus protecting against autodigestion. Recently, this recommendation has come into question. Studies in dogs, rodents and man have demonstrated that exocrine pancreatic excretion is already inhibited by the inflammation associated with pancreatitis and feeding has no impact on exocrine pancreatic secretion. A systematic review of human literature found that patients with acute pancreatitis receiving enteral nutrition have fewer episodes of death, systemic infections, multiple organ failure and operative interventions. This data suggests that enteral nutrition (EN) should be considered the standard of care for patients with acute pancreatitis requiring nutritional support.

Recent studies support the recommendation that one of the management priorities in treating acute pancreatitis should be to feed early and enterally. Prolonged fasting
leads to immunosuppression, decreased wound healing and increased bacterial translocation, sepsis and decreased survival. Ideally, canine patients should not be held NPO for more than 48 to 72 hours including the time they were anorectic prior to presentation. Once vomiting is adequately controlled, feeding should be instituted. Enteral feeding improves enterocyte health and immune function. Documented benefits of maintaining a healthy gut barrier function include reduced mucosal permeability, reduced incidence of bacteremia, endotoxemia and septic morbidity, attenuation of the acute phase response and reduced incidence of multiple organ failures, reduced catabolism and preservation of a positive nitrogen balance and perhaps most importantly improved clinical outcomes. Early enteral nutrition is superior to both starvation and total parenteral nutrition in critical illnesses associated with gut barrier dysfunction.

One recent study comparing enteral and parenteral nutrition in dogs with acute pancreatitis documented a significantly greater number of vomiting or regurgitation episodes in dogs receiving parenteral nutrition (PN) versus enteral nutrition. Additionally dogs receiving enteral nutrition did not demonstrate any noticeable postprandial pain. There were more catheter-related complications in the PN group. The authors concluded early EN delivered proximal to the pylorus is well tolerated in dogs with severe pancreatitis and resulted in fewer complications than PN.

Depending on the situation, enteral feeding can be done either per os or via esophagostomy, nasoesophageal, or jejunostomy tube. Currently, prepyloric feeding via esophagostomy tube seems to be well tolerated and this route is certainly easier than placing a jejunostomy tube. Elemental diets or polymeric diets can be fed via tube. Vanilla Ensure is specifically preferred in canine pancreatitis patients because it is lower in fat compared to other commonly used liquid diets (e.g., Clinicare). Vanilla Ensure is not designed for longterm feeding but it is fine for the initial feeding stage in pancreatitis. When beginning to feed solid food the diet should be a carbohydrate-rich and low-fat food given as small frequent meals. Continued fat restriction (longterm maintenance feeding) is usually recommended for dogs that have had more than one bout of pancreatitis. For dogs with a single bout of pancreatitis, a low fat diet should be few for the first month or two and then if desired the patient can usually be returned to its regular diet if so desired.

OTHER POTENTIAL THERAPIES
Unproven therapy should be considered only after careful evaluation of the individual case and may include corticosteroids, somatostatin, a hormone that decreases pancreatic secretion, and low dose dopamine, which was found to preserve vascular permeability during experimental pancreatitis in cats and may be a beneficial adjunctive therapy in the management of clinical pancreatitis. Antioxidants may be of benefit in the acute management of pancreatitis; vitamin E is a potent membrane antioxidant and SAMe replaces glutathione stores that may have some benefit in pancreatitis, peritoneal lavage removes inflammatory products in the peritoneal cavity before they are absorbed, and pancreatic enzyme supplementation has been reported to decrease the pain that accompanies chronic pancreatitis in humans by the feedback inhibition by endogenous pancreatic enzyme secretion. It is not known if enzymes are helpful in the acute cases but such supplementation may have some benefit in early nutrition of patients with acute pancreatitis.
Hyperbaric oxygen therapy (HBOT) can also be very helpful in patients with severe pancreatitis (shown to be beneficial in both humans and animals). Physiological effects include:

- Oxygen delivered to the alveoli under increased atmospheric pressure results in large increases in the amount dissolved in plasma
- Increase in neovascularization
- Enhancement of WBC oxidative killing and antibacterial effects
- Inhibition of neutrophil adherence to microvascular endothelia resulting in SUPPRESSION of the deleterious cascade of events that follows in ischemia-reperfusion injury
- Modification of cytokine effects (anti-inflammatory)
- Inhibition of free radical formation
- Down regulation of intercellular adhesion molecule (ICAM-1) expression

A 2012 review article on the pathophysiology of pancreatitis by Caroline Mansfield in the Journal of Veterinary Internal Medicine implicates perpetuation of inflammation in pancreatitis by the adhesion of leukocytes to endothelial walls via expression of ICAM-1 and selectins mediated by IL-8. In addition, a disturbance in pancreatic microcirculation with ensuing ischemia is implicated as a major factor in the ongoing cycle of inflammation associated with pancreatitis. For veterinarians who have access on a referral basis to a hospital with a hyperbaric chamber HBOT is an exciting modality that can be used in addition to all of the other high priority therapeutic modalities already discussed.

**Surgical Management of Pancreatitis**

Surgery is rarely indicated for pancreatitis. However, there are situations where surgery may be necessary. Indications may include pancreatic abscess, septic suppurative peritonitis secondary to severe necrotizing pancreatitis, pancreatic pseudocyst, jejunostomy feeding tube placement, and open peritoneal lavage and drainage. Surgery of the pancreas is discussed in current surgery textbooks.

**Conclusion**

Acute pancreatitis can vary in severity of signs and often results in multiple organ system involvement. Despite extensive literature on pathogenesis of pancreatitis and its complications, there have been few notable advances made in its medical and surgical management. It is possible that future research on modification of enzymatic disturbances will result in an effective treatment for acute pancreatitis. Surgical treatment of pancreatitis remains a controversial topic and is generally reserved for patients with severe necrotizing pancreatitis with septic peritonitis or pancreatitis associated with pancreatic mass.

**Further Reading:**
ACUTE AND CHRONIC DIARRHEA IN DOGS AND CATS

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Introduction

Giardia, Clostridium perfringens enterotoxin, and Cryptosporidium are important causes of diarrhea in dogs and cats. Tritrichomonas foetus is an important problem in cats. These disorders should be investigated early in the course of diarrhea, whether it is persistent or intermittent, along with evaluation for dietary causes of GI signs, nematode parasites, bacterial and viral causes, and acute idiopathic colitis. This group of disorders constitutes a thorough differential list for animals with acute and intermittent diarrhea (Table 1).

The challenge to veterinarians is in making an accurate diagnosis, so that the best therapy can be instituted as early as possible. This will then lead to the best opportunity for successful control of the medical disorder. It is also important to recognize that some animals may have several disorders at the same time, so a thorough diagnostic approach is recommended. This is why it is often best to run tests for these disorders at the same time, through use of a “fecal diagnostics panel” that is now available at many commercial laboratories. A single fecal sample is submitted to the lab, and tests for each of these disorders is done at the same time. This provides a prompt and thorough analysis for important clinical disorders of the GI tract. The clinician then has more clear direction on how to proceed with treatment, or other diagnostic tests in the event that none of these disorders is identified.

Table 1: Common Causes of Acute Diarrhea in Dogs and Cats

<table>
<thead>
<tr>
<th>Young Animals</th>
<th>Older Animals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dietary problems</td>
<td>Dietary problems</td>
</tr>
<tr>
<td>Parasites possible</td>
<td>Parasites less common but always</td>
</tr>
<tr>
<td>- nematodes</td>
<td>- nematodes</td>
</tr>
<tr>
<td>- protozoa (Giardia, Trichomonads)</td>
<td>- protozoa (Giardia, Trichomonads)</td>
</tr>
<tr>
<td>- coccidia (including Cryptosporidium)</td>
<td>- coccidia (including Cryptosporidium)</td>
</tr>
<tr>
<td>Viral and bacterial</td>
<td>Viral causes uncommon in older animals</td>
</tr>
<tr>
<td>(CPE)</td>
<td>Clostridium perfringens enterotoxidosis</td>
</tr>
<tr>
<td></td>
<td>(diarrhea often intermittent)</td>
</tr>
<tr>
<td></td>
<td>Acute colitis (fairly common cause of diarrhea in older animals)</td>
</tr>
</tbody>
</table>

Giardia is an important cause of diarrhea, and for some patients other GI signs as well. It is an important pathogen in dogs and cats, as well as humans and other species. Historically, accurate diagnosis of Giardia has posed a significant challenge to veterinary practitioners, but there are now much more sensitive tests readily available.
for veterinarians to use on a routine basis. Because of the impact that this organism can have on animals, and also humans because of its zoonotic potential, it is important that veterinarians perform accurate diagnostic testing on animals to determine whether or not an animal is infected with *Giardia*. These notes will emphasize steps for accurate diagnosis, and also management of giardiasis.

*Clostridium perfringens* enterotoxosis is a common cause of intermittent diarrhea in dogs and cats. Veterinary practitioners should test for the enterotoxin whenever faced with a patient that has unexplained diarrhea.

Cryptosporidiosis is now recognized to be a more common disorder in dogs and cats than was previously thought. It can cause significant abnormalities, and it has zoonotic potential. Cryptosporidiosis can be fatal in people that also are immunosuppressed (e.g., on chemotherapy or corticosteroids, carriers of HIV). Therefore, it is incumbent on veterinarians to test for this disorder, as there are important implications to both the patient as well as to humans who may come in contact with an infected animal.

**Early Diagnostic Screening in Animals with Diarrhea**  
**Diarrhea – Making the Correct Diagnosis(es)**

### Acute Diarrhea – DOGS (initial screening)  
### Acute Diarrhea – CATS (initial screening)

- Direct smear in house (fresh sample (perform by <1hr))
- ZnSO₄ w/centrifugation
- Giardia antigen test
- Parvo test if indicated

### Later if Persistent:

- Repeat all of the above
- Cryptosporidium IFA
- *Clostridium perfringens* perf enterotoxin assay

**Later if Persistent:**

- Repeat all of the above
- Cryptosporidium IFA
- *Clostridium perfringens* perf enterotoxin assay
- Large bowel signs??
  > R/O Tritrichomonas foetus (special tests)

**Negative results do not make a rule-out. Be persistent, as retesting can be very important in establishing the definitive diagnosis.**

**Diagnosis and Management of *Giardia***

**Diagnosis**

Standard diagnostic tests used in any practice setting should include *fresh saline fecal smears* and zinc sulfate flotation with centrifugation. *Zinc sulfate flotation with centrifugation*, rather than gravity flotation alone, is a somewhat more sensitive means of testing for *Giardia* and other parasites. Trophozoites are more likely to be found in loose stools, while cysts are more often found in semi-formed or formed stools. Performing both zinc sulfate concentration with centrifugation and a *Giardia antigen test* together constitutes the most accurate means of evaluating a patient for
the presence of *Giardia*. This has been recognized as the “gold standard” in human medicine, and is true also in veterinary medicine.

**Direct Saline Smear**
Direct smears should be performed on fresh fecal samples as soon as possible after being passed, but definitely within 1 hour. A fresh saline smear is made by mixing a drop of feces with a drop of saline on a glass slide. A coverslip is applied and the preparation is examined immediately under 40x magnification. Trophozoites are pear-shaped and have a characteristic concave ventral disk. They demonstrate rolling/wobbling motion (e.g., like a falling leaf). Adding a drop of Lugol’s solution of iodine on the edge of the coverslip can be done as an optional procedure and this will enhance the morphologic features of the organisms and make them easier to find. The iodine kills the parasite, so motion will no longer be seen if this procedure is used. Differentiation of trichomonads from *Giardia* is based on a different motion pattern (more forward motion with trichomonads versus rolling motion with *Giardia*), the absence of a concave disk, a single nucleus, and the presence of an undulating membrane. Identification of *Giardia* trophozoites is diagnostic, while their absence in fecal samples does not rule out presence of infection.

**Zinc Sulfate Concentration with Centrifugation**
Many studies have now shown that zinc sulfate concentration with centrifugation is the most reliable test available for demonstration of *Giardia* cysts in fecal samples. The test can be done in any practice setting, and the technique is described below. Alternatively, because the best accuracy in detection of *Giardia* is achieved through well trained and experienced lab personnel consistently setting up the assay and studying the microscopic specimens on time, many practices now submit fecal samples for centrifugation assays to a commercial laboratory.

Zinc sulfate centrifugation is also a very effective method for identifying nematode eggs in feces. It is therefore now used as the standard test for screening for intestinal parasites in most academic and many private practices. Studies have shown that approximately 70-75 percent of *Giardia* positive dogs can be identified on a single zinc sulfate centrifugation test (as opposed to approximately 40 percent of dogs after 3 separate saline smear preparations). Slides should be examined within 10 minutes of preparation because the cysts may begin to shrink. **Since animals shed *Giardia* on an intermittent basis it is recommended that a series of zinc sulfate concentration tests be run over a 3 to 5 day period in order to maximize chances of accurately diagnosing or ruling out *Giardia* in animals with chronic diarrhea (or, alternatively, an antigen test can be run at the same time to help increase diagnostic efficiency and accuracy – this is what I recommend now as a standard practice).** Diagnostic efficiency increases to 95 percent when 3 zinc sulfate examinations are conducted over a 3 to 5 day period. A positive result on any of the tests warrants treatment for *Giardia*.

**Caution:** It is not uncommon for plant spores, yeast bodies, and other amorphous debris to be mistaken for *Giardia* cysts. In fact, *Giardia* is frequently misdiagnosed – either it is being diagnosed incorrectly, or the wrong tests are being run and animals with *Giardia* are being missed. *Giardia* cysts are 11-13 μ in size, and the subtle characteristics of the nuclei, axostyles, and median bodies are often more easily
observed under 100X oil immersion magnification. Sometimes there are crescent shaped indentations of the cyst wall. Yeast bodies are similar to *Giardia* in size, shape, and color. Yeast bodies appear to be far more common than *Giardia*.

**Zinc Sulfate Concentration - Summary**
- Zinc sulfate is the flotation solution of choice in small animal practices (excellent for detection of *Giardia* as well as nematodes)
- Zinc sulfate concentration with centrifugation is the best test for identification of *Giardia* cysts
- Causes less distortion of *Giardia* cysts than standard salt solution

**Giardia Antigen Testing**
Other diagnostic tests for *Giardia* include an enzyme-linked immunosorbent assay (ELISA) test for *Giardia* antigen in feces, a direct immunofluorescent assay, duodenal aspiration under endoscopic guidance, and the peroral string test. The latter two tests are impractical for routine use in small animal practice, especially when the effectiveness of today’s fecal tests is recognized.

The fecal ELISA test detects *Giardia* antigen that is produced by dividing trophozoites. The test is very sensitive in humans and reportedly detects 30 percent more cases of *Giardia* than does zinc sulfate. Studies have now confirmed that this is also an excellent test for use in animals. One advantage of the ELISA test is that, since it detects *Giardia* specific antigen in the feces, it avoids the problem of intermittent cyst excretion in the feces. This test can be a significant aid in accurate diagnosis of *Giardia* in any private practice setting, and I highly recommend that veterinarians utilize this test in order to more consistently make an accurate diagnosis of giardiasis in their small animal patients.

**Indications for Running *Giardia* Antigen Test:**
- Cases of acute or chronic diarrhea in which zinc sulfate centrifugation tests are negative for parasites
  *Including young dogs with suspected viral or bacterial enteritis – *Giardia* and other parasitic infections can significantly compromise animals with these conditions. **I recommend that all puppies with parvoviral enteritis be screened early for parasites with a combination of zinc sulfate with centrifugation and a *Giardia* antigen test (both tests day one or two on a single fecal sample)**
- Cases in which it is unclear whether *Giardia* cysts are being seen on flotation tests (e.g., vs. plant spores)
- For evaluation of animals with unexplained weight loss, unthriftiness, abdominal pain
- Acute or chronic vomiting **(some animals with disease related to *Giardia* have only vomiting as a clinical sign)**
- Many hospitals are now using the ZnSO4 with centrifugation and *Giardia* antigen combination assay as a routine screening test for GI parasites and wellness testing. This is because there are animals that have *Giardia* but that do not have any GI signs (loose stools, vomiting, etc) at the time of the exam. The addition of the antigen assay significantly improves the diagnostic sensitivity for *Giardia*. In summary, this approach offers: Better more sensitive
diagnostic testing, more convenience to the client (one sample only), and ultimately it is more economical.

**Treatment of Giardia**
For many years the primary treatment for *Giardia* in dogs and cats has involved metronidazole. For dogs in which metronidazole proved ineffective, quinacrine was often used in the past. However, although quinacrine has been shown to be more effective than metronidazole, it frequently causes side effects, including lethargy, anorexia, and vomiting. It was also used in cats. Quinacrine is no longer available, however. More recently it was shown that albendazole (Valbazen) is highly effective in controlling *Giardia*. I recommended albendazole as an effective treatment for Giardia from 1993-1997, but experience with albendazole in dogs and cats has shown that it can cause bothersome side effects; including leukopenia, lethargy, and inappetence. Therefore, I have not recommended albendazole for many years. I mention it here because some veterinarians still do use it.

**Fenbendazole (Panacur)**, well known for its effectiveness against a variety of intestinal parasites, also appears to be very effective against *Giardia*. In a controlled trial at Cornell University 6/6 dogs were effectively treated in an initial study. The same dose that is used to treat roundworms, hookworms, whipworms, and the tapeworm *Taenia pisiformis* (50 mg/kg orally once daily for 5 consecutive days [there have been treatment failures occasionally when therapy is given for only 3 days]) is used to treat *Giardia*. If the infection is not cleared on this regimen, a longer course of therapy is used (7 days). Fenbendazole has a proven track record for being very safe and is thought to not have any teratogenic effects. **Fenbendazole is therefore the drug of choice for treatment of Giardia in pregnant animals.** This is now also the preferred treatment for *Giardia* in cats.

**Drontal Plus (Bayer Animal Health)** is also an excellent choice for treatment of *Giardia*. This product includes febantel in addition to praziquantel and pyrantel pamoate. Febantel is the drug component that treats *Giardia*. Febantel is metabolized into fenbendazole and oxyfenbendazole after oral administration. Drontal Plus is administered once daily for 3 to 5 consecutive days for treatment of *Giardia*. Drontal Plus has been approved for use in dogs. Drontal Plus has been administered to cats empirically at a dosage of two small dog tablets per cat (about 50 mg/kg febantel) orally for 5 days with subsequent demonstration of decreased shedding of cysts (Scorza, Radecki, and Lappin).

**Metronidazole** is still a useful drug for treating *Giardia*, and it has the added advantage of having antibacterial as well as antiinflammatory properties. In situations in which it is unclear whether diarrhea is due to giardiasis, bacterial overgrowth, or mild inflammatory bowel disease, metronidazole is an excellent choice, especially when a client requests empirical therapy rather than definitive diagnostic testing. Metronidazole is only 67-74 percent effective in eliminating *Giardia* from dogs, however, and if a positive diagnosis is made fenbendazole or febantel would also be a reasonable choice. Potential side effects of metronidazole include anorexia, vomiting, and neurologic problems (ataxia, vestibular problems, seizures). In my experience these side effects are not common. They are more likely to occur when the anti-*Giardia* dose is used (25 to 30 mg/kg orally every 12 hours for 5 to 7 days). **The total**
dose of metronidazole should not exceed 65 mg/kg per day (30 mg/lb per day). A lower dose (10 to 20 mg/kg every 12 hours) is used in treatment of intestinal bacterial overgrowth and inflammatory bowel disease. Side effects are infrequent at this dose. In the past, if a 5 to 7 day course of metronidazole failed to eliminate *Giardia*, a longer follow-up course (10 to 14 days) was often used. With the availability of fenbendazole and Drontal Plus it is recommended that one of these drugs be used instead in this situation.

Metronidazole neuro toxicity can be resolved more quickly by administering diazepam for several days. This is likely related to modulation of the GABA receptor within the cerebellar and vestibular systems.

In addition to use of pharmacotherapy to eradicate *Giardia*, it is important to consider environmental control so as to minimize chances of reinfection, especially in kennel or cattery situations. Cysts present in a cool environment can remain infective for a long period of time. Cages and runs should be thoroughly cleaned of all solid fecal material. Steam cleaning, or treatment with a quaternary ammonium compound (e.g. A 33) are both very effective measures for killing cysts. Allowing time for thorough drying is important, to desiccate any remaining cysts.

**Bathing:** Steps to prevent reinfection play an important role in resolution of giardiasis in dogs. Dogs may be reinfected with cysts from the hair or the environment, and bathing at the time that drug therapy is concluded, thereby removing cysts that could be licked from the hair coat by the animal, may be a very helpful additional step in decreasing the chances of reinfection. Changing the environment, if possible, can also be beneficial.

**Dietary Therapy and Supplementation:**
In animals that are known to be chronic carriers of *Giardia*, it may be benefical to supplement the diet with fiber. Increased dietary roughage may make it more difficult for *Giardia* trophozoites to attach to the small intestinal mucosa (use either commercial diets or simply add a fiber source such as Metamucil or pumpkin, for example, to the animal's standard diet

**Rx for Chronic Giardiasis: Will Probiotics Help?**
- *Lactobacillus johnsonii* has been shown to inhibit *Giardia* proliferation in vitro
  - Due to alterations in pH from production of lactic acid
  - In guinea pigs, in vivo, prophylactic feeding of Lj greatly reduced fecal shedding following experimental inoculation with *G. intestinalis*
- Enterococcus faecium SF68 fed to mice
  - Stimulated increase in anti-*Giardia* intestinal IgA and circulating IgG
  - Increased CD4+ immunocytes
- Reduced shedding and more rapid clearance of *Giardia*?
- Studies are ongoing

**Zoonotic Potential:** Current information indicates that zoonotic potential may exist with some *Giardia* genotypes, but certainly not all. When both animals and humans living in the same environment become infected, a common source of infection rather than direct transmission must also be considered.
Are most *Giardia* spp. infections shared between animals and man? The genus *Giardia* contains multiple species of flagellated protozoans that are indistinguishable morphologically. Host specificity was thought to be minimal for *Giardia* spp., but not all small animal isolates cause disease in human beings. There have been varying results concerning cross-infection potential of *Giardia* spp. Human *Giardia* isolates usually grow in cell culture, animal isolates often do not. Recent genetic analysis has revealed 2 major genotypes in people. Assemblage A (*G. duodenalis*) has been found in infected humans and many other mammals including dogs and cats. Assemblage B (*G. enterica*) has been found in infected humans and dogs, but not cats. It appears that there are specific genotypes of *Giardia* that infect dogs (*G. canis*; Assemblages C and D) and cats (*G. felis; Assemblage F*) but not people. Accordingly, healthy pets are not considered significant human health risks for HIV infected people by the Centers for Disease Control (www.cdc.gov/hiv/pubs/brochure/oi_pets.htm).

Should *Giardia* Positive But Asymptomatic Animals Be Treated?
The question whether animals that are asymptomatic carriers of *Giardia* should be treated is often asked. *Giardia* cysts have been found in many animals with well-formed feces. *Giardia* is clearly not pathogenic in some animals, while in others it causes significant enteritis. And there may be others that experience intermittent GI upsets that could potentially be related to chronic parasite carriage, and that may benefit in the long term from more effective parasite control. Because the public health considerations must still be considered, I do recommend that all animals with fecal samples that are positive for *Giardia* be treated, using these guidelines:

- Administer Fenbendazole (Panacur) 5 days
- Re-check fecal at 14-28 days, not later – use the zinc sulfate w/centrif assay, NOT the antigen test (we don’t know how long it takes to go negative)
- If positive on O&P, treat once more
  - Fenbendazole again, or febantel (in Drontal PLUS); could also combine with metronidazole for this second round of therapy
- If still not clinical, stop here, don’t re-check again
  - Pet is not clinical and likelihood of transmission of any infectious agent to a human is very low
  - Is the Giardia even a significant problem for the patient?

NOTE: We do not want to overtreat! The antigen test should not be used as a recheck test in the immediate post treatment phase. The idea is to use the best diagnostic approach up front and then to manage the patient judiciously.

Preventing Infection/Premises Control
In controlled environments, the following methods should be used to keep the area as decontaminated as possible:

1. Decontaminate the environment
2. Treat all animals in the environment
3. Bathe at the conclusion of drug therapy to remove cysts from haircoats
4. Prevent reintroduction of infection
In hospital and kennel/cattery situations (controlled environments) moving animals away from contaminated areas so they can be cleaned and decontaminated is very important. Steam cleaning after all fecal material has been removed is very effective. Chemical disinfection can be effectively accomplished using quaternary ammonium (QUAT) – containing disinfectants (e.g. Roccal, Totil), which will inactivate cysts in one minute at room temperature. The area should be allowed to dry completely and if possible left open for a few days. Animals should be bathed with a general cleansing shampoo before being returned. In some situations, e.g., shelters, research facilities, it may also be advisable to bathe the animals a second time, especially around the perianal area, using a quaternary ammonium compound. These can be safely left on the coat for 3 to 5 minutes, before being thoroughly rinsed off (longer exposure can cause irritation). Allow the coat to dry thoroughly before returning the animal to the clean area, and then administer one more course of anti-Giardia therapy, preferably using a different drug than was used during the initial course. Subsequently, any new animals introduced to the kennel or cattery should be tested as a matter of routine, but also bathed and treated as well, regardless of whether the fecal tests are positive or negative for *Giardia*.

**Tritrichomonas foetus**

*Tritrichomonas foetus* is a recently identified (1999) enteric protozoan of cats. It causes chronic large bowel diarrhea (loose stools, presence of blood and mucus, straining to defecate), and is most commonly seen in young cats that have resided in densely populated housing such as catteries and shelters. The diarrhea may be intermittent or persistent. Loose stool may dribble out (lack of control) and the anal area may become edematous. The organism is present in the ileum, cecum, and colon as a trophozoite. The organism does not encyst, so trophozoites are the only recognized stage. Infection in feral cats and healthy cats appears to be uncommon.

Until 2005 no effective treatment had been identified. Unfortunately, some cats with chronic diarrhea and dyschezia were euthanized due to a lack of any therapy that could control the clinical signs. It was exciting news in 2005 when Dr. Jody Gookin and colleagues at North Carolina State University reported that the nitroimidazole drug ronidazole is effective in controlling *T. foetus*. Although the diarrhea eventually resolves over a period of time (months up to one to two years) in untreated cats, ronidazole is the recommended therapy once a diagnosis has been established. It is important that an accurate diagnosis be made so that clients can be counseled appropriately, i.e., they should expect that their cat(s) will continue to have abnormal stools for some period of time. Further, there can be side effects of significant concern related to ronidazole, so this is NOT a drug that should be used empirically in lieu of testing. Also, it is not uncommon for cats to be co-infected with *Giardia* or *Cryptosporidium* or even both, so a thorough evaluation for parasites is important (run a minimum of one zinc sulfate with centrifugation and a *Giardia* antigen test and consider IFA fecal assays to check for *Cryptosporidium*). Accurate and thorough testing is essential and once any causative agents are identified they can be treated appropriately for the benefit of the patient and its owner.
Tritrichomonas foetus is commonly mistaken for Giardia trophozoites on direct smear exam. All trichomonads possess three to five anterior flagella, an undulating membrane, and a recurrent flagellum attached to the edge of the undulating membrane. All flagella originate from an anterior basal body. An axostyle extends the length of the trichomonad and extends posteriorly. A cyst stage is not known for this genus. Video clips showing both Giardia and Tritrichomonas trophozoites are available on the North Carolina State University website cited in the reference list below.

Definitive diagnosis can be made in some cases by direct smear of fresh feces in saline and examined at 200 to 400x magnification. Sensitivity is low, however, for diagnosis by direct smear (only 14% in one study), so results can often be false negative. To increase the chance of finding Tritrichomonas trophozoites on direct smear, it is recommended that multiple direct smears be done on the same day. Whenever possible, a cat with suggestive signs should be hospitalized for part or all of a day so that each fecal sample that is passed can be examined quickly via direct saline smear.

Tritrichomonas foetus can also be grown from feces via incubation at 37 degrees C in Diamond’s medium. A commercially available culture system is also available and is recommended for use in clinical practice (InPouch TF, Biomed Diagnostics Inc., San Jose, CA). The medium in InPouch does not support the growth of Giardia species or Pentatrichomonas hominis so presence of organisms is consistent with T. foetus. PCR is the most sensitive means for confirming a diagnosis. In one study of 36 cats with T. foetus infection, 20/36 were positive on the InPouch TF test and 34/36 were positive on PCR. Details on the PCR assay can be reviewed on the North Carolina State website.

Studies at North Carolina State University in 2005 showed that ronidazole is effective for treatment of T. foetus. The original dosage guidance was to administer 30 mg/kg BID for 14 days. However, a study reported in 2008 provided new guidance: 30 kg/kg once daily is effective and safer, i.e., less likely to cause neurologic adverse events (RONIDAZOLE PHARMACOKINETICS IN CATS AFTER IV ADMINISTRATION AND ORAL ADMINISTRATION OF AN IMMEDIATE RELEASE CAPSULE AND A COLON-TARGETED DELAYED RELEASE TABLET; Levine, Papich, Gookin et al).

Ronidazole is a nitroimidazole antimicrobial that is not licensed for any use in the U.S. The medication has become more readily available in the United States through compounding pharmacies. The drug has mutagenic properties, so it must be compounded the same way as chemotherapy drugs. We have had some cats experience mild neurological side effects to ronidazole, similar to what can be seen with metronidazole. These resolved upon discontinuation of the drug. It is expected that there will be fewer instances of neurotoxicity with the new schedule of 30 mg/kg on a once daily dosing schedule. It is important that an accurate diagnosis be made so that clients can be counseled appropriately, i.e., they should expect that their cat(s) will continue to have abnormal stools for some period of time until definitive treatment can be administered.

Other recommended steps during therapy include isolating cats to decrease the risk of reinfection and to discard any litter boxes the cat has used, after treatment is completed.
Follow-up testing: Dr. Gookin recommends testing by PCR at 1 to 2 weeks and 20+ weeks after treatment is completed. Negative results should be interpreted with caution since PCR cannot prove the absence of infection and prolonged symptomatic carriage of the organism after antimicrobial therapy may be common.

An alternative drug which can be tried is tinidazole. This is also a nitroimidazole antimicrobial. A dose of 15-30 mg/kg SID can be tried. It should be safe and may or may not be effective. Studies have been ongoing, however, and results have not been very impressive.

References:


**Website for periodic updates and video clips of motile trophozoites:** [www.JodyGookin.com](http://www.JodyGookin.com). There is an excellent reference section titled AN OWNERS GUIDE TO DIAGNOSIS AND TREATMENT OF CATS INFECTED WITH TRITRICHOMONAS FOETUS.

**Clostridium Perfringens Enterotoxicosis**

Over the last 12 years Clostridium perfringens enterotoxidosis (CPE) has emerged as a frequently recognized cause of chronic intermittent diarrhea in dogs. Although it is likely a less common cause of diarrhea in cats it is still diagnosed frequently enough that it should be considered in the diagnosis of diarrhea in cats as well. This is not a new disease. Frequent use of the definitive test (enterotoxin assay performed on feces) for this disorder has revealed that CPE is seen relatively commonly in clinical practice and that CPE is a disorder that should be considered in any dog or cat with intermittent or chronic persistent diarrhea.

*C. perfringens* is a normal vegetative enteric organism. Simply identifying *C. perfringens* on a fecal culture is meaningless. The pathogenesis of CPE is through an enterotoxin that is produced after certain strains of *C. perfringens* sporulate. The toxin damages epithelial cells of the distal ileum and colon. Inciting factors that promote sporulation are not clearly understood but may include stress, diet changes, concurrent disease, or inherent immune status.

The most common clinical signs are chronic intermittent or persistent diarrhea. In some animals acute diarrhea is the primary sign. In fact, some of the cases of hemorrhagic gastroenteritis (HGE syndrome), characterized by acute bloody diarrhea and an increased packed cell volume that most practitioners have seen over the years, may have been due to CPE. Many animals exhibit signs of large bowel diarrhea, but small bowel signs may be seen as well. In some cases signs may be seen for only a day
or two at a time, with persistent recurrences on a weekly, monthly, or on a less frequent basis. Stressful events or diet changes may incite flare-ups of clinical signs. In other cases *C. perfringens* enterotoxidosis is one of several problems that an animal may have concurrently and diarrhea may be persistent.

**Diagnosis**
CPE must be considered whenever more than one animal in the environment has diarrhea (e.g., household, kennel, cattery). Transmission from animal to animal can occur. A presumptive diagnosis may be suggested on fecal cytology in which more than 3-4 spores per high power oil immersion field are observed (the spores have a safety pin appearance and are larger than most bacteria). However, **definitive diagnosis** is by identification of enterotoxin which is currently done via a fecal assay. Clinicians should be aware that simply seeing spores on fecal cytology does not establish a definitive diagnosis (see JAVMA February 1, 1999). Stool is submitted to the lab for enterotoxin analysis. Fecal samples that will be shipped off from the hospital directly to a laboratory should be sent on ice via overnight express. If a courier service will be picking up samples for transport to the laboratory it is sufficient to keep the sample refrigerated until pick-up. The courier service will keep the sample properly chilled during transport. The minimum amount of stool that should be submitted is the size of a pea. Typically I submit samples in a red top tube, without serum separator. In animals with intermittent diarrhea the chances of a positive toxin finding are greater when abnormal rather than a normal stool is examined. A negative result does not definitively rule-out CPE.

**Treatment**
Several antibacterial drugs are effective in controlling CPE. Acute cases often respond well to amoxicillin (22 mg/kg BID) or metronidazole (10-20 mg/kg BID) for 7-28 days. Many clinicians have likely treated CPE with these medications empirically without knowing what they were treating. Chronic cases tend to respond best to tylosin powder. The recommended dose is: Animals greater than 23 kg ¼ tsp BID, 12 to 23 kg 1/8 tsp BID, 5 to 12 kg 1/12 tsp BID, and less than 4.5 kg 1/16 tsp BID (a “pinch”). Cats definitely do not accept the powder well at all, even when it is mixed in very tasty foods. It is best to have the powder reconstituted to capsule form for administration to cats. The medication is very safe. Some animals require treatment for several to many months (3 to 12 months or more). Over time the dose may in some cases be successfully reduced to SID and then every other day dosage (after several months or more on a BID schedule).

Dietary fiber supplementation may also help control CPE. Probable mechanisms include decreased *C. perfringens* fecal concentration, lower colonic pH, which prevents sporulation, and increased concentrations of SCFA. Some patients may respond well to dietary fiber supplementation alone.

Follow-up testing at 3-6 months can be done to determine if toxin persists. Once daily to every other day tylosin in conjunction with dietary fiber supplementation are used in chronic cases.
Cryptosporidiosis

Infection with *Cryptosporidium* is much more common than most small animal practitioners recognize. Currently it is recommended that all dogs and cats with diarrhea, whether acute or chronic, be screened for *Cryptosporidium* in addition to testing for nematode and protozoan parasites. In 2004 the American Association of Feline Practitioners adopted a position statement recommending that all kittens and adult cats with diarrhea be screened for *Cryptosporidium*. It is recommended that the same policy be followed with dogs (given that the cause is not simple diarrhea related to an acute upset due to sudden change in diet or dietary sensitivity).

*Cryptosporidium* spp. are coccidians that reside in the gastrointestinal tract. Infection can be associated with diarrhea in both immunocompetent and immunodeficient hosts. In the past, most of the cases of mammalian cryptosporidiosis were attributed to *C. parvum*. However, molecular studies have demonstrated that cats are usually infected with the host-specific *C. felis*, dogs are infected with *C. canis*, and people are infected with *C. parvum* or *C. hominus* (Scorza and Lappin). In a recent study at Colorado State University, they documented the presence of *Cryptosporidium* spp. DNA in diarrhea from 24.3% of the 292 animals tested (180 cats, 112 dogs) (Scorza and Lappin). This highlights the importance of testing dogs and cats for cryptosporidiosis. PCR is much more sensitive than the tests that are used most commonly at this time (acid fast staining of fecal smears or IFA). In this same series with 24.3% positive on PCR, only 2.7% were positive on IFA.

All dogs and cats infected with *Giardia* or *Cryptosporidium* species should be considered potentially zoonotic, even though the number of cases in which humans are infected through contact with pets is probably not high. Infection in humans is sometimes fatal in the presence of severe immunosuppression. Acute symptoms may include diarrhea, abdominal pain, vomiting, fever, and listless behavior. Infection can also be subclinical in dogs and cats. Chronic unresponsive diarrhea has been associated with cryptosporidiosis in cats with serious underlying disease as well as in dogs.

Because *Cryptosporidia* oocysts are quite small (as little as one-tenth the size of common *Isospora* oocysts) and are usually present in the feces in small numbers, they are very difficult to detect on routine fecal flotation and microscopy. The best tests currently available for routine testing for *Cryptosporidium* are fecal IFA and acid fast staining of fecal smears; however, they lack sensitivity. These tests are readily available at commercial laboratories (acid fast staining can also be done in house). PCR is a much more sensitive test but is labor intensive, expensive and is only available at a limited number of laboratories. Antigen tests for detecting *C. parvum* in human species are not sensitive for use in dogs and cats. In time there will be more sensitive tests readily available.

**Treatment**
The following treatment regimens may be used for cryptosporidiosis:

**Canine**

**Feline**
**Azithromycin** 5-10 mg/kg, BID orally, for 14-28 days **Azithromycin** 7-15 mg/kg, BID, orally, for 14-28 days

**Paromomycin** 150 mg/kg, SID orally, for 5 days **Paromomycin** 150 mg/kg, SID orally, for 5 days

**Tylosin** 15 mg/kg, BID orally, for 21-28 days **Tylosin** 15 mg/kg, BID orally, for 21-28 days

**References**

7. Scorza AV and Lappin MR. An update on three important protozoan parasitic infections of cats: cryptosporidiosis, giardiasis, and trichomoniasis. Supplement to Veterinary Medicine, March 2006; 18-32.
Inflammatory Bowel Disease

Inflammatory bowel disease (IBD) is not a specific diagnosis, rather it is a histological description of a syndrome resulting from a host hypersensitivity response to antigenic stimuli. In IBD there is an increase in the inflammatory cell population in the intestinal mucosa. The predominant inflammatory component in cats with IBD can be lymphocytic-plasmacytic (most common type), eosinophilic, or neutrophilic. Changes in mucosal architecture and cell morphology should also be noted (crypt lesions including abscesses, villus atrophy or fusion, edema, epithelial erosions or ulceration, fibrosis). The etiology of IBD is poorly understood. Primary causes of initiation and perpetuation of intestinal inflammation that should be considered include parasites, bacteria (specific agents including normal luminal bacteria or bacterial overgrowth), immune-mediated diseases, and food sensitivities. Many cases of IBD are likely idiopathic in nature.

Clinical Course
Inflammatory bowel disease (IBD) currently is recognized as a common and important medical problem in cats. Three general types of clinical presentations have been identified in cats with idiopathic IBD: (1) a clinical course characterized primarily by vomiting, (2) a clinical course characterized primarily by diarrhea, and (3) a clinical course that includes both vomiting and diarrhea as primary signs. Associated clinical signs can include change in appetite (anorexia, inappetence, or ravenousness), weight loss, and lethargy. In some cats, the clinical signs are cyclic; they seem to flare up and then abate in a predictable pattern.

Vomiting, one of the most frequent clinical signs of IBD in cats, is most often recognized as an intermittent occurrence for weeks, months, or years. As the disorder progresses, an increased frequency of vomiting often leads the owner to seek veterinary attention. In addition to vomiting, diarrhea is a common sign observed in feline IBD and most likely is due to derangement of normal mechanisms of absorption and motility caused by mucosal inflammation. In most cases, diarrhea is intermittent early in the course of the disorder, and there may be a transient response (weeks to several months) to dietary manipulation or any of a variety of medications (in some cases, however, dietary manipulation can effect excellent control and drug therapy
may ultimately not be necessary). Later, the diarrhea becomes persistent and usually responds only to specific treatment, which is determined after a definitive diagnosis is made. Signs of small bowel diarrhea predominate, but signs of large bowel diarrhea may be evident as well if there is generalized intestinal tract involvement.

Appetite changes in cats with idiopathic IBD vary from decreased appetite to complete anorexia to ravenousness. Inappetence seems to occur more commonly in cats that have vomiting as the primary clinical sign and usually occurs during exacerbation of clinical signs, and vomiting or diarrhea is not observed until later or not at all. The three leading differential diagnoses for a cat with a ravenous appetite, diarrhea, and weight loss are IBD, hyperthyroidism, and exocrine pancreatic insufficiency (uncommon).

Diagnosis
A definitive diagnosis of IBD can be made based only on intestinal biopsy (performed either at endoscopy or exploratory laparotomy, and ensuring that both upper and lower [ileum] biopsies are obtained). A definitive diagnosis of IBD cannot be made based on barium series radiography or ultrasonography. Diagnostic work-up prior to performing biopsies includes baseline testing to evaluate the overall health status of the patient and to rule out other disorders. Recommended baseline tests include a complete blood count, complete biochemical profile, urinalysis, fecal exams for parasites, serum thyroxine test, serum cobalamin level, and FeLV/FIV. Cats with chronic vomiting should be screened for heartworm disease. fTli is done to rule-out exocrine pancreatic insufficiency. Ultrasonography is useful for assessing the abdominal organs, intestinal wall thickness, searching for any masses, and examining for lymphadenopathy. Dietary sensitivity is a common problem in cats with vomiting and/or diarrhea and food trials are an important part of the diagnostic work-up, especially early in the clinical course. Hydrolyzed protein and novel protein foods should be fed for 2-3 weeks at a time to determine if dietary therapy will either reduce or resolve the problem entirely.

Abdominal Imaging in Cats – IBD vs. Lymphoma

Radiology
Radiography is important for diagnosing intestinal diseases. During evaluation of the small bowel on survey radiographs, important factors that should be evaluated include location of small intestine (normally fills the abdomen where nothing else is present, not unusual to be mostly right-sided in cats), appearance of bowel contents (gas, fluid, or mottled material), contour of small bowel, and diameter of the small intestine. The normal diameter in cats is up to 12 mm.

In normal animals, intestinal luminal contents should appear as a homogeneous fluid opacity. Disease of the small intestine may be missed on survey films unless there is a change in bowel opacity (mineralized mass or foreign material), luminal diameter (functional ileus or complete or partial mechanical obstruction), or changes in contour of the small bowel (linear foreign body).

Contrast studies (upper GI series) are often necessary to identify normal or abnormal shape, diameter, or continuity of small bowel. The transit time of barium varies greatly in cats. It usually travels from the stomach through to the ileum in about 60 minutes,
although it can take as long as 4 hours. The range of transit times for organic iodides through the small bowel is approximately 15–90 minutes. The organic iodide usually reaches the ileum and colon in less than 60 minutes.

**Small Intestinal IBD**
Diagnostic radiographs are recommended in the work-up of cats with gastrointestinal signs. Although survey and contrast radiographs are usually not specific/diagnostic for IBD, abdominal radiography is most helpful in defining extra-alimentary tract disorders causing gastroenteritis. Survey radiography might detect organomegaly (liver, kidney) unrelated to IBD or intestinal obstruction that might cause similar GI signs. Survey radiographs of inflammatory bowel disease are usually normal. There is no consistent radiological finding in cats with inflammatory bowel disease. The intestines may appear thickened (intestinal thickness cannot adequately be determined on survey radiographs), or luminal fluid maybe increased and there may be more gas than normal in the intestines, but these signs can occur in many conditions. Contrast examinations (upper GI series) are helpful in identifying a mass or obstruction. With contrast, assessment of the location and extent of the intestinal lesion may be more accurate than on survey images. Changes associated with IBD on barium study are often not present. With severe inflammatory however; changes may include: irregular mucosal lining abnormalities and thickened intestinal walls. In most cases contrast radiography is unrewarding.

**Intestinal Lymphoma**
Survey radiography might detect organomegaly (liver, kidney, lymph nodes) associated with lymphoma. Radiographic findings may reveal a mid-abdominal mass associated with the GI tract and/or mesentery, or localized or diffuse decrease, or loss, of serosal detail suggestive of peritoneal effusion. If a mass is suspected radiographically or historically, or a mass has been palpated, then compression radiography may be helpful to isolate and visualize the mass. Obstruction occurs more often with adenocarcinoma of the small intestine than with small intestinal lymphoma. Contrast examinations (upper GI series) are helpful in identifying the mass or the obstruction. With contrast, the location, bowel wall thickening, mucosal irregularity and extent of the intestinal lesion may be more accurate than on survey images.

**Ultrasonography of the Feline Small Intestines**
The small intestines can be seen throughout the abdomen, both end-on and longitudinally oriented. The duodenum has a slightly larger diameter than the rest of the small intestinal loops, and is the most lateral and ventral bowel loop in the right cranial abdomen. It can be located usually just ventral and lateral to the right kidney and followed cranially into the pylorus. The ileum has a distinct cross-sectional appearance (resembling spokes on a wheel) and can be visualized as it enters the colon, just medial to the right kidney. The colon typically is gas-filled, with poor visualization of the lumen.

The following five layers are present in the intestinal wall, from outside to inside:
- **Serosa**: Thin hyperechoic layer
- **Muscularis**: Thin hypoechoic layer
- **Submucosa**: Thin hyperechoic layer
- **Mucosa**: Prominent hypoechoic layer (typically the thickest layer)
Mucosal surface–lumen interface: Hyperechoic layer in the center of the bowel

These individual layers are best visualized with higher-frequency transducers.

Normal wall thicknesses have been established in the cat for various segments of the GI tract:
- Duodenum: 2.0–2.4 mm (mean of 2.2 mm)
- Jejunum: 2.1–2.5 mm (mean of 2.3 mm)
- Ileum: 2.5–3.2 mm (mean of 2.8 mm)
- Colon: 1.4–1.7 mm (mean of 1.5 mm)

One to three contractions per minute should be seen with normal small intestinal peristaltic activity.

Ultrasonographic features of intestinal disease include bowel wall thickening, loss of wall layers, loss of motility, and regional lymph node involvement.

**Intestinal Ultrasound: IBD versus Lymphoma**

An abdominal ultrasound examination may be helpful in cases of suspected small intestinal disease. Abdominal ultrasound is superior to radiology in defining focal versus diffuse disease, loss of layering, intestinal thickening and mesenteric lymphadenopathy seen with IBD and lymphoma. Ultrasonography also allows for precise guidance of fine needle aspiration or biopsy for cytologic or histopathologic sampling of small intestinal disease and associated lymphadenopathy.

Ultrasonography can also be used to assess response to therapy noninvasively. A limitation of ultrasonography would be the difficulty in assessing the exact anatomic location (duodenum and ilium should be more easily identified by an experienced operator). Findings may be normal, especially in cases of low-grade small cell lymphoma or mild IBD.

Changes of the small intestine may or may not be present dependent upon chronicity and/or severity. The changes may be diffuse or focal. The intestine may appear normal. Biopsy is indicated to confirm disease.

The most common finding with inflammation is normal to symmetric wall thickening with the layering retained. In comparison, neoplasia is usually localized with greater wall thickness and loss of normal layering. These categories can overlap, and therefore cytology or histopathology is required for definitive diagnosis. Acute enteritis or inflammatory bowel disease may demonstrate corrugation of the intestine on ultrasound examination.

**Ultrasound of IBD**

With inflammatory bowel disease, the intestine may be normal on ultrasound. The measurement of the intestinal wall thickness by ultrasound is neither specific or sensitive for diagnosing IBD. Changes, especially those of severe or chronic disease, have been reported as focal to diffuse thickening, altered echogenicity, poor intestinal wall layer definition, and mild enlargement of adjacent lymph nodes. Mucosal echogenicity may remain hypoechoic. Round, enlarged, hypoechoic lymph nodes may
be more consistent with neoplasia, while inflammatory lymph nodes may be enlarged but tend to maintain their normal shape.

### Ultrasonographic Measurements of Feline Abdominal Lymph Nodes

<table>
<thead>
<tr>
<th></th>
<th>US Length (mm)</th>
<th>US Diameter (mm)</th>
<th>Frequency of detection</th>
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<tbody>
<tr>
<td>Jejunal</td>
<td>20.2 (11.4-39.0)</td>
<td>5.0 (2.8-7.2)</td>
<td>90%</td>
</tr>
<tr>
<td>Colic</td>
<td>9.0 (4.6-12.1)</td>
<td>3.1 (1.9-5.2)</td>
<td>50%</td>
</tr>
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### Ultrasound of Intestinal Lymphoma

Perform abdominal ultrasonography to evaluate the extraintestinal organs in addition to GI tract wall thickness, layering, and motility. Lymphoma most commonly presents as transmural, circumferential, homogenous, hypoechoic thickening with loss of normal wall layering. Lymphoma tends to involve a long bowel segment or multiple bowel segments. Regional moderate, hypoechoic lymphadenopathy is generally present. Lymphoma is less likely to cause obstruction of the lumen.

Six major patterns of ultrasonographic features in feline lymphoma include: transmural-circumferential, symmetrical and asymmetrical, transmural-bulky, transmural-nodular, transmural-segmental, and mucosal infiltration. The transmural-circumferential pattern is most common. The transmural-bulky pattern has been described as a space occupying mass representing the thickened wall with areas of increased and decreased echogenicity. The transmural-segmental pattern has been described as wall thickening involving only a portion of the wall. The transmural-nodular pattern appeared as nodular wall infiltration and local nodular spread into the mesentery. Mucosal infiltration pattern demonstrated mild thickening of the intestinal wall associated with faint hyperechoic foci throughout thickened mucosal layer. In cats GI lymphoma can affect the intestinal tract without disrupting the wall layering.

### Ultrasonographic Evaluation of Muscularis Propria in Cats with Diffuse Small Intestinal Lymphoma or IBD

It is difficult to detect small intestinal lymphoma or IBD in cats without a mass lesion, loss of layering or thickened bowel wall. Thickening of the muscularis propria is associated with diffuse infiltrative bowel disease such as lymphoma or IBD in cats. This has also been seen in normal cats as well. The most common ultrasound descriptions of GI lymphoma in cats are as mass lesions previously discussed.

### Intestinal Biopsy Techniques

**Endoscopic Biopsy:** Endoscopy is a minimally invasive procedure in which multiple biopsies can be obtained and this procedure generally has greater client compliance than with surgery because it is less invasive and less expensive than exploratory abdominal surgical procedures. Endoscopy is considered a gold standard procedure for tissue collection. Operator experience and the quality and number of biopsy samples obtained are very important. Endoscopy offers a means of examining the upper and lower small intestine, stomach, and colon. It is especially advantageous...
because biopsies can be obtained early in the course of the disorder, at a stage when a client will likely be reluctant to agree to an exploratory surgery for their pet. The degree of intestinal changes noted on biopsy also provides useful guidelines for both type and duration of therapy that will be needed to control the specific disorder.

Clinicians need to make sure they are taking an adequate number of endoscopic biopsy samples for accurate diagnosis. Even expert endoscopists report that in some cases one-fourth to one-third of the biopsy samples they take from a patient will have some degree of damage to the tissue that may preclude the samples from being useful or representative. Therefore, it is recommended that clinicians take 8 to 12 biopsy samples from the upper small intestine so that the pathologist will have enough tissue to work with. Also, it is recommended that both upper and lower GI endoscopy be done on cats with chronic GI signs (vomiting and/or diarrhea, weight loss). In this way biopsies from the ileum can be obtained by passing the endoscope along the full length of the colon up to the level of the ileocolic orifice. It is very important that the effort be made to obtain ileum samples, since some cats with small cell lymphoma have disease in the ileum but not in the upper small intestine. The diagnosis can be missed in these cats if only upper small intestinal biopsies are obtained.

When a pediatric diameter endoscope is used it is possible in most dogs over 4 to 5 kg to advance the endoscope through the ileocolic orifice and into the ileum, where it can then be advanced along the terminal ileum for exam and biopsies. However, in cats the ileocolic orifice is very small and in most cats it is not possible to advance the endoscope through this junction and into the ileum. In cats ileum biopsies are obtained blindly by advancing the endoscopic biopsy instrument through the ileocolic orifice with the endoscope tip positioned at the ileocolic sphincter area. Usually 3 – 4 samples are procured in this way. Colon biopsies are always obtained as well during colonoscopy in order to evaluate for inflammation in the colon.

**Surgical Biopsy Techniques for Abdominal organs**

**Biopsy.** Organ biopsy is usually required to confirm feline IBD and Lymphoma. This can be accomplished using either laparoscopic techniques or open abdominal surgery. Laparoscopic techniques have been well described for organ biopsy. These techniques are minimally invasive and well suited for tissue procurement, however, laparoscopy is not yet readily available as a diagnostic tool in most small animal clinics. Surgery on the other hand is an excellent way to obtain liver, pancreatic and intestinal biopsies. In addition to biopsy the liver should be cultured as well as bile aspirates for culture and cytology. We also currently culture the pancreas as well during laparotomy.

**Intestinal Biopsy:** One can obtain intestine using several techniques. A full thickness biopsy allows the pathologist to provide the most accurate diagnosis. When taking an intestinal biopsy, the easiest way to guarantee you will get an adequate size, full thickness piece of intestine is to use a brand new 4mm or 6mm skin punch biopsy instrument. The skin punch is placed on the antimesenteric border of the proposed segment of intestine and ‘drilled’ through all layers of intestine until the biopsy punch can be felt to enter the lumen of the intestine. The skin punch is removed and the biopsy retrieved from the shaft of the skin punch biopsy. This technique is particularly useful for ileal biopsy as it is easy to biopsy between the mesenteric and antimesenteric vessels. Transverse closure of the biopsy site is recommended to
eliminate the possibility of lumen compromise. The biopsy site is closed using a simple interrupted or simple continuous suture pattern. 3-0 or 4-0 monofilament absorbable suture with a swaged-on sharp taper or taper-cut (penetrating point) needle is recommended. Care is taken to ensure that at least 3 mm bites are taken into the intestine and the sutures are no more that 2-3 mm apart. This is Dr. Seim’s preferred technique for intestinal biopsy.

An alternate technique for intestinal biopsy is to make a 2-3 mm long incision on the antimesenteric border of the intestinal segment. A #11 or #15 BP scalpel blade is used to penetrate the intestinal wall. The blade is withdrawn to create a 2-3 mm long incision. A second parallel incision is made 1 – 2 mm from the original incision. A DeBakey forcep is used to grasp one end of the parallel incisions, a Metzenbaum scissor is used to cut out the piece of intestine. The surgeon should be careful not to crush the specimen with forceps. Only handle one end of the specimen while excising the biopsy specimen. If excessive trauma is created during biopsy, the pathologist may not be able to determine if the pathology is real or surgically created. The excised piece of intestine is examined closely to ensure that all layers have been included in the specimen. The biopsy site is closed using a simple interrupted or simple continuous suture pattern. 3-0 or 4-0 monofilament absorbable suture with a swaged-on sharp taper or taper-cut (penetrating point) needle is recommended. Care is taken to ensure that at least 3 mm bites are taken into the intestine and the sutures are no more that 2-3 mm apart. Complications associated with multiple intestinal biopsies are rare. Complications in patients undergoing intestinal surgical procedures are generally related to the surgeon’s technical ability and not the patient’s preoperative status.

**Lymph node biopsy:** All lymph nodes are encased in a layer of peritoneum. When performing a lymph node biopsy it is best to tent the peritoneal covering with forceps and incise it with metzenbaum scissors. The peritoneum is then gently dissected off the lymph node. The exposed lymph node is biopsied using a #15 or #11 scalpel blade. Generally, a thin section of lymph node is ‘filleted’ off and placed in a moistened gauze sponge. The peritoneum covering the remaining lymph node is sutured to create suture pressure to help control surface hemorrhage.

**Liver Biopsy:** Surgical biopsies obtained during exploratory laparotomy are described here. The simplest method is performed by cutting a strip of liver parenchyma 5 to 6 mm thick along the border of the liver lobe. Excessive bleeding is rarely a problem with this technique; hemorrhage is controlled via cautery or direct pressure. Diffuse liver disease must be present if this method is to be diagnostic.

A second technique involves placing an encircling ligature around a pedicle of liver tissue. As the ligature is tightened, it cuts through the hepatic parenchyma, ligating hepatic vessels and bile ducts. This technique, widely known as the Guillotine technique, has been criticized for leaving excessive amounts of devitalized parenchyma. This can be avoided by inserting scissors through the cut parenchyma and cutting hepatic vessels and bile ducts just distal to the ligature. This method requires the presence of diffuse liver disease to obtain a diagnostic biopsy unless the lesion is present in the distal aspect of the liver lobe.
More localized abnormalities can be biopsied by wedge resections or partial lobectomy. Wedge resections may be performed by placing a row of overlapping, full-thickness, interrupted mattress sutures of 0 or 2-0 Maxon or Biosyn along each side of the wedge to be removed; these sutures should commence at the edge of the liver lobe and meet proximally to form a “V”. The sutures should be tied so as to compress the liver slightly but not cut into liver parenchyma. The wedge of tissue to be removed is incised about 5 mm from the suture line. Alternatively, the wedge may be removed prior to tightening the mattress sutures; preplaced mattress sutures are then gently tied with enough tension to control bleeding.

An alternate technique for use in patients with diffuse fibrotic liver disorders is performed by penetrating the affected liver lobe with a straight mosquito hemostat. The hemostat tip is placed on the surface of the liver lobe to be biopsied and gently plunged through the liver lobe until the tip of the hemostat is seen penetrating through the opposite side of the liver. The jaws of the hemostat are opened just wide enough to accept a piece of 2-0 or 3-0 Maxon or Biosyn suture. The suture is doubled on itself, the loop is passed into the jaws of the hemostats, and the loop pulled through the liver lobe. The exiting loop is cut leaving two strands of suture coursing through the liver lobe. Each strand is tied individually to “cut” through the liver. A “V” wedge is cut through the liver when both strands of suture have been tied. A number 15 BP scalpel blade is used to cut the V-shaped liver biopsy wedge from the sutures.

Pancreatic Biopsy: Samples from the pancreas should be obtained in all suspected triaditis cases. The old wife’s tale stating “don’t touch the pancreas” needs to be put to rest in veterinary medicine. Gentle manipulation and biopsy of the pancreas is a predictably successful procedure with almost no incidence of postoperative pancreatitis. Biopsy of the pancreas is performed in a similar manner as biopsy of the liver. In patients that have diffuse pancreatic disease, a segment of the right or left limb of the pancreas is identified. An encircling ligature of 3-0 Biosyn is placed around the pedicle. As the ligature is tightened, it cuts through the pancreatic parenchyma, ligating vessels and pancreatic ducts. The distal pedicle of pancreas is carefully removed with a number 15 BP scalpel blade or metzenbaum scissors. Care is taken to avoid cutting the suture.

Treatment of IBD
It is important that the clinician formulate a treatment plan based on a correlation of clinical course, laboratory and gross findings, and histologic findings (considering both cellular infiltrate and morphology) rather than relying on histologic changes alone. Since food sensitivities can be a cause of IBD, dietary trials are an essential part of both the diagnostic and therapeutic strategy, utilizing hydrolyzed protein diets and novel protein diets and treating each patient as an individual (i.e., there can be variable responses to specific diets varying from patient to patient). Regarding pharmacotherapy, while corticosteroids have long been considered the cornerstone of treatment for idiopathic inflammatory bowel disorders, antimicrobial agents may play a role as well. Bacteria have been implicated in the pathogenesis of IBD.

Guidelines for corticosteroids in cats with IBD are as follows. Mild to moderate cases of IBD often respond to prednisolone (preferred over prednisone in cats) at a starting dose of 1 to 2.2 mg/kg divided twice daily for two to four weeks followed by a gradual
decline in 50% increments at two week intervals. Cats with inflammatory changes graded as mild usually respond quite well to the lower dose and alternate day or every third day treatment can often be achieved by two to three months. Occasionally treatment can be discontinued altogether by three to six months.

If biopsies reveal disease that is moderate to severe a prednisolone dose of 2 to 4 mg/kg divided twice daily is used in cats for the first 2 to 8 weeks or until clinical signs resolve. This dose of corticosteroid is usually well tolerated in cats. In some cases a dose of 1 to 2 mg/kg per day may be necessary long term (months to years) to maintain clinical remission. Use of combination drug therapy may also be required at the outset to control clinical signs and prevent progression of the disease (e.g., metronidazole or tylosin plus prednisolone). Cats with hypoproteinemia and histologic changes graded as severe often respond quite well when an aggressive therapeutic course is undertaken.

Budesonide is a glucocorticoid that represents an alternative for management of IBD in dogs and cats, especially in severe cases that have proven to be refractory to prednisolone, metronidazole, azathioprine, chlorambucil, tylosin, and dietary management; or that are intolerant of the corticosteroids discussed above. Budesonide is one of a group of novel corticosteroids that have been in development for use in humans in an attempt to make available alternative preparations that will help limit toxicity associated with corticosteroid use.

Budesonide undergoes high first pass metabolism in the liver and 90% is converted into metabolites with low corticosteroid activity. It has minimal systemic availability. The potential for typical corticosteroid side effects is significantly reduced as a result of decreased bioavailability and the resulting limited systemic exposure, which makes this a particularly attractive drug for use in humans and animals that are poorly tolerant of other corticosteroids. Budesonide also has a high receptor-binding affinity in the mucosa. It has been referred to as a “locally acting” corticosteroid.

Therapeutic results with budesonide have been promising in humans with Crohn’s disease, collagenous colitis and lymphocytic colitis, ulcerative colitis, either when administered as a retention enema or in oral form, and primary biliary cirrhosis.

Budesonide has been used by some veterinary clinicians in recent years to treat IBD in dogs and cats. Dose recommendations vary. In humans, a range of 6 mg to 9 mg per day has been used during initial therapy. In general, budesonide is administered to cats at 1 mg administered once per day (this dose level is prepared at a compounding pharmacy).

Budesonide can be used in combination with other drugs. Since cats tolerate corticosteroids very well, there is little indication to use budesonide as initial therapy for IBD. However, this may be a very attractive option for use in diabetic cats that also have IBD, or in patients where conventional therapies have not been sufficiently effective.

Potential adverse effects include PU/PD, when budesonide is used at the high end of the dose range, and GI ulceration. These reactions have been observed in some
human patients. These problems would be more likely to occur in dogs than in cats. It appears to be very safe when used at the levels listed above.

When combination therapy is indicated metronidazole is usually the first choice to be used in conjunction with prednisolone. Metronidazole’s mechanism of action includes an antiprotozoal effect, inhibition of cell-mediated immune responses, and anaerobic antibacterial activity. A dosage of 10 to 20 mg/kg two times daily is used for IBD. Ideally, at least several months of metronidazole therapy is given once it is started. In some cats with severe disease long term consecutive use or one to two month cycles of treatment may be required. Side effects to metronidazole at this low dose are uncommon in cats. Occasionally nausea or vomiting may be seen.

If a client is unable to successfully administer oral medications, methylprednisolone acetate (Depo-Medrol) can be used as sole treatment for cats with mild to moderate IBD or as adjunctive therapy when oral prednisolone and/or metronidazole are used as the primary treatment and flare-ups of clinical signs occur. Consistent control of clinical signs in cats with moderate to severe IBD is more difficult to maintain when methylprednisolone acetate is used alone, however. It is recommended that sole use of methylprednisolone acetate be reserved for situations in which the owner is unable to consistently administer tablet or liquid prednisolone preparations. Initially 20 mg is given subcutaneously or intramuscularly and is repeated at 2-week intervals for 2 to 3 doses. Injections are then given every 2 to 4 weeks or as needed for control.

If remission cannot be maintained with use of corticosteroids and metronidazole then chlorambucil (Leukeran) should be used. Azathioprine was used more in the past but it has been largely supplanted now by chlorambucil. Chlorambucil is an alkylating agent. Alkylating agents alter DNA synthesis and inhibit rapidly proliferating cells. Chlorambucil is administered initially at 0.1 to 0.2 mg/kg/day in conjunction with prednisolone at 2.2 mg/kg/day. The small pill size of chlorambucil (2 mg) allows for easy dosing. Most cats receive one-half tablet (1 mg) per day. Various dosage schedules for cats have been published. An alternate schedule is 0.15 to 0.3 mg/kg every 72 hours. Toxicities are uncommon in cats but may include anorexia, vomiting, and diarrhea, but these problems generally resolve rapidly when chlorambucil is reduced from daily to every other day administration. Bone marrow suppression is possible but uncommon, and is mild and rapidly reversible when it does occur. Once the desired clinical response is achieved, chlorambucil is gradually tapered over several months while prednisolone is continued as the primary maintenance drug.

Cyclosporine is another immunosuppressive drug that can be used in management of IBD. Cyclosporin inactivates calcineurin phosphorylase in T cells, preventing transcription of interleukin-2 (IL-2) as well as other cytokines. Cyclosporin inhibits activation of T cells, natural killer cells, and Langerhans (i.e., antigen-presenting) cells. Suppression of the Th1 or Th2 response induces antigen tolerance. The dose is 5 mk/kg once daily. Once sufficient response is achieved the dosage interval can be reduced to administration of a full dose every 48 hours and subsequently even further, on an individual patient basis.

**Cobalamin therapy in cats:** Significant tissue level cobalamin deficiency is present in some animals with GI disease. This is usually secondary to reduced cobalamin
absorptive capacity. It is essential that all cats with any form of GI disease (including involvement of liver, stomach, pancreas, intestines) have a serum cobalamin level run to determine if the patient is hypocobalaminemic. Response to therapy will be limited if low cobalamin levels are not resolved. The reference range for cobalamin in cats is 290-1500 ng/L. Therapy is given if the value is less than 500 ng/L (i.e., in the low part of the reference interval; don’t wait until the level drops below the low end point of the reference range).

Therapy involves administering injectable cobalamin at the following schedule for cats: 250 ug subcutaneously once a week for 6 weeks, then every 2 weeks for the next 6 doses, then dose monthly. Most generic cobalamin preparations contain 1 mg/ml (1000 ug/ml). It is important to note that multi-vitamin and B-complex injectable formulations contain significantly lower concentrations of cobalamin and they also cause pain when injected. Therefore, it is recommended that these preparations not be used for cobalamin supplementation. Unless the intestinal disease is totally resolved, long-term and perhaps lifelong supplementation with cobalamin may be necessary. The frequency of injections on a long-term basis is determined by regular measurement of serum cobalamin concentration.

Because dietary allergens may play a role in the cause if IBD, specific dietary therapy may be beneficial. Often, moderate to severe degrees of IBD are either temporarily responsive or only minimally responsive to careful dietary manipulations. However, long term control of IBD with as minimal a drug administration schedule as possible may be aided by specific dietary management. This should be started as soon as a diagnosis is made and continued as drug therapy is decreased later. Feed elimination (novel protein) or hydrolyzed protein diets. Chicken, duck, lamb, fish, or venison based diets are often tried initially. Elimination diets have been found to be very beneficial in cats.

**Poor responses to treatment** of cats with IBD usually result from:

1. Inadequate initial or long-term maintenance corticosteroid dosage in cats with more severe forms of IBD (moderate to severe disease).
2. Failure to use ancillary medications (metronidazole, chlorambucil, cyclosporin) in cases where disease is moderate to severe.
3. Failure to recognize and treat a concurrent condition (e.g., gastric hypomotility disorder that may either be secondary to IBD or idiopathic in nature, hyperthyroidism, parasitism [e.g., Giardia, Cryptosporidium], Clostridium perfringens enterotoxicosis, cholangitis/cholangiohepatitis, chronic pancreatitis).
4. Treatment for only small intestinal inflammatory disease when colitis is present as well. Some cats with concurrent IBD and colitis may show minimal or no clinical signs of colitis.
5. Failure to recognize and treat low body cobalamin levels (measure serum cobalamin).
6. Failure to identify an effective diet.
7. Poor client compliance

**What If Biopsies are Not Definitive for Either IBD or Small Cell Lymphoma?**
It can be difficult to definitively differentiate benign IBD from small cell intestinal lymphoma, even when full thickness intestinal biopsies are obtained. If the biopsies were obtained via endoscopy, one option is to proceed to exploratory laparotomy to obtain full thickness samples. However, this is not practical in some cases and involves a more invasive procedure and more expense. Further, there is no guarantee that the differentiation can be made even when full thickness samples are obtained. Another option that is employed more commonly now is to perform special tests to help differentiate benign IBD from low-grade, small cell lymphocytic malignant lymphoma. Specific immunohistochemical techniques can be done to identify populations of malignant B and T lymphocytes (i.e., phenotyping) and molecular (PCR) testing is done for clonality. Clients should be given the option of ordering these additional tests if the pathologist indicates on the initial histopathology interpretation that the differentiation can’t be made definitively between IBD and lymphoma. If the client declines to have the additional tests performed, the clinician then needs to decide whether or not to just go ahead and treat for the disease that poses greater concern, i.e., lymphoma. Low grade small cell lymphoma is often treated with the combination of prednisolone and chlorambucil (see later discussion on treatment details in the next section).

**Treatment of Intestinal Lymphoma in Cats**

Lymphoma is the most common feline neoplasm. It is also the most common form of gastrointestinal neoplasia in cats. Gastrointestinal lymphoma is often referred to as either well differentiated (low grade or lymphocytic), poorly differentiated (high grade, lymphoblastic, or immunoblastic), and intermediate (or mixed). Endoscopy has been shown to be a very useful modality for diagnosis of intestinal lymphoma in cats, especially when multiple biopsies are obtained using proper technique and instruments that can procure adequate size tissue samples. Immunohistochemical stains are beneficial for differentiating IBD from intestinal lymphoma in cases where it is difficult for the pathologist to distinguish between the two. Full thickness intestinal biopsies may be required in a very limited number of cases in order to establish the correct diagnosis.

Many cats respond favorably to treatment for intestinal lymphoma, especially with the low grade or chronic lymphocytic type. Clinical signs can be very similar to cats with IBD. Therefore, it is strongly recommended that cats with chronic GI signs undergo a biopsy procedure as early as possible, so that the correct diagnosis can be established and the best course of therapy be made available for each individual cat. Biopsies should be obtained from both the upper and lower (ileum) small bowel.

Multi-agent chemotherapy is recommended for all cats with GI lymphoma. Surgery is done only if there is an isolated mass that is causing some degree of luminal obstruction. Survival times in excess of 12 to 18 months are not unusual. In some cats the response is somewhat shorter (three to six months). The prognosis for longer survival time is much better if the diagnosis is made before clinical signs become chronic and debilitation results.
One study has reported excellent results in cats with chronic lymphocytic lymphoma using a protocol of prednisone (10 mg PO per cat per day) and chlorambucil (Leukeran) at a dosage of 15 mg/m² PO, once every day for 4 days, repeated every 3 weeks (Note: prednisolone is used routinely at this time, rather than prednisone, in cats). Sixty-nine percent of the cats with lymphocytic lymphoma treated with this regimen achieved a complete remission. The median disease free interval for cats that achieved complete remission was 20.5 months (range, 5.8-49 months). The median survival for all cats with lymphocytic lymphoma treated with chemotherapy was 17 months (range, 0.33-50 months). Cyclophosphamide (Cytoxan) was used for rescue in some of the cats that were entered in this protocol (225 mg/m², PO, every 3 weeks). For further reference on this protocol, see Richter, K: Feline gastrointestinal lymphoma, ACVIM Proceedings 2001, p. 547-549.

The protocol that Dr. Tams has used most often for cats with the more aggressive lymphoblastic form of GI lymphoma was originally published by Cotter in 1983. Dosage levels have been modified slightly since that time. This protocol utilizes cyclophosphamide, oncavin, and prednisolone (COP). This protocol can be easily managed in any practice setting. Vincristine is administered intravenously at a dose of 0.5-0.75 mg/m² once weekly for 4 consecutive weeks and then once every 3 weeks. The initial doses are often decreased by approximately 25 percent for cats that are inappetent or debilitated. If well tolerated the dose can then be gradually increased. Care is taken to ensure that none of the vincristine is given extravascularly. The average volume that is administered is quite low (0.1 to 0.15 ml for many cats, using a vincristine concentration of 1 mg/ml). Cyclophosphamide is given orally at a single dose of 225 mg/m² every 3 weeks (50 mg tablets are used with dosage adjusted to the nearest 25 mg on the low side of the calculated dose). Prednisolone is given orally at 10 mg per cat per day. Although cyclophosphamide and vincristine can be given on the same day I often prefer to have the owner administer the cyclophosphamide 2 to 3 days after the oncavin. A CBC is done several times during the first month and then every 3 weeks to be sure that adequate granulocytes are present before treatment. At least 3,000 granulocytes/ul must be present before cyclophosphamide is given. If the granulocyte count drops to less than 1,000/ul 5 to 7 days after cyclophosphamide, the dose for subsequent treatments is reduced by 25 percent. The highest non-toxic dose is most likely to result in the greatest tumor cell kill.

The COP protocol is generally well tolerated, although side effects may occur and dosage or interval adjustments may be necessary. Side effects of COP in cats may include anorexia, vomiting, lethargy, and severe tissue irritation if any vincristine is given extravascularly. Also, the haircoat may become thinner, but complete hair loss does not occur. Cats do tend to lose whiskers. Cats should be carefully observed for sepsis especially during the induction phase. Prophylactic antibiotics are not indicated, but any infections that occur should be treated aggressively. Advantages of this protocol include hospital visits at only 3 week intervals after the first 4 weeks, lower cost to the owner, and a treatment interval that allows recovery of normal cells between treatments. I would like to emphasize that with careful monitoring and use of a dosage schedule that is tailored to each individual cat few problems are encountered. It is our general practice to encourage owners of most cats with GI lymphoma to pursue treatment that includes chemotherapy.
Nutritional and metabolic support are also important. If inappetence is a problem cyproheptadine can be administered as an appetite stimulant (1 to 2 mg orally every 12 to 24 hours) on an as needed basis (long-term if necessary). Mirtazapine is another appetite stimulant that can be used (one-fourth of a 15 mg tablet every three days). Intermittent vomiting, nausea, and inappetence is managed with maropitant (Cerenia) administered at 4 mg for most cats once orally daily as long as it is needed. If there is concurrent renal disease with azotemia or if dehydration is a problem owners are taught how to administer subcutaneous fluids at home (e.g., lactated Ringer’s 100 to 150 ml every 24 hours to 48 hours, based on each individual cat’s needs). Special attention is given to ensuring that low cobalamin levels are addressed, if serum tests indicate that hypocobalaminemia is present.

Rarely chemotherapy can be discontinued after one year. This is done only if follow-up endoscopic intestinal biopsies indicate that there is no remaining lymphoma. Most cats remain on treatment for the remainder of their lives. If chemotherapy is poorly tolerated and reduced dosages and increased intervals between treatment times are unsuccessful in adequately decreasing side effects chemotherapy should be suspended. Prednisolone should be continued however because it may help maintain remission for a period of time. Doxorubicin (Adriamycin) can also be used in cats.

For clinicians inexperienced in administering chemotherapy, or who have not treated many cats with intestinal lymphoma, it is recommended that a veterinary oncologist or internist be consulted for guidance on protocol selection and ongoing management. Many cats with intestinal lymphoma can be managed successfully for some period of time.

Further Reading:
THE ROLE OF DRY COW TREATMENT IN MASTITIS CONTROL

Marguerite Cameron, DVM PhD

Introduction
Good management of non-lactating cows is important for the control of intramammary infection and mastitis in subsequent lactations. From an udder health perspective, the purpose of the dry period is to provide an opportunity for mammary epithelial cells to regress, proliferate, and differentiate with the ultimate goal of maximizing milk production (Capuco et al., 1997). As a result of this regenerative process, the non-lactating period presents an ideal opportunity to improve the health of the mammary gland via the clearing of existing intramammary infections. Concurrently, the dry period is recognized as the stage of the lactation cycle with the highest incidence of new intramammary infection. Thus, the dry period is a double-edged sword of mastitis control.

Dry Period Physiology
Three phases of the dry period have been described: active involution, steady-state involution, and colostrogenesis (Smith and Todhunter, 1982). During the first phase of the dry period (involution), there is an increase in susceptibility to infection as a result of the cessation of milking and a slow transition to steady-state involution (Sordillo and Nickerson, 1988; Bradley and Green, 2004). Once fully involuted, protection against new intramammary infection is partially conferred by the presence of a keratin plug which blocks the teat orifice and teat canal (Comalli et al., 1984). During the last phase of the dry period, the mammary gland is once again at increased risk for new intramammary infection. With impending parturition, colostrum production is initiated and the accumulation of mammary secretions leads to the dilution of protective factors within the gland (Bradley and Green, 2004). Furthermore, as calving approaches, the keratin plug breaks down leaving the teat canal patent and in danger of bacterial invasion (Oldham et al., 1991).

Mastitis Control During The Dry Period
While clinical mastitis is rare in non-lactating cows, intramammary infections present at calving are a significant cause of clinical mastitis in early lactation (Bradley and Green, 2000; Green et al., 2002). The aim of mastitis control during the dry period is to minimize the prevalence of intramammary infection at the beginning of the next lactation. In order to attain that goal, both prevention of new infections and effective clearing of existing infections must occur.

Dry Cow Antibiotic Therapy
Antibiotic treatment of intramammary infection during the dry period has benefits over treatment in lactation for reasons such as the following: 1) a higher dose of antimicrobial can be used; 2) in the absence of milking, antimicrobials are maintained within the mammary gland parenchyma for longer periods; 3) dry cow therapies are formulated to be slow-release and long-acting; and 4) the risk for contamination of saleable milk is reduced (Blowey and Edmondson, 2010). Consequently, when dry cow
therapy is used to treat existing intramammary infections present at drying off, high cures rates can be achieved. Regarding protection against new intramammary infection, the effectiveness of dry cow therapy is uncertain. Despite infusion with dry cow therapy at drying off, dry period new intramammary infection risks of 13.3 to 25.4% have been reported (Godden et al., 2003; Gundelach et al., 2011; Arruda et al., 2013). As a result of declining concentration of active compound over time, dry cow therapy does not protect against new intramammary infections in the periparturient period when infection rates are known to be high (Oliver et al., 1990).

**Internal Teat Sealants**
Internal teat sealants are a non-antimicrobial alternative to dry cow therapy for the prevention new intramammary infection during the dry period. Internal teat sealants are an inert viscous paste, 65% bismuth subnitrate by weight, which forms a physical barrier within the teat cistern and canal thus preventing entry of bacteria into the mammary gland (Godden et al., 2003). Internal teat sealants are effective against all bacteria, including Gram-negative coliforms, and have been shown to persist within the teat canal for up to 100 days (Woolford et al., 1998).

**Blanket Dry Cow Therapy**
In the late 1960s, with the knowledge that cows are highly susceptible to new intramammary infections during the non-lactating period, mastitis researchers made the recommendation to infuse all quarters of all cows with dry cow therapy after the last milking prior to drying off. This practice, known as blanket or total dry cow therapy, became a mainstay of mastitis control following its inclusion in the Five Point Mastitis Control Plan and it has persisted to the current day as a control point in the National Mastitis Council’s Recommended Mastitis Control Program. Concerns regarding antimicrobial drug use in food animal production systems as a contributor to the development of antimicrobial resistance are increasing across the world (Call et al., 2008; Oliver and Murinda, 2012). Consequently, a significant challenge facing the dairy industry is the mounting pressure to reduce the amount of antimicrobials used in production.

**Selective Dry Cow Therapy**
Considering that the prevalence of intramammary infection at the end of lactation is estimated at 28 to 41% of cows, blanket dry cow therapy does result in the over-usage of antimicrobials for the purpose of eliminating existing infections (Browning et al., 1994; Sanford et al., 2006b; Torres et al., 2008; Torres et al., 2009). With the advent of internal teat sealants, producers have at their disposal an alternative for the prevention of new intramammary infection over the dry period. Choosing cows for infusion with dry cow therapy based on known or suspected infection status at the end of lactation is known as selective dry cow therapy. Selective dry cow therapy has the potential to reduce the amount of antimicrobials used in dairy production and is considered a more targeted approach to the control of intramammary infection during the dry period (Berry and Hillerton, 2002; Robert et al., 2006; Rajala-Schultz et al., 2011). Addition of an internal teat sealant to a selective treatment protocol will ensure that all quarters will have some form of protection against new intramammary infection during the dry period.

**Selection Procedures In Selective Dry Cow Therapy**
The success of a selective dry cow therapy program depends on the accurate identification of a cow’s intramammary infection status at drying off so that appropriate treatment decisions can be made. Recently, an on-farm culture system has been developed and validated for selective dry cow therapy (Cameron et al., 2013; Cameron et al., 2014). The system utilizes 3M Aerobic Count Petrifilms (3M Canada, London, Ontario) to culture composite milk samples directly on the farm and provides results within 24 hours, enabling producers to make selective treatment decisions based on the current intramammary infection status of a cow. The accuracy of historical somatic cell count and clinical mastitis criteria were greatly improved when Petrifilm-based on-farm culture was added to the protocol with a sensitivity of 85% and specificity of 73% (Cameron et al., 2013). Furthermore, when on-farm culture system-based selective therapy was applied in low bulk tank somatic cell count herds (< 250,000 cells/mL) on low somatic cell count cows (< 200,000 cells/mL), the risk of intramammary infection at calving and clinical mastitis in the first 120 days of the next lactation was equivalent to that observed in cows under blanket dry cow therapy (Cameron et al., 2014).

Thorough reviews of general mastitis prevention for non-lactating cows and heifers can be found in Bradley and Green 2004 and McDougall et al., 2009.

Further Reading:
LARGE ANIMAL BOVINE MASTITIS

LARGE ANIMAL BOVINE MASTITIS

EMERGING MASTITIS PATHOGENS

Marguerite Cameron, DVM PhD

Introduction
While coagulase negative staphylococci (CNS) and environmental streptococci are not new mastitis pathogens, with the application of Matrix-Assisted Laser Desorption/Ionisation Time-of-Flight Mass Spectrometry (MALDI-ToF MS) for the identification of bacteria isolated from bovine intramammary infection, our ability to detect members of these bacterial groups have greatly improved.

Coagulase negative staphylococci
Coagulase negative staphylococci were the most frequently isolated family of pathogens in the National Cohort of Dairy Farms study of the Canadian Bovine Mastitis and Milk Quality Research Network (Reyher et al., 2011) and have been reported to be the most prevalent in several other countries as well (Pyörälä and Taponen, 2009). While intramammary infection with CNS typically results in moderate increases in somatic cell count (SCC) and rarely develops into clinical mastitis, research has shown that the role of CNS is of greater importance in herds with a low bulk tank SCC (Schukken et al., 2009; Pyörälä and Taponen, 2009; Sampimon et al., 2010). Schukken et al. (2009) observed that in herds with an average bulk tank SCC < 200,000 cells/mL, CNS-infected quarters contributed more somatic cells to the bulk tank than quarters infected with a major pathogen. At least 24 different CNS species have been isolated from clinical milk samples, however CNS are normally not identified to species level but treated as a uniform group (Vanderhaeghen et al., 2015). While commonly considered opportunistic pathogens found on the skin of cows, research has established that CNS originate from multiple sources, including both the cow (such as infected glands and bovine skin) as well as the farm environment, and that reservoir type and mode of transmission are species-specific (Piessens et al., 2011; Piessens et al., 2012). According to the results of the NCDF, the most common species of CNS isolated on Canadian dairy farms were S. chromogenes (~48%), S. simulans (~19%), S. xylosus (~10%), S. haemolyticus (~8%), S. epidermidis (~4%), and S. cohnii (~3%; Fry et al., 2014).

DETAILS OF THE 5 MOST COMMON SPECIES OF CNS (Vanderhaeghen et al., 2015; De Vliegher, 2015)
S. chromogenes
- Most commonly isolated CNS in bovine milk
- Udder-adapted; common inhabitant of bovine skin
- Opportunistic > contagious in nature
S. haemolyticus
- Most diverse of all CNS with a variety of habitats
- Most likely opportunistic
S. epidermidis
- Colonizer of human skin; humans important source of infection
- Udder-adapted
- Variety of habitats
- Opportunistic and contagious
- Presence is farm-dependent
• Associated with SCC >200,000 cells/mL

*S. simulans*
• Most likely contagious spread
• Main source is unclear
• Presence is farm-dependent
• Associated with SCC >200,000 cells/mL

*S. xylosus*
• Inhabitant of bedding, but has been found in a variety of habitats
• Mode of infection unclear

**Other environmental streptococci**
With better control of contagious mastitis pathogens, the epidemiology of mastitis has changed and environmental pathogens have become the major cause of clinical mastitis on many dairy farms. Due to difficulties in differentiating *Lactococcus*, *Streptococcus*, and *Enterococcus* by conventional biochemical techniques, the importance of other streptococci as mastitis pathogens may have been underestimated. According to the results of the NCDF, other streptococci were the third most common pathogen isolated from clinical mastitis and follow-up samples (Reyher et al., 2011). MALDI-ToF MS analysis of a subset of 175 isolates of other streptococci from the NCDF revealed that 42% were *Aerococcus viridans*, 22% were *Enterococcus spp.*, and 19% were *Lactococcus spp.* (unpublished data).

*Aerococcus viridans* (Devriese et al., 1999; Wyder et al., 2010; Spakova et al., 2012)
• Commonly reported isolate from bovine milk;
• Generally considered to be apathogenic;
• Reported to be third most common species of Gram-positive, esculin-positive, catalase negative streptococci isolated from subclinical intramammary infections;
• Data on the importance of A. viridans in bovine intramammary infection is lacking because often misidentified as Streptococcus uberis.

*Lactococcus species* (Devriese et al., 1999; Plumed-Ferrer et al., 2013; Werner et al., 2014; Malvisi et al., 2016)
• *Lactococcus lactis* is used as a starter culture in the production of dairy products such as cheese and buttermilk;
• *L. lactis* produce bacteriocins that have been used in the treatment of bovine subclinical and clinical mastitis;
• *L. lactis* as a mastitis pathogen likely underreported due to misidentification;
• *Lactococcus spp.* have been linked to high bacteria counts in bulk tank milk;
• *L. garvieae* is closely related to *L. lactis* and has also been associated with bovine mastitis, but to a lesser degree.

As our ability to accurately identify members of CNS and other streptococci continues to improve, the role these varied bacteria play in bovine mastitis will be elucidated. With this new knowledge and species-level diagnosis, our ability to better control opportunistic and environmental pathogens will improve.
Further Reading:

THE MASTITIS TRIANGLE (Dr. Andrew Johnson, Grande Milk Marketing, WI)

COMPONENTS OF A SUCCESSFUL MILKING ROUTINE

Cows are calm prior to milking
Adrenalin blocks the action of oxytocin, resulting in poor milk letdown, reduced milk flow, and increased residual milk; calm cows enter and exit the parlour smoothly and readily, resulting in more efficient milking.

Cows are clean prior to milking
Even the best udder preparation will only reduce bacterial load on the teats by 85%, therefore if you start with 1 million bacteria, at best you can reduce this to 150,000. “Mastitis control is a numbers game” (Dr. Andrew Johnson). Plus, dirty cows take much longer to prepare for milking.

Cows are milked with a consistent milking routine
Cows are creatures of habit. Cows milked with a consistent operating routine have better milk letdown, produce more milk, and have better milk quality. Consistency in the milking routine (same procedures by every milker and at every milking) requires training, periodic refresher training, and posted Standard Operating Procedures.

All milkers wear gloves and gloves are clean
Gloves greatly reduce the risk of pathogen transfer to cows and protect the milkers’ hands. Gloves should be changed or disinfected between groups, after handling cows with mastitis, or when visibly dirty.

WHAT IS THE GOAL OF THE PREMILKING ROUTINE?
TO HAVE CLEAN, DRY, STIMULATED TEATS!
**Fore-Stripping**
The process of removing two to three streams of milk from each teat prior to milking aids in the detection of mastitis, stimulates milk letdown, and removes the worst quality milk contained within the tear cistern. Fore-stripping can be done before or after the pre-dip is applied, but *never* after drying.

**Pre-dipping**
Pre-dipping works best when teats are clean as organic matter reduces teat dip activity. Dry wiping teats before dipping can reduce the organic matter on the teats. Pre-dip should be applied in a manner that 75-90% of each teat is covered; pre-dip should have 20 to 30 seconds contact time before being wiped off.

**Drying**
Complete drying of the teats (don’t forget the teat ends!) is considered to be the most important step for reducing the bacterial load on teats. Always use clean, sanitized and dry towels, and one towel per cow. Use the twist method the get the teat ends clean.

**Lag time of 90 seconds (range 75-120)**
Lag time is the time between fore-stripping and the attachment of the milking unit. Ensuring that proper lag times are followed will lead to fast and complete emptying of the udder. Teats should be swollen with milk when the units are attached.

**Units are properly attached and aligned**
Unit attachment should follow a standardized procedure that minimizes air leaks. The milking unit should be aligned so that the cluster is directly beneath the cow and the cluster weight is evenly distributed between the teats. Proper alignment will reduce the frequency of liner slips and squawks, and will ensure that the cow is milked out evenly.

**Units are properly removed**
Units should be removed when the flow of milk falls to 0.8 to 1.0kg/minute to ensure teat health and reduce the risk of mastitis. The vacuum should always be shut off before teat cups are removed.

**Post-dipping**
Using an approved product and ensuring good coverage, post-dipping will reduce new intramammary infections by >50%. Dipping is better for achieving good coverage than spraying. Cows should remain standing for at least 30 minutes after milking until the teat sphincter closes.

**Further Reading:**
AVOIDING A DRUG BUST: MEDICATIONS IN PERFORMANCE HORSES

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To protect the health and welfare of horses, veterinarians must legitimately use therapeutic medications. Therapeutic medications have been defined by the American Association of Equine Practitioners. While these drugs are commonly and appropriately used to treat various disease conditions of horses, many of them have the ability to affect athletic performance. For performance horses, the primary reason to have drug rules is to ensure fair competition and to protect the welfare of the horses. However, the sensitivity of drug testing allows for laboratory detection of very small quantities of drug that may even be pharmacologically insignificant for performance horses. This makes it extremely difficult for veterinarians to advise owners and riders about long a treated horse must wait before being entered in a competition. Clearly, if horses are to receive proper veterinary care, suitable information on drug detection times and withdrawal times must be available to equine practitioners.

Drug Rules and Performance Horses

Equine competitions may be regulated by a local or discipline-specific group, state or provincial agency, national federation or agency, or international federation. Drug rules vary enormously between sport organizations, but there are 4 basic types of medication rules for competitive horses. The first is the “no drug” rule, which stipulates that no trace of any drug can be found in any body fluid. This is also known as the “oats, hay and water” rule. The second type of drug rule is the time rule, where the period of time before a competition during which drugs cannot be administered is defined. The third type of rule is no rule at all. Virtually all equine sport organizations have drugs whose use they disregard, as they have no influence on performance (eg, dewormers, vitamins, antibiotics, fly spray). The fourth rule is the “tolerance level” rule, where acceptable concentrations of drug are established and horses are only penalized if this concentration is exceeded at the time of competition. Many equine sport organizations have “therapeutic substance provisions” and permit the detection of small concentrations of drugs, such as nonsteroidal anti-inflammatory drugs, without penalty.

Equestrian competitions such as Federation Equestre International (FEI) sanctioned competitions and long distance riding competitions tend to have “no drug” rules. Horses are required to compete entirely on their natural abilities without being influenced by any drug, medication or veterinary treatment. When competing horses are tested, the “no drug” rule means that any trace of a drug or drug metabolite is called as a positive test. Simple confirmation of the presence of any drug is evidence that the rule has been broken (primae facia evidence). While this is a simple and straightforward policy and appeals to those concerned about horse welfare, drug testing methods are now advanced enough to detect very small amounts of drugs for long periods of time after they have been administered, at concentrations that have no effect on performance. This also puts the sport’s drug rule solely into the hands of the
analytical chemist. As they develop a more and more sensitive tests, drug violations are called for insignificant traces of drugs, causing unfair penalties to competitors and preventing veterinarians from administering valid therapeutics to horses out of fear of causing positive tests. It is actually rare in equine sports for a positive drug test to mean that someone intentionally tried to alter the outcome of the competition. Violations of the drug rules are usually due to carelessness or inattention to dose or dosage form, misunderstanding of the directions or using a therapeutic medication for a medically indicated reason, and not waiting a suitable “withdrawal time” before entering a horse in a competition, or cross-contamination from people or the environment picked up by ultra-sensitive testing methods. In some equine competitions such as flat racing, unscrupulous persons may deliberately administer drugs to horses with the goal of altering performance. With parimutual betting, large sums of money can be involved in “fixing” of racehorses, with some being drugged to win and some being drugged to lose (“nobbling”).

Under the rules of most competitive organizations, a “positive” drug test result is an unarguable fact and penalties are imposed, but there is no uniformity between organizations on the penalties imposed. The Association of Racing Commissioners International, Inc. (ARCI) classifies over 700 drugs into five classes in decreasing order of their potential to affect a horse’s athletic performance. With this system, lay persons with little knowledge of drugs can understand the performance altering potential of a detected drug and determine appropriate penalties for infractions. According to the ARCI system, Class I drugs have no recognized therapeutic use in equine medicine, stimulate or depress the central nervous system, or have a history of abuse in humans or horses. Examples include opiates and amphetamines. Class II drugs include therapeutic agents that have a high potential to affect performance. They include phenothiazines (except acepromazine and promazine), benzodiazepines, barbiturates, opioid agonist-antagonists and local anesthetics (except procaine). Class 3 drugs may have generally accepted uses in horses and less potential for altering performance than Class 2 drugs. It includes bronchodilators, vasodilators, potent diuretics affecting renal function and body fluid composition, antihistamines, and local anesthetics with minimal potential for use in nerve blocking or which have a high potential for detection in urine, such as procaine. Class 4 drugs are therapeutic medications with some potential for altering performance. It includes antiarrhythmics and cardiac glycosides, gastrointestinal antisapsmotics, nonsteroidal anti-inflammatory drugs, corticosteroids, mineralocorticoids, and skeletal muscle relaxants. Class 5 drugs are therapeutic drugs of only minor regulatory interest and include anti-ulcer medications such as cimetidine and omeprazole. Non-classified drugs are considered to be of no interest to regulatory authorities and include antimicrobials, sulfonamides, anthelmintics and vitamins.

**Drug Testing Methodology**

Drug testing laboratories for equine athletes may be privately owned or government run and predominantly deal with samples from race horses, where there are strong economic incentives for “fixing” the outcome of a race. Depending on the jurisdiction, drug testing protocols may require urine or blood samples. Blood samples are ideal in that they are easily collected and the amount of drug in the blood can be correlated with its effect on performance. Because it is an invasive procedure, collection of blood usually must be done by or under the direct supervision of a veterinarian. Urine
testing is a better method to simply determine whether or not a horse has been given a drug, since most drugs are eliminated in the urine, it tends to contain higher levels of drugs for a longer period of time than blood samples. Urine collection is not invasive and may be performed by a lay person. However, urine collection is slow and it may be difficult to collect from horses dehydrated from exercise. Urine testing provides a simple “positive” or “negative” result, as drug levels in urine do not determine if a horse had a blood concentration of drug that was therapeutic or performance altering.

One of the major problems with a “no drug” policy is that there is little standardization between testing laboratories as to which testing methodology is used and they can arbitrarily start using a more sensitive test. In effect, the testing laboratory decides how rigorous the “no drug” policy will be when they select the test method. Unfortunately, veterinarians and competitors are unlikely to know what screening test is being used or may not know that the screening test (and therefore the detection time) has been changed until notified by the sport organization of a drug rule violation.

**Drug Detection Times versus Drug Withdrawal Times**
A drug “detection time” is the time period after the drug has been administered that the testing laboratory can detect the drug with accuracy and repeatability. This is specific to the laboratory and subject to change as testing methodology changes. Detection times are also typically determined from studies using only small numbers of horses, so the reporting organization typically has clear disclaimers that this information may not be applicable to your horse (eg, the Canadian Pari-Mutuel Agency guidelines have this on every page). A “withdrawal time” is the period of time that the horse is kept from competition to make sure that a positive test does not occur. It must be determined for an individual horse by the treating veterinarian taking into account a number of factors and should always be longer than the organization’s detection time.

**Determining Drug Withdrawal Times**
The amount of a drug given to a horse has a great effect on drug withdrawal time. For some equine medications, the doses are very small yet for others, the amount of drug given is very large. For example, the butorphanol is usually dosed at 500 micrograms to a 1000 lb horse, while the dose of phenylbutazone to the same horse would be 3 grams. Obviously, the more molecules of drug given, the easier it is going to be to detect amounts of the drug in blood or urine. Most drugs are eliminated in the urine by the kidneys, but some drugs may also be eliminated in bile and feces through the intestinal tract and through sweat, tears, saliva and other body secretions. The elimination half-life (t½) is defined as the amount of time for the drug’s concentration to decrease by one half. For any drug, this is determined by administering a dose of the drug to a test group of horses then taking repeated blood samples. The concentrations of the drug in the serum or plasma are measured, and when the concentrations are graphed over time, the drug’s elimination half-life can be calculated. This rate of drug elimination stays constant over time until the drug can no longer be measured in the blood. The value for t½ depends on the chemical properties of the drug and the horse’s elimination mechanisms. From simple mathematics, after $10 \times t\frac{1}{2}$’s the drug concentration in the horse’s blood will have decreased by 99.9%, but it will take about 70 t½’s to clear all the drug molecules from the body. In general, the detectability of a drug depends on the amount of drug administered and the speed at
which the horse eliminates the drug. If the drug is administered in gram amounts, such as phenylbutazone, and it has a long t½, then it will be detectable in blood or urine for a relatively long period of time. On the other hand, if it is administered in milligram or microgram amounts and it has a very short t½, then it is likely to only be detectable for a short period of time.

One of the major reasons for the difficulty in making drug withdrawal time recommendations is the variability of drug elimination between horses. When a group of horses are given a drug and then the drug concentrations are measured in their plasma, the measured drug concentrations are spread out in a peculiarly skewed fashion, with a cluster of levels at the lower end of the distribution, but a longer tail at the higher concentrations.(5) If withdrawal time recommendations are made from studies using small numbers of horses, they will tend to miss the horses that contribute to the high tail of the distribution curve. So veterinarians should be cautious regarding withdrawal time recommendations made from studies using small numbers of horses, such as the Canadian Pari-Mutual Agency’s recommendations.(10) It appears that you have to test at least 50 horses to get an accurate picture of the skewness of the distribution of drug concentrations.(5)

So What’s a Veterinarian or Competitor to Do?
Riders, owners and trainers of equine athletes should always work with their veterinarian to provide the best medical care for their horse. Valid medical therapy should never be withheld out of fear of a positive drug test. Common sense and some understanding of the properties of drugs and the limitations of drug testing will help everyone to avoid drug rule violations.
Compounding of veterinary drugs is both necessary and beneficial for the treatment of animals. In Canada, approved drugs can be identified by a “Drug Identification Number”. Unlike the United States, where pharmaceutical compounding is federally codified, veterinary compounding in Canada is guided by Health Canada policy, provincial veterinary and pharmacy regulations, and guidelines from the National Association of Pharmacy Regulatory Authorities (NAPRA) and the Canadian Veterinary Medical Association (CVMA).

**COMPOUNDING DONE RIGHT**
The first step in valid veterinary compounding is the establishment of a valid veterinarian-client-patient relationship. The veterinarian must establish that there is no approved animal or human drug that, when used on label or extralabel, can be used to treat the diagnosed condition and that the health of the patient is threatened, or suffering or death may result from failure to treat with the compounded product. The compounding must be performed by a licensed pharmacist upon the prescription of a veterinarian or by a veterinarian if allowed by their province’s pharmacy law. The compounded product must be safe and effective and the compounding operation must be consistent with providing small quantities of product for very specific patient needs.

Preparations should only be compounded from Health Canada-approved animal or human drugs. In the United States, the FDA regulations do not permit compounding from bulk (active pharmaceutical ingredient, API) chemicals. While currently permitted in Canada, Health Canada is looking to tighten regulations on compounding from APIs for use in animals. Veterinarians may occasionally face situations where they need to treat conditions for which there are no approved products available for compounding (e.g., pergolide for horses before Prascend® was approved, cisapride for cats).

**COMPOUNDING DONE WRONG**
When drugs are compounded without adherence to current good compounding practices, veterinary patients may be harmed, the public health may be endangered and the prescribing veterinarian may be liable. Because compounded drugs are typically used in small numbers of patients, if things go wrong it usually does not draw much attention. But recent compounding mishaps were significant enough to draw international attention. In 2009, twenty-one Venezuelan polo horses died acutely in Florida after Franck’s Pharmacy, a veterinary compounding pharmacy, incorrectly prepared a vitamin-and-mineral cocktail that was injected into the horses prior to their polo match. Due to a mathematical error (the easily misplaced decimal point) in the concentration of selenium, the solution was too potent and caused fatal selenium toxicity. In October 2012, an outbreak of fungal meningitis, paraspinal/spinal infections and peripheral joint infections occurred in the United States. The US Centers for Disease Control and Prevention traced the outbreak to contamination with a black
mold called Exserohilum rostratum in three lots of preservative-free methylprednisolone acetate solution used for steroid injections. The FDA-approved methylprednisolone acetate (Depo-Medrol®) was sold by Pfizer and two generics companies, but since the compounding’s version did not contain preservatives, it sidestepped the regulatory process with tragic results. Doses from these three lots had been distributed to 75 medical facilities in 23 states, and administered to approximately 14,000 patients. As of May, 2013, 741 people have had infections from the contaminated product and 51 people have died.

Compounding Versus Manufacturing
Traditional compounding was limited to a pharmacist or a physician meeting the therapeutic needs of a specific patient. Once disconnected from individual patients, compounding essentially becomes drug manufacturing. Often compounded products are marketed as cheaper alternatives to the approved drug. All compounding guidelines state that cost is not a defensible reason for prescribing a compounded drug. But in actuality, price is the main reason for using compounded drug products when approved products are readily available. In the US, the FDA has taken action against some pharmacies for large scale compounding from bulk APIs and aggressive marketing of such products.

Problematic Packaging
Guidelines state that a compounded product should not be made to look like an approved product. Yet I frequently see packaging that looks very much like an approved veterinary product and “compounded” is often not indicated on the box or the bottle. In Canada, an approved human or veterinary drug product has a DIN (drug identification number). One compounding pharmacy puts a “CIN” number on their products, but it has no meaning other than to the compounder. I suppose it means “Canadian Identification Number”, but to me it means it’s a sin to sell this stuff!

Problematic Product
Compounding is legitimate if an approved product is available but the appropriate method for dosing or the drug concentration is not suitable and a practical alternative does not exist. One of the most frequent examples that I see violating this requirement is compounded oral liquid phenylbutazone. Considering that there are approved tablet, paste and flavoured granule formations, it is very difficult to make the case that a liquid formulation is necessary to save equine lives!

There are numerous publications documenting the poor quality of compounded drug products. Many products contain less than the stated drug concentration, but some have been to contain more drug than stated. So there is a much greater chance of under dosing (leading to therapeutic failure) or overdosing (leading to toxicity) than with an approved drug product. In performance horses, there is the additional issue of poor quality compounded drugs resulting in “positive” tests during competition, resulting in loss of purse or prize money, suspensions from competition and the public humiliation of being identified as violating competition drug rules.

Problematic Prescription Labeling
Provincial pharmacy regulations are explicit regarding the information required on a veterinary prescription. I’ve seen numerous instance of mislabelling on compounded
products and sometimes they have had no prescription labelling at all. The expiration date of an approved product should be based on known stability data, but stability testing is rarely done by compounding pharmacies for their products. If no stability data exists, products compounded for re-dispensing should have a “beyond use date” identical to the last date the treatment will be administered, as per the duration of the prescription, with a maximum dating of 180 days. A number of publications have demonstrated problems with stability of compounded veterinary drug products such as pergolide and doxycycline.

PRESCRIBER BEWARE
If you choose to prescribe compounded drugs, you need to counsel your client regarding potential adverse reactions, including therapeutic failure, and the potential for unintended human or animal exposure to the drug. You are obligated to inform your client that the compounded preparation has not been evaluated by Health Canada for potency, purity, stability, efficacy or safety, and client consent should be obtained in writing.

As a practicing veterinarian, if you choose to prescribe or dispense a compounded drug, you are responsible for its quality, safety, efficacy and potency and for following pharmacy regulations for proper labeling. You should report suspected adverse events including therapeutic failure and quality defects involving compounded drugs to the compounding pharmacist, the provincial Board of Pharmacy and Health Canada’s Veterinary Drug Directorate. Should an adverse drug reaction, toxicity or lack of therapeutic effect occur from a poorly prepared or labeled compounded product, you are responsible for the consequences, which can include client complaints and disciplinary action by your state or provincial veterinary medical regulators. Pharmacies are not required to carry product liability insurance, so you should be sure that your liability insurance will cover you. This may be difficult as there are no “registered label indications” on which an insurance company can base their decision on whether or not a product was used in accordance with best practices. If the manufacture of the compounded product violates federal regulations (e.g. compounded from bulk APIs), your liability insurance will not protect you. So maybe that approved drug product isn’t so expensive after all!

COMPOUNDING GUIDELINES

CVMA Guidelines for the Legitimate Use of Compounded Drugs in Veterinary Practice (http://www.canadianveterinarians.net/programs/national-issues.aspx#.UaeFTdj8_kc)

NAPRA Guidelines to Pharmacy Compounding (http://napra.ca/pages/Practice_Resources/guidelines_to_pharmacy_compounding.aspx)
In equine practice, antimicrobial drug use is common for treatment of local and systemic bacterial infections and surgical prophylaxis. The initial choice of antimicrobial therapy is often empirical, as delaying treatment while waiting for culture and susceptibility results can have disastrous consequences for athletic performance or even survival. Veterinarians make their empirical choices based upon information from equine medicine textbooks and published reviews and “within the practice” traditions. In our recent North American survey, we found that antimicrobial use by equine practitioners frequently deviates from published recommendations. The most obvious concerns raised by the survey were the use of antimicrobials without justification (e.g., routine castration, treatment of recurrent airway obstruction) and serious infection cases requiring broad spectrum antimicrobial therapy where there was little consensus on the ideal therapy. Despite less than prudent antimicrobial use by equine practitioners, antimicrobial susceptibility data from equine hospitals with a predominantly first opinion caseload show that equine pathogens remain consistent in their antimicrobial susceptibility and multi-drug resistance bacterial infections in horses are rare. The following recommendations for equine antimicrobial therapy are based on the bacterial culture results from cases at the Western College of Veterinary Medicine seen over the last 10 years.

**Respiratory Tract Infections**

**Upper Respiratory Tract**

Streptococcus zooepidemicus and Streptococcus equi are the primary causative agents of equine upper respiratory tract infections and bacterial sinusitis. Penicillin, ampicillin, ceftiofur and trimethoprim/sulphonamides (TMS) are the usual first line treatment choices for treatment of streptococcal bacterial sinusitis and gullet pouch infections. However, there is a high rate of resistance of *S. zooepidemicus* isolates to TMS, so it should be used only with favourable susceptibility test results. The new human macrolide-type antimicrobials, azithromycin and clarithromycin, have good activity against streptococci and can be considered in some cases.

Gullet pouch mycosis in horses is most commonly due to Aspergillus spp. Along with surgical occlusion of the carotid artery, treatment consists of topical and systemic antifungal therapy, based on susceptibility testing. The oral bioavailability of theazole antifungal drugs is typically poor in horses, with only an oral solution of itraconazole providing potentially therapeutic concentrations. Antifungal therapy is usually approached topically, by instilling miconazole or enilconazole into the gullet pouch.

**Lower Respiratory Tract**

Pneumonia and pleuropneumonia are often polymicrobial in horses, with initial colonization of the lower respiratory tract with *S. zooepidemicus* followed by invasion by Gram-negative and anaerobic pathogens. Gram-negative bacteria such as Pasteurella spp., Actinobacillus suis and A. equuli are common isolates from
pneumonia and pleuropneumonia cases after S. zooepidemicus. Anaerobes are likely to be present in cases with advanced disease. More virulent pathogens such as Pseudomonas spp. are predominantly isolated from chronic cases with severe pathology. For the Gram negative pathogens, enrofloxacin or gentamicin show excellent activity, however neither drug is efficacious against obligate anaerobes and the susceptibility of Pseudomonas spp. and Klebsiella spp. are variable to both drugs. Therefore, the most logical treatment for bacterial pneumonia or pleuropneumonia is a combination of penicillin, ampicillin or ceftiofur with gentamicin or enrofloxacin. The use of gentamicin or enrofloxacin for respiratory infections in horses is extralabel, but consistent with prudent use guidelines when it is based on culture and susceptibility testing. Practitioners should be familiar with the potential for adverse effects from either of these drugs and client consent should be obtained before initiating treatment. Although penicillins and cephalosporins are highly effective against most anaerobes, resistance by betalactamase-producing Bacteroides fragilis commonly occurs, so oral metronidazole is often added to the treatment. Metronidazole is inexpensive and has excellent activity against all anaerobes including Bacteroides fragilis. Amikacin has very poor activity against streptococci compared to gentamicin, so it should be reserved for cases complicated by Gram negative pathogens resistant to enrofloxacin and gentamicin.

Respiratory tract infections in foals due to Rhodococcus equi are commonly treated with a macrolide-type antimicrobial in combination with rifampin, in order to reduce the likelihood of antimicrobial resistance developing. While erythromycin has been the traditionally used macrolide, azithromycin and clarithromycin also appear effective. There is also evidence that oral tilmicosin and injectable tulathromycin can be used safely in foals.

**Reproductive tract infections**
The majority of reproductive tract infections are limited to the mucosa and superficial endometrium, therefore intrauterine therapy is the preferred method of treatment. Systemic therapy is limited to cases of postpartum metritis where the mare shows systemic illness or where a uterine biopsy indicates deep inflammation and infection. The most common bacteria isolated from the reproductive tract of mares are Strep. zooepidemicus. Only gentamicin and amikacin are approved for intra-uterine use in mares with endometritis, but human formulations of penicillin or ampicillin salts are also used. Because of its poor streptococcal activity, amikacin should be reserved for Gram-negative isolates, such as Pseudomonas spp., with documented resistance to gentamicin.

**Urinary tract infections**
Urinary tract infections in horses typically occur as an ascending infection from skin and gastrointestinal flora. Most antimicrobials are eliminated in high concentrations in the urine. Therefore, in vitro susceptibility results do not always predict therapeutic efficacy for bacterial cystitis, as drugs reported as “resistant” may be clinically effective. Ceftiofur is appropriate for initial therapy due to its activity against E. coli and streptococci, but gentamicin or enrofloxacin may be necessary for Pseudomonas spp. or Enterobacter spp. infections, and ampicillin is the best choice for enterococcal infections.
**Gastrointestinal Tract Infections**

The efficacy of antimicrobials in the therapy of equine gastrointestinal diseases is unknown or unproven in most clinical situations. Antimicrobials are frequently thought to be the cause of diarrhea in horses. There are a few conditions that have a known etiology for which antimicrobial therapy is indicated. Clostridial enterocolitis in horses is caused by toxin-producing strains of Clostridium difficile and C. perfringens. As resistance is very rare, metronidazole is the drug of choice for the treatment of clostridial enterocolitis. C. difficile isolates are susceptible to vancomycin, but this drug is extremely expensive and is not appropriate for routine use in horses because of the emergence of vancomycin-resistant bacteria of human health significance. Risk factors for nosocomial Salmonella enterocolitis outbreaks typically include prior antimicrobial exposure. Treatment of affected horses is controversial and usually limited to animals showing signs of septicemia. Most Salmonella strains from horses are susceptible to enrofloxacin, ceftiofur and gentamicin. Equine ehrlichial colitis (aka Potomac Horse Fever) is caused by Neorickettsia risticii and is commonly treated with intravenous oxytetracycline or oral doxycycline. Proliferative enteropathy in foals caused by Lawsonia intracellularis typically responds to oral doxycycline or erythromycin plus rifampin. Cholangiohepatitis in horses is typically caused by Gram negative enteric bacteria, so enrofloxacin, ceftiofur or gentamicin are reasonable therapeutic choices.

**Musculoskeletal infections**

**Wounds**

Bacterial isolates from the acute wounds must be cautiously interpreted as they may represent environmental contamination rather than active infection. Distinguishing between contamination and colonization is difficult and based on the type of bacteria, history of the wound and number of bacteria isolated. As infection becomes established, the bacterial populations may change. When treating a traumatic open wound that is contaminated, infected or likely to become infected, choose an antimicrobial that is active against *S. zooepidemicus*. Since mixed infections are common, a broad spectrum antimicrobial such as ampicillin or ceftiofur may be indicated while awaiting bacterial culture results. Aminoglycosides are commonly used for local antimicrobial delivery (e.g. regional perfusion). Although amikacin is very active against *S. aureus* and *Pseudomonas* spp. isolates, its activity against other common wound isolates is poor, so gentamicin is a better initial choice.

**Surgical infections**

*E.coli* and other coliforms predominate in suture line infections following soft tissue surgery but *S. aureus* may also be found. Susceptibility testing for *S. aureus* is very important to determine if these infections are the result of contamination with equine cutaneous flora which are susceptible to most antimicrobials or are multi-resistant strains associated with environmentally acquired infection. Recent data indicates that penicillin, ampicillin or ceftiofur is the treatment of choice for prophylaxis of orthopedic infections and that gentamicin is the best choice for prophylaxis of soft tissue infections. Culture and susceptibility testing is essential for post-surgical infections in order to refine antimicrobial therapy and to identify emerging nosocomial problems.

**Infectious Keratitis**
Infectious keratitis in horses can be caused by Gram positive or Gram negative bacteria or fungi. Nearly 50% of the Gram negative bacterial isolates are Pseudomonas spp. Due to the consequences of nonresponsive or inadequately treated corneal infections in horses, initiate treatment with broad spectrum antimicrobial therapy effective against staphylococci and pseudomonads. Gentamicin or Polysporin® (gramicidin and polymyxin B) or Polytrimethoprim® (trimethoprim and polymyxin B) are good initial choices. Polymyxin B is rapidly bactericidal against Gram-negative bacteria, with excellent activity against Pseudomonas spp. Polymyxin B also binds and inactivates endotoxin, reducing inflammation and tissue destruction. Due to systemic toxicity, polymyxin B is only used topically, so it is not typically included on susceptibility reports from microbiology services. Gramicidin is active against Gram positive bacteria, with a mechanism of action similar to the β-lactam antibiotics. Trimethoprim and polymyxin B are combined in the human formulation Polytrimethoprim® to provide broad spectrum activity against Gram positive and Gram negative bacteria including Pseudomonas. Penicillins and cephalosporins are not available as ophthalmic formulations because of the risk of contact sensitization, so bacitracin is their equivalent. Human ophthalmic formulations of tobramycin and ciprofloxacin should be reserved for the treatment of resistant Pseudomonas infections. Nystatin, natamycin, or clotrimazole are recommended for topical treatment of keratomycosis in horses.

**Neonatal Septicemia/Bacteremia**

Septicemia in neonatal foals is an extremely important cause of morbidity and mortality. Infected foals often have minimal or nonspecific signs, making definitive diagnosis difficult and antimicrobial therapy is often initiated without culture and susceptibility results. Because disease develops so rapidly in the neonate, broad spectrum antimicrobial therapy is initiated prior to culture and susceptibility results. A β-lactam (e.g., penicillin G, ampicillin or ceftiofur) and aminoglycoside combination is the traditional first line therapy. Although amikacin is frequently recommended as first line therapy, resistance rates to gentamicin may not be as high in practices as that reported in neonatal intensive care units. With either aminoglycoside, once-daily, high dose therapy is recommended to account for the high volume of distribution in foals and to minimize nephrotoxicity. Enrofloxacin can be used instead of an aminoglycoside and it will reach therapeutic concentrations in the central nervous system, but arthropathies have been documented to occur in neonatal foals. Fluoroquinolone arthropathy is exacerbated by weight-bearing, so treated foals should be confined and exercise limited. Third generation cephalosporins may be used in septic foals with meningitis, but are extremely expensive so they are rarely used. Two weeks of antimicrobial therapy is typically recommended for blood culture positive foals, and longer therapy may be required for foals with localized infections in bone, joints or lungs.
MIND-BODY WORKSHOP

COMPLEMENTARY MEDICINE - REKINDLING THE GIFT

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Introduction
The practice of Veterinary Medicine offers a unique and extremely rewarding professional life. It also offers many challenges that can create stress and impact on one's mental, physical and emotional health. The concept of “burn-out” within the profession has been discussed much more openly and frequently during this past decade. “Rekindling The Gift” is a unique weekend long experiential workshop based on concepts of Mind-Body Medicine. This program is for veterinarians who are searching for renewed personal and professional meaning and mission as well as for veterinarians in transition in their career.

The workshop will include lectures, exercises, training in the Relaxation Response as defined by the Mind/Body Medical Institute at Harvard Medical School along with periods of reflection, journal writing and creative dialogue. Through experiential learning, this intensive will focus on developing your inner resources to bring new levels of personal satisfaction and healing potential to your practice of veterinary medicine. It is essential that one commits for the weekend, for this is a progressive process oriented experience.

The focus will be on the utilization of these approaches to assist veterinarians in reflection, reevaluation, rejuvenation and re-creation in regards to their current role in veterinary medicine and any transitions they may be considering.

Additional Detail
The practice of Veterinary Medicine offers a unique and extremely rewarding professional life. Veterinarians have many gifts to offer others. We bring knowledge and compassion into a healing profession that cares for the helpless. The profession also offers many challenges that can create stress and impact on one's mental, physical and emotional health.

The challenges of practicing progressive medicine, staff management, practice management, client relations, and balancing these with other parts of our lives can become overwhelming at times. The experience of “burnout” is a major risk factor for veterinarians involved in the demanding area of patient care. Substance abuse, crisis management and suicide appear to be on the rise as well. These challenges are not limited to the veterinary profession, but are seen in many healing professions.

There is increased discussion regarding “compassion fatigue” in many health professions as well. Burnout is often the undesired end-point of a career that began with the noblest of intentions. Burnout sufferers begin to feel cynical, depressed, alienated, and negative about their role as a veterinarian. Some veterinarians choose to leave the profession or explore alternative career pathways within the profession.
One source of burnout may lie within the personality of the individual who may feel overly responsible for the welfare of others and use unrealistically high measures of personal performance in evaluating themselves (1). When care-givers suffer disappointments in patient care, they may become emotionally depleted, lose touch with themselves and others, second guess themselves and eventually sink into professional despair.

Developing a way to prevent compassion fatigue is a necessary component of professional development according to Chaplain S. Bryant Kendrick, Associate Professor of Internal Medicine and Gerontology at The Bowman Gray School of Medicine (1). One other perspective on burnout is that it may correlate with professional stagnation as well. Continuing education and training are key to maintain a stimulating career.

Mind/Body Medicine is a rapidly expanding field in human medicine and its applications for veterinarians are just beginning to be explored. In its simplest definition, Mind/Body medicine is the use of our mental activity, thoughts and feelings to help prevent and treat various “dis-eases”. Studies in mind/body medicine document the effects of thoughts on the release of various neurotransmitters and neurohormones and the impact that has on our physical, mental and emotional health. Many ancient traditions discuss the essential nature of our mind and its impact on our health. What we think directly impacts on our overall health. One key teaching in Buddhism is on training the mind and cultivating loving-kindness. In “The Art of Happiness, A Handbook for Living”, the Dalai Lama states “The systematic training of the mind—the cultivation of happiness, the genuine inner transformation by deliberately selecting and focusing on positive mental states and challenging negative mental states—is possible because of the very structure and function of the brain (2). The key to rekindling the gift that we as veterinarians brought into this profession is in our thoughts, our mind. Dr. James Austin, Professor Emeritus of Neurology at the University of Colorado, provides an extensive review of the effects of our thoughts on brain mechanisms and neurochemistry in “Zen and the Brain” (3).

Dr. Joel Robertson describes the impact of various neurotransmitters such as acetylcholine, norepinephrine, epinephrine, serotonin and others on our moods and performance. Through neurochemical evaluations, he offers simple approaches to maximizing our brain efficiency through nutrition, exercise and mental attitudes (4). By balancing our neurochemistry, we can enhance performance and prevent burnout.

One essential mind/body exercise to practice in order to manage the challenges of our career is what Dr. Herbert Benson of the Mind/Body Medical Institute at Harvard Medical School calls “The Relaxation Response” (5) There are two basic steps necessary to elicit The Relaxation Response. The first is the repetition of a word, sound, prayer, thought, phrase or muscular activity. The second step is the passive return to the repetition when other thoughts intrude. The Relaxation Response has been found to decrease respiratory rate, heart rate, blood pressure and oxygen consumption. It has been demonstrated to be effective in the treatment of anxiety, hostility, depression, hypertension, insomnia, chronic pain, premenstrual syndrome, infertility and stress. Other stress reducing exercises will be taught as well.
Chaplain Kendrick recommends developing a personal burnout prevention plan based on the effects that your thoughts have on your actions. It is based on realistic expectations, the ability to differentiate subjective from objective components of reality, appropriate self-love and support from others. Kendrick feels that this stewardship program “facilitates self-preservation and renewal so that you can adapt to the stresses of the modern health care environment without losing the capability of being there for others”(1). Often times we waste energy experiencing frustrations based on expectations that exceed what is really possible. We also tend to not experience the moment, always reflecting on the past or worrying about the future.

Often there is a tendency to confuse stress with fear, fear of the past, fear of the future. Simple mindfulness techniques will also be taught to assist in regaining the ability to be in the present moment. Too frequently we also fall into the trap of basing our self-worth on someone else’s opinion of our professional performance, creating a performance based self-esteem system. We need to differentiate who we truly are from what we do.

Exercises and creative dialogues will assist us in reflecting on where we began our journey as veterinarians to where we have ended up at this particular point in our career. Twelve approaches to creating a life and career that works better for us will be reviewed. These include the openness to change, self responsibility, feeling our feelings, accepting what is, establishing appropriate boundaries, transforming our self-talk, loving ourselves, exploring the power of choice, commitment, surrender, standing in our truth and re-experiencing joy (6).

Through these and other experiential processes we will begin to manage our challenging profession better, re-create our heart’s desires and create a career and life style where we will enjoy each moment and each day to its fullest. We will then be able to be of the most service and benefit possible to all beings, two-legged, four-legged and winged.

**Summary**

Techniques of mind/body medicine offer veterinarians opportunities to reflect on, rejuvenate and re-create our careers and lifestyles. These are time-tested and scientifically documented processes that help balance our brain chemistry, physical and emotional health and bring greater joy and satisfaction back into our lives.

**References**