2023 THE UNIVERSITY OF TENNESSEE
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April 29-30, 2023

UTCVM
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Annual Conference

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PROCEEDINGS

April 29-30, 2023

UTCVM INSTITUTE OF AGRICULTURE THE UNIVERSITY OF TENNESSEE

Orbital Disease in Dogs

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The appropriate diagnosis and successful therapy of orbital disorders starts with recognition of what normally occupies the orbital space. The eyeball, obviously, but also a large amount of fat (at least until advanced age), extraocular muscles, the zygomatic salivary gland, the optic nerve, a number of cranial nerves, peripheral nerves, vasculature, connective tissue, and bone. Theoretically disease or dysfunction of any of these tissues will manifest as orbital disease. Although there are exceptions, almost all orbital disorders will result in either exophthalmos, ocular deviation, or both. It is key at the outset to distinguish exophthalmia (i.e., anterior protrusion of a normally sized eyeball) from buphthalmia (i.e., an enlarged eyeball that is sitting in its normal location within the orbit). The only thing that causes buphthalmia is glaucoma….and chronic glaucoma, at that. Exophthalmia, on the other hand, indicates a retrobulbar space occupying lesion of one or more of the aforementioned tissues.

Retrobulbar Abscess

The lesion that we generally term "retrobulbar abscess" is actually more appropriately termed "retrobulbar cellulitis" in most instances because it is actually relatively uncommon to encounter an actual pocket-like accumulation of purulent material. More commonly these materials intercalate between and within orbital tissues. Most cases are caused by bacterial infection, although fungal infection and sterile cellulitis also occur. One common route of entry of bacteria is via the oral cavity, because the only thing that separates the oral and orbital cavities in the dog is a thin slip of medial pterygoid muscle. Puncture of this muscle while chewing on sticks, bones, etc. can result in deposition of bacteria into the orbital cavity. Other routes include infected molar roots (infected roots of carnassial teeth usually cause abscessation anterior and ventral to the eye, not in the orbit), ascending infection from the oral cavity up the zygomatic salivary duct, contiguous infection from adjacent nasal cavity or sinuses, and hematogenous.

The typical clinical history is of acute onset of exophthalmia, third eyelid protrusion and periocular pain. Ophthalmic and physical examinations are generally normal apart from those same observations, and these cases are almost always unilateral. On rare occasions an indentation in the back of the globe is visible on fundic exam, but this is an equivocal finding and, honestly, somewhat difficult to interpret. There is no real age, breed or gender predilection, although outdoor dogs probably predominate. A real key to differentiating this lesion from other orbital lesions is demonstration of intense pain on opening the mouth. The ramus of the mandible resides in the far posterior orbit when the mouth is closed, but moves rostrally when the mouth is open. If cellulitis is present, when the ramus moves forward into the resultant inflammatory soup intense pain results….and it is not subtle. These dogs typically come off the exam table in excruciating pain with little more than an inch or so of mouth opening. Sometimes there is a history of reluctance to eat, which stems from this same pain. Pain on opening the mouth has a diagnostic sensitivity and specificity of approximately 90%. If you are lucky enough to get the mouth open far enough, sometimes a red, fluctuant swelling is seen posterior to the last molar.

If the index of suspicion of retrobulbar cellulitis is high, I usually recommend simply starting broad spectrum antibiotics empirically. A retrospective paper (Wang AL, et al. Orbital abscess bacterial isolates and in vitro antimicrobial susceptibility patterns in dogs and cats. (*Vet Ophthalmol* 12:91-96, 2009) showed that the most common isolates in cases of bacterial retrobulbar cellulitis were *Staphylococcus*, *Escherichia*, *Bacteroides*, *Clostridium* and *Pasteurella* (note that 2 of the top 5 are anaerobes!). Sensitivity results in that study indicated that ceftiofur was the most reliable antibiotic against the aerobic infections, and would be expected to be effective against most anaerobic infections as well (even though sensitivities against the anaerobes were not evaluated). Of course, being available only in injectable form could prove problematic. Oral alternatives that were also highly effective included cefpodoxime and amoxicillin-clavulonate. Trimethoprim

sulfa and enrofloxacin were highly effective against the aerobes, but would not be expected to work well in anaerobic infections.

In most instances clinical improvement is seen quite rapidly. When improvement appears to be lacking, you may consider entering the orbital cavity from the oral cavity just behind the last molar to obtain a culture and/or establish drainage. With the patient anesthetized, prep the mucosa as best you can with dilute betadine, then make an incision into the mucosa about 1 cm long. Spread the incision with sterile hemostatic forceps, and introduce a second CLOSED hemostatic forceps into the incision and forcefully penetrate the pterygoid to enter the orbital cavity. *DO NOT FISH AROUND BLINDLY IN THE ORBIT WITH THE HEMOSTATS* as you may encounter something you'll wish you hadn't… like the optic nerve or the maxillary artery. The hemostats are only used to make entry into the orbital cavity. In rare instances purulent material may then drain into the mouth, and if this occurs place a Penrose drain to maintain drainage for several days. More often, no outright drainage occurs but an accurate culture can be obtained through the same incision, and antimicrobial treatment altered accordingly.

As mentioned above, clinical resolution is generally rapid and complete in most cases without remarkable sequelae. If the infection was present for a long period prior to veterinary intervention, there may be a loss of retrobulbar fat with subsequent fibrosis and permanent enophthalmia. Extension of retrobulbar infections into the calvarium, while considered exceedingly rare, has been reported at least twice… once with bacterial infection (Oliver JAC et al. Central nervous system infection with Staphylococcus intermedius secondary to retrobulbar abscessation in a dog. (*Vet Ophthalmol* 12:333-337, 2009) and once with an orbital blastomycosis infection (Baron ML, et al. Intracranial extension of retrobulbar blastomycosis (Blastomyces dermatitidis) in a dog. (*Vet Ophthalmol* 14:137-141, 2011).

Retrobulbar Neoplasia

Cancers of the orbital region make up the second most common cause of exophthalmia in dogs. Neoplasia and abscess together account for approximately 95% of all cases of exophthalmia. In contrast to cellulitis cases, neoplasms are generally seen in older animals (although I have encountered malignancies in dogs as young as 6 months), usually cause slowly progressive exophthalmia, are usually not terribly painful, and typically don't cause discomfort upon opening the mouth. In addition, retrobulbar neoplasms are more likely to cause deviation of the globe than cellulitis. These tumors are generally bad news…the vast majority are primary and malignant, with the most common offenders being osteosarcoma and fibrosarcoma (Hendrix DV et al. Diagnosis, treatment and outcome of orbital neoplasia in dogs: a retrospective study of 44 cases. *J Small Anim Pract* 41:105-108, 2000). Other primary tumor types represent essentially every orbital tissue. The most common secondary tumor is nasal adenocarcinoma, via direct extension through the medial orbital wall. Due to the large amount of volume in the normal orbit, these tumors are generally fairly well established by the time exopthalmia becomes apparent. This delay only makes effective treatment even more difficult.

Diagnosis is best made by advanced imaging techniques. I prefer CT scanning because it gives the best demonstration of bony involvement, with such involvement largely dictating the likelihood of effective treatment. MRI scanning is also very informative. I don't find conventional radiography helpful, and ultrasound, while better than conventional radiography, doesn't tend to give much additional information beyond what can be gleaned from the clinical picture alone. In addition to giving superb definition of tumor margins, CT scanning offers the possibility of guided biopsy for definitive diagnosis.

The wisdom of pursuing therapy is contingent upon how invasive the tumor is at the time of initial diagnosis. Cases in which the tumor is so pervasive that it has eroded the medial orbital bony tissues to enter the nasal cavity and/or calvarium are rarely pursued. If confined to the orbit, exenteration followed by radiation (usually approximately 2 weeks after surgery) may give good palliative results. For very early, small and minimally invasive tumors (these will usually be meningiomas of the optic nerve) globe-sparing orbitotomy and surgical resection may be curative.

Sterile Inflammatory Disorders

Inflammation of the muscles of mastication ("masticatory myosisitis") or extraocular muscles ("extraocular muscle myositis") both present as a sudden onset of exopthalmia, generally with periocular tenderness and pain on opening the mouth that is reminiscent of retrobulbar cellulitis. The primary clinical difference is that these cases are generally bilateral, and have strong breed predilections. Golden Retrievers, Labrador Retrievers, German Shepherd dogs, and Weimaraners are particularly overrepresented. The pathophysiology is probably auto-immune, and stems from unique antigenicity of extraocular and masticatory muscles. Large numbers of eosinophils are often seen in the inflammatory infiltrates. Diagnosis is generally made on the basis of clinical suspicion, although, in the case of masticatory muscle inflammation, biopsy of the temporalis muscle can be contemplated. Advanced imaging will corroborate muscle enlargement. Most cases respond relatively well in the short term to immunosuppressive doses of systemic corticosteroids (prednisone 1 mg/# divided BID to start, and weaned by reducing by 25% per week over 4-6 weeks) or other immunosuppressives (azathioprine, cyclosporine or mycophenolate). In the longer term, replacement of muscle tissue with fibrous tissue is almost universal, resulting in severe enophthalmia (in the case of masticatory muscle myositis) or strabismus (in the case of extraocular muscle myositis). The enophthalmia can be severe enough to lead to chronic epiphora and involutional entropion, the latter of which might need surgical therapy to prevent corneal injury. There is no simple cure for the enophthalmia itself. I do think there are cases of masticatory myositis in which the acute inflammatory stage is never obvious, and in these cases the first thing the owners notice is the enophthalmia.

Cystic Disorders

The most common orbital cystic lesion is the zygomatic salivary mucocele. In most cases the mucocele will present as a fluctuant swelling of the lower lid, with bulging of the palpebral conjunctiva spilling over the lid margin. In rare instances this conjunctival bulge is not seen, and the primary manifestation is simply exophthalmia. This creates a diagnostic dilemma in differentiating it from a retrobulbar tumor, and advanced imaging is needed to definitively diagnose the lesion. The classical therapy is surgical removal of the cyst, but more recently we have had some luck in treating cystic lesions with intracystic injection of the sclerosing agent polidocanol (1%; injection volume 0.5 – 2.0 mL per cyst; see Stuckey JA et al. Use of a sclerosing agent (1% polidocanol) to treat an orbital mucocele in a dog. (*Vet Ophthalmol* 15:188–193, 2012; obtain from peoplescustomrx.com)

Another cystic lesion that is far more common than our literature would suggest is post-enucleation orbital cyst. These occur due to remnants of secretory tissue (either conjunctiva, lacrimal gland, or third eyelid gland) being left behind following enucleation. The presence of a cyst is indicated by a soft, fluctuant swelling in the area of the enucleation site, and these cysts can appear many months after surgery. In some instances, they become infected, probably due to movement of organisms up the nasolacrimal duct from the nasal cavity. As with zygomatic mucoceles, classically these lesions have been surgically excised but more recently we have been treating them with intracystic polidocanol injection.

Vascular Anomalies

While uncommon, vascular anomalies such as arterio-venous fistulas and varices have been reported in dogs. These lesions are often diagnostic challenges, requiring advanced imaging and sometimes contrast studies to definitively diagnose. In some cases, auscultation of the orbit will produce an audible "swoosh" called bruit (pronounced BRU ee). Invasive orbital surgery has been the treatment of choice, but is associated with significant morbidity and mortality due to severe hemorrhage. Vascular coil embolization appears to be a superior therapy in most cases, but is not widely available.

Imaging the Coughing Dog

Silke Hecht, DVM, DACVR, DECVDI, Professor in Radiology

Introduction

Coughing is one of the most common presenting complaints for dogs in small animal practice. Even though there is a trend for increased use of other imaging modalities (thoracic ultrasound, computed tomography) for the evaluation of the small animal neck and thorax, radiographs remain the mainstay of at least the initial diagnostic work-up. This presentation will focus on a review of radiographic abnormalities that may be seen in coughing dogs.

Technical Considerations

Cervical radiographs may or may not be obtained dependent on physical examination findings and clinical suspicion. Lateral radiographs of the neck are most helpful in evaluating the pharynx, larynx, trachea and surrounding structures. A ventrodorsal view is often less helpful due to superimposition of the airways over the spine. Especially for full evaluation of the lung, 3 view thoracic radiographs (right lateral, left lateral and ventrodorsal or dorsoventral) are indicated in almost all cases. Acquiring radiographs under sedation aids in straight patient positioning and minimizes the need for personnel presence in the x-ray suite.

Approach to the Thoracic Radiograph

Like any thoracic radiographic examination, the assessment of radiographs in coughing dogs includes a systematic evaluation of the thoracic wall, diaphragm, pleural space, mediastinum, lung and cardiovascular structures. This presentation will focus on the latter 3 as they are most likely to be abnormal in coughing animals. When lesions are identified, lesion distribution and opacity will aid in prioritization of differential diagnoses.

Mediastinum: Of the structures that are normally seen on radiographs, the trachea and the heart are the most likely structures to appear abnormal in a coughing animal. Other mediastinal structures not normally seen (tracheobronchial lymph nodes or mediastinal mass lesions) may also cause or contribute to cough.

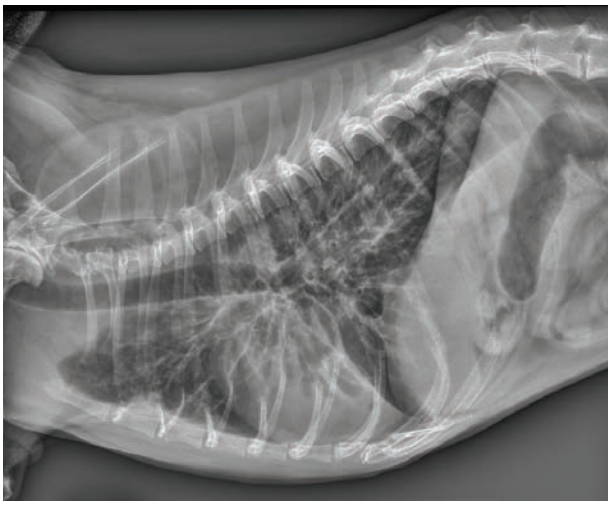
Lung: Possible pulmonary patterns that may be seen in coughing dogs include structured interstitial, unstructured interstitial, vascular, bronchial and alveolar patterns or a combination thereof.

Cardiovascular structures: Animals with left-sided congestive heart failure may present with cough and/or respiratory distress. Cough may also be caused or contributed to by heart enlargement, especially left atrial enlargement.

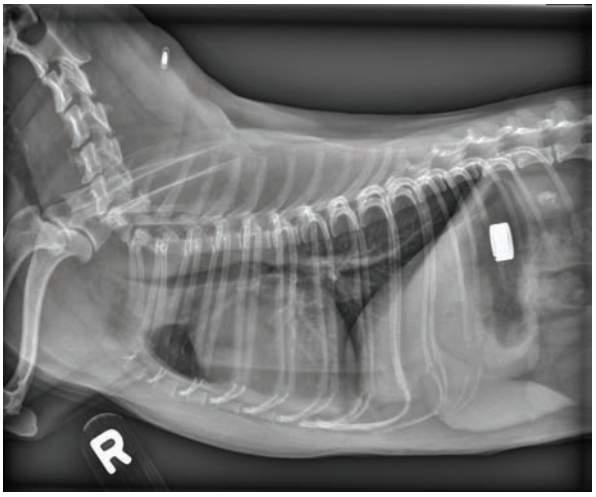
Causes of Coughing and Imaging Findings

Possible differential diagnoses in dogs with cough include inflammatory, parasitic and allergic conditions (e.g., bronchitis or asthma), neoplasia (of the trachea, lung, or other thoracic compartments), traumatic and physical causes (e.g., foreign bodies), and cardiovascular disease. Examples of different disorders will be presented during this lecture. Some dogs with cough will have normal thoracic radiographs, requiring additional tests or empirical treatment.

Example cases:



Lateral thoracic radiographs in an 11-year-old Chihuahua with chronic cough. There is a marked diffuse bronchial pattern. Some bronchi appear blunted and lobular, consistent with bronchiectasis. A focal alveolar pattern is associated with the ventral aspect of the right middle lung lobe. The imaging findings are consistent with chronic bronchitis and bronchopneumonia.



Lateral thoracic radiographs in an 11-year-old Yorkshire terrier presented with a honking cough. There is complete collapse of the caudal cervical trachea. The metal opaque structure in the cranial abdomen is consistent with an ameroid constrictor previously placed for occlusion of a portosystemic shunt.

Imaging the Small Animal Emergency Patient

Silke Hecht, DVM, DACVR, DECVDI, Professor in Radiology

Introduction

Emergency cases are commonly seen in small animal practice. There are numerous instances that require immediate veterinary consultation and or care, including but not limited to bleeding, ocular injuries, respiratory distress, seizures, or signs of an acute abdomen. Due to the multitude of emergency situations requiring diagnostic imaging, this presentation will specifically concentrate on thoracic imaging in the small animal emergency patient. Even though thoracic ultrasound is increasingly used and may occasionally be helpful in the assessment of the small animal emergency patient, this presentation will focus on thoracic radiographs.

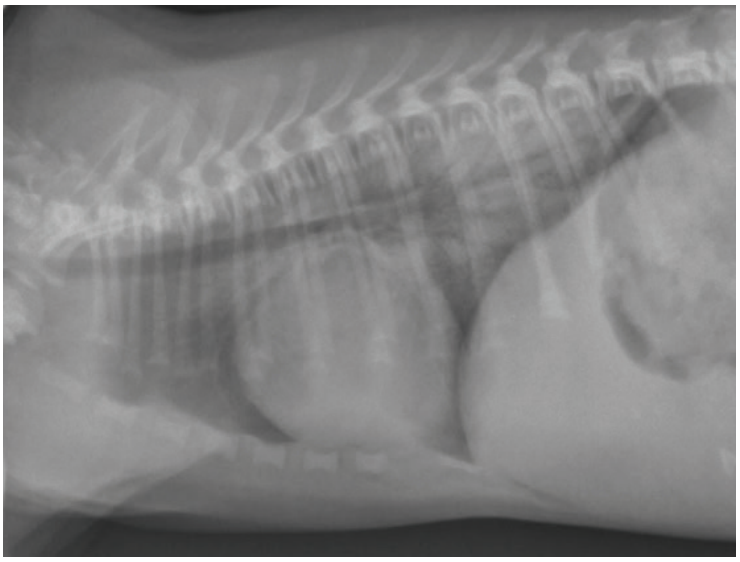
Thoracic emergencies

Thoracic emergencies can be broken down into different categories.

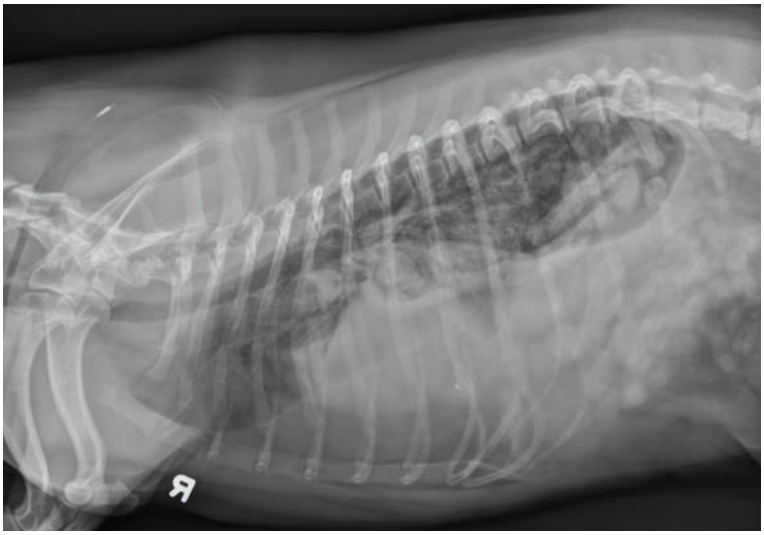
Acute dyspnea: This is a life-threatening presentation which requires immediate attention and treatment. Dyspnea can result from pleural effusion, pneumothorax, heart failure, noncardiogenic pulmonary edema, other lung diseases, acute airway obstruction, and other causes. This presentation will provide a review of common conditions (including left heart failure, asthma, and aspiration pneumonia) and less common or underrecognized diseases (including pulmonary infiltrates secondary to pulmonary hypertension, intrathoracic hemorrhage, and pulmonary thromboembolism).

Other thoracic emergencies: Examples of thoracic emergencies other than those listed above include trauma, certain types of diaphragmatic hernia, esophageal foreign bodies, and others.

Example cases:



Lateral thoracic radiograph in a 2-month-old Yorkshire terrier presented with acute onset respiratory distress after having 2 seizures. There is an alveolar pattern associated with the caudal dorsal lung fields. The cardiovascular structures are normal. A diagnosis of noncardiogenic pulmonary edema was made.



Lateral thoracic radiograph in a dog presented after being hit by car. The ventral aspect of the diaphragm is not clearly delineated, abdominal organs are shifted cranially, and the cardiac silhouette is partially obscured by superimposition of abdominal organs. The findings are consistent with a diaphragmatic rupture ("hernia").

Medical and Surgical Management of Hemoabdomen

Cassie N. Lux, DVM, DACVS-SA, Associate Professor of Surgery, Coughlin Distinguished Professor

Hemoabdomen is a pathologic accumulation of blood within the peritoneal cavity, and its true prevalence in veterinary patients is unknown due to the potential lack of diagnosis of small volumes of free blood. Two main classifications of hemoabdomen exist based on etiology: spontaneous (nontraumatic) or traumatic. Spontaneous hemoabdomen can be a result of many etiologies including congenital or acquired coagulopathies, hepatic or splenic torsions, gastric dilatation volvulus, and many benign or malignant processes of the various abdominal organs. Traumatic hemoabdomen is a result of blunt (most commonly motor vehicle accidents) or penetrating trauma.

A complete history should be taken on any emergent patient presenting with hemoabdomen. If trauma is confirmed, as much information should be obtained about the event including progression of signs following the trauma. Any access or exposure of the patient to anticoagulants should be elucidated, including rodenticides and medical anticoagulants taken by the owners. Young animals may be presenting with heritable coagulopathies (e.g. Beagle), so previous history of bleeding, surgery, or response to any trauma can be helpful. Patients presenting with spontaneous hemoabdomen can present with severe clinical sings such as collapse, or with vague waxing and waning signs such as inappetence, vomiting, and weakness.

Physical examination findings should assist the clinician in triaging the emergent status of the patient. Cardiovascular stability should first be determined by evaluating perfusion parameters, including mentation, pulse rate and quality, mucous membrane color, capillary refill time (CRT), and core vs extremity temperature. Findings that might point the clinician towards a suspicion of hemoabdomen include abdominal pain, abdominal distention, a palpable abdominal mass, or a palpable fluid wave which requires ≥ 40mL/kg of abdominal fluid. These patients may be in varying levels of shock, and those that are most at risk of sudden death and therefore requiring immediate intervention are those in early or late decompensatory shock with tachycardia or bradycardia (particularly in cats), weak to absent peripheral pulses, prolonged or absent CRT, cool extremities, pale to white mucous membranes, and abnormal mentation. Additionally, high plasma lactate, low bicarbonate, increased base deficit, and minimal urine output would support advanced stages of shock.

The goals of fluid resuscitation for a hemoabdomen patient are to support tissue perfusion by assuring effective circulating blood volume and maintaining oxygen-carrying capacity, identify the presence of hemoabdomen with systemic derangements, and to arrest the hemorrhage. Abdominocentesis is the quickest way to diagnose hemoabdomen (PCV >5-10% is diagnostic), and the abdominal fluid PCV/TS should be compared to the peripheral PCV/TS. If the abdominal PCV/TS is greater than the peripheral PCV/TS, these patients should have more intensive monitoring. Maintaining tissue oxygenation may include enhancing oxygen levels with supplementation (flow-by via face mask, nasal prongs, and oxygen cage), expansion of blood volume, or blood product transfusions.

Expanding or restoring blood volume can be performed with isotonic crystalloids, synthetic colloids, blood products, or a combination of these fluid types. Any fluid administration should be titrated to the give a dose sufficient to resolve the clinical signs of shock in the patient, and after fluid bolus a constant rate infusion of crystalloids should be administered to maintain the blood volume. Traditional fluid resuscitation consists of rapid administration of isotonic crystalloids (dogs: 80-90 mL/kg; cats: 40-60 mL/kg delivered in ¼ dose aliquots over 5-15 min); the volume given based on the fluid deficits of each patient with frequent re-evaluations of perfusion parameters. This resuscitation has been associated with dilutional coagulopathy, hypothermia, edema formation, and re-bleeding due to rapid increases in blood pressure disrupting blood clots. Another method of fluid resuscitation is known as low-volume resuscitation, in which the administration of fluid provides intravascular support, while still minimizing fluid extravasation and the rapid elevations in blood pressures. Low-volume resuscitation involves administration of small boluses of crystalloids and

sometimes hypertonic saline and/or colloids with constant monitoring of the patient to meet improved perfusion parameters and low normal blood pressure end points of 60-70 mmHg for mean arterial pressure (MAP) and 90 mmHg for systolic pressure. One must be cautious about delaying surgery for extended periods of time with low-volume resuscitation, as this methodology maintains blood pressure just at the levels required to support vital organ function.

Use of colloids can aid the clinician in providing intravascular volume, yet allowing smaller volumes of crystalloids to be administered. Combinations of hypertonic saline (up to 8mL/kg) and hydroxyethylstarch (up to 10mL/kg) have been shown in some studies to produce superior cardiovascular resuscitation. However, synthetic colloids have been associated with decreased platelet function, hypocoagulability, an increased risk of mortality, and acute kidney injury. The use of hydroxyethylstarch (670/0.7) should be limited to 20 mL/kg/day and tetrastarch (130/0.4) to 50 ml/kg/day to minimize the effects of coagulopathy. Blood product transfusions may be necessary in these patients, but the need should not be based on PCV alone but also signs associated with severe anemia such as tachycardia, tachypnea, poor pulse quality, generalized weakness, and sometimes increased lactate. Commonly used blood products include whole blood, packed red blood cells, and fresh frozen plasma. Administration of large amounts of blood products have been associated with a higher mortality rate, hypocalcemia, and thrombocytopenia. Clinicians should consider blood product transfusion when PCV < 20-25%, especially when the patient is to undergo anesthesia and surgery. Autotransfusions can be performed by sterile collection and administration of free abdominal hemorrhage through a blood filter, but is controversial if the etiology is suspected to be neoplastic. Blood within the peritoneal cavity will have rapid depletion of all clotting factors within an hour and is not a good source for this. The following doses can be administered for blood product transfusions: fresh whole blood (10-30 mL/kg), packed red blood cells (6-10 mL/kg), and fresh frozen plasma (10-30 mL/kg).

Results of diagnostic testing should guide further stabilization and medical treatment. Pulse oximetry can useful to determine the severity of the oxygen exchange abnormalities. Non-invasive blood pressure monitoring is an easy method to evaluate fluid resuscitation and continued hemodynamic instability. An electrocardiograph (ECG) can be useful to determine if cardiac arrhythmias are present that may result in cardiovascular compromise that fails to respond or worsens.

A CBC should be performed to evaluate the red cells, white cells, and platelets, and in addition quick evaluation can be performed by assessing a peripheral PCV and total solids. Metabolic and respiratory derangements may be present, and a blood gas analysis may identify some of these abnormalities to guide therapy. Coagulation assessment should be performed including prothrombin time (PT), activated partial thromboplastin time (aPTT), and if rodenticide toxicity is suspected HPLC (high performance liquid chromatography) can be performed. Finally, a serum biochemistry panel can assist in identifying any other organ system dysfunctions.

Most diagnostic imaging should be postponed until stabilization has occurred, with the exception of an A-FAST scan (abdominal-focused assessment with sonography in trauma). The A-FAST scan provides quick, accurate, and serial evaluations of abdominal fluid accumulation by identifying 4 repeatable anatomic quadrants to scan. With the patient in left lateral recumbency, the four anatomic quadrants evaluated are the cranial midline just caudal to the xiphoid, the caudal midline around the urinary bladder, the left gravity-dependent flank region around the spleen and kidney, and the right gravity-dependent flank region around the liver and kidney. Thoracic radiographs are always advisable to rule out metastatic disease and evaluate trauma patients for thoracic injury. A full abdominal ultrasound can be valuable in order to evaluate abdominal organs, identify obvious abnormalities, and potentially determine the source of hemorrhage. The use of computed tomography may be helpful for non-splenic lesions and in very large dogs in which the utility of ultrasound is diminished. There is also evidence that magnetic resonance imaging is useful to identify features that may be specific to malignant or benign disease.

Medical management will be pursued in a hemoabdomen patient until surgery is decided as the course of action, as not all hemoabdomen patients will require surgical management. For instance, although hemoabdomen following vehicular trauma is relatively common (23-45% incidence), it is reported that only up to 6% will require surgical intervention. Surgical intervention can be pursued once resuscitation end points are met and the source of hemorrhage is identified. On rare occasion, a patient may be hemorrhaging so severely that medical management is not effective, and the patient requires emergent surgery as quickly as possible. In patients with rodenticide toxicity, administration of vitamin K_1 at 2.5–5 mg/kg given every 8–12 h depending on the type of ingested anticoagulant should occur. Antifibrinolytic drugs like epsilon aminocaproic acid (EACA) and tranexamic acid have been investigated as treatments to minimize hemorrhage associated with surgery. There is evidence that antifibrionolytics can reduce mortality in humans with bleeding trauma and bleeding complications in dogs, and the risk of adverse events with EACA is very low. Yunnan Baiyao is a Chinese herbal remedy with hemostatic properties; it is well-tolerated in dogs and has been shown to increase clot strength. Antifibrinolytics and Yunnan Baiyao may be used as treatments for non-surgical patients or as adjunct in the surgical treatment of hemoabdomen. Abdominal counterpressure (a tight abdominal wrap modified to include the pelvic limbs) can be applied in the short term, and for patients which owners will not allow surgical intervention. This technique elevates intra-abdominal pressures and provides hemostasis, but can lead to decreased renal perfusion, organ ischemia, and respiratory compromise.

Surgery is pursued with spontaneous hemoabdomen to alleviate the source of hemorrhage, and also when medical management fails as shown by hypotension that is transiently responsive or unresponsive to fluid resuscitation, PCV is progressively decreasing, abdominal fluid volume is increasing, and perfusion parameters do not normalize with medical therapy. Additional reasons to pursue surgery include identification of a ruptured hollow viscus, ruptured mass, penetrating trauma, septic or bile peritonitis, and evidence of torsion of an organ.

The clinician should have various hemostatic options available when pursuing surgical intervention for hemoabdomen. With appropriate hemostatic application, blood flow should be decreased to the site of hemorrhage, leading to a blood clot to form within 2-3 minutes. Large vital vessels in the abdomen can be temporarily occluded with digital pressure, Rumel tourniquets, or vascular clamps (Bulldog, Cooley, Debakey, or Satinksy). Severe hepatic hemorrhage may require use of the Pringle maneuver, which includes digital occlusion of the hepatic artery and portal vein within the epiploic foramen. Definitive hemostatic methods include hemoclips, suture ligatures, electrocautery, and vessel-sealing devices (LigaSure™, En seal®, Harmonic scalpel®). Mechanical hemostatic agents tamponade hemorrhage or provide a barrier while providing a matrix for clot formation. The commonly used agents include porcine gelatin sponges (Gelfoam™), cellulose sheets (Surgicel™), bone wax, bovine collagen, and agents containing human thrombin (Floseal®).

Spontaneous hemoabdomen is often associated with a poor prognosis due to the high likelihood of malignancy. Various retrospective analyses have investigated this condition, and malignant neoplasia was the diagnosis in 76-87% of cases. Mortality rates have been reported for these cases from 16-59%, though non-neoplastic etiologies are considered to have a better outcome.

Hepatic origin hemoabdomen could be due to lobe torsion, hepatic abscess, cysts, and malignant neoplasia. Metastatic neoplasia is more common than primary hepatic neoplasia categorized as hepatocellular, bile duct, mesenchymal, and neuroendocrine. In the case of very large and hilar located hepatic masses, it is important to prepare the patient for potential extension of the abdominal incision either in a paracostal technique or caudal sternotomy. In cases of uncontrolled liver tumor hemorrhage, the hepatic artery can be ligated and the Pringle maneuver should be utilized; antibiotics should be administered if ligation of the hepatic artery occurs. Partial or complete liver lobectomy may be required using any of the following techniques: skeletonization of the parenchyma with ligation of the vasculature or placement of surgical staples, thoracoabdominal (TA) or directional stapling technology (DST) stapler, bipolar vessel sealing devices (LigaSure™), and direct dissection of hilar anatomy with ligation and transection. The author prefers

use of the TA or DST surgical staples but finds that other techniques can be necessary to minimize hemorrhage.

The most common organ of origin for spontaneous hemoabdomen is the spleen, and this is also the simplest organ to manage to arrest hemorrhage via a splenectomy. It is quite difficult to differentiate malignant from benign disease in the spleen, so a full splenectomy is required for spontaneous hemoabdomen. In cases of splenic torsion, de-rotation is not recommended due to the potential release of inflammatory mediators and toxins, and full splenectomy is warranted. Hemangiosarcoma is the most common splenic neoplasia in dogs, though many types of neoplasia are reported. Recent studies evaluating the presence of a splenic mass as the etiology of hemoabdomen document the prevalence of neoplasia to be as high as 76-87% with 76-92% of those tumors representing hemangiosarcoma. Hilar splenectomy is commonly performed; however, some authors suggest ligating only the splenic artery and vein and the left gastroepiploic and short gastric vessels beyond the pancreatic artery and vein to allow more of a “margin” in cases of neoplasia. Common techniques for hilar splenectomy include individual ligation and transection of each vessel, use of a 'ligate and divide stapler' (LDS), and use of bipolar vessel sealing devices (LigaSure™). The use of bipolar vessel sealing devices may provide a shorter anesthesia and surgery time, and the LigaSure™ can safely fuse and seal vessels walls of up to 7 mm in diameter. When splenectomy is performed in a large or giant breed dog, particularly those at risk for gastric dilatation volvulus, the clinician should consider performing a gastropexy at the time of surgery.

The adrenal glands and kidneys are an uncommon source of hemoabdomen. Generally, these are due to adrenal tumors (rupture of adrenal capsule) and trauma or neoplasia of the kidney. In the small number of reported cases of adrenal neoplasia as the source of hemoabdomen, the mortality rate has been documented as high as 50%. About 14% of renal hemangiosarcoma is reported to present as hemoabdomen. Both locations are likely to require advanced imaging prior to surgical intervention.

If a coagulopathy is suspected postoperatively (dilution or DIC) a fresh frozen plasma transfusion should be administered. Continuous or intermittent electrocardiography (ECG) and blood pressure is advised in the immediate post-operative period particularly in patients that demonstrated abnormal blood pressures or significant arrhythmias intra-operatively. Ventricular arrhythmias are common in the postoperative period following splenectomy and can range in severity. Treatment is recommended if ventricular tachycardia is sustained, compromises hemodynamic stability, exhibits R-on-T morphology, or if the ventricular complexes are multiform in appearance. Lidocaine HCl is administered in dogs at 2 mg/kg IV bolus followed by 30-80mcg/kg/min CRI to effect and in cats at 0.25 to 0.5 mg/k IV bolus followed by 10-20mcg/kg/min CRI to effect thereafter.

Considerations for Laceration Repair

Cassie N. Lux, DVM, DACVS-SA, Associate Professor of Surgery, Coughlin Distinguished Professor

Management of lacerations and wounds is one part of managing a trauma patient. Overall patient assessment should always come first when trauma has occurred, and the laceration can be temporarily covered with a protective bandage during patient stabilization. It is important to get a complete history from the clients, including vaccination history, current medications, knowledge of trauma, and some reference of time since the injury occurred. A full physical examination should be performed in addition to any ancillary tests, if there is concern for multi-system trauma or co-morbidities that may complicate healing. Some factors known to delay or complicate healing include infection, high tension or high motion injury location, poor nutrition state, metabolic diseases (hyperadrenocorticism, uremia, and diabetes mellitus), medications (chemotherapeutic agents or corticosteroids), previous injuries or therapy (radiation) to the site, and being feline species.

Lacerations should be approached similar to all open wounds when wound evaluation occurs. Initially, there should be a generous application of sterile, water-soluble lubricant gel, followed by a wide hair clip of the area of the laceration. A preliminary lavage and wound cleansing should be performed prior to evaluation of the tissues. Normal saline, tap water, or a dilute chlorhexidine (0.05%) or povidone iodine (1%) solution can be used to lavage the wound. The best technique to achieve the desired pressure for wound lavage (pressure required to remove debris while minimizing local tissue trauma; 7-8 psi) is with a 1-liter bag of saline in a pressure cuff set to 300 mmHg with 16-22 gauge needles (7-8 psi). Other techniques result in pressures that are less ideal but still beneficial, including use of a saline bottle punctured with a needle (4 psi), and use of 35 mL syringe attached to 16-22 gauge needles (16.5-18 psi). The local skin around the laceration site can be cleansed with a normal surgical scrub (e.g. 4% chlorhexidine scrub solution). Health of the local tissues should be considered including vascular supply. Wounds with compromised blood supply might be locally colder than the surrounding tissues, have thin, friable, or leathery tissues, may be white to yellow or purple to grey/black in color, and will not have capillary bleeding at the wound edges. Evidence of infection or severe inflammation should be noted, including hyperemia, diffuse edema of the surrounding tissues, malodorous tissues or discharge, obvious purulent debris, and significant pain in the region. If muscle, fascia, or bone is exposed, regardless of the other wound qualities, systemic antibiotics should be administered following submission of deep wound tissue for culture. The superficial and deeper tissues traumatized by the laceration should be evaluated for necrotic or severely damaged tissues, as debridement may be needed. Sharp surgical debridement may be required if closure of the laceration is expected to be immediate, but if open wound management is elected for a few days mechanical or autolytic debridement can be used.

The two questions that should come to mind when closing wound lacerations are: "should I close this wound?" and "can I close this wound?". Most lacerations will likely fall into either the primary or delayed primary wound closure categories. Primary wound closure is ideal for fresh wounds, generally within 6 hours of trauma, with minimal or no contamination. Some lacerations need to be closed as soon as possible due to the location of the injury near important or vital structures (body wall penetration), high risk of contamination if left open (wounds near anus or urogenital tract), or extremely difficulty in managing as an open wound. Delayed primary closure generally occurs within 3-4 days of wounding, prior to granulation tissue formation. If a wound has extensive contamination and requires open wound management for repeated debridement, it may fall into secondary wound closure classification, which occurs after a full bed of granulation tissue has formed. Lacerations may appear as localized injury, but cause of the injury is very important to evaluation as damage may be more extensive than is immediately apparent. For instance, animal bites may have more significant tissue injury deep to the dermis that generally requires evaluation and treatment, and crush injuries resulting in lacerations may develop vascular compromise and dermal necrosis 12-24 hours following the trauma. For these reasons, when bite wounds and crush injuries are known causes of trauma, open wound management with a delayed closure should be considered. Another reason to consider delaying closure of the wound is presence of infection or significant contamination. Animals presenting with plant debris, hair, or dirt and rocks from the environment within their laceration will benefit from open wound

management for 24-48 hours to allow debridement of the area and repeated opportunities to remove contamination. Closure over this type of contamination can result in trapping of the debris and result in progressive infection or abscess formation. If there is suspicion of remaining infection or concern that all debris/contamination has not been removed and closure of the wound is needed, then a surgical drain should be placed to allow evacuation of wound fluid. Some considerations when placing drains include: avoid placement directly under the incision site and do not exit them from the incision site, removal should be considered at 5 days or sooner to prevent ascending infection, and drains are radio-opaque to allow them to be tracked via radiography if the drain is damaged, eaten, or dislodged. There are two types of surgical drains, active and passive drains. Passive drains, such as Penrose drains, work via gravity, require a ventrally placed exit site for drainage of fluid, and best used in superficial lacerations (dermis and subcutis) or lacerations where closure of all defects is not an option. Active drains, such as Jackson-Pratt drains, apply consistent negative pressure in a closed environment, are safer for deep lacerations or those connecting to body cavities as they have less risk of ascending infection, can be more consistent at evacuating fluid, and provide negative pressure in the wound bed to promote healing between superficial and deeper layers.

A major contributor for failure of wound closures or complications is excessive tension on a wound. Most lacerations require a subcutaneous layer and a dermal layer of closure to distribute tension from the skin. If the wound is very deep and approached muscle bellies or fascia, a three-layer closure is recommended, and drains can be placed in to the deepest layer to facilitate drainage. Tension may be more of concern in areas where significant trauma has resulted in skin necrosis or where excessive motion or pressure points exist. If wound tension is encountered during laceration closure, there are a few ways to minimize complications secondary to that tension or to mitigate the tension as it is being closed. Undermining tissues local to the laceration below the subdermal plexus is an easy way to alleviate some tension. Generally, a combination of blunt and sharp dissection frees the skin and subdermal plexus from the underlying subcutaneous tissues, allowing more stretch and mobility to the skin. For mild amounts of tension, stent sutures can be placed away from the skin edges to remove some tension from the primary incision site for a maximum of 3-4 days (avoid pressure necrosis). These can include vertical or horizontal mattress sutures placed through IV tubing or a red rubber catheter to distribute pressure on the skin. Bolster bandages can be placed over a laceration closure to distribute tension. Large simple interrupted bites started away from the incision edges by 2 cm or more pass deep to the skin layer in the subcutaneous tissues, and pass out of the skin on the opposite site at approximately the same distance as was started, and then a roll of padding is placed under the knot of the suture and over the incision site. Similarly, a tie-over bandage can be used over a laceration repair to stretch skin towards the center of the incision, removing some tension, and providing compression to the area. If high tension is suspected when open wound management is required prior to closure, pre-tensioning sutures can be placed for 24-72 hours prior to closure. This technique uses the properties of mechanic creep and stress relaxation of the skin to stretch the skin. Use of a large suture size, 0 or 2-0 monofilament nonabsorbable suture, can be placed in a simple continuous pattern through the skin and hypodermis. The initial suture line should be placed loosely, under no tension, so that primary dressings can be applied to wound surface. Use of a split-shot fishing sinker will allow tensioning on the suture line, which can allow the wound edges to be progressively pulled together over 2 to 3 days. If closure of a laceration occurs and excessive tension is noted afterwards, multiple relaxing incisions (stab incisions using a scalpel blade, at least 1 cm from the incision edge, each incision approximately 1 cm long, and all incisions 1 cm apart from each other) or a single releasing incision (full thickness skin incision with a distance from the wound edge that matches the original wound width and is the same length or up to 1.5 times longer than the original length) can be performed. It is important to consider bandages over laceration repairs where compression and restriction of motion might be helpful, but use caution as compression can also result in necrosis of tissues where pressure points reside.

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Keywords: laceration, surgical drains, skin tension, wound closure, wound management

Maximizing Lymph Node Cytology

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Introduction

Lymph nodes are commonly aspirated for cytologic evaluation in companion animal general practice. The most common reasons for cytologic evaluation of lymph nodes are to differentiate a reactive from neoplastic process (i.e. lymphoma) in an enlarged lymph node or to evaluate for metastatic disease during the staging process in patients with a cancer diagnosis. Unfortunately, all too often lymph node aspirates are "inconclusive" because of either thick preparation precluding morphologic assessment or cellular rupture. Here, we'll discuss tips to optimize lymph node smear preparation and perform a cursory evaluation of your cytologic preparations in the clinic.

Sample Aspiration and Smear Preparation

Lymph nodes are very amenable to the non-aspiration or "woodpecker" technique of fine needle biopsy (FNB), using only a 20- or 22-gauge needle. Aspiration with a syringe during the FNB could result in dilution with blood or rupture of delicate lymphoid cells. Lymphoma cells, in particular, appear to be quite susceptible to rupture during the sample collection and smear preparation process.

The preferred method for making a cytology smear with lymph node aspirates is the "squash prep", although this is a misnomer as no "squashing" should be occurring with this technique! Expel the aspirated material on one slide and then place the spreader slide on top, either parallel or perpendicular to the bottom slide. Do not place any downward pressure on the spreader slide, only the weight of the slide should be used to spread the cellular material. Glide the spreader slide over the bottom slide to create an oval smear. Work quickly as aspirated lymph node material is sticky and more difficult to spread as it starts to dry or if the slides are in contact for too long. A small amount of blood in the lymph node aspirate can act as a lubricant, and easier spreading of the aspirated material. Avoid preparation techniques that use a needle to spread the cellular material or by placing the spreader slide on top of the aspirated material and then pulling it straight up off the material. Although both of these techniques are presented in text books, cells are often not adequately spread to assess cellular morphology. Always spread the aspirated material, even if there are just the tiniest speckles on the slide after expelling from the needle. Droplets that dry without being spread result thick splatters of proteinaceous fluid or blood with balled-up cells that cannot be adequately assessed.

Microscopic Evaluation

The general categories of cytologic interpretation for lymph node cytology are:

- Inconclusive/Non-diagnostic
- No cytologic abnormalities
- Reactive
- Inflammation
- Lymphoma
- Metastatic Neoplasm

There are many reasons for obtaining a non-diagnostic lymph node sample:

- **Poor cellularity of the sample:** Due to a poorly exfoliating lesion, inadequate sample collection, or sample dislodging during the staining process

- **Sampling error:** Aspiration of surrounding fat or another structure, such as aspiration of the salivary gland instead of mandibular lymph node

- **Many smudged or ruptured cells:** This may result from exuberant collection methods or smear preparation although some tumor types, such as lymphoma, are just fragile and prone to disrupted cells (see section above on smear preparation)
    - Intact cells will have crisp cytoplasmic margins and cytoplasm will be visible surrounding or adjacent to the nucleus
    - Disrupted cells will appear as lavender/purple streaks or puffy purple nuclei with a "basket-weave" appearance and no visible associated cytoplasm
    - Almost all cytology smears will have areas of disrupted cells. Ask yourself, are there sufficient regions of the smear with intact cells for cytologic assessment?

- **Thick preparation:** Some preparations are so thick that cells are piled on top of each other and morphology cannot be assessed. In a well-spread area, cells should appear like fried eggs where nuclear and cytoplasmic detail can be assessed, versus thick areas where cells appear like hard-boiled eggs and are rounded/three-dimensional.

If the sample has adequate cellularity and the cells are well-stained and well-preserved, the next step in cytologic diagnosis is the identification of the cell types and pathologic process that may be present. It may be helpful to ask a series of questions when working through lymph node cytology smears:

- Did I aspirate lymph node? Is there lymphoid tissue present?
- From low power do the lymphocytes appear homogenous or do I see a mixture of cell sizes?
- What size are the lymphocytes?
    - Lymphocyte nucleus smaller than neutrophil = small lymphocyte
    - Lymphocyte nucleus similar size to a neutrophil = intermediate lymphocyte
    - Lymphocyte nucleus larger than a neutrophil = large lymphocyte
- Am I seeing a predominance of small lymphocytes or a predominance of intermediate or large lymphocytes?
- Am I seeing plasma cells?
- Are there inflammatory cells present in excess to blood in background?
- If there is inflammation, what type is it (neutrophilic, macrophagic, mixed, etc.) and am I seeing a cause, such as an infectious organism?
- Are there any other cell populations present that either shouldn't be in a lymph node (ex: epithelial cells, malignant melanocytes) or present in excess densities (ex: mast cells)?

Common Causes of Enlarged Lymph Nodes

Reactive lymph node

- Mixed (heterogenous) lymphoid cells
- Mostly small lymphocytes (75-80% of nucleated cells)
- Increased proportion of intermediate and large lymphocytes
- Frequent plasma cells
- May have concurrent neutrophilic, eosinophilic, or macrophagic/histiocytic inflammation
- Can see mitotic figures

Large Cell Lymphoma

- Predominance of intermediate or large lymphocytes that all look similar (homogenous or monomorphic)
- Intermediate to large cell proportion >50% of lymphoid cells is suspicious for lymphoma, but severely reactive nodes can have expansions or intermediate or large lymphocytes that approach 50%.
- Large cell lymphoma can be confidently diagnosed when >80% of lymphoid cells are intermediate or large in size
- Can see frequent mitotic figures

Small Cell Lymphoma

- Difficult cytologic diagnosis
- Predominance of homogenous/monomorphic small lymphocytes (similar to what you would see in a normal or reactive node)
- Other evidence of reactivity (expanded intermediate and large lymphocytes, plasma cells, neutrophils, histiocytes) often missing
- If node is moderately to markedly enlarged and predominated by small lymphocytes without concurrent evidence of reactivity, be suspicious of small cell lymphoma
- Might need additional diagnostics such as histopathology, flow cytometry, or PCR for antigen receptor rearrangements (PARR) for definitive diagnosis

Metastatic neoplasms

- **Metastatic mast cell tumor**
  - low to moderate numbers of mast cells are normal in lymph node aspirates, particularly reactive nodes
  - Effacement of node by mast cells or mast cells present in large aggregates of 5-10+ cells lends confidence to cytologic diagnosis of metastatic mast cell tumor

- **Carcinoma**
  - Epithelial cells are not expected in normal or reactive lymph nodes
  - Need to exclude contaminants (keratinocytes), aspiration of an adjacent epithelial structure (ex: salivary gland sampled concurrently with mandibular lymph node), or aspiration of a primary epithelial neoplasm overlying the area of the lymph node
  - Evaluate for clusters of cohesive cells with tight junctions to ensure epithelial origin. Aggregates of histiocytes/macrophages in reactive nodes can mimic epithelial cells.

- **Melanoma**
  - Melanoma cells contain finely granular green to brown cytoplasmic pigment
  - Amelanotic melanoma cells can be difficult to identify
  - Need to exclude melanophages, which are macrophages containing phagocytized melanin. These usually have larger, coarser globules of green-black pigment. Melanophages can be present in reactive nodes or in nodes from animals with pigmented skin.
  - Need to exclude hemosiderophages, which are macrophages containing the blood-breakdown pigment, hemosiderin. Pigment is generally larger, coarser, and variable in size and color (golden brown to dusky blue-grey).

Helpful Tips

- Do stain 1-2 smears and take a look for yourself. Write down your thoughts and compare to the cytology report when it is returned. Include your interpretation in the history section of the submission form. This way we can address why we do or do not agree with your initial interpretation in our report. Be sure to submit the stained smears that you evaluated to the clinical pathologist. What you saw on your stained smears might not be present on the submitted unstained smears.

- Do submit several unstained smears in addition to the stained smears you already assessed. The stains we use in the clin path lab have a wider color spectrum and are helpful for subtle changes in chromatin pattern, which can be essential when diagnosing lymphoma. The benchtop stains used in practice can mask these subtle differences.

- Do submit aspirates from more than one lymph node when you are concerned about lymphoma (and label which slides represent which lymph node). The proportion of intermediate to large lymphocytes can vary from node to node. An expansion of large lymphocytes on one slide from one node, could represent aspiration of a germinal center in a reactive node, not lymphoma. At UTCVM diagnostic laboratory services, we allow submission of three lymph nodes on one cytology submission/fee.

- Do Not heat fix, refrigerate, or ship your cytology slides directly on an ice pack or with samples contained in formalin. This results in artifacts that make these slides non-diagnostic.

- Do call your clinical pathologist if you have any questions about sample preparation or the cytology report.

Regenerative Anemia: Is it Hemorrhage or Hemolysis?

Nora L. Springer, DVM, PhD, DACVP, Assistant Professor of Clinical Pathology

Introduction

Anemia is defined as decreased red blood cell mass, usually determined by evaluating hematocrit or packed cell volume. Generally, anemia is further characterized by severity, red blood cell indices, and regenerative response. This characterization is done to help identify the underlying mechanism(s) and cause for the anemia. Assessment of regeneration is the first step in evaluating an anemia. If an anemia is regenerative, it is due to hemorrhage or hemolysis. If an anemia is nonregenerative, it is due to decreased bone marrow production. Remember, the bone marrow takes 3 to 5 days to respond to an anemia, therefore some acute anemias secondary to hemorrhage or hemolysis will initially appear non-regenerative, a third category of anemia termed "pre-regenerative". In this lecture, we'll discuss the mechanisms of regenerative anemias and the blood smear findings, hematologic, and biochemical parameters that can help you differentiate between hemorrhage versus hemolysis in patients with regenerative anemias.

Identifying Regenerative Anemias

In dogs and cats, the best method to assess for a regenerative response is quantification of an absolute **reticulocyte count.** Not all hematology analyzers are capable of performing an absolute reticulocyte count, so this can also be done manually using a new methylene blue stained blood smear. Semiquantitative enumeration of **polychromatophils** on a blood smear also helps to differentiate between regenerative and non-regenerative anemias, with the exception of equids, who do not usually release polychromatophils in circulation.

Other **red blood cell morphologic changes** present in animals with a regenerative response are:

- Anisocytosis
- Increased numbers of nRBC – not equids
- Basophilic stippling – particularly in ruminants
- Increased Howell-Jolly bodies
- Macrocytes – particularly ruminants and equids

These findings are not specific to regeneration alone and should be interpreted in conjunction with other hematologic findings.

Red blood cell indices can assist in differentiating regenerative from non-regenerative anemias. Because immature red blood cells (reticulocytes, visualized as polychromatophils on a blood smear) are larger and contain less hemoglobin than mature RBC, regenerative anemias might have an increased MCV and decreased MCHC (macrocytic, hypochromic). However, remember that the "M" in these abbreviations stands for "mean" indicating that these are average values of the entire red blood cell population. Therefore, the regenerative response has to be quite robust for the mean of the RBC population to be shifted outside the reference interval. Many regenerative anemias will be normocytic, normochromic. **Take Home Point:** *A macrocytic, hypochromic anemia is most likely regenerative, but a normocytic, normochromic anemia does not exclude a regenerative response.*

Mechanisms of Regenerative Anemias

Hemorrhage

Hemorrhage occurs when RBCs are lost outside of blood vessels. This can be either internal hemorrhage, such as hemoperitoneum or external hemorrhage. In some instances, like trauma, external hemorrhage is not a diagnostic dilemma. However, other forms of external hemorrhage can be more insidious such as bleeding in the gastrointestinal, urinary, or respiratory tracts.

Hemorrhage is often associated with **low total protein concentration** due to concurrent loss of albumin and globulins. This decrease is attributed to dilution of plasma proteins due to the replacement of depleted blood volume by shifting of extravascular, interstitial fluid, which has a low protein concentration, into the intravascular space. Hypoproteinemia associated with hemorrhage is dependent on multiple factors including degree and duration of hemorrhage, the need to maintain intravascular volume, the body's ability to increase protein synthesis, and whether or not there is concurrent inflammation that affects protein concentrations. Hypoproteinemia can be present with either internal or external hemorrhage, but is often more prominent with external hemorrhage.

In cases of **chronic, external hemorrhage,** there might be concurrent **iron deficiency**. In iron deficiency, smaller red blood cells are produced that contain less hemoglobin. RBC indices can be variable in regenerative anemias due to external hemorrhage. However, the presence of a **microcytic hypochromic** anemia is highly suspicious for iron deficiency anemia. **Take Home Point**: *A microcytic, hypochromic anemia (either regenerative or nonregenerative) is most likely secondary to iron deficiency, but iron deficiency is also possible in anemias that are not microcytic, hypochromic.* In iron deficiency anemias, a subpopulation of RBCs may appear hypochromic, with increased central pallor fading into a thin rim of peripheral hemoglobinized cytoplasm. These hypochromic RBCs are more mechanically fragile and evidence of fragmentation injury, including schistocytes, keratocytes, and acanthocytes, can be observed on blood smear evaluation. Technically, fragmentation injury is a form of hemolysis (see below), thus overlapping mechanisms can be contributing to regenerative anemias. **Serum iron concentration and % transferrin saturation** can be low or normal with chronic external hemorrhage. Internal hemorrhage does not result in depletion of iron stores.

Hemolysis

Hemolysis occurs when RBCs are destroyed within the body prior to the end of their normal lifespan. This destruction can be **extravascular** due to increased removal by macrophages in the spleen, liver, and bone marrow or can be **intravascular** due to lysis in the blood vessels. All underlying causes of hemolysis will result in extravascular hemolysis, but not all will have intravascular hemolysis. Intravascular hemolysis, but not extravascular hemolysis, will result in hemoglobinuria, as free hemoglobin in plasma can escape glomerular filtration and appear in the urine.

Hemolytic anemia often results in **hyperbilirubinemia** due to production of indirect (unconjugated) bilirubin by macrophage-mediated red blood cell turnover that exceeds the liver's ability to conjugate and excrete the bilirubin. **Hyperferremia,** increased serum iron, might be present in cases of hemolytic anemia as iron is a major component of hemoglobin. Total protein is usually not decreased with hemolysis.

A cause for hemolysis might be identifiable on blood smear examination. Red blood cell morphologic changes associated with major causes of hemolysis are listed below.

Immune-mediated hemolysis (IMHA)

- Spherocytes (extravascular hemolysis)
- Agglutination
- Ghost cells (intravascular hemolysis)

Oxidative injury (zinc, onion/garlic, copper, red maple leaf, acetaminophen)

- Heinz bodies
- Eccentrocytes

Erythroparasites

- *Mycoplasma haemofelis or canis*
- *Anaplasma bovis*
- *Theileria spp.*
- *Babesia spp.*

Fragmentation injury – usually secondary to vascular disease or DIC

- Schistocytes
- Keratocytes
- Acanthocytes

Other uncommon causes of hemolytic anemia include hemophagocytic histiocytic sarcoma, inherited red blood cell defects, severe hypophosphatemia, bacterial toxins, and spider bite or snake envenomation.

SUMMARY TABLE

Finding*	Hemorrhage	Hemolysis
Evidence of bleeding severe enough to cause anemia	Yes	No
RBC morphologic changes (all changes +/-)	Hypochromasia	Spherocytes Agglutination Ghost cells Eccentrocytes Heinz bodies Erythroparasites
Total protein	Usually low	Normal to increased
Total bilirubin	Normal	Often increased
Serum iron	Low or normal	Normal or high
Urinalysis		Hemoglobinuria (intravascular only)

*These are generalizations and there will always be exceptions not listed in the table

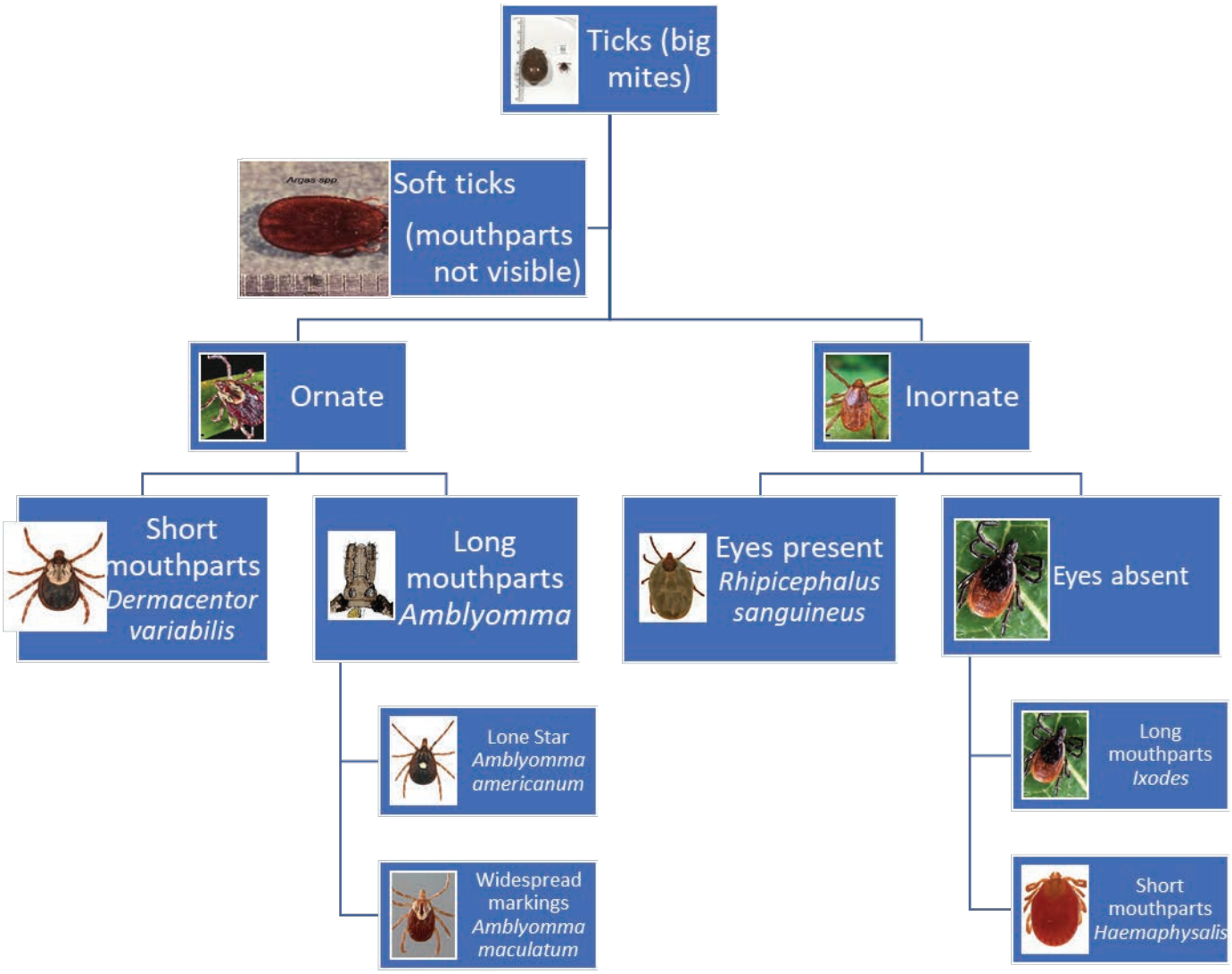
Final Caveat: Not all regenerative anemias can be neatly categorized as hemorrhage or hemolysis and we can't always determine the mechanism of anemia. Anemia can be caused by overlapping processes. For example, a patient with an inadequate regenerative response to hemorrhage or hemolysis might also have inflammatory disease suppressing the bone marrow's ability to respond. Always consider all the laboratory data, physical examination findings, and your clinical intuition! And when in doubt, feel free to contact your friendly neighborhood clinical pathologist for a consultation.

Living in the Lymelight

John J. Schaefer, DVM, MS, PhD, DACVM (Parasitology)

The lecture is a broad overview of the spectrum of ticks and tick-borne diseases that may be encountered in Tennessee. Ticks are highly specialized arachnids that rely exclusively on the acquisition of blood meals from a spectrum of higher animals to survive and perpetuate their populations. The tendency of many to feed on a large spectrum of hosts and their generally hardy nature make them ideal candidates for transmitting a variety of disease-causing pathogens, notably the causative agent of Lyme disease *Borrelia burgdorferi*. Tennessee is in the unfortunate position of being a state with a number of severe entrenched tick-borne diseases and being at the edge of what is potentially a wave of increased cases of Lyme disease, as has been realized in Southwestern Virginia. This lecture will explore the biology of some of the most common ticks that we encounter in our state, as well as enumerating the pathogens that are spread by them. Brief summaries of these diseases and their recognition will conclude the lecture. Please find below an extremely simplified flowchart for distinguishing the most commonly encountered ticks on domestic animals in our state.

Common Ticks of Domestic Animals in Tennessee (females)



The Down Dog: Diagnosis and Treatment of Acute Lower Motor Neuron Tetraparesis.

Talisha M. Moore, DVM, DACVIM (Neurology)

Every 'down dog' is not a consequent of spinal cord disease. In fact, diseases causing acute lower motor clinical signs may present as a "down dog' to the untrained eye. Lower motor neuron (LMN) disease is a consequence of disorders that preferentially affect the motor neuron within the ventral horn of the spinal cord grey matter, the peripheral motor nerve (neuropathy), the muscle fiber (myopathy), or the neuromuscular junction (junctionopathy). Clinically, patients with acute LMN disease present with generalized neuromuscular weakness (e.g., paresis), and if severe, complete loss of motor function (e.g., paralysis). Muscle tone and myotactic reflexes are diminished. And, concurrent cranial nerve abnormalities, autonomic dysfunction, megaesophagus, and respiratory compromise due to phrenic and/or intercostal nerve involvement may be present.

There are numerous etiologies of lower motor neuron disease ranging from toxins to neoplasia. However, the most common etiologies for acute LMN tetraparesis include myasthenia gravis, idiopathic polyradiculoneuritis (Coonhound paralysis), botulism, and tick paralysis. With such an array of differential diagnoses and disease-specific treatments (e.g., acetylcholinesterase inhibitors), it is imperative that an accurate diagnosis be made. Clinical differentiation and successful treatment of these diseases hinges on thorough history-taking, accurate neuroanatomic localization, and performing common diagnostics (i.e., complete blood cell count, serum biochemistry, and thoracic radiographs). The purpose of this session is to briefly review lower motor neuron disease, common neurologic findings, diagnosis and treatment.

Rabbit Dental Disease

Emi Knafo, DVM, DACZM, Clinical Assistant Professor of Zoological Medicine

Rabbits are classified in a separate mammalian order, Lagomorpha. The presence of sharp rostral incisors resulted in their classification alongside rodents. However, the second pair of reduced upper incisors present behind the major set (peg teeth) is what sets them apart from rodents.

The dental formula of the rabbit is 2(I 2/1, C 0/0, PM 3/2, M 3/3) =28. The lack of canine teeth creates an elongated diastema between the incisors and premolars. Rabbit teeth are classified as elodont (meaning continuous growth with no anatomic root) and hypsodont (for having a long crown). Rabbit mouths exhibit anisognathism, meaning that the lower jaw is narrower than the upper, and have a maximum gape of about 20-25 degrees, which makes evaluation of the teeth and dental procedures more difficult. The portion of the tooth that can be visualized above the gum line is called the clinical crown, while the portion that remains below the surface is the reserve crown. Since the teeth are continuously growing, they also do not have a true root, but instead have an apex (or germinal center) from which new growth emerges.

At rest, the opposing incisors and cheek teeth (premolars and molars) remain in contact. When eating, rabbits will use the incisors to cut the food into manageable lengths using a vertical, chopping motion. Once the food is passed to the cheek teeth, a rotating horizontal grinding motion is used to process the food prior to ingestion. This horizontal motion is a large contributor to proper dental wear, which is only achieved when coarse roughage such as hay is consumed. During this process, every lower cheek tooth occludes with 2 upper cheek teeth with the exception of the first mandibular premolar (PM 3) and the last mandibular molar (M 3). This dual occlusion allows for normal wear even in the absence of one opposing cheek tooth.

Dental disease is divided into four main classes: congenital, traumatic, metabolic bone disease, and abnormal wear. Dental disease is one of the most common reasons companion rabbits present to veterinarians. It is usually acquired but can be congenital, and is always progressive. As veterinarians, we do not cure dental disease, we treat specific disease events and manage the patient's dental disease over time. This is an important expectation to set for clients.

Acquired Dental Disease

The most common causes of abnormal wear are low fiber diets and excessive carbohydrates. The natural diet of wild lagomorphs is high in fiber and silicates which require exaggerated horizontal movement and appropriate wear of the clinical crown. In captivity, a diet high in grass hay and fresh greens is the best substitute, but will not eliminate the risk of acquired disease. Rabbits that lack fiber in their diet are far more likely to end up developing dental disease over time. Without fiber and normal masticatory movements, the elodont teeth continue to grow and create pressure on the apex. The apex will be pushed further into the socket, elongating the reserve crown and bending the apex. This changes the direction of growth in the clinical crown and creates gaps between teeth where food and debris can accumulate (and ultimately lead to infection and abscessation). Over time, lingual points will develop on the mandibular cheek teeth and buccal points will develop on the maxillary cheek teeth. The horizontal chewing motion required to grind hay will aggravate lesions in the mouth resulting from these acquired points, and as a result of oral pain, rabbits will begin to prefer pellets that can be crushed using vertical masticatory motions, further exacerbating the problem.

Incisor Malocclusion

Incisor malocclusion can be treated by either routine trimming of the affected teeth or by extraction. Deciding which plan to offer is based on signalment, history and diagnostic work up. Young rabbits with congenital incisor malocclusion are better candidates for extraction as the frequency and cost of trimming can become overwhelming every 4-6 weeks for the next 7-10 years. Extraction is the only option for rabbits presenting with tooth root infection. Trimming can be performed awake in calm rabbits who allow for proper manual restraint. Sedation or gas anesthesia will be necessary for some rabbits to prevent iatrogenic injury to the surrounding soft tissues during a trim. A Dremel™ tool with a diamond wheel is the preferred instrument to reduce the likelihood of longitudinal fractures. A tongue depressor is used on the mesial surface to protect the soft tissues once the disc has transected the incisors. Incisor trimming with nail trimmers, wire cutters or rongeurs should all be used with caution. Crushing the teeth with these tools can lead to longitudinal fractures that will cause introduction of bacteria to the reserve crown and surrounding soft tissues. Incisor extraction is always performed under anesthesia. The use of a Crossley Incisor Luxator™ as well as flattened and curved large gauge hypodermic needles are required. Ensuring that all the dental pulp is removed is important to reduce the incidence of the incisors growing back in. Using properly curved needles to curettage the alveoli after extraction can reduce the incidence of regrowth.

Extraction of the Incisor Teeth

Molar points not able to be effectively or safely trimmed in the awake patient. Inhalant anesthesia is required to allow for a patient to be properly placed on a dental board where full oral examination can be performed. A low-speed drill is the ideal tool to quickly and effectively reduce molar spurs or malocclusion, and restore the occlusal angle. Hand tools, such as rongeurs and dental rasp, can be useful in conjunction with a low-speed drill, but would prolong a dental procedure if used alone. Finding loose or diseased teeth may indicate the need for extraction. Intraoral extraction can be challenging given the limited gape of the rabbit mouth and the caudal location of the cheek teeth in the mouth. If indicated, a Crossley Molar Extractor™ and extraction forceps will be necessary. If only one molar is removed then removal of the opposing tooth is not necessary. Each cheek tooth engages with two opposing teeth and will allow for normal wear.

- Incise gingival attachment circumferentially around the tooth with tip of No. 11 or 15 scalpel
- Insert a luxator in periodontal space on medial side of the tooth until resistance is felt
- Hold in position for a few seconds to stretch periodontal ligament
- Gradually move the tip of the luxator toward the apex of the tooth
- Use free hand to hold and stabilize the jaw at all times
- Perform the same procedure on the lateral aspect of the tooth
- Use a contoured, 18-gauge hypodermic needle to break down the periodontal ligament on the lingual and labial sides of the tooth, if needed
- The tooth should be loose and very mobile at this point
- Grasp the tooth with extraction forceps close to gingival margin
- Extract using steady, slowly increasing force, following the curvature of the root
- Once loosened, advance the tooth in the socket to destroy germinal tissues at bottom of alveolus
- If periodontal ligament has been completely and correctly severed and the tooth is not severely deformed, it can be extracted without the use of significant force
- Once extracted, examine the tooth to ensure that the entire tooth and pulp have been removed
- After extraction, insert a needle into the alveolus to curette the alveolar walls and damage any germinal tissue to prevent tooth regrowth
- Flush alveolar cavity with saline solution to remove any debris and dental or bony fragments
- If periapical infection is present, use dilute 2% povidone iodine or 0.05% to 0.1% chlorhexidine solution
- Use a contoured needle to sever the periodontal ligament on the labial and palatal sides of the maxillary incisor teeth, similar to the procedure described for mandibular incisors. Lift the upper lip with your free hand, which is used to hold and stabilize the patient's head without obstructing the nares (if the patient is not intubated).

- Sever the periodontal ligament on the medial and lateral aspects of the teeth with Crossley's luxator. The periodontal ligament is particularly tight on the medial aspect of the tooth.
- Once it is loose, gently grasp the tooth with suitable extraction forceps or a pair of needle holders. To avoid dental and bony fractures, gently extract the tooth following the natural shape of the tooth, applying slight distal rotation.
- Completely extract the tooth and examine it for the presence of pulp tissues. Repeat the procedure on the contralateral incisor tooth. Control bleeding with sterile cotton swabs.
- Use a thin (22-gauge) hypodermic needle to loosen the second incisor teeth. When the tooth is completely luxated, extract it with small extraction forceps or thin hemostats, taking care to avoid crushing the tooth.
- After extraction, curette the alveoli to remove any remaining pulp tissues and rinse with saline or 0.1% chlorhexidine solution.
- To promote gingival healing, close the extraction site with simple interrupted sutures or a purse-string suture pattern with 3-0 or 4-0 absorbable suture material. Do not suture the extraction site when infection is present. In using a purse-string pattern, fix the suture material at a minimum of six points before tightening it.
- Obtain a lateral view postoperative radiograph to confirm complete extraction of all six incisor teeth.

Procedure for Treatment of Surgical Abscesses and Osteomyelitis

- Perform a head CT with contrast or diagnostic radiographs in five standard projections before the dental procedure.
- For optional adjunct analgesia, perform local nerve blocks of the mental and inferior alveolar nerves for extracting mandibular cheek teeth and surgical debridement of the mandible. Perform a local nerve block of the rostral infraorbital nerve for extracting the maxillary cheek teeth and surgical debridement of the maxilla.
- Place the patient under general anesthesia in dorsal or lateral recumbency, depending on necessary intraoral procedures and the site of infection.
- Perform intraoral dental procedures as needed; in particular, extract the tooth/teeth affected by periapical infection.
- Shave and aseptically prepare the surgical site. Place an adhesive transparent drape on the surgical field, facilitating the view of the orientation of the head.
- Make a 1- to 2-cm skin incision over the mass, taking care not to enter the underlying abscess.
- Gently dissect subcutaneous tissue and muscle layers to free as much of the abscess capsule as possible, taking care not to disrupt connection to the cortical bone.
- Incise the junction between the capsule and mandibular bone with the tip of a No. 11 scalpel blade. Portions of the wall of the abscess are typically composed of thick connective tissue and/or thin cortical bone, which usually prevents removal of the entire capsule in one piece. Carefully dissect the wall of the abscess free from the bone and remove it.
- Remove the purulent exudate and flush the bone cavity. Collect samples for culture and sensitivity from the capsule wall, as the purulent material itself is often sterile. Remove remaining debris or purulent material with a bone curette.
- Debride infected or necrotic cortical bone to the point of bleeding with a bone curette.
- After thorough debridement, the fragment of the diseased tooth/teeth, if present, can be seen. Use a hypodermic needle to gently free the attachment of the fragment to the bone. Extract the tooth and fragment of necrotic alveolar bone.
- Debride the bone cavity again and thoroughly flush with saline and dilute povidone iodine or 0.1% chlorhexidine solution.
- Marsupialize the surgical site with 3-0 or smaller monofilament long-lasting absorbable or nonabsorbable suture material.

- Obtain postoperative radiographs with lateral and oblique views to confirm proper coronal reduction of the cheek teeth, bone debridement, and extraction of tooth fragments.
- Show and discuss with the owner detailed instructions for flushing. Schedule frequent rechecks.

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Introduction to Honey Bee Medicine

Emi Knafo, DVM, DACZM, Clinical Assistant Professor of Zoological Medicine

Honey Bee Anatomy

Honey bees are invertebrates with exoskeleton exterior, a flat, triangular head, which contains a brain and sensory organs (sight, tough, taste, smell), glands to produce royal jelly and pheromones. There are 2 compound eyes for distance, 3 simple eyes (ocelli) for low light vision within the hive, 2 antennae with thousands of sensors to detect smell, moisture, distance when flying, mouth parts include mandibles for feeding larvae, collecting pollen, manipulating wax, and carrying objects. The proboscis is retracted at rest and unfolded to allow feeding, drinking.

The thorax is located between the head and abdomen with 2 pairs of wings attached to each side. The wings are hooked together in flight and separated at rest. Each of the 3 pairs of legs has 6 segments with taste receptors on the tips. Each pair of legs has a unique function: the forelegs clean the antennae, the middle legs walk and pack pollen onto pollen baskets (on hind legs), and the hind legs contain combs and pollen press used to brush, collect, pack, carry pollen and propolis. The trachea is connected to the spiracles, which are holes in the side of the thorax through which the bee breathes.

The abdomen contains all the digestive organs, heart, wax glands, reproductive organs, scent glands (in workers), and stinger (workers and queen).

Honey Bee Language

Chemical:

Pheromones are chemical scents produced by animals to trigger behavioral responses from other members of the same species. In honey bee colonies, queen pheromones (also called queen substance) is used to inform the colony that the queen is present and stimulates worker bee activities. Worker activities include comb building, brood rearing, foraging, and food storage. Queen pheromone also acts to attract male drone suitors from other colonies (outside the hive), and regulates the drone population inside the hive.

Worker bees use the Nasonov gland at the tip of the abdomen to produce pheromones. The major worker bee pheromones are ones released at the hive entrance to guide foraging bees back to the hive, as well as alarm pheromones to trigger aggression from the colony. Brood also secretes pheromone to help workers recognize the brood's gender, stage of development, and feeding needs

Choreographic:

Foraging worker bees perform dances on the comb in precise patterns to share information with other workers regarding water, nectar, and pollen sources. There are 2 main dances: the Round Dance and the Waggle Dance. The Round Dance is used to communicate when food sources are near the hive (10-80 yards), whereas the Waggle Dance is used for food sources farther from the hive. The Waggle Dance includes shivering side to side movements of the abdomen in a figure of 8 pattern. The number of times this is repeated, the direction, and the sound made communicate precise information. Pauses between performances allow other worker bees to taste the resources brought back to the hive. This likely also provides information about location, food type, and flower type.

Roles Within the Hive

Queen:

Queen honey bees are the largest bee and the only bee without which the colony cannot survive. A strong queen is reflected in a strong and productive hive. Therefore, at each hive inspection the beekeeper should ask the question "is a queen present?" and "is she healthy?" There will only ever be one queen per hive (if a second queen, a successor, were to hatch the two queens would fight to the death). The queen is the only female with fully developed ovaries and she produces ~1500 eggs/day at 30 second intervals. The queen is unable to feed or groom herself, and relies on attendants to perform these basic needs as she moves through the hive. She has a stinger, yet it is rarely used. Only to sting rival queen bees that may emerge or be introduced. Queens live 2+ years, but are generally replaced every 1-2 years to maximize productivity since an old queen results in slower egg production, a smaller colony, and lower honey yield. The major pheromone that queens produce is called Queen substance. It is produced in the mandibular glands and has several functions:

- Inhibit worker bees from making new queen
- Prevent development of worker bees' ovaries
- Chemical communication that all is well

As the queen ages, her pheromones diminish, and the colony knows to produce a new queen to supersede. Queen attendants pick up the pheromone and transfer it by contact to neighbors. They pass it along similarly to spread the message throughout the colony. If a queen is removed from a hive, the entire colony knows within hours. In the absence of a queen, workers lose productivity as they have no directions.

Workers:

Worker bees are all female and comprise the majority of the hive population. This cast of bees can be identified by their smaller stature than the queen and shorter abdomen. They have functional ovaries but are infertile as they never mate and have no capacity for sperm storage. The hind legs have pollen baskets. The stinger consists of 3 barbed shafts. The barbs cause the stinger, venom sac, and part of bee GI to remain in a mammal after delivering a sting (this is fatal to the bee). Workers can repeatedly sting non-mammals (i.e. another invertebrate inside the hive) as the barbs do not stick as they do in mammals. The life span of worker bees is 6 weeks during the active season, and 4-8 months during the less active winter season.

Worker bee duties vary with age:

- Housekeeping, days 1-3
    - Emerge from cell, self-grooms, feeds on pollen and honey
    - Clean cell from which she emerged, then cleans out other cells so they can receive new eggs

- Undertaking, days 3-16
    - Important to prevent disease
    - Removes dead bees, brood. Dispose of as far from hive as possible
    - Invaders like mice are stung to death. Too large to remove, so encased in propolis
    - Propolis: sticky resin, "bee glue" that has antibacterial properties
    - Mummifies mouse corpse

- Nursery, days 4-12
    - Tend to developing larvae by feeding mix of pollen, honey, royal jelly
    - Royal jelly produced from hypopharyngeal glands in worker bees head

- Queen attendant, 7-12
    - Groom, feed, and remove excrement from the hive
    - Coax queen to lay eggs as she moves around hive

- Foraging, days 12-18
    - Take nectar from field bees as they return to the hive. House bees deposit it into cells, add enzyme and start fanning to evaporate water and turn nectar into honey
    - Take pollen from field bees and pack them into cells. Honey and pollen, "bee bread" are food for the colony

- Fanning, days 12-18
    - Control temp and humidity inside hive
    - Line up at one side of entrance facing hive, fan furiously to draw air into hive
    - Additional fanners inside hive
    - Maintain constant temp 93-95F for brood development
    - Fanning aids evaporation of excess moisture from curing honey
    - Communicative fanning:
        - Nasonov (scent) gland at end of abdomen
        - Arch abdomen, expose moist pink gland tissue while fanning wings to release sweet odor into air
        - Pheromone attractive to other bees and helps orient returning foragers

- Architects and builders, days 12-35
    - At 12 days old, produce beeswax
    - Secreted from wax glands at underside abdomen
    - Build new wax comb and cap ripe honey and cells with developing pupae

- Guards, days 18-21
    - Sting glands are mature and contain venom
    - Guard bees stay at hive entrance poised and alert
    - Check each bee that returns to the hive for familiar scent
    - Only family members allowed inside
    - Strange bees, wasps, hornets, etc are driven off
    - Occasionally, guard bees are bribed with nectar by bees from other hives. These strange bees are allowed to pass and steal honey, nectar or pollen, then leave

Roles Outside the Hive

Workers:

- Field bees, days 21-42
    - Last, most important job
    - Collect pollen, nectar, and propolis to sustain hive
    - Orientation flight
        - Bees face hive, dart up and down all around entrance
        - Imprinting look, location of hive, then encircle it in progressively wider circles
        - Learn landmarks
        - Visit 5 million flowers to produce single pint of honey
        - Forage 2-3 mile radius from hive to search for food (~8,000 acres)
        - Can get cold at dusk before returning to hive, be eaten by birds, other, or wings can be traumatized

Drones:

Drones are the only male bee in the entire colony. They comprise a small percentage of the total population (up to ~1,000 individuals). Drones are distinguished by their large eyes and large, stout body. They are often mistaken for a queen bee, though a queen has a tapered abdomen compared to the drone's barrel. Drones don't forage as they dont have pollen baskets. They do not contribute to building comb as they have no wax glands, and they don't defend the hive as they have no stinger. Worker bees care for drones, and the single function of a drone is to mate with new virgin queen bees when the old queen dies or is superseded. As the weather cools, workers become intolerant of drones as they consume a significant amount of food over winter months. Drones are expelled from the hive as the climate cools (end of nectar producing season in temperate climates).

Life Cycle in the Hive

In the winter, bee hives are dormant. Adult bees cluster in the center of the hive for warmth and the queen is kept deep in the middle. As spring begins and the weather warms, bees start feeding the queen royal jelly produced from their mandibular glands. This protein rich material stimulates egg production in the queen.

Honey bee eggs develop in 4 stages: egg, larva, pupa, adult:

- Queens develop in 16 days
- Workers develop in 21 days
- Drones develop in 24 days

When a queen lays fertilized eggs into smaller cells, these eggs become workers. When the queen lays unfertilized eggs into larger cells, these eggs become drones. Workers create comb cells and therefore control the ratio of females:males in the hive.

Basics of Honey Bee Practice

In 2017, the Food and Drug Administration began requiring veterinary oversight of antimicrobials used in food-producing animals that also are important in human medicine. This effectively created the field of honey bee medicine as beekeepers were suddenly required to source antibiotics through veterinarians.

The profit margins within the honey industry are slim, so large-scale apiarists often need to be convinced that veterinarians can add value to their business. It's important to remind producers that veterinarians are trained in biosecurity, disease management, herd health, and toxicology, and therefore can help improve the health of honey bee colonies.

Conversely, honey bee hobbyists, or backyard beekeepers usually have two or three hives. These beekeepers aren't usually using their hives to generate income, and view the bees more as pets than a production animal. Therefore, they may be more receptive to a veterinarian keeping their colonies healthy.

In order to prescribe antibiotics for a bee hive, there must be a valid Veterinarian Client Patient Relationship (VCPR). In order for a veterinarian to write a lawful VFD, the veterinarian must issue the VFD in the context of a VCPR as defined by the state. However, if applicable VCPR requirements as defined by that state do not include the key elements of a valid VCPR as identified in FDA's regulations (21 CFR 530.3(i)), the veterinarian must instead follow federal VCPR requirements and issue the VFD within the context of a valid VCPR as defined by FDA. In general, a VCPR is present when all of the following are met:

- The veterinarian has assumed responsibility for making clinical judgments regarding health of the patient and the client has agreed to follow the veterinarians' instructions
- The veterinarian has sufficient knowledge of the patient to initiate at least a general or preliminary diagnosis of the medical condition of the patient.
- The veterinarian is personally acquainted with the keeping and care of the patient by virtue of a timely examination of the patient by the veterinarian, or medically appropriate and timely visits by the veterinarian to the operation where the patient is managed
- The veterinarian is readily available for follow-up evaluation or has arranged for the following: veterinary emergency coverage, and continuing care and treatment
- The veterinarian provides oversight of treatment, compliance, and outcome
- Patient records are maintained
- The veterinarian must visit the bee yard and personally inspect the hive

Veterinary Feed Directive (VFD)

The veterinarian must completely fill out the VFD form in compliance with the conditions for the specific drugs. It's important to enter additional information to specifically ID animals treated. It's a requirement to provide the client with a copy of the VFD, and the veterinarian must retain the original VFD for 2 years. The veterinarian must be able to provide VFD orders for inspection and copy to FDA upon request. When filling out the VFD, pay attention to the "Indications for Use" section, and enter indication VERBATIM from product approval off the drug label. In the "Caution" section, enter a cautionary statement from the drug label (if there is no cautionary statement, enter "NONE").

Parasites and Vector-Borne Disease in Small Animals: Prevention is Best!

Bryan Clarke, DVM, Zoetis

Parasitic diseases, including those resulting directly from the parasite as well as vector-borne conditions, have long been one of the most common groups of diseases faced in veterinary medicine. The prevalence of parasitic and vector-borne diseases is such that preventive parasiticides and routine screening are critical aspects of wellness care for general practitioners. Despite the development newer generations of parasiticide agents which offer greater efficacy and convenience, compliance rates amongst pet owners remain well below recommendations. Not surprisingly, as parasiticide compliance remains low, the incidence of some vector-borne diseases is increasing. Veterinarians must familiarize themselves with these trends as geographical regions previously considered low or no risk for some conditions are seeing substantial increases in incidence. This lecture will cover data and trends in parasitic disease, parasiticide compliance, and vector-borne diseases, as well as focus on using the most effective parasiticide agents in order to maximize the protection for each pet. We will also discuss some strategies for the individual practitioner and for the practice to help increase their parasiticide compliance rates.

Creating a Veterinary Community of Care in your Clinic:

Tools for Wellbeing and Peace

Elizabeth B. Strand, Ph.D., LCSW Director, Veterinary Social Work

This presentation will review tools that you can use at macro and micro levels to create a community of care in you clinic. Starting with macro (i.e. community) skills, we will use interactive tools to collectively define "a community of care." Then the presentation will offer an array of resource tools such as free workbooks or training to take or download, book recommendations, and communication habits and skills to consider implementing within the clinic. Then the presentation will begin to explore micro (i.e. individual skills) such as knowing your brain state, knowing your conflict style, knowing how and when to set boundaries, and developing a personal mission statement. Lastly, the session will end with a thoughtful action plan activity to help you implement ideas that match yourself and community care goals.

Resources:

- Seven Challenges of Cooperative Communication Workbook Set Boundaries Find Peace, Neda Glover Tawwab
- United Nations Educational, Scientific and Cultural Organization Handbook for Intercultural Story Circles
- Animal Depopulation Resiliency Check In Tool Description Worksheet
- Ten Communication Guidelines
- Take the free conflict styles assessment: https://www.usip.org/public-education/students/conflict-styles-assessment /
    - What is it?
- Coherent Breathing Exercise
- Coherent Breathing App: https://www.breathing.zone/
- Ascending Descending Bells: https://drive.google.com/file/d/1Q33evgeTxl33RXBI-44v-8VGN-Z798eM/view?usp=sharing
- Another recording for coherent breath: https://drive.google.com/file/d/1qxTKi70BHBY2sWtpaXHrM0XTAhESJ-QM/view?usp=sharing
- Breath-Body-Mind: https://www.breath-body-mind.com/
- Dan Siegel Hand Model of the Brain: https://youtu.be/f-m2YcdMdFw
- Watch Ted Talk: https://www.ted.com/talks/kwame_christian_finding_confidence_in_conflict?utm_campaign=tedspread&utm_medium=referral&utm_source=tedcomshare

Compassionate Curiosity 3-part Framework:

1. Acknowledge and Validate feelings
    a) Acknowledge: "It sounds like…." or "It seems like…."
    b) Validate: "That makes sense"

2. Getting curious with compassionate tone
    a) "Out of curiosity…."
    b) Use open ended questions

i) Who
ii) What
iii) Where
iv) When
v) How
vi) **NOT** Why ("Why" can sound judgmental)

3. Joint problem solving
    a) You have something to contribute to solving problem and so do I
    b) Collaboration builds commitment

4. Use these three steps for internal conflict as well
    a) Acknowledge and validate your own feelings
    b) Become curious with the reason the is driving your feelings. What is it that is actually bothering you?
    c) Joint problem solve by including both (a) and (b) in your next steps

- Christian, K. (n.d.). *Finding Confidence in Conflict: How to Negotiate Anything and Live Your Best Life.*

- **Non-Violent Communication:**
    a) Feelings Inventory
    b) Needs Inventory

- Feelings Wheel
- https://howwefeel.org/

What's Your Number? Understanding Financials to Reach Your Goals

Missy Tasky, DVM, Medical Team Coach, Blue Heron Consulting

If someone has financial security, it means they have the ability to pay their bills. If someone has financial independence, it means they work because they want to, not because they have to. Do you know how to achieve financial independence? Do you know when you want to get there? Do not miss the opportunity to let Blue Heron Consulting show you the way.

VETERINARY NURSE PROGRAM

PROCEEDINGS

Saturday, April 29, 2023

UTCVM INSTITUTE OF AGRICULTURE THE UNIVERSITY OF TENNESSEE

A Chicken Just Walked in Through the Front Door, Now What?

Janet L. Pezzi-Jones, LVMT

Abstract

Chickens have become a popular backyard pet. Many cities have now allowed people to keep chickens in their backyard. It has become more common for the veterinary practice to have a chicken present for illness or emergency. A chicken is not triaged any differently than any other domestic animal. It is important to understand what is normal, including behavior, before being able to identify what is abnormal. The history is the most important starting point. The physical exam starts with visual observation, then with gentle restraint, start with the head and work your way to the back end. Treatments will vary depending on if the bird is for egg production, meat production, show, or as a pet only.

Chicken Diseases – What the Backyard Chicken Owner Needs to Know

Janet L. Pezzi- Jones, LVMT

Abstract

When chickens present at the clinic, they are often showing general signs of disease. A compete and through history are the most important first step in diagnosis of the patient. Respiratory diseases are the most common diseases of poultry with non-specific respiratory disease being extremely common. Other very common presentations are trauma and husbandry issues. Other conditions range from musculoskeletal, dermatologic, gastrointestinal, reproductive and zoonotic diseases.

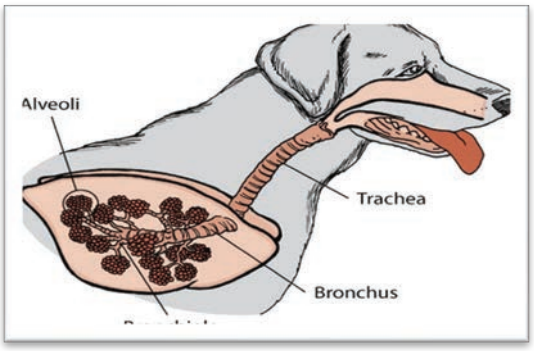
The Anesthetist's Guide to Capnography

Alyssa Stair, LVMT, VTS (Anesthesia & Analgesia)

Capnography is a noninvasive, continuous, and practical method of monitoring CO2 levels in patients, without the need to catheterize an artery. EtCO2 closely mirrors PaCO2 (~2-5mmHg < PaCo2).

Anatomy + Physiology

The respiratory anatomy is made up of the lungs, which take in O2 while inspiring and remove CO2 while expiring. The flow of air in and out of the lungs goes through the following anatomy: Pharynx > trachea > main bronchi > bronchi > smaller bronchi > bronchioles > alveolar ducts > alveoli. Within the alveoli is where all the gas exchange happens.



Respiration itself is a process in which O2 is supplied to the lungs and CO2 is eliminated from the tissues. This cycle consists of 3 main events:

1. Cellular metabolism

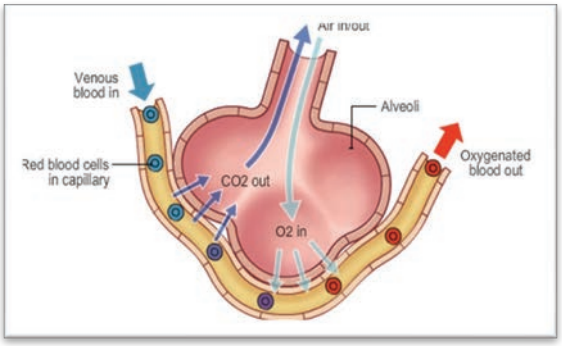
CO2 is created as a byproduct of anaerobic metabolism. With that in mind, we can appreciate that an increase in metabolism = increase in CO2 production, and a decrease in metabolism = decrease in CO2 production.

2. Transport of CO2

Majority of CO2 is converted to bicarbonate ions (60-70%), some CO2 is bound to hemoglobin (20-30%), and the remainder is dissolved into plasma (5-10%)

3. Ventilation

CO2 is carried by the RBC to the lungs, where it crosses the blood-gas barrier and diffuses into the alveolar gas. This barrier consists of the alveolar wall, interstitial fluid, and pulmonary capillary endothelium.



Vocabulary

Capnometry: measurement and display of CO2 in numeric form only
Capnography: measurement and display of CO2 and waveform (capnogram) of ETCO2

Benefits:

- Assessment in airway integrity
- Confirms tracheal intubation
- Aids in prevention of hypoxia
- Shows efficacy of CPR
- Able to assess multiple complications from mechanical to cellular

How It Works

- Absorption of infrared light by carbon dioxide molecules
- Absorbs any gas with at least more than two different molecules
- Not only CO2, but also inhalants
- Infrared light beam passes through gas sample
- Electronic signal is obtained by a photodetector, which measures remaining light energy
- That measurement is then compared to the original energy of the infrared light

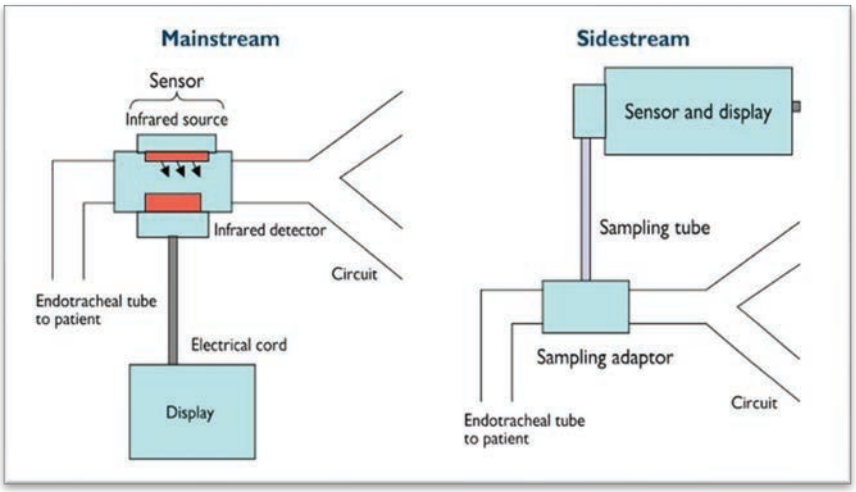
Types of Capnometers

Mainstream:

- Sensor chamber placed directly between patient and breathing circuit
- **Benefits:** immediate readings (no delays), no removed air for sampling, IR sensor cannot be contaminated by patient secretions
- **Disadvantages:** heavy/bulky, heats up to prevent condensation, risk extubation/kink in tube

Sidestream:

- Sensor chamber is located in the computerized monitor
- Air is pulled through attached sampling line placed between the patient and breathing circuit
- **Benefits:** lightweight, small, little deadspace
- **Disadvantages:** slight delay in readings, large amount of sampled gas taken, contamination of IR sensor



Capnographs

Phases of Capnometry:

A-B: Baseline

B-C: Expiratory Upstroke

C-D: Expiratory Plateau

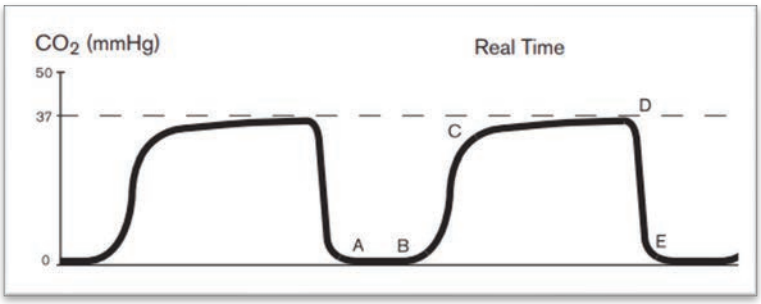
D: End-Tidal Concentration

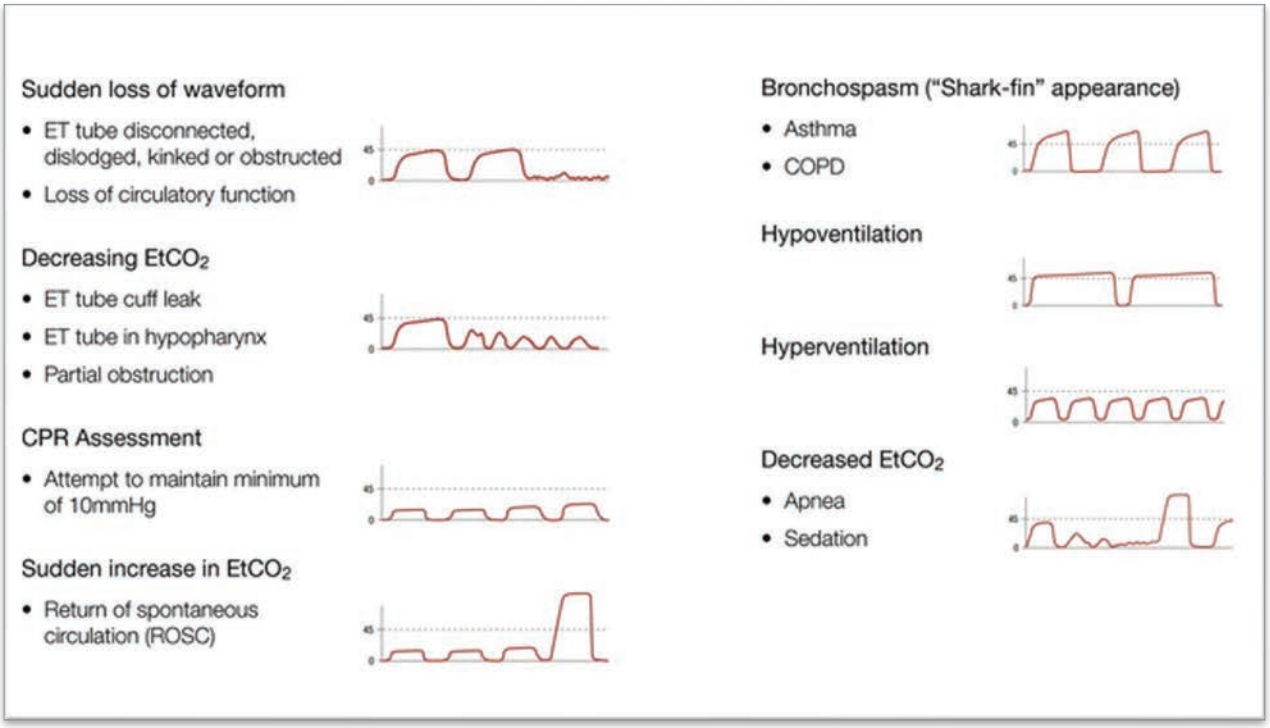
D-E: Inspiration

Normal EtCO2: 35-45 mmHG

EtCO2 < 35mmHg = Hypocapnia

EtCO2 > 45mmHg = Hypercapnia





The Art of Bandages: Do's and Don'ts

Jessica Wiley Montoya, LVMT, CCRP, Small Animal Orthopedics

Any coaptation is an art form. It takes properly trained personnel to place an appropriate bandage to avoid morbidity to the limb(s). Each person involved when placing coaptations plays an important and vital role. Proper planning prevents frustrations among team members as well as avoids multiple administrations of sedatives to the patient during placement. Most importantly at the end of placing these the patient should be comfortable and ambulate without any difficulties.[1,2]

Generalities

Patient Preparation:

Gathering the necessary supplies ahead of time will help the process move along smoothly, in addition to avoiding any frustration that may come along with placing the coaptation itself. Never place clean bandaging supplies on a floor or counter surface, as it provides a venue to introduce bacteria into the affected area, especially if there are open wounds.

It's important to have a clean tray with all of your supplies gathered in one place.[2] Ensure that the tray is wiped clean using 70% isopropyl alcohol and waft in the air to all complete drying before placing the materials. *Important note:* If you know that the patient is going to be returning for several bandage changes, the recommendation is to place any remaining wound or bandage supplies in a clear container labelled with the patient's name and date in which the items were opened. Never use expired products on patients.[2] Before beginning the placement of the coaptation either have the digital radiographs up on a computer screen so that you can refer back to them or have the radiographic films up on the view box in the area where you are placing the bandage.

Supplies:

Most bandages require the same starting supplies. It's better to get multiple items of each material than not enough and become frustrated while looking for more. The basic supplies for any coaptation are 1 in zonas tape, roll cotton, conform cling gauze, Vetrap® and a porous waterproof tape or Elasticon.[2,3]

Robert Jones bandage:

A true Robert Jones bandage is 40-60% the diameter of the other three limbs.[1,3]

Incorporating supplies:

Quick splints, Metaspoon splints, spoon splints, aluminum rods, fiber glass casting tape, or Thermaplast™ can be added to any coaptation after placing the first two primary layers.[3,4] After adding these, an added minimum of two layers of conform cling gauze should be added to secure these in place. Either porous waterproof tape or Elasticon® can be added to the distal edges of the bandage. Lastly, the bandage should be covered with Vetrap® with a 50% overlap of each layer placed. Vetrap® should never cover the digits. If the limb swells and the digits are covered there is nowhere for the digits to go except through the Vetrap®. This can lead to a cascade of events with digits and limb, in addition the owner cannot monitor the limb or digits for swelling.

Coverings:

Bandages should always be kept clean and dry. In order to achieve this an Elizabethan collar should be worn at all times until the bandage is permanently removed. Dogs with coaptation should have it covered when taking outdoors either with a plastic bag, Mediboot, Medipaw, Ziplock bag, etc. and removed upon reentry.[4] It's imperative to tell the owner to remove this once indoors. Failure to do so will inevitably lead to moisture buildup within the bandage and could lead to bandage disease and other complications. In cats, checking their bandage after they return from the litter box is important. Any bandage that is wet, dirty, falling off or compromised should be removed and replaced.

Final remarks:

Poor placement of any coaptation has the potential to endanger the limb, cause vascular compromise, necrosis or bandage disease. If unsure of how to place a coaptation please seek help from your orthopedic surgeon, trained personnel, or from online resources such as books (e.g. Atlas of Small Animal Wound Management and Reconstructive Surgery) or magazines such as Clinician's Brief, Today's Veterinary Practice, or DVM 360. These are all excellent means and have examples and step-by-step instructions of how to place these properly.

References:

1. Pavletic, MM. Basic Princles of Wound Healing, Basic Principles of Wound Management, Common Complications in Wound Healing. In: Pavletic MM. *Atlas of Small Animal Wound Management and Reconstructive Surgery.* Fourth. Hoboken, NJ: Wiley Blackwell; 2018

2. Tobias KM, Johnston SA. *Veterinary Surgery: Small Animal: 2-Volume Set*. Canada: Saunders, an imprint of Elsevier Inc; 2012: *ProQuest Ebook Centeral,* https://ebookcentral.proquest.com/lib/utk/detail.action?docID=1585260.

3. Swaim, Steven F., Walter C. Renberg, and Kathy M. Shike. *Small Animal Bandaging, Casting, and Splinting Techniques.* 1st ed. Ames, Iowa: Wiley-Blackwell, 2011.

4. Millis, Darryl L., David Levine, and Caroline P. Adamson Adrian. *Canine Rehabilitation and Physical Therapy*. 2nd ed. Philadelphia, Pennsylvania: Elsevier, 2014.

Medical Math Boot Camp

Ashley Self, BS, LVMT, VTS (Nutrition)

No need to be intimidated by math! Veterinary nurses are the first line for patient safety and having the ability to calculate medications correctly and in a timely manner provides a great benefit to your veterinary patient(s). As a veterinary nurse, being able to accurately and efficiently calculate the broad range of medical math applications to obtain the correct concentration, dose, rate, and route is part of a gold-standard practice. Our veterinary patients' safety depends on the veterinary nurses and their ability to not only correctly calculate medical math but also catch medical math errors!

Come and strengthen your veterinary nursing skills as it relates to medical math.

*** This session will be interactive and case-based so please bring your phones and/or calculators to work through the math and participate in the friendly competition! ***

We will cover the following topics:

- Units of measure/converting units
- Dosage calculation (drugs/fluid additives)
- Percent solutions
- Reconstitution of medications
- Constant rate infusions (CRI)
- Feeding calculations (RER, MER)

Quick Reference: Common Conversions Among Systems of Measurement

UNIT OF MEASUREMENT	APPROXIMATE EQUIVALENT(S)
1 teaspoon	1 teaspoon = 60 drops 1 teaspoon = 5 mL
1 tablespoon	1 tablespoon = 3 teaspoons 1 tablespoon = 15 mL
1 fluid ounce	1 fluid ounce = 2 tablespoons 1 fluid ounce = 30 mL
1 ounce (weight)	16 ounces = 1 pound 1 ounce 30 g
1 cup	1 cup = 8 ounces 1 cup = 16 tablespoons 1 cup = 240 mL
1 pint	1 pint = 2 cups 1 pint = 480 mL
1 quart	1 quart = 2 pints 1 quart = 4 cups
1 gallon	1 gallon = 4 quarts 1 gallon = 8 pints 1 gallon = 3,785 mL
1 pound 1 mL	1 pound = 16 ounces 1 pound = 480 g 15-16 drops
1 kilogram	2.2 pounds
1 kilogram	1,000 grams
1 gram	1,000 milligrams (mg)
1 mg	1,000 micrograms (mcg)

Nutrition for the Inappetent Patient: Energy Calculations, Diet Selection, Slurry Preparation & Feeding Tubes Wet Lab

Ashley Self, BS, LVMT, VTS (Nutrition)

Come to this session ready to jump into nutrition! Nutrition is a great place for veterinary nurses to be heavily involved in the creation of a feeding plan and the follow-up. We will discuss different enteral feeding devices, how to perform the best nutrition assessment to help evaluate how to initiate nutrition support, understanding when to intervene, selecting diets, creating slurry-diets, and calculating the math to determine feeding guidelines. This will be interactive and have some cases to discuss. Bring your calculator and/or phone to work through creating feeding plans for these inappetent patient(s).

Selecting a Diet for Nasal Tubes

Liquid diets are available from both the human and veterinary market. When using a nasal tube, liquid diets are the only option due to the small diameter of the feeding tube. These diets are generally well tolerated and when targeting appropriate calorie goals facilitate weight maintenance while hospitalized. While some veterinary liquid diets are formulated for maintenance, most commercially available liquid diets are not complete and balanced, meaning they do not meet every nutrient need of the dog or cat. Since assisted feeding with a nasal tube generally does not exceed 7 days, using these diets will likely not result in clinically significant nutritional deficiencies. However, when using human enteral products in cats, note that these products may not have sufficient levels of protein, more specifically, taurine required to support maintenance feeding. For many veterinary patients, their disease will limit the dietary choices that can be incorporated into their plan, therefore, there are several veterinary liquid diets targeting specific conditions.

Nasal Tube Feeding Case Example:

Patient History:

5-year-old, female (spayed), mixed breed canine, BCS 5/9 (estimated 20 - 25% body fat), adequate muscling, 21.8 kgs (48 lbs.), 3-day history of anorexia, hospitalized for pancreatitis. Nasogastric tube placed to provide supplemental nutrition support while hospitalized.

RER at current body weight (kg):

$70\ (21.8)^{0.75}$ = 706 kcal per day

Diet selection:

Royal Canin Gastrointestinal Low-Fat Liquid - Canine – 8-ounce bottle

0.9 kcal/mL

*Prior to initiating a feeding plan, consider evaluating electrolytes associated with refeeding syndrome (magnesium, phosphorus, potassium) to determine if supplementation is warranted prior to the introduction of diet.

Feeding Plan:

Day 1:

33% Current BW RER: 233 kcal/day = (233 kcal ÷ 0.9 kcal/mL) = 259 mLs/day

CRI rate (24 hours) = 10.8 mL/hour ~ 11 mL/hour

*Consider evaluating electrolytes associated with refeeding syndrome (magnesium, phosphorus, potassium) 12- 24 hours after the introduction of food to determine if supplementation or diet adjustment is warranted.

Day 2:

66% Current BW RER: 471 kcal/day = (471 kcal ÷ 0.9 kcal/mL) = 523 mLs/day

CRI rate (24 hours) = 21.8 mL/hour ~ 22 mL/hour

Day 3 and beyond (until NG tube removal):

100% Current BW RER: 706 kcal/day = (706 kcal ÷ 0.9 kcal/mL) = 784 mLs/day

CRI rate (24 hours) = 32.7 mL/hour ~33 mL/hour

Quick check:

< 1 kcal/mL energy density, should be feeding more mLs per day than calories per day. If > 1 kcal/mL energy density, should have more calories per day than mLs per day.

Selecting a Diet for Esophageal Tubes

A major advantage to E-tubes is the ability to use commercially available canned products, generally, allowing for targeted nutrient profiles supporting a variety of needs. These canned diets can be made into blenderized slurries by use of additional water or enteral liquid products. It is necessary to experiment with different water/liquid ratios to achieve the desired consistency that will easily flow through the selected E-tube size. This will help to avoid tube obstructions and aid in calculating the most calorically dense slurry, limiting overall volume necessary to meet the patient's caloric demands.

Esophageal Tube Feeding Case Example:

13-year-old, female (spayed), standard poodle, 5/9 BCS (estimated 20-25% body fat), mild muscle wasting, 25 kg (55 lbs.), hospitalized for hyporexia and chronic kidney disease, IRIS stage II, normotensive, non-proteinuric.

RER at ideal body weight (kg):

$70\ (25)^{0.75}$ = 783 kcal per day

MER at ideal body weight (kg):

Maintenance energy needs can be compared to prior caloric intake of the patient when eating and maintaining an ideal weight or estimates can be quantified based upon common life stage factors.

MER = RER * life stage factor

(783 * 1.5) = 1,175 kcal/day

Slurry Calculation – using water:

Diet Selection:

Royal Canin Veterinary Diet Canine Renal Support T (Tasty), canned, pate

13.5 oz. can (385 grams), 596 kcal/can

Option 1:

1 gram = 1 mL

1 can RC Renal T 385 grams = 385 mL

Option 2 (if grams per can is NOT available):

1 ounce = 30 mL

1 can RC Renal T 13.5 oz. = 13.5 ounces * 30 = 405 mL

Place an entire can of RC Renal T into blender.

Add 180 mLs water into blender.

Blend until smooth.

Contents in Blender	
(1) can RC Renal T	Water
385 mL	180 mL
596 kcal	0 kcal

**Water amounts added to create a slurry will vary based upon the texture/thickness of the pate and stew canned products. It is recommended to test blend diets to determine best water volume addition for each new slurry type.*

Determine energy density of blender components:

596 kcal ÷ 565 mL = 1.05 kcal/mL = 1.1 kcal/mL

Slurry Calculation – using enteral product**:**

Diet Selection:

Royal Canin Veterinary Diet Canine Renal Support T (Tasty), canned, pate

13.5 oz. can (385 grams), 596 kcal/can

Royal Canin Veterinary Renal Support Liquid Canine Diet, bottle

8-ounce bottle (237 mL), 1.3 kcal/mL

237 mL * 1.3 kcal/mL = 308 kcal/bottle

Option 1:

1 gram = 1 mL

1 can RC Renal T 385 grams = 385 mL

Option 2 (if grams per can is NOT available):

1 ounce = 30 mL

1 can RC Renal T 13.5 oz. = 13.5 ounces * 30 = 405 mL

Place an entire can of RC Renal T into blender.

Place an entire bottle of RC Renal Support Liquid into blender.

Blend until smooth.

**For ease of measure, consider using ½ bottle or 1 bottle of the added enteral product. Final additive amount based upon desired slurry consistency.*

Contents in Blender	
(1) can RC Renal T	(1) bottle RC Renal Support
385 mL	237 mL
596 kcal	308 kcal

Determine energy density of blender components:

595 kcal from RC Renal T pate + 308 kcal from RC Renal Support Liquid = 903 kcal

385 mL from RC Renal T pate + 237 mL from RC Renal Support Liquid = 622 mL

904 kcal ÷ 622 mL = 1.45 kcal/mL = 1.5 kcal/mL

Feeding Plan:

Day 1:

50% ideal BW RER: 392 kcal/day

RC Renal T + water = 392 kcal/1.1 kcal per mL = 356 mL per day

RC Rental T + RC Renal Support Liquid = 392 kcal/1.5 kcal per mL = 261 mL per day

Tube feed every 6 hours (QID):

RC Renal T + water = 392 mL/4 = 98 mL per feeding

RC Renal T + RC Renal Support Liquid = 261 mL/4 = 65 mL per feeding

Day 2:

100% ideal BW RER: 783 kcal/day

RC Renal T + water = 783 kcal/1.1 kcal per mL = 712 mL per day

RC Rental T + RC Renal Support Liquid = 712 kcal/1.5 kcal per mL = 475 mL per day

Tube feed every 6 hours (QID):

RC Renal T + water = 712 mL/4 = 178 mL per feeding

RC Renal T + RC Renal Support Liquid = 475 mL/4 = 114 mL per feeding

Day 3:

1.3 * RER at ideal BW: 1,018 kcal/day

RC Renal T + water = 1,018 kcal/1.1 kcal per mL = 925 mL per day

RC Rental T + RC Renal Support Liquid = 1,018 kcal/1.5 kcal per mL = 677 mL per day

Tube feed every 6 hours (QID):

RC Renal T + water = 925 mL/4 = 231 mL per feeding

RC Renal T + RC Renal Support Liquid = 677 mL/4 = 169 mL per feeding

Day 4 and beyond:

100% ideal BW RER: 1,175 kcal/day

RC Renal T + water = 1,175 kcal/1.1 kcal per mL = 1,068 mL per day

RC Rental T + RC Renal Support Liquid = 1,175 kcal/1.5 kcal per mL = 783 mL per day

Tube feed every 6 hours (QID):

RC Renal T + water = 1,068 mL/4 = 267 mL per feeding

RC Renal T + RC Renal Support Liquid = 783 mL/4 = 196 mL per feeding

Upon patient discharge, the feeding schedule may require adjustments to support three meals per day (TID) versus four meals per day (QID), to ease owner commitment, if the pet can tolerate the increased feeding volumes.