FOREWORD

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Again, thank you to our speakers and our sponsors.

Kathleen Norman, DVM
2017 Conference Chair
Ontario Veterinary Medical Association
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SMALL ANIMAL PROGRAM
Metabolic Nightmares
Tricks for Managing Acute Renal Failure and Diabetic Emergencies

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Appropriate management of acute renal failure and diabetic emergencies requires that appropriate attention be paid to the patient’s metabolic abnormalities. This lecture will discuss treatment of these two diseases with a focus on how understanding the electrolyte and acid-base abnormalities can lead to decreased morbidity and mortality.

Chloride
Chloride values must be corrected to take into account any changes in plasma free water before assessing whether or not the chloride concentration is low, normal or elevated. This is calculated by the following formula:

\[ [\text{Cl}^-]_{\text{corrected}} = [\text{Cl}^-]_{\text{measured}} \times \frac{156}{[\text{Na}^+]_{\text{measured}}} \]

The number of 156 is used in cats under the assumption that 156 mEq/L is the mean sodium concentration in cats. The number 146 should be used in dogs. This corrected value can have a significant impact on the choice of fluid therapy in certain conditions. For instance a ketoacidotic diabetic cat with a sodium of 140 mEq/L and a chloride of 118 mEq/L may become even more acidotic if 0.9% saline is infused. The corrected chloride in this situation is 131 mEq/L which is already a contributing factor to this cat’s metabolic acidosis. There is a potential that this will only be worsened if 0.9% saline is infused.

Renal Failure
The primary goals of therapy are the following:
1) normalize renal blood flow, 2) Establish urine output, 3) Normalize electrolytes and acid-base status, 4) Diagnose the cause of the real failure and provide specific treatment, 5) Provide nutritional support

Mean arterial blood pressure must be maintained at greater than 65 mm Hg to ensure renal perfusion. Systolic pressures less than 110 mm Hg can be associated with mean arterial pressures less than 60 mm Hg. Blood pressure can be normalized in most patients using fluid therapy. In order to ensure blood volume (preload) is optimized a jugular catheter should be placed so that central venous pressure can be measured. If a jugular catheter is not an option then the jugular veins should be clipped and carefully assessed.

Fluid therapy must be closely tailored to the animals needs and should be based on cardiac status, volume status, dehydration, sodium, potassium, albumin, and type of renal failure (polyuric, oliguric or anuric). Dehydration should be restored using crystalloids. Patients who are hypernatremic may require fluid with low sodium concentration or even no sodium. Patients who are hyponatremic may be volume overloaded and both sodium and fluid rates need to be monitored carefully. Ongoing losses in patients with polyuria can be significant and urine output should be measured. Anuric patients may not even tolerate the amount of fluids required to restore dehydration until urine output is restored. If diuresis is the end goal then fluid will need to be adjusted to ensure urine output is greater than 2 ml/kg/hr.
Urine output in hypovolemic or hypotensive patients hopefully will be restored once perfusion is improved. If the patient is not responding several options are available. Mannitol is a hyperosmotic agent that causes an osmotic diuresis, which flushes the tubules and it has benefits as a free radical scavenger. It is dosed at 0.25 g/kg over 15-20 minutes. This dose can be repeated if it is effective and it can be followed with a constant rate infusion at 1 mg/kg/min until it is no longer effective. It should be used with extreme caution in patients that might be oliguric or anuric. Dextrose at 5% -20% concentrations can be used as an osmotic diuretic if mannitol is not available. The dextrose will be metabolized ultimately making this perhaps a safer choice than mannitol.

Furosemide is a loop diuretic that is most effective when given as a constant rate infusion. A bolus dose of 1-2 mg/kg can be given intravenously followed by a constant rate infusion of 1 mg/kg/hr. If there is no response after 4 hours it is less likely to be effective.

Dopamine at dopaminergic doses (0.5-3 mcg/kg/min) is designed to impact dopaminergic receptors in the kidneys and improve renal perfusion and thus renal function. There is no clear evidence this occurs. The cat lacks the appropriate dopaminergic receptors in the kidneys and one study showed no improvement in urine output, sodium excretion or glomerular filtration in cats given dopamine.

Patients in renal failure may have significant acid-base abnormalities. Acidemia related to perfusion abnormalities should correct once perfusion is restored. Dogs and cats with chronic renal failure often lose large amounts of bicarbonate in their urine due to kidney dysfunction. This is more common in the cat than the dog. These cats must be supplemented with bicarbonate or their values will never return to normal. In the author’s experience these cats often require much higher doses of bicarbonate than is typically recommended. In addition the anorexia and nausea seen in cats often improves once the acidemia is improved. These cats often will require oral supplementation to maintain normal acid-base status. Bicarbonate is typically supplemented at 0.3 x kg body weight x deficit (18- measured bicarbonate level) with ¼ of this dose given over 1 hour and the remainder over the next 12 hours. If blood gas assessment is readily available this dose can be given more quickly. If blood gases are not available but total carbon dioxide levels are the bicarbonate can be estimated by the total carbon dioxide minus 1. Bicarbonate supplementation should not be provided if measured levels are not available since the side effects can seriously outweigh the benefits.

Patients in anuric renal failure will develop hyperkalemia. Hyperkalemia should be treated if it is lifethreatening as determined by evidence of significant electrocardiographic abnormalities in combination with clinical signs. Ultimately unless the animal is able to urinate the hyperkalemia will persist. If anuria persists dialysis is indicated.

Vomiting can be a substantial problem in patients with renal failure. Placement of a nasogastric tube permits decompression and nutritional support. Uremia triggers vomiting centrally and central acting antiemetics are likely to be the most effective at controlling vomiting. Uremia can be associated with gastric erosions and ulcerations. Administration of an H-2 blocker or a proton pump inhibitor and sucralfate may be indicated and are definitely indicated if there is evidence of hematemesis. Enteral nutrition has been shown to be as effective as, if not more effective than, antacids at preventing gastric ulceration. Oral ulcers are not uncommon in patients with renal failure. Oral rinses using 0.005% chlorhexidine can help prevent infection. Rinses with topical anesthetic agents have also been used anecdotally by the author to try and minimize the pain associated with the ulcers.
Nephrotoxic drugs should be discontinued and specific treatment aimed at the cause of the renal failure should be instituted as soon as possible. Hyperphosphatemia may resolve with fluid therapy; however if the hyperphosphatemia persists then phosphate binders should be administered orally once the patient is eating.

Patients with chronic renal failure may be hypertensive. Amlodipine, a calcium channel blocker, has been found to be the most effective in treating hypertension caused by renal disease. Blood pressures greater than 200 mm Hg systolic can lead to retinal detachment and should be aggressively managed.

Diltiazem has been recommended as an agent to help with renal failure associated with ischemia. It appears to decrease the intracellular calcium levels following ischemia thus decreasing the production of reactive oxygen species. It also prevents apoptosis. The dose recommended is 0.3-0.5 mg/kg over 10 minutes followed by 1-5 mcg/kg/min for 48-96 hours or as long as it takes to decrease the serum creatinine concentration to normal. The dose should be decreased if bradycardia or hypotension develops.

Angiotensin-converting enzyme (ACE) inhibitors (enalapril) are used to treat protein-losing glomerulonephropathy. The drug appears to attenuate glomerular hypertrophy and preserve glomerular function. Angiotensin-converting enzyme inhibitors should be used with caution in the acute stages of renal failure since the drug can decrease glomerular filtration rate and worsen azotemia. It can also cause hypotension and should be avoided in hypotensive patents.

Antibiotics should be administered based on culture and sensitivity results; however, occasionally, antibiotics may need to be administered without confirmation. Prophylactic antibiotics should be avoided in patients with indwelling urinary catheters since these patients are predisposed to infection and the use of antibiotics will lead to microbial resistance.

Analgesics should be provide to all patients showing signs of pain. Nonsteroidal antiinflammatory agents should be avoided. Opioids should be given intravenously to effect and constant rate infusions may be required.

**Diabetic Ketoacidosis**

Diabetic ketoacidosis is a life-threatening complication of diabetes mellitus characterized by metabolic acidosis, hyperosmolality, and electrolyte disorders. These patients have diabetes mellitus and another illness such as a urinary tract infection, pancreatitis, cholangiohepatitis, hyperadrenocorticism, pneumonia or an abscess leading to an increase in diabetogenic hormones. The insulin deficiency and insulin resistance leads to a significant cellular energy deficit. In an attempt to keep up with energy requirements the ensuing lipolysis leads to excessive hepatic production of ketoacids. The metabolic acidosis is primarily related to the ketoacids but can be complicated by a lactic acidosis that develops secondary to perfusion abnormalities. Ultimately the body’s normal buffer systems are overwhelmed.

Electrolyte disorders include hyponatremia, hypokalemia, and occasionally hypomagnesemia. Hyponatremia may be due in part to the hyperglycemia but is usually compounded by urinary losses as well as the possibility of third spacing into the gastrointestinal tract or peritoneal cavity. Hypokalemia can occur secondary to loss in the urine from the osmotic diuresis as well as vomiting. The presence of hypokalemia in the face of a significant acidosis should alert the clinician to a severe total body potassium depletion which will worsen substantially when insulin therapy is instituted. The electrolyte disorders are compounded by the ketoacids which worsen the osmotic diuresis of a normal diabetic as well as the aggressive fluid therapy that is provided.
during treatment. Hypomagnesemia may develop secondary to increased free fatty acids which bind the magnesium as well as insulin which can shift magnesium intracellularly. Because measurement of serum levels (total magnesium or ionized) may not reflect total body stores, hypomagnesemia should be considered in patients with refractory hypokalemia or hypocalcemia or unexplained weakness or gastrointestinal motility abnormalities.

The diagnosis of diabetic ketoacidosis is made based on the history as well as laboratory findings of hyperglycemia, ketonuria and a metabolic acidosis. Patients that are known diabetics or are ketotic with normal or only mildly elevated blood glucose levels are usually in severe decompensatory shock and mortality rates are high. There are three ketoacids – acetoacetic acid, β-hydroxybutyric acid and acetone. The urine test strips only confirm the presence of acetoacetic acid and acetone as do the routine serum tests. The β-hydroxybutyrate is the most common ketoacid formed in shock states and not until insulin treatment is instituted, which converts the β-hydroxybutyric acid to acetoacetic acid, will the urine dipsticks show a positive result. Blood gas results showing a metabolic acidosis with a severe base deficit but evidence of only a mild or moderate decrease in the bicarbonate concentration are consistent with unmeasured acids such as ketoacids.

The goals of treatment are to restore normal perfusion, provide insulin to ensure the cells are no longer energy deficient and the ketoacids become metabolized, correct electrolyte abnormalities, and treat any underlying additional illness.

Oxygen should be provided to patients in shock. Intravenous fluids (balanced electrolyte solution) should be provided and dehydration should be corrected over 4 to 12 hours unless the patient is at risk for fluid overload in which case hydration may need to be corrected over 24 hours. Normal saline may not be the ideal choice since the hyponatremia will correct as the glucose concentrations normalize which can lead to a hypernatremia and the chloride load can lead to a worsening of the metabolic acidosis. Synthetic colloids may be required if the patient is hypoalbuminemic secondary to concurrent disease process. Insulin therapy is generally begun several hours following initiation of fluid therapy. Constant rate infusions are easier to regulate than intermittent injections but require closer monitoring. Giving insulin into the intramuscular and subcutaneous tissues can lead to unpredictable uptake due to altered perfusion. This can lead to a depot effect followed by a rapid uptake of insulin at some later stage leading to significant rapid decreases in glucose levels. It would be ideal to maintain tight glycemic control with glucose levels ideally maintained between 5.5 and 11 mmol/l (100 and 200 mg/dl) but this usually is not possible. Glucose levels should be maintained below 16 mmol/l (300 mg/dl) and rapid shifts in glucose levels should be avoided in order to minimize the likelihood of causing cerebral edema. Hypokalemia, secondary to intracellular fluid shifts from correction of the metabolic acidosis as well as insulin therapy which drives both potassium and glucose intracellularly, is not uncommon. Hypophosphatemia can develop secondary to dilution, diuresis, and the increased production of ATP as glucose is driven intracellularly and can be a life threatening complication. Requirements for phosphorus supplementation are often much higher than what is recommended. Ultimately nutrition is essential, preferably via the enteral route. Long acting insulin is not recommended until the patient is well hydrated and eating and the ketoacids have been metabolized. Close monitoring of these patients is essential if patient morbidity – and mortality – is to be minimized.

**Hyperglycemic Hyperosmolar Syndrome**

Hypoglycemic hyperosmolar syndrome or hyperosmolar hyperglycemia nonketotic syndrome is an uncommon condition that can be diagnosed in preexisting diabetics and in those who have been never been diagnosed with diabetes. It is characterized by a hyperglycemia of greater
than 33 mmol/l (600 mg/dL) and an osmolality of greater than 350 mOsm/L. Ketonuria is not present. Common clinical findings include severe dehydration, renal dysfunction and brain dysfunction. The disease has a very poor prognosis in large part because congestive heart failure and renal failure are frequently found in association with the syndrome.

The goal of treatment is to reduce the glucose levels slowly to avoid rapid shifts in cerebral osmolality. Idiogenic osmoles have developed over time in the brain to counteract the effects of the hyperglycemia; therefore, if the plasma osmolality is decreased too rapidly cerebral edema will develop. Hyponatremia is commonly found in association with hyperglycemia. This is a normal physiologic response by the body to the hyperosmolality although other causes for hyponatremia must be ruled out. Changes of greater than 1 mEq/hr in the plasma sodium concentration should be avoided so close monitoring of electrolytes (hourly initially) is essential. Fluid therapy should be tailored according to the response of the patient to treatment. The cornerstone of therapy is to increase the glomerular filtration rate to allow the kidneys to filter the glucose. Due to the fact that heart disease and renal disease are often present in these patients, high fluid rates are often not tolerated making management of this condition complicated. If the patient is not tolerant of high rate intravenous fluid therapy, enteral fluid therapy may also need to be initiated. Insulin therapy should be instituted once the patient is rehydrated. The recommended starting dose is 50% of that used for treatment of diabetic ketoacidosis.

**Hypoglycemic Crisis**
Severe hypoglycemia can occur in diabetics that have been overdosed with insulin. The overdose may be an acute problem where the patient is double-dosed inadvertently. It is also associated with an inadequate decrease in the face of decreased food intake or a Somogyi effect. The latter is very common when glucose curves are not being performed and insulin doses are increased based solely on the finding of a single elevated blood glucose level or when changes in insulin dosing are based on laboratory results as opposed to clinical status (i.e., resolution of polyuria, polydipsia and weight loss. In cats it can occur when the animal reverts back spontaneously to being a non-diabetic or a non-insulin-dependent diabetic.

The brain is an obligate user of glucose and has a very poor ability to use other energy sources, nor does it have a significant store of glycogen. This means that neural tissue can be severely and sometime permanently damaged by neuroglycopenia. The degree of damage will depend on the severity of the hypoglycemia as well the duration of the hypoglycemia. Abnormal mentation, seizures and blindness can persist even after resolution of the hypoglycemia.

Hypoglycemic patients should receive an intravenous bolus of 1-2 ml/kg of 25% dextrose. This should be followed immediately with a constant rate infusion of 2.5% to 5% depending on the severity of the hypoglycemia. If seizure activity does not resolve a blood glucose level should be checked immediately since hypoglycemia may not be the sole cause of the seizure activity. A blood glucose level should be checked within 15 minutes to ensure the patient’s glucose has normalized. If it is still low another bolus should be given and the infusion should be increased by 2.5%. This process should be repeated until the blood glucose has normalized. Some patients may require 10% dextrose infusions. Infusions of greater than 5% dextrose ideally should be administered via central lines due to the hyperosmolality. If placement of a central line is not an option then the smallest gauge catheter possible should be inserted. This should improve the blood flow around the catheter relative to one of a larger diameter which should help decrease the likelihood that phlebitis will develop.

References available on request.
Why is nutrition support important in the critically ill or injured patient?
Adequate nutrient intake is necessary to provide energy for cellular function, substrates for
protein synthesis and, vitamins and minerals for daily metabolic processes and maintenance of
homeostasis. Hypermetabolism is characteristic of the acute illness or injury and a catabolic
state rapidly develops. Anorexia associated with severe injury or sepsis has been shown to lead
to: glycogen depletion within 8 to 12 hours leading to muscle weakness, substantially decreased
fibronectin levels within 48 hours of anorexia contributing to immune dysfunction, and
decreased protein synthesis. The deleterious effects of malnutrition are more pronounced in the
acutely ill or injured patient due to the higher metabolic rate necessary to increase liver and
immune function as well as provide substrates for wound healing. Experimental and clinical
research has emphasized the importance of beginning enteral nutritional support as early as
possible to prevent immune system depression, serum albumin decreases, muscle weakness,
bacterial translocation, infection, major organ failure, and death.

How do I decide to feed enterally or parenterally?
Enteral feeding is preferred over parenteral feeding whenever possible as it is more
physiologic and less expensive than total parenteral nutrition. It also avoids the risk of
catheter-related sepsis. When compared with total parenteral nutrition, enteral feeding has
been shown to maintain gut mucosal integrity thus decreasing bacterial translocation,
improve lymphocyte function, improve wound healing and improve survival from peritonitis.
The adage of "if the gut works use it" should be followed as much as possible. During severe
injury or infection gut perfusion may be inadequate and the gut mucosal barrier may become
compromised leading to bacterial translocation. Lack of luminal nutrients leads to mucosal
atrophy, which also enhances bacterial translocation. Early enteral feeding has been found to
blunt the release of stress hormones thus reducing the elevation in metabolic rate. Parenteral
nutrition should only be used if the gut is not accessible or is not functioning adequately. This
includes patients with GI obstruction, peritonitis, intractable vomiting, severe pancreatitis,
short bowel syndrome and ileus. Parenteral therapy has no beneficial effects on the course
of inflammatory bowel disease or chronic pancreatitis and enteral feeding should be
considered as a first line of therapy.

What is meant by parenteral nutrition and how is it administered?
Parenteral nutrition is comprised of total parenteral nutrition (TPN) and partial parenteral
nutrition (PPN). PPN provides a 3% amino acid solution and is usually supplemented with
carbohydrate source to prevent all the amino acids from being used as a glucose source.
TPN provides amino acids, glucose and fat. Both solutions can be given by peripheral
intravenous lines however central lines are preferred for TPN and in both cases lines should
be kept dedicated.

Why should a feeding tube be placed?
Many ill or injured patients are unwilling or unable to eat; yet the gastrointestinal tract is still able
to digest and absorb nutrients. By providing a more palatable diet, the anorectic animal may be
encouraged to eat. By utilizing tube feeding the problems associated with anorexia can be
avoided.
What types of tubes exist?
Tube enteral nutrition in the ICU is provided primarily via nasoesophageal, nasogastric, esophagostomy, gastrostomy and jejunostomy tubes. Nasal tubes can be placed under local anesthesia. Occasionally mild sedation will be required. All other tubes require general anesthesia.

What are these feeding tubes used for?
In general the tubes are used for enteral feeding of the patient. They can be used to deliver electrolyte infusions for replacement or maintenance therapy or liquid diets for nutritional therapy. Radiographs should always be taken of nasal and esophageal tubes to confirm their location prior to their use. All tubes can be used for suctioning purposes, which is especially important in the immediate postoperative period to remove gas and fluid from the esophagus and the stomach to prevent ileus and viscus distension. Decompression is also essential in conditions such as acute megaesophagus and post gastric dilatation-volvulus syndromes. Jejunostomy tubes can also be used for suction if there are concerns about ileus; however, this should be done with caution as the small internal diameter of the tube may cause it to become clogged. Suction can be done intermittently with a syringe or continuously with a thermotic pump. Radiographs are also recommended if the animal vomits, if the tube appears obstructed, or if suctioning is producing much lower volumes of gas or air than expected.

How do I decide what kind of tube to place?
Often the type of tube placed is determined by the patient’s underlying disease, whether or not anesthesia is an option and whether or not abdominal surgery is being performed. In the postoperative patient esophageal or gastric feeding may be contraindicated due to vomiting and higher risks of aspiration in the depressed patient. Gastric motility and absorptive capacity may not return for 1 to 2 days post laparotomy precluding the use of this route of feeding in the early postoperative period. Studies have shown that the small intestine has both motility and absorptive capacity within hours of surgery, and jejunal feedings in human medicine have been shown to be an effective means of providing nutrition in the immediate post-operative period.

How do I take care of these tubes?
Any dressing must be changed as soon as it gets wet, every 24 hours for the first 3-5 days and then a minimum of every 72 hours. All ostomy and incision sites must be examined on a daily basis while the patient is in hospital. If the bandage is still clean and dry this can be done through a window cut in the bandage - creating a 'trap door' that can be replaced when the old dressing is removed. When removing the bandage it is extremely easy to cut the tube; therefore, the bandage should be removed slowly and layer by layer. An indelible marker can be used on the bandage to denote the approximate location of the tube to facilitate removal of the dressing. In general the tube should be looped up over the back of the patient, taking care not to kink the tube at the ostomy site. If a small bore tube is cut it can often be salvaged by placing a hypodermic needle into the cut end after using hemostats or needle holders to break off the needle tip with a bending motion. The needle is taped carefully into the tube and an extension set attached to the hub.

The ostomy site should be cleaned with a dilute (0.05%) chlorhexidine solution and examined closely for signs of discharge or inflammation. The area around the tube should be thoroughly palpated to check for any signs of swelling or crepitus. An antibacterial ointment should be placed over the ostomy site and the bandage replaced.

When do feedings start?
Early enteral feeding has been shown to be beneficial and should start as soon as possible—generally within the first 12 hours. If a jejunostomy tube has been placed then postoperative feeding can be started in the immediate postoperative period—as soon as the patient is normothermic and hemodynamically stable. If an esophagostomy or gastrostomy tube is being used the patient should have full control of its airway before feeding is started. Feeding into the stomach should be performed with caution in postoperative patients, as gastric motility often does not return for 1-2 days.

What should I feed?
Only liquid diets can be fed through small bore tubes (less than approximately 8 French). Blenderized canned foods can be fed through larger bore tubes; however, it is recommended that liquid be used initially until it has been determined that the patient is tolerating the food. The choice of diet may depend on the patient’s underlying disease but in most cases feeding the patient is more important than what is being fed.

How fast should the liquid be delivered?
Initially the delivery rate should be 0.125-0.25 ml/kg/hr. This can be used in all patients including those who have had gastrointestinal surgery. These rates may even be too fast for those patients who have had massive bowel resection, severe pancreatitis or prolonged anorexia. If the patient is tolerating the rate of delivery the rate can be increased as frequently as every 8-12 hours by increments of 30% until 100% of the required volume is being delivered. The volume of feeding required is calculated using the basal energy requirement (BER) in kcal for the weight of the animal and knowledge of the caloric density of the food. A commonly used linear formula for BER for adult dogs is \(30 \times \text{kg} + 70\). BER Some references suggest this number should be multiplied by stress factors depending on the disease state: 1.25 for mild stress such as cage rest, postoperative recovery from minor surgery, 1.5 for moderate stress such as trauma, postoperative recovery from major surgery, and 2.0 for severe stress such as third degree burns and sepsis. Clinical studies suggest these stress factors may significantly overestimate the caloric intake required for a patient in a cage. In general it is unlikely that most veterinary patients will require more than 100 to 125 per cent of BER. Overfeeding should be avoided since this can lead to increased carbon dioxide production and potentially to ventilatory stress. Intravenous fluid rates should be decreased in proportion to the amount being delivered enterally. Cats often will not tolerate greater than 20 ml/kg per feeding when being fed intragastrically.

What are the exceptions to the above delivery rates?
If a patient has been anorectic for longer than a few days the lower rate of 0.125 ml/kg/hr may be better tolerated as the stomach, duodenal and pancreatic atrophy that may have occurred during this time can be significant. The atrophy is associated with down regulation of the digestive enzyme systems and decreased absorptive surface area in the small intestine. The length of time the bowel takes to return to normal will vary depending on how long the patient has been anorectic. If a large percentage of the small bowel (generally greater than 80% of the small intestine) was removed at surgery the animal may suffer from "short bowel syndrome" and increases in rates may need to be adjusted even further as the absorptive surface area will have been significantly reduced.

What about microenteral nutrition?
Microenteral nutrition is the delivery of small amounts of 0.05 to 0.125 ml/kg/hr of a glucose and electrolyte solution into the gut. This is provided for the patient who is fairly intolerant of oral nutrition e.g. the vomiting patient who is continuing to vomit intermittently, pancreatitis patients who are no longer vomiting, the patient with severe esophageal injury and patients with short
bowel syndrome. This form of nutritional support will hopefully help "feed the gut", and "wake it up" functionally and help prevent further down regulation of the enzyme systems.

**How do I deliver the feedings?**

All diets should be fed at room temperature. Most patients tolerate a constant rate infusion much better than bolus feedings. Constant rate infusions may alter homeostasis and fuel use as it does not allow for the normal cascade and feedback mechanisms to occur. Enteral feeding pumps are available although some of the diets can also be given via the regular fluid infusion pumps. Eight hours worth of feeding is placed in an administration bag and the bag labeled with the product name, date, time of hanging and initials of person hanging the bag. All patients must have their tubes flushed with 2 to 5 ml of warm water (depending on the size of the tube) every 6 hours. If the patient is going to be tube fed at home a gradual switch to bolus feedings must be done, ideally over a period of 2 to 3 days. The first day the hourly feedings are delivered over 5 minutes. If this is being tolerated then the amount of the feeding can be doubled in 12 hours and the feeding given every 2 hours. The second day the amount is increased and the frequency decreased to every 3 to 4 hours. (Due to volume restrictions it is rare to be able to deliver the required number of calories in fewer than 6 to 8 feedings per day if total caloric requirements are being met by tubes requiring liquid diets.) Bolus feedings should always be given over at least 5 minutes. After a bolus feeding an attempt should be made to hold a column of water in the tube. This is done by flushing the tube and clamping the tube by kinking it prior to removing the syringe. The syringe is removed and the cap placed prior to unkinking the tube. Hypodermic needle caps make excellent caps for most larger bore feeding tubes.

**How do I tell if the diet is not being tolerated?**

If the patient starts coughing discontinue feeding, administer oxygen if required, and take a chest radiograph. The patient may show signs of abdominal discomfort, nausea, hypersalivation, vomiting or diarrhea. On occasion gastric feedings will reflux into the esophagus and jejunal feedings will reflux into the stomach and be subsequently vomited. Gastrointestinal motility modifying drugs may be required (i.e., metoclopramide). If none of these signs discussed is observed then the patient is believed to be tolerating the administration well.

**What about enteral feeding in the face of pancreatitis?**

Traditionally TPN has been used for feeding patients with pancreatitis. A number of studies have looked at enteral feeding in the face of pancreatitis. The presence or absence of ileus is a good predictor of tolerance for enteral nutrition. The problem with enteral feeding may not be the stomach. Jejunal feeding has been recommended since there is minimal effect on pancreatic secretion in patients (in laboratory dogs) fed intrajejunally. More recent studies in humans suggest that intragastric feedings may be used as long as the patient tolerates the diet and there is no worsening of the clinical signs. There is no difference in outcome from pancreatitis with bowel rest except perhaps decreasing pain and nutritional support may decrease morbidity and mortality in patients with severe pancreatitis.

**What are common complications and how do I prevent them?**

1. **Kinking:** This usually only happens with small diameter tubes and is the first thing that should be checked if the feeding solution is not flowing. Careful looping of the tube and ensuring encircling sutures are not too tight should minimize this complication.

2. **Clogging:** When blenderizing diets, the consistency should flush easily through a tube of similar diameter. All tubes should be flushed every 6 hours with warm water to prevent buildup of the diet on the sides of the tube. If clogging does occur tubes can be usually be unclogged.
using water, Diet Coke, or meat tenderizer. Flushing with small syringes (tuberculin) builds up higher pressure than larger syringes. The passage of an angiographic wire down the lumen may be needed to unclog the tube.

3. Infection: Signs of inflammation with or without discharge or fever may indicate an early infection. This must be differentiated from fasciitis since a simple wound infection can usually be treated locally with gentle cleaning with an antibacterial solution, use of topical antibacterial ointments and more frequent dressing changes. The application of warm compresses may also be recommended. The systemic administration of antibiotics should be reserved for patients with systemic signs of infection.

5. Tube dislodgement: This is most common with nasal tubes. An Elizabethan collar may be required. Nasal or esophageal tubes that pass through the lower esophageal sphincter in cats are more likely to trigger vomiting and should be backed out to just caudal to the base of the heart (ninth intercostal space.) Surgically placed tubes should be covered with a light bandage to prevent the animal from scratching or chewing at the tube. Gastric or jejunal tubes that are dislodged prior to a seal forming at the ostomy site can cause peritonitis.

9. Diarrhea: Studies in human medicine have shown diarrhea to be a major complication often necessitating discontinuing enteral feedings. Diarrhea is usually defined as the presence of 3 or more liquid bowel movements per day. The incidence of diarrhea in human patients is decreased if the diet fed is isotonic, lactose free and is delivered by a constant infusion rather than bolus feedings. Feeding through acute diarrhea in people has shown better maintenance of mucosal barrier. Rice-based oral rehydration solutions decrease stool volume relative to glucose based oral rehydration solutions as the glucose and peptides in rice provide the substrates for electrolyte pumps thus improving water absorption. The true incidence of diarrhea in veterinary patients is unknown.

10. Refeeding syndrome: Refeeding syndrome is usually thought of as the severe hypophosphatemia and its associated complications that occurs in malnourished patients receiving aggressive nutritional support. It is characterized by acute cardiopulmonary decompensation leading to death. Refeeding leads to fluid retention, increases in heart rate and blood pressure and oxygen consumption that may cause the demands on the heart to exceed supply, increased carbon dioxide production leading to respiratory distress, Central nervous system dysfunction including seizures, diarrhea, red blood cell dysfunction and leukocyte dysfunction. The rapid hypophosphatemia is in response to a rapid intracellular shift due to the demands for phosphorylated compounds and increased insulin activity, which promotes uptake of phosphorus.

Remember no critical animal has ever benefited from acute malnutrition - only the opposite - therefore, feed early, and increase gradually, and most nutritionally related complications will be avoided.
RESPIRATORY EMERGENCIES

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Not infrequently dogs and cats present with signs of respiratory distress. This can be a primary respiratory problem or can be secondary to another problem such as congestive heart failure or a diaphragmatic hernia. This lecture will provide an overview of how to diagnose the problem as well as emergency care of the patient presenting with some of the more common respiratory emergencies. Due to the scope of this topic the reader is referred to other sources for more detailed information about individual disease processes.

Physical Examination

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

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

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

The breathing pattern should be closely observed. Symmetry of chest movement and the presence of any abdominal component to the breathing pattern should be noted. Rapid shallow respiration typically is associated with pain (especially related to chest wall trauma) or pleural space disease where the patient is unable to expand its lungs. Pneumothorax, hemothorax, chylothorax, and pyothorax all can be associated with a restrictive breathing pattern. If paradoxical chest wall movement is observed a flail chest should be suspected. Increased chest wall expansion often is associated with lower airway disease although can indicate a collapsing trachea. Prolonged or forced expiration is associated with trapping of air in the lower airways such as occurs with allergic bronchitis or other diseases causing bronchospasm. Respiratory muscle abnormalities are associated with a significantly increased effort on inhalation with decreased chest wall expansion. This is most commonly seen with neuromuscular diseases and diaphragmatic hernia. Patients with paradoxical abdominal movement have severe respiratory compromise. These patients also should be assumed to be close to collapse due to exhaustion and ventilatory failure until proven otherwise.
The chest should be ausculted for the presence of breath sounds, areas of dullness, crackles or wheezes in at least 4 quadrants (upper and lower right and left sides). Crackles indicate alveolar exudate – typically pulmonary edema or pneumonia. Crackles may be very difficult to auscult in cats. Wheezes are consistent with obstructive lower airway disease. Foreign bodies in the lower airways also can cause similar sounds. Areas of dullness may indicate severe pulmonary infiltrate, pleural fluid, intrathoracic masses, or the presence of abdominal contents in the thorax. The heart always should be ausculted after the lungs have been ausculted since once the ear has accustomed itself to louder sounds quieter sounds can be more difficult to hear. Because of the narrow chest wall of the cat lung sound can be referred easily across both hemithoraces making it difficult to pick up unilateral abnormalities in this species.

Cardiovascular examination includes an assessment of heart rate and rhythm, pulse rate and quality, mucous membrane colour and temperature. Dogs in shock typically become tachycardic. Cats in shock or with underlying cardiomyopathy frequently are bradycardic. Gallop rhythms or murmurs are consistent with underlying cardiac disease, not infrequently cardiomyopathy in the cat. Low rectal temperature often is an indicator of poor perfusion in the cat.

Cyanosis is an indication of hypoxemia or a PaO₂ of less than 60 mm Hg. Cyanosis can be difficult to detect if the patient has a hemoglobin less than 5 g/dl or with certain fluorescent overhead lights.

**Respiratory Support**

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

**Oxygen**

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

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

Nasal oxygen is the most effective way to provide supplemental oxygen. Tubes between 3.5Fr and 10 Fr are usually placed in dogs. Cats will usually tolerate 5-8 Fr tubes. The catheter is measured from the tip of the nose to the lateral canthus of the eye so that the tip will be in the nasopharynx. The tube is placed in the ventral nasal meatus and sutured to the nose as well as on the bridge of the nose between the eyes. At flow rates of 100 ml/kg/min the FiO₂ will be a minimum of 0.4 and may reach as high as 0.65. Nasal oxygen should be avoided in the patient with severe nasal or pharyngeal disease, thrombocytopenia and when concerns for elevated intracranial pressure exist.

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

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

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

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

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

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

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

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

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

**Thoracentesis**

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

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

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

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

Continuous Positive Pressure Airway Support (CPAP)
CPAP helps to decrease the work of breathing and improve gas exchange. It is defined as maintaining the pressure above atmospheric pressure throughout the respiratory cycle. This can be used as a bridge in patients that do not fully respond to oxygen support but positive pressure ventilation is not an option or if it is felt that some assisted ventilation may help avoid the need to positive pressure ventilation. A modified form of CPAP can be fairly easily provided to most awake dogs. A fairly tight fitting mask attached to an anesthetic circuit is placed on the patient. The pop-off valve is tightened down and the oxygen flow rate is increased until the pressure on the circuit registers at 5 cm H₂O. The patient breathes this oxygen under high pressure.

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

References available on request.
PEDIATRIC EMERGENCIES

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The term pediatric usually refers to the first 12 weeks of life in dogs and cats. The neonatal stage is from birth to 2 weeks of age, infant from 2 to 6 weeks of age, and juvenile from 6 to 12 weeks of age.

Healthy neonates are active and have good muscle tone. Crying is normal in response to pain, cold, hunger, or loss of contact with the mother; however, crying should not continue longer than 20 minutes. Hyperemic mucous membranes are normal until day 4 to 7. Renal function does not mature until about 8 weeks of age although by 4 weeks of age they are able to excrete drugs at the same rate as adults. By 6 weeks of age hepatic metabolizing enzyme systems are functioning at nearly adult levels.

TREATMENT

Environment
Neonates lack the ability to regulate their temperature well and easily become hypothermic. If they are separated from the mother they should be placed in an incubator that is kept at 29-32C (84-90F). Humidity should be kept between 55-65%. External warming devices such as hot water bottles and heating pads can be used but the neonate should be able to get away from the heat and the source should be covered in a towel or blanket to prevent burns.

Oxygen
Neonates may require oxygen supplementation. This can be provided by placing the neonate in an oxygen-rich environment or by placing a small nasal catheter (intravenous catheter in very small patients).

Fluid Therapy
Intravenous or intraosseous fluids are preferred over subcutaneous or intraperitoneal fluids. Intraosseous access is performed using hypodermic needles or commercial intraosseous needles. Commonly used access points include the greater tubercle of the humerus and trochanteric fossa of the femur. The tibia can also be used. If only rehydration and maintenance fluids are indicated then fluids can be given into the gastrointestinal tract if it is functional. A long intravenous catheter can be used as a nasogastric tube to deliver fluids. If the patient is not eating the fluid will need to be supplemented with dextrose at concentrations between 2.5% to 10% to maintain euglycemia. Maintenance fluid requirements are between 2-4 ml/kg/hour for pediatric patients.

Blood Transfusions
Blood transfusions may be required to treat parasitic infestations, trauma, or rodenticide coagulopathies. They should be given intravenously or intraosseously. The same rules apply to transfusion medicine in pediatric patients as in adult patients. As a last resort intraperitoneal transfusion can be given; however, only about 70% of the blood will be absorbed over 72 hours.

Nutrition
Nutrition is key in the treatment of neonates since they lack sufficient glycogen stores to maintain glucose levels for extended periods of time. Hypothermic neonates should not be fed until they are warm since hypothermia leads to ileus and poor digestion, often causing curdling
of milk in the stomach. The patient should be weighed on a daily basis and should gain approximately 10% of its body weight daily. A goal of 2.5-4 g/kg of anticipated adult weight also is an acceptable estimation.

Hypoglycemic neonates should be treated with intravenous or intraosseous dextrose (1-2 ml/kg of 25% dextrose). Dextrose given orally is rarely effective at reversing a crisis.

Milk replacers should be used until weaning (which can be done as early as 2.5-3 weeks of age). Patients can be fed with nursing bottles or eye droppers. Some nursing bottles have nipples that require the neonate to generate a lot of negative suction. Expanding the hole a little using a hypodermic needle will help the weaker patient nurse more effectively. If the patient does not have bowel sounds oral feeding should proceed very slowly. If the patient is too weak to nurse tube feeding may be required.

Tube feeding can be accomplished with an orogastric tube (short term) or with nasogastric tubes (longer term). For orogastric feeding a red rubber or other infant feeding tube approximately 5-10 Fr in size is premeasured from the tip of the nose to the last rib. The tube can be cut short if necessary. A small amount of water soluble gel is used to lubricate the tube and the tube is passed with the neck held in a flexed position. The gag reflex does not develop until approximately day 10 so the tube always should be checked for proper placement prior to infusing liquids or food. This can be done by aspirating to check for gastric contents or by confirming the location with a radiograph. Injecting a small amount of air into the stomach and listening over the stomach with a stethoscope or injecting fluid and observing for a cough are very inaccurate ways of determining accurate placement. A recently described option suggests measuring the tube to the level of the wing of the atlas. The tube is aspirated when it reaches that level. Continuous air aspiration indicates the tube is in the trachea. Once accurate placement has been confirmed the tube is inserted to the indicated level. Once the feeding has been completed the tube should be kinked and then withdrawn. This will help prevent liquid from being aspirated during tube removal.

If a nasogastric tube is indicated and the smallest commercially available tubes are too large, intravascular catheters that are designed to be used as central lines can be placed. A radiograph should be taken prior to feeding to confirm the location of the tip of the tube.

The stomach capacity is approximately 50 ml/kg. Initially feedings may need to start at 1-2 ml/kg every 1-2 hours. This can progress to larger volume feedings every 4 hours. Gastric overdistention should be avoided since this will lead to delayed emptying, nausea, and sometimes compromised ventilation. Feedings should always be done at body temperature.

If diarrhea is present in the neonate milk replacer may need to be diluted 1:1 with an oral electrolyte solution.

**Analgesia**

Pain kills. No matter how small the patient analgesics should be provided. Opioids can be used safely in pediatric patients. However, it should be kept in mind that the effects of the drugs may be much more magnified in the pediatric patient especially if the animal is sick or injured. Doses should be reduced significantly (20-25% of normal) and titrated upwards. Depending on the age of the patient and the drug, some non steroidal antiinflammatory drugs may be usable.
Antibiotics
Due to an increase in body water neonates and infants may need a higher dose and a longer
dosing interval of drugs such as penicillins, cephalosporins, and aminoglycosides. Because they
have lower albumin levels, drugs that are protein bound may be more active in pediatric
patients. Tetracyclines and trimethoprim-sulfonamide antibiotics should be avoided.
Fluoroquinolone antibiotics also should be avoided since they can cause a developmental
arthropathy.

Miscellaneous Medications
Vitamin K1 may be indicated in any sick neonate less than 48 hours old or any neonate showing
signs of bleeding since they have decreased thrombin levels.

Diagnostics
A drop of blood retrieved from an intravenous or intraosseous catheter or from a jugular stick
can be placed on a portable glucometer reagent stick. Larger blood samples should be taken
from the jugular; however, the total blood volume of a 300g patient may be less than 30 ml and,
in very small animals iatrogenic anemia is a possible complication of blood sampling, especially
if the patient is anemic to begin with.

Radiographs of pediatric patients are indicated as for adult patients. Abdominal radiographs
may be difficult to interpret due to the lack of body fat.

SPECIFIC CONDITIONS
Sick neonates often cry incessantly until they become too weak. Mucous membranes may be
pale or cyanotic. Dehydration is not uncommon secondary to lack of intake or disease causing
increased losses and can be difficult to assess due to the lack of body fat. Mucous membranes
should be moist. Due to their increased maintenance fluid requirements it is often easy to
underestimate their fluid needs.

Diarrhea is common in pediatric patients. This can be due to dietary changes, maternal disease,
and infections (virus, bacteria, parasites). Bowel sounds may be absent in these patients which
often is consistent with generalized gastrointestinal dysfunction. If severe enough this can lead
to problems such as prolapsed rectum and intussusception.

Whelping and Cesarean Section
Hypoxia and trauma can develop during whelping secondary to early placental separation,
entrapment in the birth canal, and difficulty in passage through the birth canal. Fortunately these
complications are not common. More commonly problems occur during delivery of puppies or
kittens during a cesarean section. In order to minimize problems the hospital should be ready to
deal with the arrival of the neonates. An incubator or box lined with a heating pad or hot water
bottles should be made ready. Plenty of sterile (ideally) towels should be available to dry off the
neonates. Oxygen should be available as well as small masks and an AMBU bag. Over-the-
needle catheters (14-20 ga) can be used as tracheotomy tubes to provide oxygen and assist
ventilate the newborns. The adaptor from 2.0-3.5 mm endotracheal tubes fits onto the end of the
catheter so that an AMBU bag can be attached to aid in resuscitation. Suction may be
necessary in order to be able to remove meconium and other secretions from the oropharynx
and sometimes from the trachea. An ear bulb syringe can be used if mechanical or electric
suction is not available. Extra hemostats and suture should be available to tie off the umbilicus
which should be tied about 0.5 cm from the body wall.
If an animal is delivered by cesarean section the surgeon can administer naloxone to reverse any of the opioid effects present from the maternal circulation. Naloxone also appears to have other stimulatory effects in the brain. Naloxone can be given sublingually but uptake is unpredictable and ideally injections should be given into the umbilical vein. Doxapram is only useful if the animal is breathing and can lead to a worse outcome if the patient is not breathing. The neonate should not be swung back and forth between the legs since this can cause head trauma.

Neonates should be encouraged to nurse as soon as the bitch or queen is alert.

**Congenital Abnormalities**
A variety of congenital abnormalities can lead to dysfunction of multiple organ systems and the reader is encouraged to consult other texts for detailed information. Serious abnormalities often lead to mortality in the early hours or days of life. Some abnormalities may not show up immediately. These include problems such as atresia ani and megaesophagus secondary to a persistent right aortic arch (which may not be evident until the animal is weaned). Cleft palate can lead to nursing problems, swallowing disorders, and aspiration pneumonia. Isolated swallowing abnormalities can lead to aspiration pneumonia and an unthrifty neonate. Other neurologic abnormalities can lead to abnormal ambulation.

**Fading Puppy/Kitten Syndrome**
Fading puppy or kitten syndrome can be caused by malnutrition, hypoglycemia, and septicemia (see below). Common signs associated with hypoglycemia include weakness, collapse, stupor, and hypothermia. Tremors and seizures can be seen but often are absent. Intravenous or intraosseous access should be established immediately and 1-2 ml/kg of 25% dextrose infused. Vitamin B should be supplemented whenever glucose is used to control seizures since vitamin B is required for aerobic metabolism of glucose. If a drop of blood is retrieved it can be used for blood glucose determination. If the puppy or kitten responds or if hypoglycemia is diagnosed the animal should be placed on a constant rate infusion of 5-10% dextrose until it is eating normally.

**INFECTION**
Many neonatal infections caused by viruses or bacteria can be rapidly fatal. As the healthy puppy or kitten becomes older their ability to fight off infection improves; however, aggressive supportive care may be needed to improve the chance of survival. Particularly susceptible times for viral infections include the neonatal period and the juvenile period when the maternal antibody protection has worn off but the protection from vaccination is not yet effective.

**Neonatal Conjunctivitis**
Neonatal conjunctivitis results from accumulation of purulent exudate behind the eyelids before they open. The eyelids should be separated to encourage drainage. A scalpel blade is rarely necessary and should be used with extreme caution due to the possibility for damaging the eyelids. If the eyelids are very stuck warm compresses will often cause them to separate. Gentle cleansing should be performed using warm compresses, saline lavage and a broad spectrum topic antibiotic drop or ointment should be instilled.

**Umbilical Infection**
Umbilical infections can develop in the first 4 days of life. They are often associated with the neonate’s environment. A streptococcal infection is most likely. Any abscess should be lanced and drained and the umbilicus should be treated with warm compresses and systemic antibiotics. Fluid therapy and other supportive care may be indicated.
**Neonatal Septicemia**

Neonatal septicemia can be associated with staphylococcal, streptococcal, E. coli and Pseudomonas infections. Lack of colostrum intake and maternal infections (mastitis, metritis) can lead to the development of septicemia. The neonate should be separated from the mother and the bitch or queen should be treated for any infection. Intravenous fluids, antibiotics, nutritional support, and supportive care are key if the neonate is to survive.

**Toxic Milk Syndrome**

Mastitis and metritis in the bitch or queen can lead to toxic milk syndrome which is characterized by signs of bloating and green diarrhea. The neonate should be treated with fluids and a milk replacer until the infection in the bitch or queen has resolved.

**Parasites**

Intestinal parasites can start to become a problem as early as 2-4 weeks of age. Some parasites can be transmitted transplacentally and some can be transmitted in the milk. Most parasites will cause vomiting, diarrhea, and lack of appetite. Hookworms can cause significant anemia as can fleas. Pyrantel pamoate can be used as early as 2-3 weeks of age. Giardia is treated with metronidazole or fenbendazole. Coccidiosis is common in infant and juvenile patients and is often found in addition to other infections. It is treated with sulfadimethoxine. External parasites can be treated with pyrethrin once the animal is old enough. Label directions should be followed closely. Flea combs can be used in patients too young for insecticides. Cleaning the environment is essential in the control of parasitic infections.

**Juvenile Cellulitis**

This is an idiopathic disease that occurs between 3-16 weeks of age. Facial swelling, lymphadenopathy, fever, and anorexia are hallmarks of the disease. Deep pyoderma may be present. Immunosuppressive doses of prednisone are required until clinical signs resolve. Antibiotics are recommended and are essential if a deep pyoderma is present. Topical antibacterial shampoos also may be needed.

**Trauma**

Trauma in early life can be caused by the bitch or queen lying on or stepping on the neonate. This can lead to head trauma, pulmonary contusions, and fractures. Later in life trauma can lead to lacerations, head injury, and fractures most commonly. Treatment should be directed to the specific injury. Fortunately most young patients have an incredible ability to heal.

**Electric Cord Injury**

Electric cord injuries can be seen in juveniles as their natural curiosity leads them to chew on objects. The electrical injury causes oral burns, which are usually seen on the tongue and commissures of the lips, as well as neurogenic pulmonary edema. Supplemental oxygen should be provided until the patient is breathing normally and eating well. One to two doses of furosemide (1-2 mg/kg intramuscularly or intravenously) may help relieve the pulmonary edema although this is controversial. Bronchodilators also may be useful. An esophagostomy tube may be needed if several oral burns are present. Cage rest is indicated until the pulmonary edema has cleared which generally takes 48-96 hours.

**Foreign Body Ingestion**

Juvenile patients often ingest foreign materials. Inducing vomiting may be helpful in removing objects still in the stomach although this should be done with care if the object is sharp or large since it may cause lacerations or obstruction respectively during emesis. Other material will require endoscopic or surgical removal.
Intussusception
Chronic diarrhea (viral, bacterial, parasitic, dietary indiscretion-related) can lead to intussusception. Patients with chronic diarrhea should be palpated 3-4 times daily for the presence of a tubular structure that may indicated an intussusception. This is a surgical emergency. The intussusception is reduced if possible, nonviable intestine should be resected and, enteroplication of the intestine should be performed to prevent recurrence. Even with enteroplication recurrence is possible – especially if the cause of the diarrhea is not diagnosed and treated appropriately.

Prolapsed Rectum
A prolapsed rectum is not uncommon with severe diarrhea. A fecal exam is always warranted to help determine the underlying etiology. If the rectum is prolapsed it should be manual reduced. The blunt end of a pencil should be placed about 1 cm into the rectum and a pursestring should be placed and tied tightly round the pencil. Once the suture is tied the pencil is removed. This ensures the pursestring is tight enough to prevent a recurrence of the prolapse while ensuring the animal can still defecate.

References available on request.
TOXICOLOGIC EMERGENCIES

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

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

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

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

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

Xylazine (0.44 mg/kg intramuscularly) can be used to induce vomiting in cats if hydrogen peroxide does not work; however, this drug can have serious cardiovascular side effects and the patient must be closely assessed prior to administration of the drug. It will also cause sedation which may or may not be desirable. In addition it is not always successful.

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

Activated charcoal should be administered via a gavage or nasogastric tube if it is indicated. Ideally a cathartic should be administered with the charcoal to hasten removal of the toxin. Many activated charcoal compounds are manufactured with cathartic (sorbitol magnesium sulfate) already present. The charcoal may need to be repeated over as along as a 48 hour period since some toxins undergo enterohepatic cycling. The decision to do this should be on a case-by-case basis. Activated charcoal often seems to stimulate vomiting which should be kept in mind when a decision is being made to administer the compound.

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

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

If the patient has evidence of bronchospasm (increased respiratory rate and effort, wheezes or fine crackles on auscultation) then supplemental oxygen should be provided and bronchodilators should be administered. Aminophylline and β–2 agonists can be given parenterally; however, in the author’s experience nebulized β–2 agonists tend to be superior to parenterally administered agents. Aminophylline can cause anxiety and tachycardia whereas side effects of β–2 agonists are rare.
Circulation
Patients that are hypotensive may require fluid resuscitation. Animals that are significantly anemic should receive red cells. Patients with coagulopathies should receive fresh whole blood (if also anemic) or fresh frozen plasma – ideally until clotting tests are within a normal range. Blood pressure and perfusion status should be returned to normal. Some toxins may cause hypotension by depressing cardiac function or by causing excessive vasodilation. In this case positive inotropic drugs, β-blockers, antiarrhythmic drugs, or vasopressors may be indicated depending on the toxin. Patients that are dehydrated should have their fluid deficit calculated and administered over an 8-12 hour period.

Certain toxins can cause hypertension. Systolic blood pressure greater than 200 mm Hg can lead to significant patient morbidity. The underlying cause should be identified if possible in order to treat with the appropriate drug. Acepromazine will cause hypotension through vasodilation but can be difficult to titrate. If hypertension is associated with tachycardia then a β-blocker (propranolol at 0.02-0.06 mg/kg IV over 5 minutes) should be given. Hydralazine, angiotensin-converting enzyme inhibitors (enalapril, benazepril) and calcium channel blockers (diltiazem) may also be helpful in controlling hypertension depending on the underlying cause. Amlodipine is useful in controlling hypertension associated with renal disease. Unfortunately many of these medications are in an oral form only which may limit their usefulness in the acute stages. Hydralazine can be dissolved in water and given as a suspension in a crisis situation.

Seizure Management
Seizures should be controlled using intravenous diazepam initially (0.5-2.0 mg/kg). If intravenous access is not possible diazepam can be given intranasally. If this is unsuccessful intravenous phenobarbital should be given. Both diazepam constant rate infusions (0.1-2.0 mg/kg/hr) and phenobarbital constant rate infusions (2-10 mg/hr) can be given to help maintain control of seizures. The two drugs are synergistic when given together. Phenobarbital loading may be required to achieve therapeutic phenobarbital levels. If the animal has never received phenobarbital before this generally can be achieved by giving 16 mg/kg divided into 4 doses given every 20 minutes. (A dose of 3 mg/kg will raise the blood level by approximately 5 mcg/ml.) If the patient becomes excessively sedate or loses a gag reflex the clinician may prefer not to give further doses of phenobarbital until the patient is more alert. Pentobarbital can be used to control motor movement but it should be kept in mind that pentobarbital does not control seizure activity in the brain until a very deep coma level is reached. Muscle activity during recovery from pentobarbital can be easily confused with seizure activity.

Management of Stupor and Coma
Patients who do not have a gag reflex should be intubated and positive pressure ventilation should be instituted if the animal is not ventilating adequately. The patient should be placed in a 30 degree body tilt to help minimize the risk for aspiration. Pressure on the jugular veins should be avoided. Patients should be rotated every 2-4 hours to prevent atelectasis and reduce the
risk for pneumonia. Pressure points should be padded to minimize the risk of pressure sores developing. The eyes should be kept lubricated with ocular ointments and the tongue may need to be kept moistened. Chlorhexidine rinses may help minimize the colonization of the mouth with potentially pathogenic bacteria. Mannitol may be useful in helping treat cerebral edema at a dose of 0.1-0.5 g/kg intravenously. This dose can be repeated.

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

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

Management of Temperature Abnormalities
Hyperthermia may result from excessive seizure activity, muscle rigidity, malignant hyperthermia, or a hypothalamic disorder. The patient should be actively cooled if the temperature is above 40.5°C. While the patient is being cooled appropriate measures to secure the airway, provide oxygen, fluids and control seizures or muscle activity should be taken. Cooling can be done by running the fluids through an ice bath, and placing ice packs around the head and over superficial major vessels such as the femoral and brachial arteries. Spraying the patient with water and then placing a fan on the patient will cause evaporative heat loss. Application of topical alcohol should be avoided since it can be absorbed systemically leading potentially to alcohol intoxication. Cooling should be stopped once the patient’s temperature reaches 39.5°C. If the patient’s temperature is in an extreme danger zone (greater than 41.5°C) active core cooling may be indicated. This can be done by administering cold water enemas and cold water gastric lavage. These patients frequently develop the systemic inflammatory response syndrome with all of its accompanying complications (hypotension, vasculitis with secondary albumin loss and third-spacing of fluids, coagulopathy, and multiple organ failure).

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

References available on request.
Decline in kidney function can result from a variety of causes including pyelonephritis, amyloidosis, polycystic kidney disease, neoplasia, nephrotoxicosis, hydronephrosis and chronic glomerulonephritis (Scherk, 2011). Although acute insult can lead to chronic kidney disease (CKD), age seems to be the only major, consistent risk factor associated with chronic renal insufficiency (White, 2011).

Mature cat visits ideally include a complete physical examination/consultation as well as data collection in the form of a minimum database (MDB) every 4 to 6 months. A minimum database for mature cats includes a full clinical chemistry, a total thyroid test (TT4), a complete blood count, a urinalysis and a blood pressure (BP) series. Blood urea nitrogen (BUN) and creatinine have traditionally been the go-to serum values for diagnosis of kidney disease. Early diagnosis can be challenging utilizing only these values, as azotemia does not develop until there is 75% loss of nephron function. The BUN can be influenced by factors other than renal disease, including dehydration, dietary protein content, gastrointestinal bleeding and hepatic insufficiency. Creatinine is a more reliable indicator of glomerular filtration rate (GFR). However, creatinine can be influenced by muscle wasting and by dehydration. Routine screening of these values can assist the clinician in documenting upward trends in these values. Symmetrical dimethylarginine (SDMA) measures the methylated form of the amino acid arginine. This is a by-product of protein degradation excreted by the kidneys. SDMA increases with about 40% loss of kidney function. It can be impacted by dehydration. Symmetrical dimethylarginine is not a stand-alone test and should always be interpreted in light of patient status as well as other laboratory findings. Elevated SDMA in the absence of any other evidence of renal disease should be re-evaluated.

### Table 1 Urine Specific Gravity Varies with Age & Diet (Scherk, 2011)

<table>
<thead>
<tr>
<th>Age or condition</th>
<th>Expected USG</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>4-8 weeks of age</td>
<td>1.020-1.038</td>
<td></td>
</tr>
<tr>
<td>8+ weeks of age</td>
<td>Up to 1.080</td>
<td>Denotes age at which full concentrating ability is reached</td>
</tr>
<tr>
<td>Dehydrates/normal renal function</td>
<td>&gt;1.040</td>
<td>Diet dependent (wet vs dry)</td>
</tr>
<tr>
<td>Canned food only</td>
<td>&gt;1.025</td>
<td></td>
</tr>
<tr>
<td>Dry food only</td>
<td>&gt;1.035</td>
<td></td>
</tr>
<tr>
<td>Inability to concentrate urine</td>
<td>1.008-1.012</td>
<td>Nephrons no longer able to modify glomerular filtrate</td>
</tr>
<tr>
<td>Dehydrated/unknown renal function</td>
<td>1.007-1.039</td>
<td>Suggestive of renal insufficiency with or without azotemia</td>
</tr>
</tbody>
</table>
It is recommended that urine samples be collected by cystocentesis and tested immediately in the clinic laboratory. Urine testing should include chemistry testing using testing strips, measurement of urine specific gravity (USG) by refractometer and sediment analysis. Urine specific gravity can be impacted by age, diet and hydration status. Urine specific gravity varies throughout the day, such that a single low USG is not reliable evidence of a loss of concentrating ability (Scherk, 2011). Samples with a low urine specific gravity (USG; less than 1.035) should be submitted for culture.

**International Renal Interest Society (IRIS)**

For cats that are diagnosed with CKD, it is critical for practitioners to develop and promote a relationship with clients that will allow continued monitoring of the disease, including disease staging. The application of human IRIS staging guidelines to the study of feline renal disease has dramatically advanced our ability to tailor our patient therapy, thereby improving quantity and quality of life. In addition to the MDB as discussed above, imaging is likely to be beneficial.

**Table 2. IRIS Staging Guidelines**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Renal Azotemia</th>
<th>Creatinine</th>
<th>Clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Non-azotemic</td>
<td>&lt;140 µmol/L</td>
<td>Absent</td>
</tr>
<tr>
<td>2</td>
<td>Mild</td>
<td>140-249 µmol/L</td>
<td>Mild or absent</td>
</tr>
<tr>
<td>3</td>
<td>Moderate</td>
<td>250-439 µmol/L</td>
<td>Moderate</td>
</tr>
<tr>
<td>4</td>
<td>severe</td>
<td>&gt;440 µmol/L</td>
<td>Severe</td>
</tr>
</tbody>
</table>

Adapted from AAFP 2015 Dru Forrester, DVM, MS, DACVIM & Jane Robertson, DVM, DACVIM *Chronic Kidney Disease: Making the most of early diagnosis*

**Table 3. Subclassifications of IRIS staging: Proteinuria**

<table>
<thead>
<tr>
<th>Urine Protein:Creatinine Ratio (UPC)</th>
<th>Substage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.2</td>
<td>Non-proteinuric (NP)</td>
</tr>
<tr>
<td>0.2-0.4</td>
<td>Borderline proteinuric (BP)</td>
</tr>
<tr>
<td>&gt;0.4</td>
<td>Proteinuric (P)</td>
</tr>
</tbody>
</table>

Taken from AAFP 2015 Dru Forrester, DVM, MS, DACVIM & Jane Robertson, DVM, DACVIM *Chronic Kidney Disease: Making the most of early diagnosis*

**Table 4. Subclassifications of IRIS staging: Blood pressure**

<table>
<thead>
<tr>
<th>Systolic BP (mmHg)</th>
<th>Diastolic BP (mmHg)</th>
<th>Substage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;150</td>
<td>&lt;95</td>
<td>Minimal risk (N)</td>
</tr>
<tr>
<td>150-159</td>
<td>95-99</td>
<td>Low Risk (L)</td>
</tr>
<tr>
<td>160-179</td>
<td>100-119</td>
<td>Moderate Risk (M)</td>
</tr>
<tr>
<td>&gt;180</td>
<td>&gt;120</td>
<td>High Risk (H)</td>
</tr>
</tbody>
</table>

Taken from AAFP 2015 Dru Forrester, DVM, MS, DACVIM & Jane Robertson, DVM, DACVIM *Chronic Kidney Disease: Making the most of early diagnosis*
True proteinuria in cats is a known marker of poor prognosis in renal disease (Syme, H.M. et al, 2006; Syme, H.M., 2009). If proteinuria is established on the chemistry stick in the absence of active sediment, the sample will need to be submitted for a urine protein creatinine ratio (UPCR). The result should be used to direct therapy with medications to reduce the loss of protein into the urine. Ratios over 0.4 are significant and therapy is needed. If there is active sediment in the presence of proteinuria on the chemistry stick, and the UPCR is very high (>0.5), then the value may be significant and therapy may be indicated.

Blood pressure changes can be impacted by and/or impact the renal state of health (Brown, 2011). Sixty-five to 100% of cats with hypertension have evidence of reduced renal function (Jepson, 2011). The gold standard for blood pressure assessment in any species is central venous catheter assessment. Blood pressures can be measured non-invasively either by Doppler or oscillometric methods. Patient stress can be a limiting factor. Proper use of pain management in advance, as well as following cat friendly practice and handling guidelines will significantly reduce stress.

Therapeutics, Monitoring and Maintenance
With the utilization of IRIS staging, the clinician gains significant ground in combatting chronic renal disease in cats. The data collected for the purpose of IRIS staging allows a tailored, individual approach to patient therapy.

Table 5: Survival time by IRIS Stage

<table>
<thead>
<tr>
<th>IRIS Stage</th>
<th>2b*</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median Survival (days)</td>
<td>1151</td>
<td>778</td>
<td>103</td>
</tr>
<tr>
<td>Range (days)</td>
<td>2-3107</td>
<td>22-2100</td>
<td>1-1920</td>
</tr>
</tbody>
</table>

*2b Creatinine of 203-249 µmol/L

Taken from AAFP 2015 Dru Forrester, DVM, MS, DACVIM & Jane Robertson, DVM, DACVIM Chronic Kidney Disease: Making the most of early diagnosis

Treatment for pain is essential. Medications such as gabapentin should be prescribed at a dosage of 15-20 mg/kg PO q12h. In debilitated cats, a dosage of 10 mg/kg POq12h is the initial chosen dosage. This medication is safe for use in all diseased states and the only initial side effect is sedation. After 1-2 weeks, any sedation will wear off and the cat will continue to be more comfortable. Injectable products such as cartrophen or adequan are also beneficial. Additional pain medications such as buprenorphine, amantadine and non-steroidal anti-inflammatory drugs (NSAIDs) may also require consideration. Elevated stages of CKD may preclude safe use of NSAIDs.

Dietary changes recommended for cats with renal disease will vary depending on the IRIS staging results. Many renal specific diets are formulated with reduced phosphorus and protein. These diets are not necessarily considered ideal in early disease states (Scherk and Laflamme, 2016). Increasing water intake may be a key
factor in improving renal function and overall patient hydration status. Indirectly this can reduce pain from dehydration and constipation.

Identification of BP values over 160-180, with or without retinal changes indicate the need for BP-controlling drugs. Calcium channel blockers such as amlodipine are the most effective at controlling blood pressure in the feline species. Some patients will have partially or uncontrollable hypertension with amlodipine and may require additional medications. Benazepril (Fortekor) is not effective in the control of hypertension. The newly available drug telmisartran (Semintra) may be effective at controlling hypertension at higher doses, but has yet to be evaluated for further benefit to hypertension cats.

The indiscriminate use of antibiotics in the absence of evidence of urinary tract infection is not recommended. Antibiotics should be selected based on urine culture and sensitivity patterns. A repeat urine culture 7 days following cessation of therapy is critical. In cases where urine culture is negative, but a low USG exists in the face of renal disease, ultrasound is recommended.

Patients who exhibit even mild decreases in potassium levels in their serum require supplementation with potassium gluconate. The majority of body potassium is held in the intracellular or interstitial space. The serum potassium represents only 2% of body potassium. Therefore any decrease noted in the serum is significant of a major decrease in the overall body stores.

Elevated UPCR indicates abnormalities with the renin angiotensin aldosterone system (RAAS). These changes alter intraglomerular pressures and result in protein loss into the filtrate/urine. The use of benazepril has been recommended in the past. However, this drug is not targeted to the particular pathway of RAAS that is affected and impacting the glomerulus. Over time, the RAAS can escape the control that benazepril exerts, resulting in resumed proteinuria. Telmisartran is a newer alternative that is more targeted and not likely to lead to escape mechanisms over time.

Improving hydration status in renal patients is generally considered to be beneficial to renal function and overall patient health. Addition of 20-40 mEq/L of potassium chloride to the fluids should be considered in the case of hypokalemia and/or where regular subcutaneous fluids will be administered.

Patients identified with elevated total calcium, elevated ionized calcium and/or elevated phosphorus may require phosphorus-binding agents to reduce phosphorus levels. The use of agents such as aluminum hydroxide can be challenging, as palatability is less than optimal. The use of phosphorus binding agents containing calcium should be minimized unless serial monitoring of ionized calcium can be pursued.
Calcitriol is a drug that is recommended frequently in renal patients. It’s primary indication for use is following diagnosis of renal secondary hyperparathyroidism. In these cases, the use of calcitriol, with regulated serum phosphorus levels, may benefit the patient in the short and long term. Calcitriol supplementation at low doses may be recommended by some experts as a means of improving quality of life. More detailed studies on this mode of utilization are warranted (Sparkes et al, 2016).

Chronic kidney disease can lead to a reduced production of erythropoietin. The result is a reduced production of new red blood cells from the bone marrow. Some patients will also have iron deficiencies reducing production of new RBC. Evaluation of iron levels with consideration for supplementation is needed. These patients may also require injectable erythropoietin or darbropoietin to stimulate bone marrow production of RBC.

References


Feline idiopathic cystitis (FIC) is a complex disease process in cats that is not fully understood at this time. It results in the clinical signs of feline lower urinary tract disease (FLUTD). Cats may exhibit pollakiuria, periuria, dysuria, hematuria and stranguria, vocalization when urinating and sometime hair loss on the lower abdomen. The severity of signs and the frequency with which they recur is variable. FIC can be obstructive or non-obstructive in its presentation. It is the most common cause of non-obstructive feline lower urinary disease.¹ This disease is generally seen in younger and middle aged cats and is uncommonly diagnosed in cats greater than 10 years of age. In reported studies, excessive body weight, decreased activity, multiple cat households and indoor housing have been associated with increased of FIC. FIC is a diagnosis by exclusion of other known causes of FLUTD. Affected cats can suffer recurrent episodes of FLUTD, which generally resolve without treatment over the course of 3–7 days. FIC can present as an acute episode or develop into a chronic re-occurring condition.

Identifying the underlying cause of FLUTD can problematic. The main differential diagnoses of FLUTD are FIC, urolithiasis, neoplasia and a bacterial infection. Bacterial infections are rare and most likely seen in older female cats with a low urine specific gravity or cats with glucosuria. The take home message from this lecture is DO NOT prescribe antibiotics routinely to cats with FLUTD. Rarely do cats have a bacterial infection, even when they have a urolith. The need for antibiotics should be based on a positive urine culture and treated according to the sensitivity results.

When a cat is presented with FLUTD a detailed history including information about the cat’s age, home environment, census of other pets in the home, behaviour, diet (including treats), water intake and other concerns are critical. History is followed up with a thorough physical examination with attention to the palpation of the bladder. Is the bladder painful? Does palpation illicit urination? Does the bladder feel soft, thickened, firm? Does the cat have a urinary obstruction which requires immediate resolution? All cats with FLUTD require a basic urinalysis which includes visual assessment, specific gravity, dipstick analysis, and sediment microscopy. Often cats with FIC present with an empty and uncomfortable bladder so that collection of a sample by cystocentesis may not be possible at the time of presentation. However, frequently these cats will pass a small amount of urine when their bladder is palpated or in a collection box. While not ideal, this is often enough urine to start ranking the differential diagnosis. Depending on the above findings, further investigations should then include radiographs of the abdomen and if needed ultrasound imaging of the bladder to identify urolithiasis or neoplasia. Urine culture and sensitivity should be carried out for any cat with a low specific gravity or glucosuria. and blood tests if there is evidence of systemic disease. Urine cultures should be obtained only by cystocentesis to prevent false positive results from contamination during a
free-flow sample Older cats and immature cats that develop signs of FLUTD have a higher index of suspicion for the other potential causes of FLUTD. Cats with FIC tend to have highly concentrated urine (SG. > 1.045)². There is usually haematuria and proteinuria, often mild pyuria and there may be a generalized thickening of the bladder wall.

While the condition currently remains, by definition, an idiopathic disorder, recent developments in understanding of the neuro-hormonal abnormalities that exist in affected cats suggest that the signs develop from an inability to cope with chronic stress. This may manifest in a number of ways, including the development of bladder inflammation and pain.² No cure is currently available for FIC, and treatment options are aimed at keeping the cat's clinical signs to a minimum, and increasing the disease-free interval. FIC can become a chronic, frustrating disease. Excellent client communication with the client is required. MEMO therapy, analgesics, diet and possibly other pharmacologic agents can be of benefit in treating acute and chronic cases.

Environmental modification is a key factor in the management of FIC, since stress clearly plays an important part in the pathophysiology of the disease. Multi-modal environmental modification (MEMO) was evaluated in client-owned cats with FIC. Implementing MEMO as the sole management strategy with FIC was found to be successful in the majority of cats followed over a one-year period of time.³ Meeting the environmental needs of the cat and understanding the cat as a species is critical. Cats are not inherently social and in the wild are solitary hunters. They tend to be solitary and are territorial and although they are hunters, they are also prey. These traits make it challenging for cats to live in close proximity to other cats. 'Silent bullying' often goes unnoticed, but it is a major cause of chronic stress to the less dominant cat. After the diagnosis of FIC is made, a questionnaire should be completed by the client to establish a thorough environmental history followed by the recommendations for MEMO. For suggestions on developing a questionnaire, as well as a good client resource, the reader is referred to the following websites: http://www.indoorcat.org/ and http://www.cathealthy.ca.

To meet the needs of each cat within the house, each individual cat must have free access to its own key resources, ideally positioned out of sight of the other cats. Key resources are food and water bowls that are sited apart from each other, clean uncovered litter trays (one box per cat, plus one) in various locations around the home, resting places at different vertical heights with some that only fit one individual cat, and scratching posts and scratching resources. Cats need mental and physical activity several times a day and cat families need to make time in their day to play with their cat as they would their dog. Putting the hunt back in meal-time using feeding toys is a good form of entertainment for the “predator” in the cat. As cats that develop FIC tend to be overweight, a weight loss program with a strict calorie counted amount of food fed per day is critical.

Although a statistically significant difference was not found when Feliway® was used in a home compared to placebo in cats with FIC, cats that had Feliway® used in the environment had a trend for fewer bouts of FIC and reduced negative behavioral traits. Calming nutraceuticals such as Zylkene® or Anxitane® may be helpful. Diets such as Royal Canin Calm® or Royal Canin Urinary/Calm® and Hill’s Multicare C/D Stress® can be helpful in the long-term management of FIC by reducing the frequency and intensity of recurring episodes of lower urinary tract signs. To achieve this aim, they need to be fed as the cat's sole source of nutrition and used consistently in the long term.
A primary objective in managing the painful signs of FIC is to encourage the production of large volumes of dilute urine (SG < 1.035). Any measures which will increase the cat's water intake are likely to be helpful. Feeding canned food is particularly effective, as is offering the cat palatable fluids to drink (chicken or fish stock, water from tinned fish, etc.). Adding extra water to canned or dry foods works well. Monitoring of the success of the owner’s attempts to increase water intake can be done via regular analysis of the urine samples collected at home or in the clinic. Aim is to keep the urine SG below 1.035.

Clinical signs of acute FIC resolve spontaneously in as many as 85% of cats within 2-3 days, with or without treatment. Assessing the efficacy of any medical treatment for FIC is made difficult by the self-limiting nature of this disease. When a cat is diagnosed with FIC, analgesic therapy should be initiated for the acute management of the disease. These cats are painful and the pain needs to be treated in a multi-modal fashion with opioids and NSAIDS. Prazosin hydrochloride may be helpful to relieve urethral spasm, but it is not generally useful in the non-obstructed FIC patient. It is important that the client appreciates that all current treatments for FIC are merely palliative and that without application of multi-modal environmental modification (MEMO) and measures to increase water intake, the FIC episodes will recur and will require continued management.

A variety of other drugs have been tried in cats with chronic re-occurring FIC, but little evidence exists as to their efficacy. These drugs need to be used only after environmental strategies, diet changes (if necessary), and behavior modifications have failed. Parenteral and oral glycosaminoglycan precursor (GAG) supplements have been recommended for management of FIC and for interstitial cystitis in women, but to date, published studies have not shown significant effect in cats. From the proposed mechanism of action, it can be predicted that if treatment is used, it will need to be long term. Amitriptyline showed no evidence of benefit in short-term use in cats, but has been reported in uncontrolled trials to successfully decrease clinical signs of severe, recurrent FIC. Clomipramine (Clomicalm®) used in recurrent cases of FIC has shown anecdotal improvements in some patients. Other drugs such as fluoxetine (Prozac®) have been reported to help cats with inappropriate urinations with variable success rates.

DO NOT USE ANTIBIOTICS! Antibiotics are not indicated in cats with FIC and should be reserved for cats with bacterial infections. Corticosteroids are one of the few classes of drug that have undergone placebo controlled trials for treatment of FLUTD. Despite much anecdotal evidence to the contrary, they did not appear to have any effect in reducing the severity of the signs of FLUTD or the duration of the episodes.

References


**CALCIUM HOMEOSTASIS: UNDERSTANDING THE HYPERCALCEMIC CAT**

Kelly A. St. Denis, MSc, DVM, DABVP (feline practice)
Dr. Liz O’Brien, DVM, DABVP (feline practice)

**Calcium Homeostasis**

Calcium plays multiple roles in the body including skeletal support, muscle contraction, transmission of nerve impulses and blood clotting. It is an important ion in some cellular trans-membrane channels. Calcium is available in three forms in the body: ionized or free calcium, protein bound calcium and complexed calcium. Ionized calcium is the only physiologically active form and makes up 50-60% of extracellular fluid (ECF) calcium. Protein bound calcium makes up 10% of the ECF calcium, while calcium complexed to phosphate, bicarbonate, or lactate makes up 30-40% of the ECF calcium. The majority of total body calcium is stored in the bones (99%).

Regulation of calcium is a complex process. When there is a need for ECF calcium, it can be increased by absorption from the gastrointestinal (GI) tract, release from bone and/or reabsorption by the kidneys. Calcium is excreted via the GI tract (90%) and the kidneys (10%). When necessary, the kidneys are capable of resorbing 99% of calcium filtered into the urine.

The need for more or less ECF calcium is mediated by parathyroid hormone (PTH) and calcitriol. At times that ionized calcium levels are low, the chief cells of the parathyroid gland are stimulated to release PTH. Within the kidneys, PTH acts to stimulate the conversion of vitamin D to calcitriol.

In situations where ECF calcium is in excess, a negative feedback loop reduces PTH production and subsequently calcitriol production. Elevated phosphorus also has a negative impact on this loop, decreasing PTH production. An additional hormone made in the thyroid gland, calcitonin, plays a minor role in decreasing ECF calcium.
Clinical signs
Evidence of hypercalcemia in a cat may be non-specific and may be related to the disease associated with the hypercalcemia. Signs of high calcium may include weakness, depression and mental dullness as the excitability of muscular and nervous tissue becomes depressed. Cats may have GI tract signs including anorexia, vomiting and constipation as a result of reduced contractility of smooth muscle in the GI tract. Central nervous system effects may include muscle twitching, shivering or seizures. Cardiac arrhythmias may occur. The cat may experience renal disease as a result of nephroliths or lower urinary tract disease signs as a result of urolithiasis. Polyuria and polydipsia can be observed, particularly in cases where renal disease has occurred as a result of hypercalcemia (and nephrolithiasis) or in cases of renal secondary hyperparathyroidism.

Testing
Routine blood screening should include measurements of total calcium (tCa++) in the minimum database. In cases where an elevation in tCa++ is observed, confirmation of this elevation should be confirmed approximately 2 weeks later. Persistent elevations in tCa++ need to be followed up with further testing, even in the situation where renal secondary hyperparathyroidism is strongly suspected. Further tests recommended include ionized calcium (iCa++) and PTH. In some cases, the clinician may also wish to request measurement of PTH related proteins (PTHrp) and Vitamin D metabolites. Cats should be fasted more than 12 hours prior to additional testing. A serum sample should be collected and handled in an anaerobic manner, with chilling of the sample instituted immediately after collection. Centrifugation in a refrigerated or chilled centrifuge is recommended. The lab should receive the chilled sample within 6 hours of collection if possible. If chilling is not possible, the sample should be received and analyzed within 2 hours.

Differential Diagnoses
Differential diagnoses of hypercalcemia in cats is most easily remembered by the two mnemonics SHIRT and GOSHDARNIT. The mnemonic SHIRT represents the most common causes of hypercalcemia in cats including Spurious, Hyperparathyroidism, Idiopathic, Renal disease and Tumours. For a more exhaustive list, GOSHDARNIT is helpful:
• Granulomatous disease
• Osteolysis
• Spurious (lab error)
• Hyperparathyroidism, house plant ingestion, hyperthyroidism
• D toxicosis, Dehydration
• Addisons, Aluminum toxicity
• Renal disease
• Neoplasia, Nutrition
• Idiopathic
• Temperature (hyperthermia)
## Diagnosis

Once an elevation in ECF calcium has been confirmed by iCa++ testing, the clinician will need to review the case, as well as the relevant data in order to shorten the list of differentials and recommend either further testing or therapeutics.

<table>
<thead>
<tr>
<th></th>
<th>tCa++</th>
<th>iCa++</th>
<th>PTH</th>
<th>PTHrp</th>
<th>25-OH Vit D</th>
<th>1,25(OH) Vit D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neoplasia</td>
<td>H</td>
<td>Often very HIGH</td>
<td>0 to L to LN</td>
<td>N or high</td>
<td>N</td>
<td>varies</td>
</tr>
<tr>
<td>Renal 2° Hyperparathyroid</td>
<td>H</td>
<td>N or H</td>
<td>H</td>
<td>N</td>
<td>N or L</td>
<td>N or L</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>H</td>
<td>H</td>
<td>N or L</td>
<td>N</td>
<td>N</td>
<td>varies</td>
</tr>
</tbody>
</table>

Patients with hypercalcemia parameters that suggest idiopathic or neoplastic disease will require further diagnostic testing. As noted in the table, there is sufficient overlap in the patterns observed with neoplasia and idiopathic hypercalcemia to require further clarification. In order to rule out neoplasia, the patient should be assessed via imaging for evidence of tumours. This should include a full abdominal ultrasound screening as well as 3-4 radiographic views of the thorax.

Patients with known renal disease confirmed to have renal secondary hyperparathyroidism will need to be assessed and treated for their renal disease. This includes complete IRIS staging (International Renal Interest Society) with assessment for comorbidities such as hypertension, proteinuria, renal or lower urinary tract infection. Treatment with calcitriol is an option provided the patient phosphorus levels are low normal. There is popularity in the use of calcitriol in all renal patients, which still requires further testing and publication of further evidence based medicine. At this time, the author uses calcitriol only in cases of renal secondary hyperparathyroidism. If this patient is hyperphosphatemic, or the levels are above an acceptable minimum for calcitriol use, phosphate binders should be employed to reduce the phosphorus levels. Calcitriol may be started once the phosphorus levels can be reduced and maintained at or below the accepted level. Ionized calcium and phosphorus levels should be monitored regularly.

The presence of nephroliths and/or uroliths will also need to be addressed. Patients with uroliths will require surgical removal of the stones, while patients with nephroliths may benefit from analgesia, medical and dietary management.
Treatment Options for Chronic Hypercalcemia

Patients with acute hypercalcemia are not addressed in this lecture. The following treatment options are not necessarily applicable to acute situations.

1. Identify and treat underlying cause
2. Dietary therapy: a consultation with a reputable veterinary diet company such as Royal Canin can be very beneficial in these cases. A diet change from dry to all canned may be sufficient. The use of high fibre diets, renal diets and calcium oxalate preventing/therapeutic diets may be recommended. These diets should be recommended on a case by case basis.
3. Subcutaneous fluids. This has not been assessed as a modality for treatment of hypercalcemia in cats. It is not likely to be harmful and may benefit cats with renal disease
4. Low dose diuretics. These have not been assessed in chronic hypercalcemia in cats. They increase the risk of dehydration and may negatively impact kidney health. This is not currently recommended by the author
5. Glucocorticoids. These may be effective but should not be used until all diagnostic testing is complete and the cat has a confirmed negative urinary culture. These drugs can decrease the efficacy of chemotherapy, increase the risk of diabetes mellitus and increase the risk of calcium in the urine. The latter will increase the risk of nephrolithiasis and urolithiasis.
6. Bisphophonates. These are not reported well in the literature but some anecdotal evidence has been shared and accumulated.

Reference

HAIRBALLS ARE NOT NORMAL: A PRACTICAL APPROACH TO THE VOMITING CAT
Kelly A. St. Denis, MSc, DVM, DABVP (feline practice)
Dr. Liz O’Brien, DVM, DABVP (feline practice)

Clients and veterinarians often consider that vomiting in cats is a regular occurrence that is not significant of health problems. This is a particularly common assumption with regard to vomit containing hairballs. Cats spend approximately 25% of their waking hours grooming (Panaman et al, 1981). The majority of ingested hair passes through the cat’s digestive tract into the feces with no negative side effects (Panaman et al, 1981). Cats that vomit occasionally may not be considered to have any specific underlying gastrointestinal disease (GID). However, cats that are vomiting more often than every 2 weeks are significantly more likely to have some baseline underlying GID (Norsworthy et al, 2015).

During routine preventive care examinations, detailed questioning about diet, diet changes, vomiting and hairballs is essential. When clients are uncertain about vomiting and/or hairball frequency, a calendar recording system should be recommended. In addition to regular vomiting, the patient may be showing signs of nausea that are not obvious to the client. These signs might include a finicky appetite, occasional loss of appetite or periods of anorexia, licking of the lips, gagging, and/or ingestion of grass to stimulate vomiting.

A history of abnormal bowel movements should also be investigated. Diarrhea can occur in conjunction with upper GID, or as a manifestation of lower GID. The veterinarian should also carefully question the client to identify evidence of constipation. Conditions such as inflammatory bowel disease (IBD) can exist as a problem within the small intestine, combined small intestine/large intestine or solely the large intestine. Vomiting, diarrhea and/or constipation may manifest as a result.

A thorough physical examination of the vomiting cat will help elucidate signs of nausea. The patient should be observed for signs of lip licking and frequent swallowing. A thorough oral health examination may reveal foreign objects looped under the tongue, oral ulceration or other oral or dental disease that may impact appetite and vomiting. Feline patient weights should be recorded on every visit to the clinic, as subtle weight loss can be one of the first signs of disease. The documentation of weight loss in a cat with frequent vomiting may be the only physical examination change noted. This change can be a hallmark of mild to significant GID.

The abdomen should be examined in quadrants and the patient carefully observed for evidence of nausea or pain during palpation of each quadrant. Evidence of pain during abdominal palpation may include very subtle changes. The patient’s face should be monitored closely for evidence of lip licking, wincing, blinking or other
facial expression changes that could indicate pain. The patient may growl or hiss, although this is rare. Guarding of the abdomen during palpation of the painful quadrant(s) may also be observed. Abnormal findings during the palpation may include evidence of an enlarged liver, distended stomach, thickened/ropy intestines, abdominal fluid, masses and/or enlarged lymph nodes.

Making a Diagnosis
The list of differential diagnoses in the adult and senior feline patient with chronic vomiting is long and complex. In all cases, a minimum database (MDB) plus a gastrointestinal (GI) profile is ideal for diagnostic testing. The GI profile should include cobalamin (B12), folate, feline specific pancreatic lipase (sfPL) & in some cases, trypsin-like immunoreactivity (TLI).

The patient’s feline leukemia virus (FeLV) and feline immunodeficiency virus (FIV) status should be determined. Feline leukemia virus is a known cause of lymphoma in the feline patient. However, with the introduction of vaccination against FeLV, there has been a shift in the types of intestinal lymphoma in cats (Cotter et al, 2011; Louwrens et al, 2005). This shift does not change the value of knowing the patient’s retroviral status, as disease management will be impacted by retrovirus infection.

Radiography is beneficial in elimination of some differential diagnoses in the vomiting cat. In older cats, the presence of neoplastic lesions within the thorax may be the only identifiable source of vomiting. Abdominal radiographs will be beneficial in identifying some foreign bodies, masses, intestinal accidents, and other changes. Evaluation of skeletal structures may indicate the presence of painful spondylosis, osteoarthritis and/or degenerative joint disease.

Ultrasonographic imaging is beneficial in identifying GI organ abnormalities (liver, gall bladder, spleen, pancreas) as well as the urinary tract. The intestines can be evaluated for abnormal gut motility, obstruction, or other intestinal accidents (ex. intussusception). The abdomen can be evaluated for a discrete mass or masses, including evidence of lymph node enlargement. Evaluation of intestinal wall thickness, as well as thickness and integrity of the four intestinal wall layers may help identify the presence of intramural disease such as IBD and lymphoma. Ultrasound changes associated with pancreatitis may be evident (Forman et al, 2004). The sensitivity of ultrasound in the diagnosis of pancreatitis is low (Cosford et al, 2010; Forman et al, 2004; Gerhardt et al, 2001).

Where clinical signs and laboratory studies are strongly indicative of disease such as IBD, lymphoma (diffuse neoplasia), discrete neoplasia, hepatitis, cholangitis, cholangiohepatitis and/or pancreatitis, biopsy is warranted. The decision to pursue endoscopy versus full abdominal exploratory may be impacted by the findings, the relative invasiveness of each procedure and cost. Exploratory surgery permits full visual assessment of all intra abdominal organs, biopsy of extra-intestinal tissues (liver,
pancreas, lymph nodes etc) and full thickness intestinal biopsy (Kleinschmidt et al, 2010).

**Symptomatic, Targeted and Empirical Therapies**
Dietary changes may be beneficial to the patient with GID. Changing dietary format, such as dry to canned food, may improve digestion. The use of veterinary formulations that are easy to digest such as Royal Canin Gastro, Hill’s i/d or PVD EN may reduce or in some cases eliminate active GID signs. The role of dietary allergens in IBD and other GID is difficult to confirm. Food-responsive enteropathy is characterized by signs similar to other GID, although large bowel signs are more often observed and cutaneous disease may also be present. (Jergens et al, 2012). Anti-emetics may be beneficial to the vomiting patient. Drugs which also have prokinetic effects should be used with caution in case of obstruction. Gastric acid blockers such as ranitidine and omeprazole are less likely to play a beneficial role in feline patients with GID.

Appetite stimulants for loss of appetite or anorexia may be beneficial in improving intake, but in the presence of nausea and GI inflammation, these drugs are likely to be of little utility until underlying disease is addressed.

Patients with GID may be experiencing pain as a result of or concurrent to their GID. As the signs of pain in the feline patient can be subtle at best, any conditions identified as potentially painful should be treated as such. Gabapentin, buprenorphine and non-steroidal anti-inflammatories are all beneficial in pain management. Multimodal analgesic protocols are most effective over single drug therapy.

It has been recommended that all cats with signs of GID and a serum cobalamin of <300ng/L should receive parenteral supplementation of cobalamin (Ruaux et al, 2005). The current supplementation dosage recommendations from Texas A&M University (TAMU) are 250 micrograms cobalamin SQ once weekly for 6 weeks followed by 250 micrograms SQ monthly long-term. Repeat measurements of B12 are recommended after the first monthly dose (http://vetmed.tamu.edu/gilab/research/cobalamin-information).

The empirical use of steroids is generally not recommended in any situation in feline medicine, however, this is a frequently used therapeutic in feline GID patients. Limitations of finances and client willingness to pursue diagnostic biopsy may impact the treatment selection process. Empirical steroid usage precludes or limits usefulness of ultrasound or biopsy, as the drugs will change the local inflammatory pattern, thus confounding diagnosis. Where steroids are to be employed, urine culture should be considered prior to drug initiation, in order to rule out occult UTI. Prednisolone or dexamethasone are the steroids of choice in cases of IBD or GI lymphoma. The author does not recommend the use of depot steroids such as methylprednisolone acetate. The usefulness of budesonide is questionable, although it may offer benefits as an adjunct therapy. Empirical use of cyclosporine or chlorambucil is not recommended.
REFERENCES


Diabetes Mellitus: Is Remission a Reasonable and Achievable Goal?

Kelly A. St. Denis, MSc, DVM, DABVP (feline practice)

Dr. Liz O’Brien, DVM, DABVP (feline practice)

Diabetes Mellitus (DM) is a common feline endocrinopathy which is probably increasing in prevalence. Most cases are primary and similar to type II diabetes in humans, which results from abnormal secretion of insulin from the pancreatic B cells and peripheral insulin resistance. The diagnosis of DM is made based on characteristic clinical signs of diabetes mellitus (polyuria, polydipsia, polyphagia, and weight loss), and documentation of hyperglycemia and glycosuria. In cats it may be complicated by the occurrence of marked stress hyperglycemia. When making a diagnosis of DM in cats, it is important not only to document persistent hyperglycemia and glucosuria, but also to rule out other diseases that may cause similar clinical signs. Measurement of fructosamine concentrations or urine glucose of samples collected in the home environment may allow the clinician to distinguish between stress induced hyperglycemia (and resultant glycosuria) and persistent hyperglycemia due to diabetes mellitus. Therapy for diabetes should be instituted as soon as possible after diagnosis.

The main goal of therapy is to achieve normal blood glucose levels without the need for insulin therapy, commonly termed diabetic remission. Diabetic remission is usually defined as the ability to maintain normal blood glucose without insulin treatment for 4 weeks without the reappearance of clinical signs. Clinicians need to accept that not all cats will achieve remission and in these patients the goal is to minimize the clinical signs without causing hypoglycemia and improve the patient’s quality of life. The duration of remission is highly variable and unfortunately, at least 25% of cats that achieve remission subsequently become overtly diabetic and must receive insulin again. Since tight glycemic control is required to achieve remission, there is an increased chance of hypoglycemic episodes. This risk/benefit needs to be discussed with the client. Some client’s busy lifestyles can make this a challenging situation. Successful management of cats with DM includes minimizing clinical signs, improving quality of life, preventing complications such as DKA and diabetic neuropathies and achieving remission when possible.

Administration of insulin and dietary modification are the principal therapies used for management of diabetic cats. A recent study showed that cats with newly diagnosed DM have a fair to good prognosis, with 46% living longer than 2 years. However, since 30 % of cats affected with DM are euthanized within their first year of treatment due to the emotional and financial burden of insulin treatment and the required veterinary care, achieving diabetic remission is the ideal goal for every feline patient faced with this disease. Intensive glycemic control after diagnosis has been shown in humans with DM type11 to improve long – term remission rates. It appears that the same holds true for our feline patients. Cats receiving
treatment for diabetes within 6 months of diagnosis with twice daily insulin treatment aimed at euglycemia in conjunction with the cats been fed an ultra-low carbohydrate diet have the best chance of remission.

Which cat will go into remission??? Studies are suggestive that DM remission in the cat is likely to occur through reversal of glucose toxicity. As in humans, cats that have experienced more prolonged hyperglycemia will have experienced a greater deterioration of beta-cell function resulting in a lower chance of remission. There is no factor that consistently predicts diabetic remission in the cat but the shorter the duration of DM, the faster glycemic control is achieved and those patients with less severe hyperglycemia when starting appear to be factors that are favorable. A retrospective cohort study showed that cats without hypercholesterolaemia were more likely to achieve remission. In one study, diabetes as a potential result of recent corticosteroid treatment was associated with nearly 50 percent remission. A lack of diabetic neuropathy has also been associated with future remission, but neuropathy is a result of prolonged hyperglycemia so this should not be a surprise. Early client recognition, early diagnosis and intensive treatment with BID insulin and ultra-low carbohydrate diet are key.

One of the challenges we face as veterinarians is the opportunity to diagnose this disease in the early stages. Cats are “masters of disguise” They also do not receive regular veterinary care. Often by the time we see the patient and diagnose the disease, the cat already has lost weight and muscle mass, has a poor hair coat, glucotoxicity, diabetic neuropathy and possibly is in DKA. Using every opportunity, a veterinary team has to teach cat owners the importance of early disease diagnosis by receiving regular veterinary care and teaching the subtle signs of illness is critical. The author recommends using Cat Healthy as a resource to educate every client that comes through our doors. In addition, once diagnosed with diabetes, the Cat Healthy website  http://www.cathealthy.ca has a series of educational videos about diagnosis, treatment and outcome for the newly diagnosed diabetic cat family. The Cat Healthy Protocols contain a compliance section which lists other useful resources for the family as they start the journey of insulin treatment and blood glucose monitoring for their cat. The earlier we diagnose and treat the disease, the better chance we have of remission.

The use of an ultra-low carbohydrate diet has been mentioned already. Low carbohydrate diets reduce post prandial hyperglycemia in people. It seems the importance of a low carbohydrate diet in the cat is equally important. A study giving twice daily showed a 12-week remission rate of 17% in cats fed diets with variable carbohydrate content and a 12-week remission rate of 40% in diabetic cats fed an ultra-low to low carbohydrate diet. The Bennett study reported a greater chance of remission in diabetic cats fed a low CHO diet than those fed a high fibre diet. Obesity is common in DM cats. If present, it should be addressed with a therapeutic weight-loss diet and an energy-restriction plan. Listed below in Table 1 are the calorie distribution of the veterinary prescription diets commonly fed to our diabetic patients.
Table 1. Calorie distribution (% of metabolizable energy from protein, fat and carbohydrates) and crude fiber content (g/1000 kcal) of U.S. feline diets for the management of diabetes mellitus (data from manufacturer's product guides)

<table>
<thead>
<tr>
<th>Name</th>
<th>Caloric distribution (% of metabolizable energy)</th>
<th>Crude fiber (g/1000 kcal)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hill's Prescription Diet m/d dry</td>
<td>43.3 41.5 15.2</td>
<td>11</td>
</tr>
<tr>
<td>Hill's Prescription Diet m/d canned</td>
<td>45.7 40.7 13.6</td>
<td>15</td>
</tr>
<tr>
<td>Royal Canin Veterinary Diet Diabetic dry</td>
<td>46 29 25</td>
<td>12.6</td>
</tr>
<tr>
<td>Royal Canin Veterinary Diet Diabetic canned</td>
<td>51 35 14</td>
<td>18</td>
</tr>
<tr>
<td>Purina Veterinary Diet DM dry</td>
<td>49.7 37.4 12.9</td>
<td>2.8</td>
</tr>
<tr>
<td>Purina Veterinary Diet DM canned</td>
<td>38.8 58 3.2</td>
<td>6.1</td>
</tr>
</tbody>
</table>

The clinician’s choice of insulin is always an interesting discussion. Cats can go into remission with the use of any insulin. The type of insulin used for the best chance at achieving remission may be less important than factors such as the presence of concurrent diseases, initiating the treatment as soon as possible and the plan for close monitoring. Diseases such as Acromegaly and Cushings disease can be causes of a lack of response to insulin. Co-existing pancreatitis can also have an effect on the blood glucose levels and requirement for insulin. Clinicians should be familiar with at least two types of insulins that are appropriate for treating cats as it is difficult to predict in advance which insulin is best for an individual cat. Glargine has been proposed as the
optimum insulin for diabetic cats based on the relatively high remission rate reported in some studies using this insulin, but this may be because it is the most frequently studied insulin. The predominant use of PZI in a study assessing the influence of low CHO diets achieved similar remission to a study examining twice daily glargine. Further studies are required to compare if there are different rates of remissions between the different insulins. The availability of a PZI insulin licensed for cats in Canada with a long expiry date is a benefit to our feline patients. The resources available with a veterinary licensed insulin is of great benefit to the veterinary team and the client. Listed in table 2 are the commonly used insulins for cats with diabetes in Canada.

Table 2. Comparison of insulin products for treatment of feline diabetes mellitus

<table>
<thead>
<tr>
<th>Insulin</th>
<th>Licensed in cats</th>
<th>Manufacturer</th>
<th>Formulation</th>
<th>Action</th>
<th>Dose*</th>
</tr>
</thead>
<tbody>
<tr>
<td>ProZinc</td>
<td>Yes</td>
<td>Boehringer Ingelheim</td>
<td>U40 recombinant PZI</td>
<td>Nadir 5–7 hours Duration 8–9 hours</td>
<td>Start 0.25–0.5 U/kg, BID Median maintenance dose 0.6 U/kg, BID</td>
</tr>
<tr>
<td>Vetsulin, Caninsulin</td>
<td>Yes</td>
<td>Merck</td>
<td>U40 Porcine zinc</td>
<td>Nadir 4 hours Duration 8–12 hours</td>
<td>Start 0.25–0.5 U/kg, BID Median maintenance dose 0.5 U/kg, BID</td>
</tr>
<tr>
<td>Lantus</td>
<td>No</td>
<td>Sanofi Aventis</td>
<td>U100 Insulin glargine (recombinant human analog)</td>
<td>Nadir and duration not determined in diabetic cats</td>
<td>Start 0.25–0.50 U/kg, BID Median maintenance dose 2.5 U/cat, BID</td>
</tr>
<tr>
<td>Levemir</td>
<td>No</td>
<td>Novo Nordisk</td>
<td>U100 Insulin detemir (recombinant human analog)</td>
<td>Nadir and duration not determined in diabetic cats</td>
<td>Start 0.25–0.50 U/kg, BID Median maintenance dose 1.75 U/cat, BID</td>
</tr>
</tbody>
</table>

*Based on lean body weight
Teaching our clients to be comfortable to take blood glucose levels at home is critical for remission. “In clinic” blood glucose curves are inaccurate and a diagnostic method of the past. Having a few team members on staff that can guide the clients through the early stages of diabetic monitoring and treatment is critical and will greatly improve the chance of remission. Commonly used protocols are to “Spot Check”, do home blood glucose curves or multiple daily monitoring. What protocol is needed will be determined by the client’s schedule and lifestyle and the individual patient’s needs. It appears that remission is likely only achieved in those cats that received long term glucose monitoring.

In conclusion, the earlier we diagnose DM in our feline patients and initiate treatment with twice daily insulin in conjunction with an ultra-low carbohydrate diet, the better chance we have of diabetic remission. Teaching clients to monitor blood glucose levels at home is a critical part of the plan as well. Remission in the diabetic cat is possible! The majority of the studies referenced in these lecture notes were taken from an article in The Veterinary Journal which did a systematic review of twenty-two studies on factors influencing remission rates and predictors of remission in the feline. The journal article is referenced below.

References

RADIOGRAPHY IN THE ACUTE ABDOMEN
Anthony Pease, DVM, MS, DACVR

Abdominal radiographs are a rapid, readily available method to give an overview of the abdomen. Though most people believe ultrasound is the new modality of choice for abdominal evaluation, the limitations of ultrasound not being able to penetrate gas as well as the technical ability and time to acquire images still make abdominal radiographs a great first modality in the patient with acute abdominal pain.

Ultimately, the question for the clinician with an abdominal patient is whether surgery is indicated or if medical management is the best course of action. With radiographs providing an overview of the entire abdomen, and the use of the gas within the bowel to provide contrast, abdominal radiographs can be useful as a triage tool that can be augmented and finding further characterized using abdominal ultrasound.

When evaluating the stomach, generally most abdominal radiographs include a right lateral and ventrodorsal projection. The question always arises on why this is performed. These two views have become the standard since a right lateral projection places gas in the fundus of the stomach and fluid in the pyloric antrum. To evaluate the pylorus, a ventrodorsal projection is used to put fluid in the fundus and gas in the pyloric antrum. At Michigan State University, we take 3 view radiographs of all abdomens to include a right lateral to seen the fundus, a left lateral to evaluate the pylorus and look for pyloric outflow obstructions and a ventrodorsal to provide more information about the pylorus and to better evaluate the colon.

With the availability of ultrasound, the use of contrast medium for upper gastrointestinal contrast medium procedures is not routinely performed. However, in clinics without the benefits of ultrasound, barium or iodinated contrast medium procedures still provide some use to evaluate if a luminal obstruction exists, if the bowel wall is think or infiltrated, look at overall motility or assess for a rupture. The main drawback to this procedure is that if any of those differential diagnoses are suspected, an exploratory laparotomy is indicated rather than a contrast procedure that could delay surgery by 3-6 hours.

Barium contrast medium is the most universally used for gastrointestinal imaging. It is safe, the dose is 6-10 milliters per pound and generally is administered through a gastric tube. If aspirated, barium causes physical obstruction of the airways with no inflammatory component, but may cause granulomas if it leaks into the peritoneal or pleural cavity. For this reason, barium is contra-indicated if a ruptured bowel or ruptured esophagus is suspected. Iodinated contrast medium is generally used intravenously but can be administered orally. The main limitation is that it has a bad taste, is hypertonic so it will draw fluid into the bowel and since it is hypertonic, will cause an inflammatory reaction if aspirated into the lungs.

Positional radiography can also be used to evaluate for free gas in the abdomen. Since an air/fluid interface is needed to help to see gas within the peritoneal space, a horizontal
beam projection with the dog on its left side and obtaining a ventrodorsal projection will put the gas in the right lateral abdomen near the pyloric antrum. Since the pylorus is small, the gas accumulation will be identified caudal to the diaphragm.

For gastric dilation with volvulus, the main feature is to obtain a right lateral radiograph. No other projection is needed. If the pylorus is seen in the craniodorsal abdomen, a GDV is confirmed. Numerous times people have been fooled by the normal appearance of the ventrodorsal projection and decided the case was just gastric dilation. Nothing else can put the pylorus in the craniodorsal abdomen except for a GDV.

Small intestinal wall thickness is also something frequently evaluated on survey radiographs. This cannot be done. Since soft tissue and fluid are the same opacity, it is impossible to know if the structure observed is a thick wall or just a combination of fluid summating with the small intestinal wall.

The abdomen is divided into two spaces, peritoneal and retroperitoneal. The retroperitoneal space contains the adrenal glands, kidneys and sublumbar lymph nodes and the peritoneal space contains the remaining organs. This determination is important since it will aid in the differential diagnoses of a mass that is present or the cause for gas within the abdomen. The retroperitoneal space is dorsal to the colon. Therefore if a soft tissue mass displaces the colon ventrally, then the mass is likely retroperitoneal indicating it is either arising from the kidney or adrenal glands. If gas is present in the retroperitoneum, this is likely secondary to a pneumomediastinum rather than a rupture of the gastrointestinal tract.

Radiographs are useful to determine if a surgical obstruction or mass is present or at least provides a general overview of the abdomen. Though barium contrast medium can be used, this has largely been replaced with ultrasound or exploratory surgery. By the end of this lecture, the audience will seen numerous examples of radiographs for surgical and non-surgical lesions and how a better understanding of the limitations and benefits of abdominal radiography.
THORACIC RADIOGRAPHY CASES: REAL WORLD EXAMPLES THAT MAKE YOU THINK
Anthony Pease, DVM, MS, DACVR

Thoracic radiographs are the mainstay of diagnostic imaging. The debate between two view and three view thoracic radiographs may continue, but no one argues that imaging the thoracic is the most complicated and more informative radiographic procedure available. With the contrast provided by the lungs, soft tissue opacities and radiographic changes within the lungs are easy to see, but hard to interpret. By far, a normal thoracic radiograph is still the most difficult to interpret.

Radiographic technique and positioning is the most important thing to thoracic radiographic interpretation. The first priority is the proper radiographic technique. Due to respiratory motion, the kVp setting is set high (generally 100 or 120) and the mA is also maximized to keep a small exposure time to minimize motion artifacts. Recumbency is also a major factor in radiographic interpretation. The lung needs to be aerated in order to see radiographic changes since the soft tissue opacity of the lesion needs to contrast with the aerated lung. Therefore if a lesion is in the right cranial lung lobe, then a left lateral radiograph is needed. Alternatively if the lesion is in the left caudal lung lobe, especially in the dorsal aspect, a dorsoventral projection should be performed.

Once the radiograph is obtained, the next step is to determine if the lungs are too white, too dark or normal. The second question is if this change is secondary to technique or pathology. To determine if the increased opacity is secondary to technique, one should evaluate the degree that the first and second thoracic spinous process can be seen, also the degree of contact between the diaphragm and the heart as well as the the ability to see the pulmonary vasculature and the superimposed triceps musculature on the thoracic inlet.

Once you decide a lesion is present, the next debate that is currently going on is the importance of pulmonary patterns versus location. Pulmonary patterns are divided into alveolar, interstitial and bronchial lung patterns. These patterns were based on air bronchograms, increased opacity to the lung fields or increased thickness of the bronchial wall creating increased lines and rings, respectively. That said, generally, it is easier to consider the radiographic pattern as a degree of severity with alveolar being the most severe, interstitial is moderate and bronchial being mild pulmonary disease. The alternative way to evaluate the lungs is to decide on the location and the distribution of the pathology identified.

For location, you can divide pulmonary disease into cranioventral, caudodorsal or diffuse disease. Cranioventral disease has 3 differential diagnoses: bronchopneumonia, hemorrhage or neoplasia. If it is caudodorsal there are 2 differential diagnoses: cardiogenic and non-cardiogenic pulmonary edema. Diffuse can be any of the five diagnoses. If the lesion is not occupying a lung lobe and is more structured, it can have a focal or multifocal distribution. A focal pulmonary lesion can be a tumor, granuloma, abscess or bulla (if radiolucent), whereas multifocal lesions tend to be neoplasia, fungal
granulomas or pulmonary osteomas (which are < 5 mm soft tissue to mineral opacities throughout the lungs, generally seen in Collies).

If pleural fluid is present, retraction of the lung lobes away from the body wall can be seen. In cats, if this retraction remains after the fluid is removed, restrictive cardiomyopathy is considered most likely. If a cranial mediastinal mass is suspected, a standing horizontal beam radiograph can be obtained with the dog or cat standing on their hindlimbs and a ventrodorsal projection obtained to cause the fluid to be caudal to the heart. If pleural fluid is seen, the first thing to evaluate is the ribs, as rib tumors are a frequent, overlooked cause for pleural fluid. Also, radiographs can help identify a site to obtain a sample of the fluid, which can provide insight to the cause.

The cardiovascular structures of the lungs can also be evaluated to provide further information if a cardiogenic pulmonary edema is suspected. The cardiac silhouette is comprised of the heart and the blood within the heart as well as the surrounding pericardium. Since fluid and soft tissue have the same opacity, a difference between these structures cannot be identified. If the heart is enlarged, generally chamber enlargement is seen such as the left atrium or right atrium. Cardiac changes are generally vague and only occur when the changes are severe. When the heart hypertrophies, it undergoes concentric or eccentric hypertrophy. Concentric hypertrophy is secondary to a pressure overload. If the heart can compress hard enough, it can push the blood out of the chamber. The heart then hypertrophies the muscle to create a smaller lumen. The heart shape remains the same and therefore cats with hypertrophic cardiomyopathy and dogs with pulmonic or subaortic stenosis will not have radiographic signs of cardiomegaly until the disease is very advanced. Alternatively, eccentric hypertrophy is secondary to a volume overload. No matter how strong the contraction, the fluid cannot clear the chamber so the hypertrophic muscle is formed on the outside of the lumen. This change can be seen radiographically, but is a rare condition, mainly occurring with dilated cardiomyopathy.

Pulmonary vasculature can also be evaluated to help to determine the cause for a caudodorsal lung pattern. If the pulmonary artery is dilated, the primary cause is pulmonary hypertension from any cause. In adult dogs, the main cause is secondary to heartworm disease or pulmonary thromboembolic disease. If the pulmonary vein is enlarged, then generally it is a sign of left-sided heart failure. This vein enlargement is first seen in the right caudal lung lobe and then progresses to the remaining lung lobes with time. If both the arteries and veins are enlarged, then that is caused by over circulation, such as a patent ductus arteriosus or ventricular septal defect. Small vasculature is a rare finding, but may be secondary to hypovolemia, hypoadrenocorticism or severe pulmonic stenosis.

Radiographic interpretation of cardiac disease is considered difficult and numerous studies have tried to identify the easiest methods to simplify the interpretation. Using vertebral heart score, inverting the image so that black is white and white is black, even rotating the image to look for rib lesions. All these methods have found that nothing is better than experience at image interpretation and practice. In addition, with the rapid
expansion of digital imaging, bronchial lung patterns are being over diagnosed due to the increased image resolution. Having normal radiographs and evaluating the entire image, included the surrounding musculature and skeletal structures is essential to make accurate diagnoses.

Thoracic radiography is considered a challenging region to interpret not because the lesions are difficult to see, but rather because the lesions identified are generally non-specific and are difficult to interpret. Generally, most practitioners see a cranioventral alveolar lung pattern and diagnose aspiration pneumonia and a caudodorsal lung pattern as pulmonary edema. In truth, the thoracic radiographs should be evaluated as a whole, is there a megaesophagus or history of vomiting? Is there are heart murmur, enlarged pulmonary veins or enlargement of the left atrium of the heart? These questions should be asked prior to starting therapy with the hope the diagnosis is correct. Thoracic radiographs are not obtained to determine if a disease is present, but rather to identify the extent of disease and determine the progression or regression. Bearing in mind that radiographic improvement may lag behind clinical improvement by several days.

Although thoracic radiography is challenging, this lecture will provide an overview of normal anatomy as well as case examples of common disease processes to help provide the participant with an increase knowledge and level of comfort interpreting pulmonary and cardiac changes.
AGGRESSIVE VS. NON-AGGRESSIVE BONE LESIONS
Anthony Pease, DVM, MS, DACVR

The evaluation of the musculoskeletal system is difficult due to the numerous soft tissues as well as the bone structures involved. Rapid assessment of the bone structure is routinely performed using radiographs; however, the subtlety of disease and joint compared to bone pathology can be confusing. The purpose of this lecture is to cover the identification of aggressive compared to non-aggressive bone lesions as well as erosive compared to non-erosive joint pathology.

When evaluating the skeletal system, the first thing to determine is if the lesion is aggressive or non-aggressive. A non-aggressive lesion diagnoses include callous, malunion fractures, bone cysts, osteomas, osteochondritis dessicans, panosteitis, fragmented medial coronoid process, osteoarthritis or metabolic disorders. Aggressive lesions are due to neoplasia or osteomyelitis.

When deciding about aggressive lesions, there are 6 radiographic signs that are used: bone lysis, periosteal reaction, rate of progression, zone of transition, cortical lysis. Bone lysis has three different appearances, geographic (focal) moth-eaten and permeative. The difference between the degree of lysis is mainly on the rate of progression. It requires approximately 50% of the bone per unit area to be destroyed before it is visible on radiographs. This is because the bone is a three dimensional object viewed from two dimensions. Because of this, bone is superimposed on itself, making subtle lesions hard to detect. The more lysis that is present, the easier it is to see on radiographs. Also, by the time lysis is seen on a radiograph, the lesion is quite severe.

Periosteal reaction can either be smooth (continuous) or interrupted. The easiest way to determine this is if you could trace the outline of the periosteal reaction with a pencil and never have to lift the pencil from the radiograph. Smooth periosteal reactions are generally associated with trauma whereas interrupted periosteal reactions are due to an aggressive process.

Rate of progression is probably the most overlooked method to assess an aggressive lesion. By the time a questionable aggressive lesion is seen on a radiograph, the lysis is quite substantial. Therefore, the rate of progression in 2-4 weeks will also be dramatic. If a question exists between an aggressive and non-aggressive lesions, supportive medical management for 2-4 weeks then repeat radiographs to look for progression can aid in determining if the lesion is aggressive.

Zone of transition is a more nebulous sign, but the idea is that if a clear-cut demarcation between normal and abnormal bone is seen, then the lesion is more likely non-aggressive. If there is a long zone of transition, the difference between normal and abnormal bone is blurred and the lesion is more likely to be aggressive. In addition, cortical lysis as opposed to overall bone lysis can be used to determine aggressive bone lesions. If the cortex is thin, but no lysis is present, then it is more likely that the lesion is non-aggressive.
After determining these radiographic signs, the next clue is based on the location of the lesion. If the lesion is generalize in that it affects all bones equally, then the primary differential diagnosis is a metabolic or nutritional abnormality. If only one bone is involved, this is a focal or monostotic lesion and a primary bone tumor or soft tissue tumor with secondary bone involvement is considered most likely. If multiple bones in the same region (locally extensive), different bones that are not in close proximity or multiple areas in the same bone are involved, this generally indicates a hematogenous spread disease as bacterial osteomyelitis or metastatic neoplasia. A soft tissue tumor with secondary bone involvement is possible with locally extensive lesions, such as aggressive lesions that cross a joint.

Anatomic location is also a key into the differential diagnoses. If the lesion is epiphyseal or physeal in origin, then it is likely secondary to infection, trauma or potentially a nutritional abnormality. These lesions are generally in juvenile dogs and cats. If the lesion is in the metaphyseal region, then a primary bone tumor or hematogenous infection is most likely due to the proximity of the nutrient foramen. If the lesion is diaphyseal, then the lesion is likely metastatic neoplasia, a soft tissue mass with secondary bone involvement or a focal infection related to a penetrating trauma.

After all these signs and locations are taken into account, then the differential diagnoses are prioritized based on the signalment and history of the patient. A 2 year old hunting dog with an aggressive bone lesion in the proximal metaphysis of the humerus is more likely to have a fungal infection; however an 8 year old Rottweiler with the exact same radiographic findings is more likely to have osteosarcoma. These considerations should be made when assessing aggressive lesions. Since osteosarcoma is a common tumor type, it is not uncommon for clinicians to see an aggressive lesion, even if it is locally extensive and crosses a joint, and consider a primary bone tumor like osteosarcoma. However, other tumors such as malignant histocytosis, synovial cell (histocytic) sarcoma, or even fibrosarcoma, chondrosarcoma and metastatic neoplasia can all be considered possible. Biopsy (excisional or incisional), thoracic radiographs and history may aid in further prioritizing the lesion.

Lesions centered on joints are similar to those in bone. These lesions are centered on the epiphysis of both sides of the joint. Just as aggressive and non-aggressive lesions exist in bone, erosive and non-erosive lesions are in joints. A non-erosive lesion is osteoarthritis. Everything else is considered erosive. Osteoarthritis is a degenerative condition due to joint instability or trauma. It is characterized by the presence of osteophytes and enthesiophytes. An osteophyte is smooth bone production within the joint capsule that serves as a buttress to tighten ligaments and stabilize the joint. Enthesiophytes are bone production at the attachment of the joint capsule and ligaments due to abnormal tension that is present on the soft tissues from joint instability.

Erosive lesions in small animals are usually infectious and mostly autoimmune in origin. Causes of erosive arthropathy also include chronic hemarthrosis or neoplasia, but these are less likely in small animals. Just as with bones, joint lesions are characterized by the number involved. A monoarthrosis (one joint) is usually osteoarthritis or a traumatic
infection, such as a puncture wound. A polyarthritis (multiple joints involved) usually indicates a hematogenous infection or immune mediated disease.

The radiographic signs for an erosive arthropathy include subchondral bone lysis, presence of osteoarthritis, decreased joint space (especially when weightbearing), luxation or subluxation of the joint and fragmentation of adjacent bone. Based on these signs, and the presence of one or multiple joints involved, an arthrocentesis can be performed to determine the cause for the erosive arthropathy.

Radiographic findings of joint and bone lesions can be confusing if one does not consider the vast number of differential diagnoses possible and then makes an educated decision to prioritize the lesion. This is generally done by the clinician automatically due to the geographic location and the likelihood of disease in a given area. If a dog in southern Michigan presents to Michigan State University for an aggressive bone lesion, neoplasia is more likely. However, if the patient is from northern Michigan, then fungal osteomyelitis should be considered possible. At the end of this lecture the hope is that the veterinarian will have numerous examples and a better overall appreciation of how to evaluate a radiograph for aggressive and erosive lesions.
A PAIN IN THE NECK: DIAGNOSING AND TREATING NECK AND BACK PAIN IN SMALL ANIMALS

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North Carolina State University

Neck and back pain are common presenting complaints in veterinary medicine and occur in animals with a variety of signalments. Animals may present with a chronic history of lower-grade discomfort, although acute presentations of moderate to severe pain are common and extremely distressing to owner and pet alike. Pain may originate from a variety of locations associated with the spine, including vertebrae, nerve roots, meninges and possibly the intervertebral disk. A variety of etiologies are possible, although most patients fit within “the big 5” causes listed below.

Neck and Back Pain – The Big Five

1) Intervertebral disk disease (IVDD)
2) Diskospondylitis
3) Meningitis and meningomyelitis
4) Trauma
5) Neoplasia

Neck and Back Pain – Other Etiologies

6) Lumbosacral disease (LSD)
7) Caudal cervical spondylomyelopathy (CCSM, “Wobbler’s” syndrome)
8) Atlantoaxial subluxation (AAS)
9) Caudal occipital malformation syndrome (COMS, “Chiari-like” malformation)
10) Space-occupying intracranial disease (e.g., Brain tumor)
11) Spondylosis deformans (rare cause of neck or back pain)
12) Polyarthritis (rare cause of neck or back pain)
13) Polymyositis (rare cause of neck or back pain)

Diagnostic Considerations for Patients with Spinal Pain

The diagnosis of the cause underlying neck and back pain revolves around imaging of the spine and spinal cord and examination of cerebrospinal fluid (CSF). Survey radiographs of the affected area are the typical starting point, and are usually diagnostic in cases of diskospondylitis, revealing lysis and malformation of the vertebral endplates next to the affected disk space. Discovery of diskospondylitis should initiate radiographic examination of the entire spine, as multiple lesions are often seen. Bacterial culture of the urine and blood and serum titers for infectious organisms (e.g., *Brucella canis, Aspergillus spp.*) may help to determine the offending organism and site of origin. Examination of urine sediment may reveal fungal hyphae in some *Aspergillus* infections. Fractures and luxations of the spine are usually seen with plain radiographs, although the changes may be subtle in some cases. Atlantoaxial subluxation is usually obvious, demonstrated by an increased distance between the dorsal arch of C1 and the dorsal spinous process of C2. Occasionally radiographs taken with the neck in mild flexion may facilitate the diagnosis, but should be obtained with extreme caution (particularly in anesthetized animals) to avoid further spinal cord damage. The dens (odontoid process) is
often hypoplastic or absent in these animals, a finding which can also facilitate diagnosis. Neoplasms involving the bone may show lytic and/or proliferative changes on survey radiographs if a sufficient portion of the bone is involved. Animals with IVDD often show typical changes on radiographs, including calcified disks in situ, narrowed intervertebral disk spaces and radiodense material overlying the intervertebral foramen, although definitive diagnosis usually requires additional imaging techniques.

Additional diagnostic imaging techniques applicable to spinal disease include contrast studies (e.g., myelography, epidurography), nuclear scintigraphy, computed tomography (CT) and magnetic resonance imaging (MRI). Myelography demonstrates compressive spinal lesions, such as IVDD, CCSM, and neoplastic disease. A CSF sample should be collected before performing myelography, as the contrast material creates an inflammatory reaction, complicating later analysis. Myelography is rarely useful for LSD, as the spinal cord typically ends over L5-6 in the dog. Epidurography may show compression of the cauda equina, but is rarely used with the current availability of CT and MRI.

CT can be useful in LSD or after myelography to further delineate the location and character of compressive lesions. CT is much more sensitive than survey radiography, and may show obvious evidence of disease in animals with neoplastic lesions or diskospondylitis when radiographs are equivocal. MRI is superior to other imaging techniques for soft tissue disease involving the spinal cord or nerve roots, and is indispensable for certain conditions, such as COMS.

Analysis of CSF is critical for making a diagnosis of meningomyelitis, and typically shows elevations in cell counts (pleocytosis) and protein levels. Specific serum titers or other tests (e.g., PCR) for infectious diseases may be considered for animals with meningomyelitis. Most compressive spinal cord disease will result in elevations of protein levels without pleocytosis. Occasionally neoplastic cells are seen in CSF, although this is rare. If AAS is a consideration, survey radiographs must be performed before collection of CSF from the cerebellomedullary cistern to avoid serious spinal cord injury.

Electromyography (EMG) and other electrodiagnostic tests are occasionally useful in the identification of focal spinal lesions or to demonstrate neuromuscular diseases (e.g., polymyositis).

**Treatment Considerations for Neck and Back Pain Patients**

Compressive disease involving the spinal cord (e.g., IVDD, AAS, CCSM, LSD, fractures/luxations, neoplasia) and/or nerve roots is best treated with surgical decompression in many cases. Certain of these conditions, such as fractures, AAS and in some cases CCSM and LSD, require surgical stabilization as well. However, many patients with compressive diseases can be managed medically, usually with a combination of strict confinement, analgesics and occasionally glucocorticoids. NSAIDS are often ineffective for nerve root pain, although they may be useful for lesions of the spine itself. NSAIDS should not be used in combination with glucocorticoids. Diazepam and methocarbamol are useful to control the pain associated with muscle spasm secondary to nerve root compression. Gabapentin can be very useful for pain of neuropathic origin. Ketamine and amantadine are NMDA receptor antagonists that can be useful adjuncts to address some of the pathophysiologic processes associated with pain (“windup”), and may be beneficial for acute and chronic pain patients respectively. Pregabalin is a relatively new medication with a mechanism of action similar to gabapentin, but with a greater potency. Experience with veterinary patients is relatively limited thus far, although preliminary
results in some patients with neuropathic pain are encouraging. Combinations of the above medications can take advantage of multiple mechanisms of action and may be more effective than single medications. Acupuncture can also be a useful adjunct, particularly in chronic cases.

Appropriate antibiotic therapy (ideally directed by culture and sensitivity) for a minimum of 6-8 weeks is required for diskospondylitis cases. Meningomyelitis may require specific antimicrobial therapy if an infectious etiology is identified. Patients without an apparent infectious etiology usually respond well to glucocorticoid therapy, which is tapered to the lowest controlling dose, and may eventually be discontinued in some cases. Additional immunosuppressive or cytotoxic drugs may be useful in animals not controlled with glucocorticoids alone, or intolerant of their side effects. Animals with neoplasia involving the spine, meninges, nerve roots or spinal cord may benefit from surgical debulking and radiation therapy, or chemotherapy in some cases.

**Some Potentially Beneficial Medications in the Therapy of Neck and Back Pain in Small Animals**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose/Route</th>
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<tbody>
<tr>
<td>Diazepam</td>
<td>2-10 mg/animal PO q 8 hours</td>
</tr>
<tr>
<td>Methocarbamol</td>
<td>15-20 mg/kg PO q 8 hours</td>
</tr>
<tr>
<td>Gabapentin</td>
<td>3-10 mg/kg PO q 6-12 hours</td>
</tr>
<tr>
<td>Pregabalin</td>
<td>2-4 mg/kg PO q 8-12 hours (start at low end &amp; escalate as needed)</td>
</tr>
<tr>
<td>Ketamine</td>
<td>0.5 mg/kg bolus IV, then 0.1-0.3 mg/kg/hour constant rate infusion</td>
</tr>
<tr>
<td>Amantadine</td>
<td>1.25-4 mg/kg PO q 12-24 hours</td>
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<tr>
<td>NSAIDs</td>
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</tbody>
</table>

**References**

CATS ARE NOT SMALL DOGS: FELINE NEUROLOGY IN A NUTSHELL

Christopher L. Mariani, DVM, PhD, DACVIM (Neurology)
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Overview and Objectives

As most of us were taught early in veterinary school, cats are not small dogs; this is as true for neurologic patients as it is in other veterinary fields. This talk will review how cats are unique in terms of the diseases that affect their nervous systems with a focus on practical aspects of the neurologic examination, generation of a differential diagnosis, diagnostic evaluation and therapeutic interventions.

Presentation and Neurologic Examination

Veterinarians generally see fewer purebred cats than dogs and therefore breed-specific feline diseases are often less familiar to us. However, it is important to realize that certain signalments may be quite informative in arriving at a final diagnosis. For example, paraparesis and urinary and/or fecal incontinence in a Manx cat should alert the clinician to the possibility of a spinal cord anomaly (e.g., meningomyelocele). Progressive neurologic dysfunction in a young (< 1 year-old), purebred cat may increase the suspicion for a neurodegenerative disorder (e.g., storage disorder), although these are rare diseases.

Although cats are certainly highly variable in their receptiveness to examination, in general cats will tolerate fewer of the examination procedures routinely used in dogs. As a result, observation of a patient’s gait and behavior, which is important in dogs, becomes even more crucial in cats. Such observation is important to assess the cat’s mentation and interaction with its environment, as well as to look for evidence of gait abnormalities such as ataxia, paresis or lameness. Most of the cranial nerve testing performed in dogs can be completed in cats. However, postural reaction testing is more challenging. Some cats will allow assessment of proprioceptive placing (“conscious proprioception”) although many will simply lift up their feet when touched or resent handling of their feet. Such observation is important to assess the cat’s mentation and interaction with its environment, as well as to look for evidence of gait abnormalities such as ataxia, paresis or lameness. Most of the cranial nerve testing performed in dogs can be completed in cats. However, postural reaction testing is more challenging. Some cats will allow assessment of proprioceptive placing (“conscious proprioception”) although many will simply lift up their feet when touched or resent handling of their feet. Similarly, hopping can be performed in many cats, although a cat will often drop to their side or roll after a few hops. Wheelbarrowing and extensor postural thrust are usually reliable and can be used to detect discrepancies in one side versus the other. Segmental spinal reflexes can be assessed in most cats, and the most reliable are the patellar and withdrawal reflexes. In dogs the cutaneous trunci reflex is best elicited using a pair of mosquito hemostats to pinch the skin but this seldom works in cats (other than to really irritate them!). A better technique is to pull on/pluck the hair over the back to produce the desired twitch of the cutaneous trunci muscle. Finally, palpation of the spine to detect discomfort can be accomplished in some cats although many normal cats will resent this test, particularly over the thoracolumbar region.

The principles of localization of neurologic lesions remain the same in cats as in dogs and other species. One point to note is that the spinal cord terminates further caudally in the cat than in the dog, usually extending down to L7 or even to the sacrum in some individuals.

Differential Diagnoses by Presenting Signs

Seizures
The list of differential diagnoses for seizures is relatively similar in the cat when compared to dogs with one large exception; cats do not develop any significant degree of genetic (“idiopathic”) epilepsy. Therefore, any cat with seizures would ideally receive a complete diagnostic workup including brain imaging and potentially cerebrospinal fluid (CSF) analysis. The main differentials for cats with seizures are metabolic disorders and inflammatory conditions (i.e., meningoencephalitis) although neoplastic, anomalous, traumatic and vascular etiologies must also be considered. Cats with hepatic encephalopathy can have intermittent seizures without other associated neurologic signs, a scenario that is very uncommon in dogs; these cats often have a striking, copper colored iris. Despite the concern for underlying metabolic or structural diseases, some cats with seizures do not have an identifiable cause, and are best classified as unknown (cryptogenic) epileptics.

**Altered Mentation**

Mentation changes usually indicate forebrain dysfunction, although a primary brainstem lesion is possible. Differential diagnoses to consider include metabolic disease (e.g., hypoglycemia, electrolyte disorders, hepatic encephalopathy), meningoencephalitis, neoplastic disease, cerebrovascular disease, trauma and toxins. Meningiomas are the most common primary brain tumor in older cats and typically lead to altered mentation as the primary clinical sign, often without seizures or other obvious neurologic signs.

**Vestibular Dysfunction**

Vestibular dysfunction is common in cats and can be caused by a variety of etiologies. Diseases leading to peripheral vestibular dysfunction (i.e., affecting the vestibular nerve or semicircular canals) include otitis media-interna, inflammatory polyps, neoplastic disease, trauma and toxins (e.g., chlorhexadine, aminoglycosides). An idiopathic peripheral vestibular syndrome has also been described. Central vestibular disease (i.e., brainstem lesion) can arise from inflammatory, neoplastic or vascular etiologies. Thiamine deficiency is rarely seen with the well-supplemented diets currently fed to most cats, but can cause brainstem dysfunction including vestibular signs.

**Cerebellar Dysfunction**

A classic lesion is cerebellar hypoplasia secondary to in utero infection with feline panleukopenia virus. This disorder manifests itself from the time of birth as profound ataxia, intention tremors and dysmetria without obvious paresis. Other causes of cerebellar dysfunction include meningoencephalitis, cerebrovascular accidents, neoplastic disease and neurodegenerative disorders.

**Ataxia, Tetraparesis & Paraparesis (Spinal Cord Dysfunction)**

Spinal cord lesions are relatively less common in cats when compared with dogs. The main reason for this discrepancy is the paucity of intervertebral disk disease (IVDD) in feline patients. When it occurs, IVDD typically presents as a chronic paresis in cats and acute disk extrusions are quite rare. Important differentials for cats with spinal cord lesions include meningomyelitis, neoplastic disease and less commonly vascular accidents involving the spinal cord. Of course, thromboembolic events affecting the vasculature of the limbs (e.g., distal aorta or “saddle” thrombus) must be kept in mind and appropriately ruled out.

**Spinal Pain**

Spinal pain is harder to appreciate in cats than in dogs as their signs are often nonspecific and include behaviors such as avoiding contact with owners or other pets, inappetence and lethargy or general
malaise. Disorders that can cause cervical or thoracolumbar pain in cats include meningomyelitis, diskospondylitis, intervertebral disk disease, trauma and neoplastic disease involving the vertebrae, spinal nerve roots or meninges.

Neuromuscular Disease

As in dogs, neuromuscular disorders are uncommonly diagnosed, but are underappreciated. These disorders encompass peripheral neuropathies, neuromuscular junction disorders and myopathies. One classic presentation is pelvic limb paresis characterized by a plantigrade stance, which is highly suggestive (but not pathognomonic) of poorly controlled diabetes mellitus. Most neuropathies present as chronic paraparesis or tetraparesis and may be first recognized by a reluctance of the cat to jump or vigorously play. There is typically no ataxia and segmental spinal reflexes may or may not be appreciably depressed. Differential diagnosis includes metabolic disorders, paraneoplastic polyneuropathy, infectious disease (e.g., Toxoplasmosis) and immune-mediated polyneuropathy. Acute onset of severe, progressive (ascending) tetraparesis can be seen with several disorders including acute polyradiculoneuritis ("Coonhound paralysis"), tick paralysis and coral snake envenomation. Myasthenia gravis can also occur in cats and causes weakness that typically worsens with activity. In cats this disorder is frequently associated with a cranial mediastinal mass (typically a thymoma) and has also been associated with methimazole therapy for hyperthyroidism.

Diagnostic Considerations

A complete blood count, serum biochemistry and urinalysis are often the first tests to consider in cats with neurologic disorders. These tests can help to identify metabolic disorders, certain infectious diseases and occasionally neoplastic conditions and are also useful as a preanesthetic workup if advanced diagnostic testing is pursued. Bloodwork also serves as an important initial baseline in animals receiving anticonvulsant therapy (particularly phenobarbital). Radiographs of the spine are an important diagnostic test for cats presenting with signs of spinal cord disease or spinal pain, and may show lytic or occasionally proliferative changes in patients with neoplastic or infectious conditions (e.g., diskospondylitis) affecting the vertebrae. Imaging of the thoracic and abdominal cavities (radiographs and/or ultrasonography) can be useful to further define systemic diseases or to detect metastases or primary neoplasms. Additional tests available to the general practitioner include specific metabolic tests (e.g., T4, serum bile acids, fructosamine), infectious disease tests (e.g., Toxoplasma gondii titers, coronavirus [FIP] assays), coagulation testing, blood pressure evaluation and assays for specific toxins (e.g., ethylene glycol, THC, lead).

Despite the utility of the tests discussed above, advanced diagnostic testing is frequently required in order to arrive at a diagnosis in cats with neurologic dysfunction, and such testing often requires referral to a specialty hospital. As in other species, the main neurodiagnostic testing modalities are imaging, CSF analysis and electrodiagnostic testing. Magnetic resonance imaging (MRI) is almost always the preferred imaging test to evaluate the brain, spinal cord and associated structures, although computed tomography (CT) also has some utility. Myelography can identify spinal cord compression, and can be used in conjunction with CT. Cerebrospinal fluid analysis is primarily used to document inflammatory CNS conditions, although there are nonspecific changes seen with compressive diseases and on rare occasions neoplastic cells can be identified (e.g., lymphoma). Electrodiagnostic tests include electromyography (EMG), assessment of nerve conduction velocity and repetitive nerve stimulation and are primarily used to investigate neuromuscular diseases although electroencephalography (EEG) is occasionally helpful in defining seizure disorders. Finally, biopsy of muscle and/or nerve tissue for histopathological analysis can be very helpful in documenting peripheral neuropathies or myopathic disease.
Therapeutic Considerations

When considering traditional anticonvulsant medications, phenobarbital is the most frequently used medication, is relatively well tolerated and is effective in controlling seizures in most cats. Although an effective anticonvulsant, bromide causes inflammatory lung disease in a large proportion of cats and is not recommended for feline patients. Oral diazepam can also be effective as a maintenance anticonvulsant in cats but is not recommended due to a rare but devastating adverse effect (idiosyncratic hepatic necrosis). Several newer generation anticonvulsants can be utilized for seizure control in cats including levetiracetam, pregabalin, gabapentin and zonisamide.

If infection of the CNS is a potential concern, the antibiotics chosen would ideally achieve high concentrations within the CNS as well as show efficacy against documented or suspected organisms. Sulfa medications (trimethoprim-sulfa and related drugs) are a good option, as they have good CNS penetration, a broad spectrum of activity and activity against protozoal organisms (e.g., *Toxoplasma gondii*). Clindamycin can also be used effectively although it has relatively poor CNS penetration. Doxycycline is another good choice with a broad antibacterial spectrum as well as activity against rickettsial organisms. When treating infections involving bony structures (e.g., diskospondylitis or otitis media-interna) antibiotic therapy should be continued for at least 2 weeks past the clinical resolution of signs and for a minimum of 8 weeks.

Glucocorticoid therapy is helpful to control inflammation in patients with inflammatory CNS disease or to reduce peritumoral edema in patients with brain or spinal cord tumors. Nonsteroidal anti-inflammatory drugs (NSAIDS) can be effective in some cats to control pain associated with vertebral or nerve root lesions; NSAIDS and glucocorticoids should never be used together in these patients. Gabapentin or pregabalin can be quite helpful as adjunctive analgesic therapy for cats with painful conditions.

References

Definitions

Seizures are an important clinical problem in both dogs and cats and account for a substantial proportion of admissions to both general and referral veterinary hospitals. Although most veterinarians are very familiar with seizures, it helps to define this term explicitly as follows:

Seizure – the clinical manifestation of excessive and/or hypersynchronous neuronal discharge within the brain; may be manifested as episodic impairment or loss of consciousness, abnormal motor phenomena, psychic or sensory disturbances, or autonomic signs such as salivation, vomiting, urination or defecation. Synonyms – convulsion, ictus, fit.

Seizures have been classified in the past according to the areas of the brain that they affect. Thus, they have been termed generalized or focal (partial) seizures, defined as follows:

Generalized – abnormal electrical discharges affecting the cerebral hemispheres bilaterally and typically causing symmetric signs. Several manifestations are possible, including generalized tonic-clonic (“Grand mal”), or exclusively tonic, clonic or atonic activity.

Focal (Partial) – abnormal electrical discharges of neurons in a focal area of the brain; may result in asymmetric motor or sensory signs such as twitching of the eyelids, lips, ears or one limb. Focal seizures may secondarily generalize to involve both hemispheres; that is, the electrical activity spreads across the cerebral cortex. This often gives rise to focal motor activity, which then evolves; this can often be recognized by owners.

Epilepsy – a chronic neurologic condition characterized by recurrent seizures, with an intracranial origin.

Seizures versus Seizure Impostors

A number of other clinical conditions can mimic seizures, including syncope, acute vestibular dysfunction, tremors, narcolepsy/cataplexy, rapid eye movement (REM) behavior disorder, movement disorders (e.g., distemper myoclonus), and behavioral disorders. The majority of patients presenting for seizures are normal in the veterinary hospital, and the clinician’s first task is to decide whether the events in question are true seizures, or seizure “mimics”. Some guidelines for this determination follow:

- Seizures frequently involve alterations in consciousness and autonomic signs
- Most seizures occur when animals are at rest or sleeping; syncope and narcolepsy/cataplexy occur predominantly with exercise or excitement
- Animals with syncope and narcolepsy/cataplexy have brief episodes with loss of consciousness, but are usually normal on recovery. Animals with seizures and loss of consciousness usually have post-ictal alterations of mentation
• REM behavior disorder occurs during sleep, but the animal is normal when awakened/roused
• Vestibular dysfunction usually has characteristic signs including head tilt and nystagmus
• Animals with tremors, movement disorders, and behavioral disorders will have a normal mentation and level of consciousness

Differential Diagnosis for Seizures

Once the clinician is reasonably certain that the events are true seizures, differential diagnoses may be considered. These can be broadly categorized into extracranial and intracranial etiologies. A seizure disorder indicates dysfunction of the forebrain (cerebrum or diencephalon). Extracranial causes of seizures originate from outside the central nervous system, causing dysfunction in an otherwise normal brain, and include metabolic, nutritional and toxic causes. Intracranial etiologies cause structural alterations of the forebrain itself, and include degenerative, anomalous, neoplastic, inflammatory, infectious, idiopathic, and traumatic causes.

Extracranial Causes of Seizures

• **Hypoglycemia** – main causes are juvenile hypoglycemia, hyperinsulinemia (secondary to insulin overdose or an insulin-secreting tumor), and sepsis.
• **Hypocalcemia** – causes include eclampsia, hypoparathyroidism and ethylene glycol intoxication.
• **Hyponatremia** – results from loss of sodium rich fluids (e.g. vomiting, diarrhea, diuretics), hypoadrenocorticism or increased water intake (psychogenic polydipsia).
• **Hypernatremia** – results from the loss of free water or sodium poor fluids (diabetes insipidus, excessive panting, high temperatures), decreased water intake (primary adipsia or limited access) or rarely through ingestion of high levels of salt.
• **Hepatic encephalopathy** – results from altered filtration of gastrointestinal portal blood by the liver. Main causes are the presence of an anomalous portosystemic shunting vessel or severe parenchymal hepatic disease with secondary shunting.
• **Hypertriglyceridemia** – primarily a disease of miniature Schnauzers, which have a congenital enzyme deficiency, allowing triglyceride accumulation in the blood.
• **Nutritional disease** – rare cause of seizures; historically thiamine deficiency has been implicated.
• **Intoxication** – a wide variety of toxins may lead to nervous system dysfunction and seizures. Examples include ethylene glycol, lead, heavy metals, metaldehyde, strychnine, and organophosphates.

Intracranial Causes of Seizures

• **Degenerative disease** – very rare causes of seizures; include lysosomal storage disorders and neuronal abiotrophies
• **Anomalous conditions** – hydrocephalus is the most common and occurs predominantly in toy and brachycephalic breeds. Less common conditions include epidermoid, dermoid, and arachnoid cysts, and lissencephaly.
• **Neoplasia** – common cause of seizures in older animals. Brain tumors may be primary (arise from brain itself) or secondary (metastatic or arise from adjacent structures [e.g., skull]). It is common to see seizures as the sole clinical sign of a brain tumor.
• **Inflammatory disease** – common cause of seizures in animals of any age. Encephalitis may be infectious (viral, fungal, protozoal, bacterial, rickettsial, or parasitic) or more
frequently, non-infectious. Common non-infectious causes in dogs include granulomatous meningoencephalitis (GME) and necrotizing meningoencephalitis.

- **Idiopathic disease** – common cause of seizures – see below.
- **Traumatic disease** – seizures can occur immediately at the time of head trauma or as a late sequela after recovery.

**Idiopathic (Genetic) Epilepsy** – Idiopathic epilepsy denotes “a syndrome unto itself”; that is, a well-recognized clinical condition of recurrent seizures without other neurologic signs and without any identifiable underlying cause. It is considered to be an inherited or familial condition, although the details of the genetic transmission are unknown in veterinary medicine except for a few families of dogs. Although somewhat controversial, inherited epilepsy appears to be very rare in cats.

Seizures have generally been considered to be of the generalized tonic-clonic variety, although this is not always be the case. The seizures almost always begin between 1 and 5 years of age, although a few dogs may have an onset between 6 and 12 months or up to 7 years of age. One of the hallmarks of idiopathic epilepsy is its insidious onset; seizures initially occur weeks to months apart and gradually become more frequent. Many dogs progress to develop cluster seizures or status epilepticus. In rare cases, this may be the first known seizure activity. Although certain breeds of dogs are predisposed to the development of genetic epilepsy, this condition can probably occur in any dog.

In the absence of a genetic test, diagnosis is based upon an appropriate signalment and description of seizure onset and character, and by ruling out other potential etiologic diagnoses with appropriate tests (typically cerebrospinal fluid [CSF] evaluation and brain imaging). Outside of the ictal and immediate post-ictal periods, the neurologic examination should be normal.

**Unknown Epilepsy** – This condition is synonymous with acquired, cryptogenic, and probably symptomatic epilepsy. It implies that an acquired structural lesion within the brain is suspected to be causing the seizures, but cannot be detected with the available diagnostic tests (e.g., patients with a previous intracranial disease such as head trauma or meningoencephalitis that have since recovered, but are left with recurrent seizures because of assumed scarring or fibrosis within the brain). Seizures may be focal, focal with secondary generalization, or generalized, and may occur at any age. All breeds of dog and cat can be affected. Although occasional focal neurologic deficits (e.g., reduced conscious proprioception on one side) are noted, the majority of these patients have a normal neurologic examination. The diagnosis is based on signalment, neurologic examination, and by ruling out other causes of seizures with appropriate diagnostic testing. Unless concerned about the potential for breeding a patient and passing on the trait, it is not that critical to differentiate unknown epilepsy from genetic/idiopathic epilepsy, as the treatment for both is identical.

**Signalment and Commonly Associated Etiologies**

- **Less than 1 year of age** – Head trauma, intoxication, hypoglycemia, meningoencephalitis, hepatic encephalopathy, hydrocephalus, lysosomal storage disease, other congenital disorders

- **From 1 to 5 years of age** – Genetic epilepsy, unknown epilepsy, head trauma, intoxication, meningoencephalitis, hydrocephalus, hepatic encephalopathy, cerebrovascular disease, cerebral neoplasia
Greater than 5 years of age – Cerebral neoplasia, unknown epilepsy, meningoencephalitis, cerebrovascular disease, hypoglycemia

**Diagnostic Testing and Plan**

Diagnostic testing for seizures can be divided into three steps:

**Step 1** – CBC, serum biochemistry, urinalysis

**Step 2** – Thoracic and abdominal imaging, serum bile acids, toxin assays (e.g., lead, ethylene glycol, cholinesterase), infectious disease titers, skull radiographs

**Step 3** – CSF evaluation, computed tomography (CT) or magnetic resonance imaging (MRI), electroencephalography (EEG)

Step 1 should probably be performed on any patient with seizures. Step 2 consists of tests that are readily available to most veterinary practitioners and may be chosen in certain cases depending on the index of suspicion for a certain disease. Step 3 consists of specialized tests that specifically evaluate the nervous system, and are usually only available at specialty referral hospitals. The signalment, history and physical and neurologic examinations are critical when deciding which diagnostic tests are appropriate.

**Important History Questions to Ask** – Character, onset, frequency and progression of seizures; evidence of asymmetry; evidence of prodrome or post-ictal period, neurologic status between seizures, vaccination status and travel history, previous illness and medications

**Important Physical Examination Findings** – Evidence of cardiovascular disease, evidence of hepatic disease, retinal (fundic) examination

**Important Neurologic Examination Findings** – Altered mentation, focal cranial nerve deficits, circling or turning in one direction, focal postural reaction or conscious proprioceptive deficits, visual deficits, cervical hyperesthesia

**When to choose Step 3?** An important decision point is when to refer a patient with seizures to a specialty hospital to receive the tests listed above in Step 3. In the author’s opinion, this may be considered in any animal with seizures where extracranial etiologies have been ruled out, if the owners are so inclined. However, other patient circumstances may increase the suspicion of an active intracranial process, and thus the urgency for referral. These include focal seizure activity, focal neurologic deficits, dogs less than 1 year or greater than 5 years of age, and all cats.

**References**

SEIZE THE DAY! TREATMENT PLANS FOR THE ROUTINE AND DIFFICULT-TO-CONTROL EPILEPTIC

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Deciding on a treatment plan for an animal with seizures depends on a number of factors, including the suspected etiologic cause of the seizures, the frequency and severity of the observed seizures, and the financial constraints or intentions of the owner.

Address the Underlying Cause

If an underlying cause of the seizures is known or suspected, it should be appropriately addressed, if possible. Thus, animals with hypoglycemia or electrolyte abnormalities may require no therapy other than correction of these deficiencies (or excesses). Likewise, patients with hepatic encephalopathy, hypertriglyceridemia or various intoxications may not require long-term anticonvulsant therapy if the primary disease is appropriately addressed, although they may benefit from shorter-term treatment with these drugs. In some cases, damage to the brain may lead to acquired (probably symptomatic) epilepsy, requiring long-term treatment.

Animals with intracranial diseases also benefit from addressing the underlying condition, although these patients are more likely to require maintenance anticonvulsant therapy. Thus, placement of a ventriculoperitoneal shunt for hydrocephalus, anti-inflammatory and/or antimicrobial medications for meningoencephalitis, surgery or radiation therapy for brain tumors, and other specific therapies address the underlying disease process and may reduce or eliminate the need for anticonvulsant therapy.

Maintenance Anticonvulsant Therapy

Maintenance anticonvulsant therapy is used as an adjunct in symptomatic epilepsy and is the cornerstone of therapy for patients with idiopathic or probably symptomatic (acquired, cryptogenic) epilepsy. The first question to address is: When to start anticonvulsant therapy? There are no hard and fast rules on this issue, and each patient much be approached individually. However, some guidelines apply. In general, maintenance therapy should be considered if:

- Seizures are more frequent than once every 6-8 weeks
- Seizures are obviously increasing in frequency
- Status epilepticus or cluster seizures occur
- Seizures last longer than 5 minutes
- Seizures are very severe or involve aggression towards the owner

The second question to address is: Which anticonvulsant should I choose? Historically in dogs, the two main initial options for therapy have been phenobarbital and (potassium) bromide. These medications are chosen because of their long history of use, apparent efficacy, ease of dosing, favorable pharmacokinetics and inexpensiveness. There is limited evidence to suggest that phenobarbital may be slightly more efficacious as a first line agent in the dog. Diazepam is not effective as a maintenance anticonvulsant in the dog due to a very short elimination half-life,
and the development of tolerance within several weeks. Some of the newer anticonvulsant medications can be effective as initial therapy, and the author uses zonisamide and levetiracetam with some frequency as first-line agents in dogs. Zonisamide is particularly attractive in this setting due to its low incidence of side effects and its relatively long half-life, allowing twice daily administration. Recently, a generic extended release formulation of levetiracetam has become available, and pharmacokinetic studies suggest that twice daily administration may be effective in canine patients. However, published reports of efficacy in this setting are lacking in veterinary patients. Use of these newer drugs has been limited in the past by their expense when compared with traditional anticonvulsants, although generic versions of most of these newer generation drugs are now available at reduced costs. In the cat, phenobarbital (preferred) and diazepam are the historical maintenance drugs of choice. Bromide is an effective anticonvulsant in the cat, but is associated with a very high incidence of inflammatory lung disease, and is not recommended. Diazepam should be used with extreme caution in cats and is generally not recommended, as it has been associated with idiosyncratic hepatic necrosis after oral administration. Levetiracetam may be a reasonable choice in the cat if phenobarbital is not an option, although the drug must be administered three times daily. There is limited information available on zonisamide in cats, although side effects seem to occur more frequently in this species.

Initial Maintenance Therapy for Epileptic Animals

- Phenobarbital – 2.5-3 mg/kg q 12 hours (Dogs or cats)
- Potassium bromide – 40-50 mg/kg/day q 24 hours or divided (q 12 hours) (Dogs)
- Zonisamide – 3-5 mg/kg q 12 hours (Dogs and cats)
- Levetiracetam – 20 mg/kg q 8 hours (Dogs and Cats)
- Levetiracetam (extended release) – 30 mg/kg q 12 hours (Dogs)
- Diazepam – 0.2-1.0 mg/kg q 12 hours (Cats, use with caution and not recommended)

Phenobarbital is available in generic tablets (15, 30, 60, 90, 100 mg) or suspension (3 and 4 mg/ml) formulations, as is diazepam (2, 5, 10 mg tabs; 1 and 5 mg/ml suspension). Zonisamide is available as 25, 50 and 100 mg capsules. Levetiracetam is available as 250, 500, 750 and 1000 mg tablets and a 100 mg/ml suspension. Extended release levetiracetam is available as 500 and 750 mg tablets; these should not be broken, as it interferes with the extended release mechanism. Bromide is typically compounded from the chemical grade salt, and complexed with potassium (KBr) or less frequently with sodium (NaBr). It should be noted that due to molecular weight differences between the cation, equal amounts of KBr and NaBr do not contain the same amounts of bromide, and therefore have different anticonvulsant potencies (250 mg KBr = 211 mg NaBr). KBr is available from a number of compounding pharmacies. Liquid formulations are preferred over capsules, as they facilitate dosage adjustments, and KBr is best administered with food to reduce gastrointestinal irritation. Dietary salt affects serum levels of bromide, and a constant salt level should be maintained in the diet.

Monitoring Maintenance Therapy

A complete blood count (CBC), serum biochemical evaluation and urinalysis should be performed before starting maintenance anticonvulsant therapy, both as part of the diagnostic evaluation (see previous talk) and as a baseline before starting therapy. In addition, the metabolism of these drugs varies between patients. Blood levels are essential to guide therapy for phenobarbital and bromide, and may be indicated for some of the newer drugs, depending on the response to therapy. Steady state of a drug after regular oral dosing depends on its half-
life in the body, and varies between medications and species. Monitoring times and desired blood levels are shown below.

**Table 1. Monitoring Anticonvulsant Therapy**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Time to Steady State</th>
<th>Desired Blood Levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenobarbital</td>
<td>10-14 days</td>
<td>15-35 µg/ml</td>
</tr>
<tr>
<td>Bromide</td>
<td>3-4 months</td>
<td>1000-3000 µg/ml (or 1-3 mg/ml or 100-300 mg/dl)</td>
</tr>
<tr>
<td>Diazepam</td>
<td>5-10 days</td>
<td>Monitoring not typically performed</td>
</tr>
<tr>
<td>Zonisamide</td>
<td>3-5 days</td>
<td>10-40 µg/ml (extrapolated from humans)</td>
</tr>
<tr>
<td>Levetiracetam</td>
<td>1-2 days</td>
<td>5-45 µg/ml (extrapolated from humans)</td>
</tr>
</tbody>
</table>

These desired blood levels are a guide only, and must be interpreted in light of the resulting seizure frequency and clinical condition of the patient. When measuring therapeutic blood levels, as with any medications, a serum separator tube (“tiger top”) should be avoided, as the separator device may bind the drug and artificially decrease the serum levels. After the establishment of acceptable therapeutic levels of the medication, it is generally recommended that blood levels along with a CBC, serum chemistry and urinalysis be monitored every 6-12 months or in the event of an acute change in seizure frequency or new onset of sedation, weakness or ataxia. Animals receiving phenobarbital may also benefit from pre- and post-prandial serum bile acid evaluation at these times to detect changes in hepatic function. Therapeutic monitoring is very important for phenobarbital and bromide, but has been utilized less frequently for the newer generation of drugs. As these newer medications are quite safe, they have generally been used to effect, and until recently, routine therapeutic monitoring for these drugs was not available.

**Potential Adverse Effects of Maintenance Therapy**

Adverse effects of phenobarbital include sedation, polyphagia, polyuria, polydipsia, weight gain, pelvic limb ataxia, weakness, and in rare cases hepatic failure and blood dyscrasias. Overt hepatic damage is an unusual sequel to this medication, particularly if the serum levels are maintained below 35 µg/ml. It should be noted that increases in liver enzyme levels are common with this medication, and this does not indicate hepatic failure. If this is a concern, serum bile acids should be evaluated. Side effects of bromide are similar for the most part, including sedation, polyphagia, polyuria, polydipsia, and weight gain but in rare cases also includes pancreatitis. Vomiting related to the salt content can be minimized by administration with food. Diazepam may lead to sedation and polyphagia, and rare idiosyncratic reactions causing acute hepatic necrosis have been described in the cat. Zonisamide and levetiracetam may both cause sedation, while the former may also cause vomiting and diarrhea. A reversible idiosyncratic hepatic failure has recently been reported in dogs that received zonisamide, but this appears to be a very rare sequela.

**Dealing with the Refractory Epileptic**

Monotherapy with one of the medications above controls an estimated 60-80% of epileptic dogs and the majority of cats. However, a number of animals will have their condition remain
unchanged or worsen in the face of this therapy. In this situation, a number of additional steps may be considered:

- Ensure owner is administering drug correctly
- Ask about dietary changes, other medications or herbal preparations, and topical anti-parasite medications that may interfere with seizure control
- Reconsider diagnosis, pursue additional diagnostic testing
- Ensure optimal blood levels of maintenance drug
- Increase dosing frequency (Phenobarbital - from q12 h to q 8 h) if seizures occur at times corresponding to “trough” blood levels (based on therapeutic monitoring)
- Ensure female dogs have been spayed
- Add a second anticonvulsant drug

Regarding these points, many animals require blood levels of phenobarbital above 25 µg/ml for seizure control, although levels exceeding 35 µg/ml should be avoided. Although unusual, some animals receiving phenobarbital metabolize the drug very rapidly, and may benefit from dosing every 8 hours. Having the owner maintain a seizure diary is useful to document these cases, as seizures may occur during the expected “trough” period of drug metabolism and peak and trough serum levels may be beneficial in guiding therapy. Serum levels of bromide above 3000 µg/ml are tolerated in some dogs, especially when used as a monotherapy.

Adding a Second Anticonvulsant Drug

In the cat, diazepam may be added successfully to phenobarbital to control seizures (although as mentioned above, this drug should be used with extreme caution in this species). In the dog, a combination of phenobarbital and KBr (starting dose 20-30 mg/kg daily) is effective in controlling the majority of patients refractory to monotherapy with either drug alone. However, side effects are common with this protocol, and may be unacceptable to the owner. These include sedation, pelvic limb weakness and ataxia, polyphagia, polyuria and polydipsia. It should be noted that side effects may subside approximately 1-2 weeks after initiating the new drug, and so patience can pay off. Generally, a balance must be achieved between an acceptable seizure frequency and these side effects, although this may be impossible in some dogs. Generally, the best success is achieved by aiming for a serum bromide level between 2000-3000 mg/l and maintaining a lower phenobarbital level (e.g. 10-20 µg/ml).

If seizure control cannot be obtained with this combination of drugs, then other options exist. Many refractory dogs experience cluster seizures at varying time intervals, with relatively good control between cluster episodes. In this case, administration of rectal or nasal diazepam or other benzodiazepines (see next talk) may help to control the cluster events and avoid an emergency visit to the hospital. A third anticonvulsant medication may be added, and includes the following choices:

- Zonisamide (Zonegran) – 6-10 mg/kg q 12 hours (dogs only [dose doubled when administered with phenobarbital])
- Levetiracetam (Keppra) – 20 mg/kg q 8 hours
- Felbamat (Felbatol) – 15-20 mg/kg q 8 hours (dogs only)
- Gabapentin (Neurontin) – 10-30 mg/kg q 8 hours (dog) or q 8-12 hours (cat)
- Pregabalin (Lyrica) – 2 mg/kg q 8-12 hours, increasing 1 mg/kg/dose each week to a total of 3-4 mg/kg (dogs only)
These drugs have a variety of mechanisms of action, which appear to be different from phenobarbital and bromide, and patients may receive additional benefit from a multimodal antiseizure effect. Another advantage of the newer drugs is their improved side effect profile, as side effects are essentially limited to sedation (which tends to be less severe than that seen with either phenobarbital or bromide) and gastrointestinal side effects (vomiting, diarrhea) for some drugs. Felbamate is an exception, as there is concern with hepatic dysfunction, particularly when used in combination with phenobarbital. Elimination half-lives are relatively short, and drug steady state levels are reached relatively quickly with administration of a regular oral dose. The main disadvantages of these newer drugs are their expense (although most are now available generically, and costs are decreasing) and requirement for administration every 8-12 hours.

Of these newer generation drugs, the author generally prefers to use zonisamide or levetiracetam. Assays to measure blood levels of these drugs are available at a few select laboratories, but these drugs are often administered to effect. In some cases, success with the addition of an anticonvulsant drug may allow the eventual withdrawal of the initial medication, although this must be accomplished gradually and with caution. The author frequently uses these newer anticonvulsant medications (particularly zonisamide and levetiracetam) as the second drug choice (typically instead of bromide). Other potential interventions to consider in select situations include acupuncture and the administration of a hypoallergenic diet.

References

TREATMENT OF CLUSTER SEIZURES AND STATUS EPILEPTICUS

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Definitions

- **Cluster seizures** – two or more seizures occurring within a 24-hour period.
- **Status epilepticus** – continuous seizure activity lasting longer than 5 minutes, or the occurrence of multiple seizures without recovery of baseline neurologic function between episodes.\(^1,2\) Status epilepticus can be generalized or focal in nature, or in rare cases, can be nonconvulsive.\(^3\)

Pathophysiology and Goals of Treatment

The vast majority of seizures are self-limiting events, with eventual spontaneous return to resting or baseline neurologic function. However, during status epilepticus, a variety of changes occur within cells and networks of cells that result in a situation where the seizure activity becomes self-sustaining. These changes may be independent of the initiating cause of the seizure, and involves a variety of molecular mechanisms. Repetitive seizures cause inhibitory GABA\(_{A}\) receptors to move from the synaptic membrane to the cell interior, while excitatory N-methyl-D-aspartate (NMDA) receptors may be recruited to the cell surface.\(^1\) Stores of inhibitory neurotransmitters may become depleted and increased expression of drug efflux transporters such as P-glycoprotein may occur.\(^4\) After a period of time, these cellular alterations may lead to pharmacoresistance to first-line agents that would normally be effective in seizure termination at earlier phases, such as the benzodiazepines.

Generalized status epilepticus can cause profound acidosis, hyperthermia, cardiac arrhythmias, hypoxia, neurogenic pulmonary edema, rhabdomyolysis, myoglobinuria, renal failure, cerebral edema, elevated intracranial pressure and neuronal necrosis, and therefore constitutes a medical emergency.\(^5\) The goals of treatment are to stop the seizures, support systemic organ functions, and protect brain function. Finally, ongoing seizure activity/seizure control should be closely monitored. These goals are described in greater detail below.

1) Stop the Seizures

The most critical and pressing goal of therapy is to stop the seizures, by any means necessary. The initial drug chosen is usually a benzodiazepine (diazepam or midazolam), but depends on the suspected underlying cause.

- If hypoglycemia is suspected (juvenile toy breed dog, hunting dog or insulin overdose), administer 1-2 ml/kg of 50% dextrose intravenously (IV) diluted 1:1 in saline. Oral dextrose may be used in animals able to swallow when intravenous access is not readily achieved.
- In small or toy breed dogs that have recently whelped and are nursing puppies, the administration of calcium gluconate may be considered to address potential hypocalcemia.
Animals with known idiopathic, symptomatic or possibly symptomatic (acquired/cryptogenic) epilepsy and those with unknown etiologies typically receive a benzodiazepine as the first-line drug.

- **Diazepam (0.5 mg/kg)** can be administered IV, intranasally or rectally to control seizures. The dose can be repeated twice, if necessary. Anticonvulsant action only lasts about 15-30 minutes, and therefore some form of longer acting therapy is required if the seizures stop. Midazolam can be substituted for diazepam in this scenario, and lorazepam (0.2 mg/kg) may also be considered. These drugs may also be administered IV or intranasally, but are not likely to be effective with rectal administration. In addition, unlike the others, midazolam can be successfully administered intramuscularly (IM).

- If the animal responds to a benzodiazepine bolus, phenobarbital may be considered for longer-term control. Naïve animals not previously receiving anticonvulsants can be loaded with 16-20 mg/kg divided into 4 doses and administered every 30-120 minutes (i.e., 4-5 mg/kg q 30-120 minutes). Epileptics already receiving phenobarbital may benefit from an additional “mini-loading dose” (5-10 mg/kg) depending on their serum levels of the drug. Phenobarbital should be continued at regular maintenance intervals (2-3 mg/kg IV, IM or PO q 12 hours or at the animals regular dose) after this.

- Animals with severe cluster seizures or status epilepticus with some inter-ictal time (i.e., non-continuous) usually respond to a constant rate infusion (CRI) of diazepam (0.1-2.0 mg/kg/hour IV). The CRI can be started at the low end of the range (0.1-0.25 mg/kg) and gradually increased as necessary to control seizure activity. Once controlled, a seizure-free state is maintained for 12 hours, after which the infusion is gradually tapered (usually reduce dose by half every 4-6 hours) and stopped. The CRI can be administered with a syringe pump, if available, or by mixing with 0.9% saline in a small IV bag or Buretrol system. Diazepam is degraded by light and binds to plastic, and the syringe and tubing should be covered with brown plastic or aluminum foil, if possible. Midazolam can again be substituted in this scenario, and is less likely to cause thrombophlebitis.

- Animals with continuous, prolonged seizure activity or those refractory to benzodiazepines may receive pentobarbital (3-15 mg/kg IV to effect), if available. This drug induces general anesthesia, and is extremely effective in stopping the outward manifestation of the seizure. However, respiratory and cardiovascular function may be depressed, and these systems must be monitored very closely. Although intermittent bolus doses can be used, a CRI (2 mg/kg/hr adjusted to effect) may be more effective. Similar to benzodiazepine CRIs, animals may be kept seizure free for approximately 12 hours, and then weaned from the drug. It can be difficult to distinguish recovery from pentobarbital anesthesia from overt seizure activity. However, paddling movements of the limbs typically indicate the former, while seizure activity is usually characterized by overt tonic or clonic muscle contractions. Electroencephalography, if available, can help to differentiate these two scenarios. This medication also reduces the metabolic requirements of the brain, and is considered to have neuroprotective effects.

- Propofol may be used as a substitute for pentobarbital if general anesthesia is required to control seizure activity. Due to its short duration of action, this drug must be given as a CRI (6 mg/kg initial bolus followed by 0.1-0.6 mg/kg/min). Substantial respiratory depression is common with this medication, and anesthesia must be closely monitored. In addition, propofol can have pro-convulsant effects in some patients. Some consider this to be the treatment of choice for patients in status epilepticus secondary to hepatic encephalopathy (typically after surgical repair of a portosystemic shunting vessel).
• If pentobarbital and propofol are not available, the use of an inhalant anesthetic (e.g., isoflurane or sevoflurane) to maintain general anesthesia should be considered as a last resort. Both require close monitoring of respiratory and cardiovascular parameters.

• A parenteral formulation of levetiracetam is also available. Although its use in animals with status epilepticus or cluster seizures has been limited to date, it may prove useful in this role, based on reports in humans and preliminary experience in canine patients. Pharmacokinetic studies in dogs suggest that a dose of 20-60 mg/kg IV results in blood concentrations within the range considered to be effective in humans (5-45 µg/ml) for greater than 8 hours. Levetiracetam is approximately 100% bioavailable after IM administration and results in similar blood levels, although peak concentrations are not reached until about 40 minutes after the drug is given.

• Reports of other medications for refractory status epilepticus are infrequent in veterinary medicine. There is a report of a dog with granulomatous meningoencephalitis and status epilepticus responding to intravenous ketamine infusion after failure to respond to diazepam and propofol. This report follows several human case reports reporting similar efficacy for ketamine in the scenario of refractory status epilepticus, the rationale being blocking of NMDA receptors which may be responsible for the self-sustaining nature of this condition. Additional therapies reported in refractory human cases include valproic acid, lidocaine, and topiramate.

2) Support and Monitor Systemic Functions

As described above, status epilepticus can have profound effects on many body systems, and systemic functions must be closely monitored. These include:

- Mental status and level of consciousness
- Respiration, oxygen saturation and blood gases (if available)
- Cardiac rate and rhythm, blood pressure
- Body temperature
- Serum electrolytes, glucose, BUN and creatinine
- Fluid status and hydration
- Muscle damage and evidence of myoglobinuria (which may cause renal failure)

Intravenous fluid therapy is often indicated in order to maintain hydration, and may help prevent renal damage if myoglobinuria is a concern. As severe seizure activity may lead to non-cardiogenic pulmonary edema, thoracic radiographs, pulse oximetry, and blood gas analysis should be considered in animals with compromised respiration. Aspiration pneumonia is also a concern, particularly in large recumbent dogs. Oxygen therapy may be administered in some of these patients. Active cooling should be considered in animals that are severely hyperthermic. Basic supportive nursing care must be performed in recumbent and stuporous animals, including applying artificial tears/lubrication to the eyes, providing adequate bedding/padding, periodically changing body position, turning from side to side, and passive range of motion of the limbs.

3) Protect Brain Function

Prolonged, severe seizure activity can lead to cerebral edema, increases in intracranial pressure and neuronal necrosis. Select cases may benefit from oxygen therapy, mannitol (0.25-1.0 g/kg IV over 10-20 minutes) and furosemide (0.7 mg/kg IV, 15 minutes after mannitol) in order to address these effects. Compression of the jugular veins, coughing and sneezing all
increase intracranial pressure, and should be avoided in animals where this is suspected to be increased. Therefore, jugular catheters, collection of blood from the jugular vein, neck bandages, nasogastric tubes, and nasal oxygen catheters should all be avoided. Intravenous lidocaine should be considered to reduce the coughing reflex if intubation is required. Elevation of the head approximately 30 degrees from the horizontal is a simple way to promote venous return from the brain and potentially reduce intracranial pressure. Pentobarbital administration, in addition to stopping seizure activity, also has the advantage of reducing cerebral metabolism, which can have neuroprotective effects in patients with status epilepticus.

4) Monitor ongoing seizure activity

Patients should be closely monitored to ensure the cessation of seizures and for the recurrence of seizure activity after initial therapy. This is typically done by visual observation and examination of animals for motor activity consistent with seizures. Whenever possible, cessation of seizure activity should be confirmed electrophysiologically with the aid of electroencephalography (EEG). This may detect animals whose outward motor manifestations of the seizure activity have stopped, but who continue to have abnormal electrical brain activity, known as nonconvulsive status epilepticus (NSE). Although NSE has rarely been reported in veterinary patients, this is likely a reflection of the infrequency with which veterinary clinicians perform EEG in this setting. The author has documented a number of canine patients with apparent NSE after prolonged convulsive status epilepticus or presenting with a primary complaint of altered mentation (Mariani, unpublished observations). An EEG is part of the routine diagnostic evaluation of human patients presenting with stupor or coma, and in the author’s opinion, the same should be offered to veterinary patients wherever possible.

At-Home Therapy for Cluster Seizures

Some owners can be taught to administer benzodiazepines in the home environment in order to reduce the number of seizures in dogs (or cats) prone to cluster seizure events. The goal is usually to prevent further seizures, reduce the number and severity of subsequent seizures, and avoid an emergency visit to the veterinary hospital. Diazepam has been used most often via the rectal route; a standard dose (0.5 mg/kg) can be administered, although in some animals on chronic phenobarbital therapy, a higher dose (1-2 mg/kg) may be required due to increased metabolism of the drug. Drugs administered rectally in the dog undergo a substantial first-pass effect and hepatic metabolism as the majority of absorbed drug enters the portal circulation. As a result, the bioavailability of diazepam after rectal administration is only about 2.7-7.4% at doses of 0.5 and 2.0 mg/kg respectively in dogs not receiving phenobarbital. However, some anticonvulsant effect is achieved as the main metabolites of diazepam (desmethyldiazepam and oxazepam) possess 20-50% of the activity of the parent drug. Lorazepam is unsuitable for rectal administration, as its primary metabolite (lorazepam glucuronide) does not have anticonvulsant activity. Midazolam does have a metabolite with reported pharmacologic activity (1-hydroxymidazolam), although the contribution of this reported activity is controversial.

Intranasal (IN) administration of benzodiazepines avoids several of the shortcomings of the rectal route. The IN route avoids substantial first pass metabolism, and drug is directly absorbed into the systemic circulation thorough the dense vascular plexus present in the nasal passages. In addition, there is evidence for direct movement of drug through the cribriform plate and into the central nervous system. Several studies suggest that the bioavailability of diazepam is much higher after IN administration than after rectal, and this route has been used successfully by the author in several emergent clinical cases (0.5 mg/kg). Preliminary
experience suggests that intranasal lorazepam (0.2 mg/kg) may also be useful as an alternative to rectal diazepam for at-home use by owners.\(^\text{27}\) Intranasal or IM midazolam (0.5 mg/kg) is another option available for these scenarios.

References

WHAT TO DO WITH LUMPS & BUMPS: SEE SOMETHING, DO SOMETHING. WHY WAIT? ASPIRATE.®
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WHAT IS “SEE SOMETHING, DO SOMETHING. WHY WAIT? ASPIRATE. DR SUE CANCER VET?”
“See Something, Do Something” (SSDS) is a lumps and bumps cancer awareness program that provides guidelines for evaluating superficial masses in dogs and cats. We hope these guidelines to increase client awareness will promote early cancer detection and diagnosis, as well as early surgical intervention. In veterinary medicine, most skin and subcutaneous tumors can be cured with surgery alone if diagnosed early when tumors are small.

See Something: If a skin mass is the size of a pea (1 cm) and has been there 1 month,

Do Something: Aspirate or biopsy, and treat appropriately!

WHY DO WE NEED SSDS?
It is well documented that cytologic and histologic evaluations are important diagnostic tools in veterinary oncology and that obtaining a preliminary diagnosis optimizes treatment planning. It is also recommended to evaluate masses that are growing, changing in appearance, or irritating to the patient. At this time, no specific guidelines exist for determining when to aspirate or biopsy or when to monitor canine and feline skin and subcutaneous masses.

Without standard of care guidelines, superficial masses may be monitored for too long. This can negatively impact our patient’s prognosis as well as limit their treatment options. Larger tumors that are diagnosed later may require more advanced treatments. Surgical excision of larger masses may result in less than adequate surgical margins (narrow or incomplete), leading to recurrence and additional costly therapy (second more aggressive local surgery, radiation therapy and/or chemotherapy).

With significant time delays and prolonged monitoring, there may be no reasonable surgical treatment options to remove large advanced tumors. These are often the most frustrating and heartbreaking cases.

WHY DIAGNOSE EARLY?
Obtaining a definitive diagnosis with cytology or biopsy early and before excision will lead to improved patient outcomes for superficial masses. When smaller, superficial tumors are detected early, surgery is likely curative - especially benign lesions and tumors that are only locally invasive with a low probability of metastasis. If tumors are removed with complete surgical margins, the prognosis is often good with no additional treatments needed.

- Visual monitoring is not enough.
- Pet owners need to be aware of the “pea” size requirement to have masses evaluated
- Veterinarians must measure and document the size of the mass in order to compare growth.
- If > 1 cm (or size of large pea) and present for a month, the mass should be aspirated or biopsied.
- Knowing the tumor type prior to the FIRST surgery will increase success of a curative-intent surgery.

WHAT ARE THE MOST COMMON TUMORS?
Primary skin and subcutaneous tumors are common in dogs and cats. While the overall incidence in dogs and cats is difficult to determine, approximately 25 to 43% of biopsies submitted in dogs and cats are of the skin. Of submitted samples, 20 to 40% are reported to be malignant.

The most common malignant skin tumors in dogs are mast cell tumors (MCT) (10-17%), soft tissue sarcomas (including fibrosarcomas [2-6%], malignant nerve sheath tumors [4-7%]), and squamous cell carcinomas (2-6%). The most common benign canine skin and subcutaneous benign tumors include lipomas (8%), histiocytomas (8-12%), perianal gland adenomas (8-12%), sebaceous gland adenomas/hyperplasia (4-6%), trichoepitheliomas (4%), papillomas (3%), and basal cell tumors (4-5%).

In cats, the most common superficial tumors include basal cell tumors (BCT) (15-26%), mast cell tumors (13-21%), squamous cell carcinomas (10-15%), fibrosarcomas (15-17%). These four tumor types make up about 70% of all skin tumors in cats. Sebaceous gland adenomas are much less common (2-4%). If BCT
are excluded, the percentage of malignant skin tumors in cats is higher than dogs, with studies reporting 70 to 80%.

**IS VISUAL MONITORING ACCEPTABLE?**

Even the most experienced veterinarian or oncologist cannot look at or palpate a mass and know whether it is malignant or not. Cancer is a cellular diagnosis! It is always recommended to evaluate masses that are growing, changing in appearance, or irritating to the patient. But these guidelines are not enough. All skin and SQ masses that are >1 cm and have been present for 1 month should be aspirated for cytologic evaluation. Biopsy is indicated if cytology does not provide a diagnosis.

**METHODS OF DIAGNOSIS**

**Aspirate and Cytology**

Fine needle aspiration (FNA) and cytology provide a diagnosis for many skin and SQ masses, especially those that that exfoliate well. FNA is useful to distinguish neoplasia from inflammation. Cellular morphology may also allow for the determination of benign or malignant phenotype. FNA is useful for identifying benign masses including lipomas and sebaceous adenomas. For malignant tumors, cytology provides information that assists in formulating diagnostic and treatment plans.

The advantages of cytology include: minimally invasive approach, low risk, low cost procedure, and results are available more quickly than biopsy results. The disadvantages of cytology are that it may be non-diagnostic or equivocal. This may be due to a small number of cells in the sample, poor exfoliation of the cells, or poor sample quality. If the sample is non-diagnostic or equivocal, histopathological confirmation may be required for definitive diagnosis.

Unless the sample is comprised exclusively of only fat, clear cystic fluid, or acellular debris, the sample should be submitted to a trained cytopathologist. **WHEN IN DOUBT, SEND IT OUT.** Including an adequate history helps the pathologist in diagnostic accuracy.

**Biopsy**

If cytology is non-diagnostic, a pre-treatment biopsy is recommended PRIOR to complete tumor removal. The pre-treatment biopsy will determine the optimal treatment plan.

The role of excisional biopsy is controversial, even among oncologic surgeons. A practical recommendation for non-diagnostic cytology and the lesion fits in an 8 mm punch biopsy, then PUNCH IT OUT. If the mass is larger than an 8 mm punch biopsy, an incisional biopsy (wedge, tru-cut, punch) is required for diagnostic confirmation.

It is tempting to remove the mass right away. An excisional biopsy establishes a diagnosis and removes the tumor at the same time. However it is not recommended for undiagnosed skin and superficial masses. Malignant tumors often require 2 to 3 cm margins. When an excisional biopsy (or debulking surgery) leads to incomplete margins for malignant tumors, more treatment, more morbidity, and more expense ensue. **Thus removing the mass entirely is not recommended without a cellular diagnosis prior to definitive excision.** Surgical approaches vary with different tumor types. Research confirms that the first surgery is the best chance for a cure.

Staging diagnostics are often indicated prior to curative intent surgery. Consultation with a veterinary oncologist is recommended.

**AFTER THE ASPIRATE/BIOPSY**

If the mass is benign:
Benign tumors may not need to be removed. A variety of factors, including mass location should be considered. Surgery should be recommended when a benign tumor is causing pain, irritation, bleeding, or infection. Surgery should also be recommended if an increase in growth would prevent a surgery in the future.

Alternatively, if removing the tumor requires a complicated surgery (i.e. near a joint, on the distal limb with minimal surrounding tissue for reconstruction) or the pet has other pre-exiting issues, you and the pet owner may make an educated decision as to whether proceeding to surgical removal is warranted. **PETS**
WITH MASSES NOT REMOVED SHOULD BE MONITORED (via measurement) BY THE VETERINARIAN EVERY 3 TO 6 MONTHS.

If surgery is performed, most benign masses require smaller surgeries, as wide margins are typically not needed.

If the mass is malignant:

If the aspirate/biopsy reveals malignancy, consult with veterinary oncologist for appropriate staging recommendations. For malignant tumors, the first surgery should be a wide excisional surgery.

If wide excisional surgery is not possible due to the size or anatomic location of the mass, consultation with a veterinary oncologist or board-certified surgeon is indicated. Surgeons may be able to perform specialized surgeries such as axial pattern flaps to remove the tumor completely. Debulking (cytoreductive) surgery may not be recommended, as this will not obtain margins, and additional post-operative treatments such as radiation will be required to prevent recurrence. In some cases, cytoreductive surgery may be performed for palliation, or with an understanding that adjunctive therapy such as radiation therapy will follow the procedure.

After surgery:
- Review the histopathology report – tumor type, grade, vascular and lymphatic invasion.
- Consult with a veterinary oncologist for additional therapeutic considerations for malignant tumors.
- Assess the QUANTITY of surgical margins in consideration of tumor type and biologic behavior. (One mm margins for a malignant tumor may be called “clean” on a biopsy report, but size of margins must be considered in light of tumor histology.)
- If margins are inadequate, recommend adjunctive treatment before tumor recurrence for optimum patient outcome. Post-operative options include scar revision (second surgery), radiation to prevent regrowth, or chemotherapy which may slow recurrence in some cases.
- It is important to consult a board certified surgeon before attempting scar revision.
- Monitor for local tumor recurrence and metastasis as indicated by the histologic diagnosis and margin assessment.

RECURRENT AND MONITORING

Patients with reported complete surgical margins can potentially suffer tumor recurrence due to microscopic cancer extension that is not seen in the evaluated sections. Therefore, it is essential to monitor for local regrowth, and to recruit the pet owner to monitor the surgical scar as well, to identify early relapse.

For malignant tumors with wide, clean margins and low metastatic potential, follow-up rechecks are recommended every two to three months after the surgery for as much as one year of follow up. Early detection is key to addressing recurrence and metastasis to ensure the highest possible chance of success.

Owners are encouraged to check their pets regularly at home for new masses.
- Owners should check their pet monthly for superficial masses by noting their location and size.
- Create a “body map” with size and location of superficial masses recorded, along with fine needle aspiration cytology results. This body map can serve as an objective medical record document and owner guide to follow masses longitudinally, and to allow for identification of new masses over time.
- All masses should be aspirated and submitted for cytology. Masses that do not need cytologic assessment include lipomas, cysts, and those containing acellular debris.
- If cytology is non-diagnostic, discuss repeating the aspirate, or proceeding to biopsy.
- Know the tumor type prior to surgery. The first surgery is your patient’s best chance for cure.

SURGERY MAY BE ALL THAT IS NEEDED

We all must be proactive to advocate for early cancer detection. Visual monitoring of superficial masses is not enough. Obtaining a definitive diagnosis via either cytology or biopsy early and before excision will lead to improved patient outcomes for superficial masses. Surgery is likely curative for the majority of these cases, especially for benign masses and those locally invasive malignancies that are non-metastatic. If tumors are detected and removed earlier – when they are small and with clean margins, the prognosis is often good and the patient may not require additional therapy.

See Something: When a skin mass is the size of a pea (1 cm) and has been present for 1 month,
Do Something: Aspirate or biopsy, and treat appropriately!

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References/Suggested Reading
Cancer is not a death sentence in pets. Chemotherapy is well tolerated in the majority of dogs and cats undergoing treatment. With treatment, many cancer patients are not only living longer, but living well.

The pet is a family member, and owners often want same standard of care for their dogs and cats as they do themselves. Sadly, cancer is leading cause of death in pets. “Cancer” is a scary word that is often equated with death. There is often a visceral fear of cancer, and people think cancer equals pain and suffering. Owners think cancer treatment will just make the patient sicker. With cancer, there is no hope. I disagree. Cancer is not a death sentence. While we all want a cure for cancer, I encourage thinking about many cancers as chronic conditions that may require chronic therapy, such as kidney or heart disease. As an oncologist, I recommend treatment when the pet is likely to live longer with it than without. Thankfully, most pets feel good, if not great, during treatment. I believe it is important to approach the topic of cancer with knowledge, compassion, and a positive attitude.

CHEMOTHERAPY

Conventional Chemotherapy

Conventional chemotherapy is typically given at high dosages, known as maximum tolerated dose, or MTD. The goal is to kill the rapidly dividing cancer cells. But some normal cells that also have high turnover often can be temporarily damaged by MTD chemotherapy. The normal tissues that typically are most sensitive to chemotherapy are the bone marrow, hair follicles (alopecia), and the gastrointestinal lining. This is often referred to as “BAG”. As a result there is a break period to allow these cell populations to recover. MTD is typically given weekly to every 3 weeks.

The overall toxicity rate is very low in veterinary chemotherapy patients. In my experience, only 15-20% experience side effects, and this is even less common in cats than dogs. The primary goal is to provide the best quality of life possible for as long as possible. As I say, live longer, live well. Most side effects are mild and medically manageable.

Metronomic chemotherapy

In contrast to MTD chemotherapy, metronomic chemotherapy is pulse or low-dose continuous chemotherapy. This is typically administered daily or every other day. The target is endothelial cells in that line tumor blood vessel. The goal may be tumor is stabilized, but this prevents further growth and spread. Common chemotherapy drugs include Palladia, cyclophosphamide, and chlorambucil and also with NSAIDS. There is still much to be learned including best drugs, dose, schedule, tumor types, and toxicity. This can be considered for some dogs and cats with advanced metastatic disease.

SIDE EFFECTS

Alopecia

Alopecia (hair loss) is due to damaging the rapidly dividing hair follicle. In dogs, potentially affected breeds have continuously growing coats and include Poodles, Scottish Terriers, and Westies. In cats, alopecia is rare, but shaved areas tend to grow back more slowly (limb catheters, abdominal ultrasounds). Cats more commonly lose their whiskers. The good news is that hair and whiskers will re-grow once the treatments have completed.

Occasionally, hair will grow back a different texture or color. In cats it is typically softer, aka the “chemo coat”. It is important to remember pets do not care about this cosmetic side effect, and it does not impact the quality of life. However, pet owners like to be advised about the whiskers and coat so they are not surprised.
**Gastrointestinal (GI) toxicity**

Gastrointestinal (GI) toxicity includes vomiting, diarrhea, decreased appetite, nausea. It typically 1 to 5 days after chemotherapy and is self-limiting – lasting on average 2-3 days. These side effects are less common in feline chemotherapy patients than dogs. I recommend being very proactive with nausea/anti-emetic drugs. I often will use Cerenia or mirtazapine preventatively and as needed.

I recommend giving Cerenia at administration with the following drugs: doxorubicin, vincristine, vinblastine, carboplatin, mitoxantrone, dacarbazine, and the MOPP protocol. If the pet has nausea/vomiting event within 24 hours of administration, I will add Cerenia SQ or IV at the time of administration at the subsequent treatment. For oral chemotherapy being given at home, I advise the owner give oral Cerenia 1 hour before chemotherapy pill dosing.

I always recommend oral Cerenia for 4 days after doxorubicin in dogs to prevent nausea and vomiting. If there are side effects with other chemotherapeutics, I also typically will add prophylactic medications to prevent side effects like nausea, vomiting or diarrhea as indicated. If the GI side effects are more severe in a patient, the drug type or dosage may be adjusted at subsequent treatments to minimize the chance of side effects recurring.

Unlike dogs, I do not routinely use GI medications unless the cat had issues with a prior treatment or had GI clinical signs prior to treatment (i.e. GI lymphoma).

For diarrhea, I typically send my patients home with metronidazole and a probiotic. Metronidazole is a synthetic nitroimidazole with antibacterial, anti-protozoal and anti-inflammatory properties and is commonly prescribed for acute and chronic diarrhea. It is metabolized and excreted by the liver, so take care with patients with impaired liver function. Neurotoxicity is associated with higher doses and chronic use, so I do not recommend chronic use. Dose: 15 mg/kg PO BID for 5 days. Rx Clay is a good option for chronic diarrhea and patients needing multiple courses of metronidazole. Rx Clay is a calcium aluminosilicate (CAS), which is geological nanomaterial that adsorbs bacterial enterotoxins and increases reabsorption of intraluminal water in GIT.

**VOMITING AND DIARRHEA**

Acute vomiting is typically associated with cisplatin, doxorubicin (Adriamycin), dacarbazine (DTIC), cyclophosphamide, actinomycin, 5-FU streptozoticin. This can typically be prevented with pre-treatment.

Delayed vomiting is more common in our patients. This is due to direct damage to rapidly dividing GIT cells (crypt cells) or via the centrally mediated CRTZ stimulated via gut vagal efferents. Delayed vomiting is most commonly 2 to 5 days post-chemo and seen with doxorubicin and the vinca alkaloids. Clinical signs include vomiting, diarrhea, anorexia, lethargy, weakness, ± dehydration.

For work up, I recommend CBC, chemistry panel, UA, +/- fecal floatation and cultures. If abdominal pain is present, consider AXR or AUS to rule out foreign body, obstruction, and intussusception. For patients with GI neoplasia, it can be challenging to differentiate chemotherapy side effects vs. disease, and a good history can be key.

For outpatient treatment, I recommend NPO, food & water trial, bland diet, anti-emetics, antibiotics with severe diarrhea and a probiotic. Do not forget to discontinue oral chemotherapy or delay chemotherapy treatment. In addition, I recommend prophylactic therapy with the next chemotherapy.

For inpatient, I add injectable antiemetics, IV fluid therapy, and IV antibiotics. An important note, I strongly encourage owners to NOT EUTHANIZE at this time. It is amazing with 1 to 2 days of
good supportive care how quickly these patients improve. And with prophylactic therapy and a dose reduction, these patients can tolerate the same chemotherapy drug.

MYELOSUPPRESSION AND NEUTROPENIA
Bone marrow suppression most commonly results in a neutropenia but cats seem to be more tolerant than dogs. Neutrophils and platelets are at greatest risk due to the shorter circulating lifespan and shorter bone marrow transit times. Neutropenia is the dose-limiting toxicity in veterinary oncology.

In addition to the chemotherapy targeting rapidly dividing bone marrow stem cells, other mechanisms for neutropenia includes bone marrow infiltration with neoplastic cells (leukemia, advanced stage lymphoma, multiple myeloma) and increased consumption due to infection. When a chemotherapy drug is used that is known to have a high potential for bone marrow suppression (like doxorubicin, carboplatin and Lomustine), a complete blood count (CBC) is often checked after the treatment to check the expected nadir (low neutrophil count) and see if antibiotics and/or a dose reduction are needed. I recommend a nadir be checked with all chemotherapy drugs except L-asparaginase.

The nadir typically occurs 7 days after chemotherapy administration. Pay attention to the neutrophil count, not the total white blood cell count. For some chemotherapy drugs the nadir is more variable such as carboplatin and Lomustine. For cats, the nadir is can occur 7 to 28 days after treatment. In dogs the nadir for carboplatin in day 10-14. Chlorambucil tends to cause delayed neutropenias and thrombocytopenias after chronic use. Subsequent doses of chemotherapy are adjusted based on the results of the CBC.

Antibiotics may be prescribed as a preventive measure but its use is controversial. Common antibiotics are TMS and Clavamox. I recommend prophylactic use with the more myelosuppressive drugs (doxorubicin, carboplatin and Lomustine) or if the previous nadir was <1500 neutrophils. Unlike dogs, I do not routinely use prophylactic antibiotics unless the cat had issues with a prior treatment.

In my experience, there is less than a 5% chance that a patient will need hospitalization. If this does occur, these patients are usually hospitalized for typically 24-48 hours with supportive care including IV fluids and antibiotics. In my experience most chemotherapy patients can successfully receive that drug again with a dose reduction.

WHAT TO DO AT THE NADIR VISIT?
In addition to running a CBC, it is important to get a good history, TPR (fever is so important in neutropenic patients), and a complete physical examination. I am always interested in knowing how the patient handled chemotherapy — did she eat well, any vomiting/diarrhea, did the owner use any nausea or diarrheal medications? For the exam, did he lose weight, was she febrile? The nadir CBC should not be a technician appointment to just pull the blood sample. The history and exam are very important.

Pay attention to the neutrophil count, not the total white blood cell count. The nadir typically occurs 7 days after chemotherapy administration, but can vary (see above). I recommend antibiotics if the neutrophil count is <1500. If the patient has <1500 neutrophils and is afebrile and feeling well, I recommend managing as an outpatient. However, if the patient has <1500 neutrophils and is febrile and sick, I recommend admitting for supportive care. Remember a febrile neutropenic is an oncologic emergency.

Also, I prefer that we get blood samples from the jugular veins for patients getting IV chemotherapy (unless thrombocytopenic). Save those peripheral veins for treatment please. Finally many times the oncologist has run a recent chemistry panel, so check with the oncologist, and try not to repeat unneeded blood work to keep costs down.
**Neutrophil count (per uL)** | **Fever, systemic signs** | **Plan**
---|---|---
1500-2500 | No | Monitor +/- treatment delay 2 to 4 days
<1500 | No | Oral antibiotics treatment delay Consider dose change
<1500 | Yes | ATH for IVF & IV antibiotics treatment delay Dose reduction

**SEPSIS**
Sepsis in chemotherapy patients is typically due to patient’s own flora - Gram negative from GI bacteria: *E. coli, Klebsiella, Pseudomonas*; Gram positive from skin bacteria: *Staphylococcus epidermitidis and aureus*, Anaerobes from oral bacteria. Predisposing factors include neutropenia (infection risk well correlated with degree and duration), cellular immune dysfunction, humoral immune dysfunction, prolonged hospitalizations, indwelling catheters, and poor nutrition.

History and clinical signs are typically straightforward - cytotoxic chemotherapy was administered typically 5 to 7 days ago. Remember, the **febrile neutropenic patient is an oncologic emergency!!!** In addition the patient may have an inability to mount an inflammatory response, so the lack of fever, pyuria, or radiographic changes of pneumonia does not rule out sepsis. Signs of illness are unrelated to absolute neutrophil count, but are related to an increased susceptibility to local and systemic infections when neutropenic. Gastrointestinal, urogenital, and respiratory infections are most common. Shock is also possible

The sepsis work up includes: CBC, Chemistry panel, UA & UCS (if >50,000 platelets). If respiratory signs are present, chest radiographs are recommended, and TTW should be considered. Blood cultures may be needed, but uncommon in my experience. Culture any catheters suspected as the infection source.

Treatment for sepsis includes: IVF and broad-spectrum IV antibiotics. Neupogen is human recombinant G-CSF. The MOA is stimulation of proliferation & maturation of neutrophil precursors, and monocyte precursors to a lesser extent. It also primes neutrophil for cell killing & neutrophil migration. The benefit for the febrile & febrile neutropenic patient is contradictory, and in my experience, Neupogen is rarely needed. The recommended dose is 5 ug/kg SQ SID until neutrophil >1000.

**WHEN SHOULD I LOWER CHEMOTHERAPY DOSE?**
**Dose Intensity** is chemotherapy given at MTD & shortest possible interval. It is important to remember than small dose changes can have significant impact on cancer control. Dose reductions as small as 20% can decrease drug efficacy up to 50%. **Dose reductions should not be considered lightly.**

**DON’T TREAT CATS LIKE SMALL DOGS WHEN IT COMES TO CHEMOTHERAPY**
Some chemotherapy drugs are dosed differently in cats. In dogs, weight and body surface area are used to determine the carboplatin dose. In cats there is now a more accurate method to dose carboplatin in cats based on glomerular filtration rate, which is determined with an iohexol clearance test.
Side effects in cats are also different. Cardiotoxicity is a well-described adverse effect in dogs treated with doxorubicin, but it has not been reported in cats. Sterile hemorrhagic cystitis (SHC) is a relatively uncommon complication of cyclophosphamide in dogs and ifosfamide therapy in dogs and cats. SHC is typically associated with long-term use, but possible after single dose, and can progress to bladder fibrosis. The incidence with cyclophosphamide has been reported to be 9% in dogs (7-24%), 3% in cats, and 24% in humans. Unlike dogs, concurrent administration of furosemide with cyclophosphamide is not recommended in cats. Mesna, which binds the SHC-inducing acrolein, is recommended for cats and dogs when administering ifosfamide.

CHEMOTHERAPY SAFETY
Chemotherapy requires careful prescription preparation, drug dispensing, drug administration, client education, and safe handing of patients by ALL staff. Chemotherapy exposure has been documented in nurses and pharmacy workers. It is important to protect your team, our clients, & follow protocols.

To protect your staff, the following are recommended a hood, closed system transfer device, dedicated counting equipment, dedicated chemo fridge, and Personal protective equipment (PPE) including gloves, gowns, chemo mat, and eye protection. Closed system transfer device such as PhaSeal® are leak-proof and airtight closed system transfer devices. Studies show closed systems reduce contamination and should be combined with other safe handling practices.

Active drug & metabolites are excreted in urine & feces, and there is some in saliva but research six limited. In the hospital identify patients after chemotherapy and dispose of wastes separately. Spill kits should be on hand and stocked.

Protect your entire staff and make sure staff is aware patient is getting chemotherapy. Special precautions are recommended for staff and clients that are pregnant, trying to become pregnant, breast-feeding, immunosuppressed, ir taking immunosuppressive medication. Recommend they talk to their physician.

Protect your client and discuss safety tips including common sense precautions, good basic hygiene, and provide an information sheet. Recommend they wear gloves when handling urine, feces, or vomit for at least 72 hours after treatment and when cleaning litter box. Wash soiled bedding separately & 2 wash cycles before use again, use detergent to clean floors, carpets, or countertops, and wear gloves when cleaning.

It is safe to be around pets undergoing chemotherapy. Metabolites are far less active than original drug. Being around family members – human and other pets in the home - is an important part of a pet's life. Normal activities are safe, but owners need to be careful with excretions.

REFERENCES
TOP ONCOLOGY MISTAKES AND HOW TO AVOID THEM
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DON’T ASSUME YOUR CLIENT WOULD NEVER TREAT THE PET FOR CANCER, ESPECIALLY WITH CHEMOTHERAPY, OR BECAUSE THE PET IS TOO OLD.
When first told their pet has cancer, a reflex reaction of pet owner is to say they are against chemotherapy. But it is not uncommon for that same pet guardian to decide to treat after meeting me. Do I convince them to treat? Of course not; but I do replace myths with facts and fears with hope and knowledge.

There is a visceral fear of cancer. I understand that some people want to treat, and others do not. But some people change their mind once they learn about the diagnosis and the treatment options. When talking about cancer and treatment, we need to approach with knowledge, compassion, and a positive attitude.

Cancer in people is not cancer in pets. Cancer is not a death sentence. Cancer is often a treatable disease. Treatment is typically well-tolerated and we can also offer a range of treatment options. I think it is helpful to think of cancer like a chronic condition like, chronic kidney failure – we may not be able to cure it, but we can manage it and maintain a good or acceptable quality of life. Pets with cancer can live longer and live well.

Some of the myths include: My pet will suffer while on chemotherapy; I don’t want to contaminate my family or other pets; there are no options for pets with cancer; there’s no point in treating; my pet will spend most of his time in the hospital; my pet will get sick on chemotherapy; and my pet will go bald.

However, most pets have a great quality of life during and after a chemotherapy protocol. The urine and feces pose minimal risk, and we can educate our clients with simple common sense precautions we can educate our clients. Most breeds have little to no hair loss and this is only cosmetic. For many cancers, there is a spectrum of options and protocols. In my experience, 80% of animals that receive chemotherapy have no side effects. Most patients are out-patients. Only 15-20% of chemotherapy patients have side effects. The incidence of severe side effects is even lower at <5%. Most of these will resolve with supportive care in 24-72 hours, and the chemotherapy related death < 1 in 200. In addition, protocol modifications are effective 90% of the time. With dose reductions and medications to prevent repeat side effects, most of my patients can receive that same chemotherapy drug again without any issues – the same chemotherapy drug that may have caused hospitalization the last time.

Giving chemotherapy to a pet is not a contract. If the owners do not like how the pet is handling it, we can stop. I never force people to continue, but I do encourage them to try a dose or two if they are considering treating. Most pet Guardians are so surprised and pleased with the results of the chemotherapy and the quality of life while undergoing chemotherapy, they return with their pet for more treatments. So it can be useful for the owners to meet with the specialist and learn about the options. Even if they decide not to treat, they will have made an educated decision.

Don’t say the pet is too old. Age is not a disease. I have many 12-plus year old patients that are otherwise healthy and strong. They may have some early kidney disease, a heart murmur, thyroid disease or arthritis, but they are still “treatable” cancer patients. Don’t assume the pet won’t tolerate treatment, and more importantly won’t, live longer and live better with treatment than without.

DON’T THINK THERE ARE NO OPTIONS FOR PETS WITH ADVANCED DISEASE LIKE METASTASIS.
Traditional chemotherapy is given at the maximum tolerated dose (MTD) chemotherapy. MTD chemotherapy is the highest dose level for an individual chemotherapy drug that produces acceptable
dose limiting toxicity (DLT). The DLT is considered the clinically acceptable/ manageable side effects at cytotoxic doses. Chemotherapy is most active in rapidly proliferating cells. The cellular targets include DNA, RNA, and proteins. The result is irreparable damage that leads to apoptosis and necrosis. MTD is typically not effective for dogs with advanced macroscopic metastatic disease.

On the other hand, metronomic chemotherapy is pulse or low-dose continuous chemotherapy. This is typically administered daily or every other day. The target for this treatment protocol is endothelial cells that line tumor blood vessels. The goal may only be to stabilize the tumor but at least this may prevent the tumor from spreading. Common chemotherapy drugs include: Palladia, cyclophosphamide and chlorambucil. There is still much to be learned, including: best drugs, dose, schedule, tumor types, and toxicity.

DON'T BE AFRAID TO REFER TO A SPECIALIST.

Surgery: Boarded surgeons are more aggressive at surgery - in a good way. Studies show that for malignant tumors, patients often have better outcomes when the surgery is done by a specialist. Why? By removing more tissue the first time, they are more likely to get those clean and WIDE margins and prevent the need for a second scar revision surgery. Surgeons are trained in advanced techniques like flaps, which can help get margins. In the long run, this will save the client money (1 surgery vs. 2), reduce treatment, reduce morbidity, and improve recovery. Boarded surgeons are often trained in proper biopsy and surgery techniques to avoid tumor contamination and spread at time of surgery.

Oncology: The oncologist provides the most current and comprehensive treatment options and newest available prognostic data. I will counsel the client about the most appropriate options. I will adjust staging based on budget and the individual case. I will adjust treatment protocols based on response and tolerance. And I will work with the primary care veterinarian and other specialists to provide comprehensive care.

As an oncologist, I have advanced training in cancer. My expertise and experience is the management of cancer patients. And I give chemotherapy – lots of it. You want an expert when it comes to these drugs, side effects, and current protocols. When it comes to general health – you, the primary care veterinarian, is the expert and the one to turn to. The primary care veterinarians are the experts in preventative health care, nutrition, vaccines, thyroid disease, kidney failure, diabetes, general surgery, and geriatric pet health. And I send my cases back to the primary care veterinarian for management of those issues. But when it comes to cancer, in my opinion, you should refer to an oncologist.

DON'T THINK YOU MUST PERFORM THE STAGING DIAGNOSTICS BEFORE YOU REFER TO THE ONCOLOGIST.

I sometimes see lymphoma patients who are told that all staging diagnostics need to be done before they come see me. But then they decide not to treat, so those tests were a waste of money and time.

In other cases, the pet guardian may have a limited budget for diagnostics and treatment. Depending on the case and the cancer, I will discuss the pros and cons on eliminating certain staging tests. If a dog with lymphoma is having difficulty breathing, I will strongly encourage we get the chest radiographs. If not, I may choose immunophenotyping or abdominal ultrasound over chest radiographs. If we are going to do a CT scan of one area of the body (such as of the head for an oral or nasal tumor), we can also CT the chest, which is a better test to look for chest metastasis. So consider holding on doing the chest X-rays if CT is recommended for a case. If you are taking chest radiographs, don’t forget the 3rd view.

For other cases, I may skip certain tests and apply those funds toward treatment. It’s less about a checklist of tests, and more about what is important and needed in each case. I will adjust staging based on budget and patient needs.

DON'T START STEROIDS (SUCH AS PREDNISONE) BEFORE CHEMOTHERAPY OR BEFORE THE DIAGNOSIS OF LYMPHOMA IS CONFIRMED. TWO REASONS:
One: Steroids complicate diagnostics. If you start prednisone before we complete staging with other testing like chest X-rays and abdominal ultrasound, those tests will be less accurate. Staging is not only prognostic, but I find having a baseline very helpful in monitoring response and sometimes distinguishing symptoms due to the chemotherapy or the cancer itself. An important prognostic predictor for lymphoma is phenotype (B-cell vs T-cell). If the dog is on prednisone and the lymph nodes are going into remission, we cannot run this test. Not only is it the best predictor we have for response and survival, it does change the chemotherapy protocol used. The UW CHOP multi-agent protocol does not work as well for T-cell lymphoma as is does for B-cell lymphomas. Along the same lines, if the lymph node aspirates were inconclusive, I will need to re-aspirate or biopsy a lymph node to confirm the diagnosis. If the dog is on prednisone, getting a diagnosis becomes a challenge.

Two: Steroids potentially make treatment with chemotherapy less effective. Unfortunately, prednisone started first can interfere with chemotherapy and can trigger a mechanism called Multi Drug Resistance. Pre-treatment prednisone has been demonstrated to be a strong negative prognostic factor for dogs with lymphoma.

DON’T LET THE PET OWNERS DECIDE ABOUT BIOPSY SUBMISSION. DO SUBMIT ANY MASS YOU REMOVE TO THE LAB.
Every mass removed should be submitted and examined by a pathologist at the lab. If you remove had 4 masses, all 4 should go to the lab—even if you think the masses look benign. Histopathology is the only way to know tumor type, grade, and for margin evaluation. And do not assume the owner won’t treat if its cancer; many reconsider once they learn that cancer is often treatable and treatment can be well tolerated.

If it is important enough to remove, it is important enough to submit for histopathology.

DON’T: ASSUME CLEAN SURGICAL MARGINS WERE CLEAN.
DO: ASK THE PATHOLOGIST TO REPORT MARGINS AND CONSIDER IF THIS IS WIDE ENOUGH FOR THIS TUMOR TYPE.

I often see dogs and cats months after a tumor was removed and then grew back. When I read the original biopsy report, it will often read: margins were clean. Good news, right? BUT when I read the microscopic decision, the measurement will say <1mm. For a malignant tumor, this is not clean. For many malignant tumors 2 to 3 cm are recommended. For STS, incomplete margins are 10X more likely to recur.

I am surprised how often this important detail is overlooked. For a benign skin tumor like a sebaceous adenoma or a benign liver tumor, margins of a few millimeters may be adequate. But malignant tumors require wider margins. So, remember, size matters! Ask how wide the margins were (get numbers) and ask if that is wide enough for this tumor type. If not sure, consult with an oncologist.

DON’T THINK RADIATION = BAD SIDE EFFECTS

CyberKnife Radiosurgery or Stereotactic RadioSurgery (SRS) is non-invasive, frameless system that uses multiple beams to deliver single fraction or hypofractionated RT with submillimeter accuracy. The large number of beams with continuous image-guidance during treatment allows high conformation around the tumor and greater precision than traditional linear accelerators and IMRT machines.

The unique robotic delivery decreases toxicity, improves targeting with submillimeter accuracy, and allows treatment in fewer fractions - 1 or 3 treatments (conventional radiation is usually 20 treatments). Since less normal tissue is irradiated, there are fewer treatments, less anesthesia, decreased side effects, good tumor control, fewer hospital visits, and less worry by the pet owner. CyberKnife is used instead of surgery and treats macroscopic disease, not microscopic. CyberKnife is used when “blade” surgery impossible, would cause unacceptable morbidity, or is refused. CyberKnife can also be used if conventional radiation has failed and can be used to retreat cases that have already had SRS.
Which tumors can we treat with CyberKnife Stereotactic RadioSurgery?

Treatment of Choice:
- Brain tumors, Nasal Tumors,
- Oral tumors especially maxillary, Spinal tumors, Thyroid tumors (non-surgical), Prostate tumors, Re-treatments

Useful case by case:
- Appendicular and axial OSAs, Soft tissue sarcomas, Lung Tumors, AMMs (thymoma).
ESSENTIAL TIPS FOR SPLENIC MASSES

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KEY POINTS

- Dogs with splenomegaly and splenic masses generally follow the “double two-thirds rule”: 2/3 have splenic neoplasia, and 2/3 of those have hemangiosarcoma. So 1/3 do not have cancer! Hemangiosarcoma is not the only differential for a dog with a splenic mass.
- Hemangiosarcoma (HSA) is the most common primary canine splenic cancer in dogs, and it is locally aggressive and highly metastatic.
- The likelihood of splenic tumor increases with anemia, nucleated RBC, abnormal RBC morphology, or splenic rupture.
- Prognosis for splenic masses cannot be determined without histology which usually requires surgery. A common clinical error is to assume HSA based on the presence of a splenic mass. Large masses are not necessarily malignant. Several splenic lesions have similar ultrasound and gross appearances.
- Except for lymphoma, splenectomy is the treatment of choice for splenic tumors when there is no evidence of metastasis based on staging tests. Even at surgery, it is often impossible to distinguish various diseases based on gross appearance of the spleen or liver.
- Dogs with HSA treated with local therapy and chemotherapy live longer than dogs without treatment and with local therapy only but 1-year survival rates are still low (10%). Chemotherapy is generally well-tolerated in most dogs, and only a minority develops significant toxicity.

WHO, WHAT, WHERE, WHY

Splenic neoplasia can arise from any of the normal splenic tissues including blood vessels, lymphoid tissue, smooth muscle and connective tissues. Common splenic tumors include HSA, mast cell tumor, lymphoma and various sarcomas. Hematomas are the most common benign splenic masses. Splenic tumors usually occur in large breed dogs. Breeds most at risk are German shepherd dog, golden Retrievers, and Labradors. German shepherds also have a high prevalence of hyperplastic nodules and hematoma.

Clinical signs are typically vague, non-specific and include enlarged abdomen, anorexia, lethargy, depression, vomiting, and diarrhea. Clinical signs also vary with how advanced disease is, so dogs may have acute and often dramatic acute signs including collapse and hypovolemic shock. In one study 80% of dogs with acute abdomen and no history of trauma had malignant cancer and 88% were HSA. Splenomegaly is readily detectable through abdominal palpation, radiography and ultrasonography.

Differentials Diagnoses: Hemangiosarcoma is not the only differential for a dog with a splenic mass. A common clinical error is to assume HSA based on the presence of a splenic mass. Large masses are not necessarily malignant. Several splenic lesions including HSA, hemangioma and hematoma have similar ultrasound and gross appearances.

Lymphoma (LSA): LSA that involves the spleen is most commonly part of multicentric LSA and typically is a diffusely infiltrative disorder. Some lymphomas may occur as solitary splenic nodules, especially marginal zone lymphoma and mantle subtypes of the indolent form. Similarly acute and chronic leukemias can also diffusely infiltrate the spleen.
**Malignant Histiocytosis (MH):** MH is an uncommon cancer of atypical histiocytes and has progressive, multicentric involvement of multiple organs, including the spleen, liver, lymph nodes, and bone marrow. The Bernese mountain dog has a familial predilection.

**Mast Cell tumors (MCT):** Tumors of primary visceral organs including the spleen are rare in dogs. Visceral mastocytosis is typically preceded by a poorly differentiated cutaneous MCT.

**Splenic sarcoma:** Splenic sarcomas are non-angiomatous, non-lymphoid tumors of connective tissues and include fibrosarcoma, leiomyosarcoma, extraskeletal osteosarcoma, and undifferentiated sarcomas. A high mitotic index (MI) of >9 is a negative prognostic factor for survival. Splenic sarcomas tend to be fatal within 1 year. Splenic leiomyosarcoma have a high metastatic rate but dogs that survive the initial post-surgical period have a MST of 8 months.

**Hemangioma:** Hemangiomas are benign tumors of blood vessels. Surgery is curative.

**Non-neoplastic:** hematoma, abscess, nodular hyperplasia, granuloma

**HEMANGIOSARCOMA (HSA)**
HSA is an aggressive malignant cancer of transformed vascular endothelial cells. It causes local infiltration and rapid systemic metastasis. German shepherds and golden Retrievers are at greatest risk. Gross metastasis is present at diagnosis in more than 50% of cases. Excluding cutaneous cancers, it accounts for about 5% of primary cancers in the dog.

Spleen is the most common primary site, but other common sites include right atrium, liver, skin and subcutis. HSA may be solitary, multifocal in an organ, or widely disseminated. Metastasis is typically hematogenously or via transabdominal transplantation. Metastasis is most commonly to the liver and lungs. Less common sites of metastasis include the omentum, mesentery, brain, muscle and bone. HSA is considered the most common metastatic tumor to the brain.

**DIAGNOSTIC WORK UP FOR HSA**

**CBC and chemistry panel:** The likelihood of splenic tumor increases with anemia, nucleated RBC, abnormal RBC morphology, or splenic rupture. The anemia may be regenerative with splenic rupture depending on the duration. Neutrophilic leukocytosis may also be present. Other abnormalities include Howell-Jolly bodies, poikilocytosis, acanthocytosis, schistocytosis and/or thrombocytopenia. Thrombocytopenia is common in 75-97% cases and ranges from mild to severe. A coagulation panel should be run if HSA is suspected.

**Imaging:** Three-view chest radiographs are mandatory to rule out pulmonary metastasis and pleural fluid. Three-views significantly decreases the false-negative rate. Abdominal ultrasound confirms the mass and allows detection of abdominal effusion, defines splenic architecture, and provides detailed evaluation of the abdominal organs and is less affected by abdominal effusion than radiographs.

**FNA and cytology:** Ultrasound-guided FNA is relatively simple, cost-effective and typically a safe procedure. It is most helpful for cases where the diagnosis eliminates the need for surgery, such as lymphoma. For diffuse splenomegaly, the spleen is accessible for cytology. But even with ultrasound-guidance, if non-representative tissues are sampled, you may get a false negative of benign or reactive. In one study, only 61% of cases did cytology match histologic diagnoses. FNA is not recommended for mixed echogenicity masses suspicious of HSA as the masses are often extremely friable so there is an increased risk of hemorrhage in addition to the low diagnostic yield due to hemodilution.
HSA effusions are serosanguinous or frank blood and usually do not clot. Unfortunately cytology is typically non-diagnostic.

**Cardiac evaluation:** Since 25 to 45% of dogs with splenic HSA have concurrent right atrial HSA, an echocardiogram is recommended. In my experience this is lower at presentation. Arrhythmias can occur with benign and malignant lesions.

**TREATMENT MODALITIES FOR HSA**

**Treatment pearls:** Treatment for HSA is ideally both local and systemic. Chemotherapy improves the MST, but HSA is still a frustrating cancer for owners and veterinarians with shorter survival times than many malignant cancers in dogs. The majority of dogs tolerate chemotherapy quite well and will maintain a good to excellent quality of life even during chemotherapy.

**Treatment: Surgery** Except for lymphoma, splenectomy is the treatment of choice for splenic tumors when there is no evidence of metastasis based on staging tests. Even at surgery, it is often impossible to distinguish various diseases based on gross appearance of the spleen or liver – including hematoma, nodular hyperplasia, hemangioma and HSA. Ideally the entire spleen should be submitted fresh on cold packs or in formalin. Biopsy of normal liver is controversial and may not be useful. The abdomen should be thoroughly explored and any suspicious lesions removed or biopsied. About 25% of dogs develop arrhythmias post-op. An ECG should be monitored during and after surgery, and they usually resolve within 24-48 hours.

**Treatment: Chemotherapy** The goal of chemotherapy is to achieve is to delay the metastatic disease that develops quickly after splenectomy. Since chemotherapy improves the MST, it is considered part of the standard of care. Single agent doxorubicin and combination protocols are most common.

Recently low dose oral chemotherapy (metronomic) was comparable to conventional doxorubicin. This protocol included low dose cyclophosphamide, piroxicam and etoposide. Current studies are evaluating whether conventional chemotherapy followed by maintenance metronomic chemotherapy for VEGF-receptor kinase inhibitors such as tocerinib will improve outcome.

**PROGNOSIS FOR HSA:** Overall the prognosis with surgery alone is poor and reported MST in dogs treated with surgery alone ranges from 1 to 3 months, and less than 10% survives 1 year. Adjunctive chemotherapy improves the MST to 4 to 6 months, and doxorubicin-based protocols are the mainstay. Stage I, non-ruptured tumors may have an improved prognosis when chemotherapy is administered after surgery. Low grade tumors may also have a better prognosis.

**Additional Resources**

WHY IS ONCOLOGY IMPORTANT?
The statistics are overwhelming. One in 3 dogs of any age will get cancer, and 50% of dogs over 10 years old will be affected by some tumor. Cancer is the number one cause of canine death. Many pets are considered important family members, and owners have increasing expectations. They want the same standard of care, highest quality medical care, compassionate care, and respectful communication. But when the pet is sick and has cancer, the human animal bond becomes stressed and fragile, making communication between the veterinarian and the owner more challenging.

CANCER COMMUNICATION CHALLENGES
Veterinarian/client communication is critical to optimal patient care. Yet there is a lack of guidelines and training to help veterinarians and clients broach difficult conversations about prognosis, treatment and palliative care options for pets diagnosed with cancer. As veterinarians and oncologists, we need clinical guidance to help initiate these conversations and better integrate both definitive and palliative therapy into our oncology practice.

Cancer communication training varies with regard to content, duration and methods. There is often a skills gap between veterinary school curriculum content and actual skills to be successful in practice. As a result, many veterinarians feel unprepared for difficult conversations about cancer.

Since the cancer diagnosis is typically made by the primary care veterinarian, The primary care veterinarians often have the more difficult job compared to the oncologist. The primary care veterinarians have the relationship with the client. On the other hand, when the client visits the oncologist, the owner usually knows cancer is the diagnosis, and the focus is treatment options and decision making, but there is no pre-existing relationship, familiarity, or trust.

Many barriers currently prevent veterinarians and clients from engaging in cancer conversations and optimal cancer care planning. To address barriers to advanced cancer care planning, we must first identify the challenges.

For the veterinarian, challenges may include general discomfort in talking about cancer and death, lack of training, shortness of time, practice culture, feelings of responsibility for cancer or a late diagnosis, perception of failure, unease with death and dying, uncertainty of outcome, impact on relationship with client, and the worry about patient quality of life (QOL), about client response, about costs, and about the veterinarian's own response.

For the client, conversations are challenging as clients are often emotional and dealing with their feelings of self-blame, guilt, anxiety, fear, and frustration. There is the unease with death and dying, anticipatory grief, and concerns about the effect on human-animal bond. The client is also concerned for the pet’s QOL, the costs, the time required to treat the cancer.

HOW DO YOU GIVE YOUR INFORMATION?
The first method is called Data Dump, and is often nick-named the Shot-put technique. The oncologist does most of the talking like a monologue, and the client is more passive. The intent is on delivery of information, but it is often too much information for the client to absorb and too challenging to receive the message. One way to improve this is to add open ended questions, so we know we are all on same page with cancer information.
A better method is the collaborative approach, is often nick-named the Frisbee technique. This reciprocal interaction focuses on a dialogue. The delivery is light and airy, and information delivery is given in small pieces. Here the emphasis is on eliciting client feedback.

CORE CANCER COMMUNICATION SKILLS
Gathering information
It is very helpful to identify the client’s full agenda and help the client identify concerns. Use open ended questions that start with: how, what, and tell me. Examples are “What other questions do you have?” or “Anything else you’d like to discuss?”

Elicit the client’s perspective. Does the client have a previous experience with cancer in people or another pet? It is important to identify misconceptions of cancer and barriers to care. Examples are “What are your goals with treating Bo’s cancer?” “What are your hopes?” and “What are your fears?”

Explaining and Planning
Assess the client’s knowledge level and what level of information to give. It is also important to determine what degree the client wants information, and be aware this may change with time. Since many clients are overwhelmed in the beginning, it is often helpful to start with the big picture and ask what they know already and to what additional information they are seeking. “Chunks and check” is very helpful when having cancer conversations. You give information in small chunks, and then follow with checks for understanding. This is less lecturing and aims to increase recall, understanding and commitment. Use questions like: “What questions do you have?” or “What part of the plan is most difficult?”

Building Relationships
Offer partnership and use inclusive language like let’s, we, our, us, such as “We’ll work together for Teddy. “Asking permission during the conversation will allow you to assess the client’s readiness to take next step with questions like: Would you like to schedule surgery? Are you ready to start treatment?

Express Empathy: Acknowledge clients emotions and put yourself in their shoes and communicate that you know where they are coming from. Example: I can only imagine how hard this is. Nemo has been part of your family for so long.

Demonstrate appropriate nonverbal behavior: This can be helped with an attentive body posture, sit at same level, sit close, and maintain good eye contact. Use a slow pace, lean forward, reach out to touch.

Provide Structure and Summarize
It is useful to take time to reflect what the client heard, to repeat key aspects of diagnostics and treatment, and to provide a summary at end of appointment. For example, “I recommend these tests and this treatment for Bo’s melanoma but there are options. What questions do you have?”

“I don’t have time for this!” Core communication skills actually save time and allows for more efficient veterinarian-client-patient interaction. If you spend time to build a relationship early, it will pay dividends through diagnosis and treatment.

DELIVERING BAD NEWS
I was never taught this in school, my internship, or my residency. Most oncologists learn to break bad news by observing more experienced colleagues in clinical situations, typically during their residencies. Many veterinarians report a lack of confidence in their ability to break bad news.

The specific lack of training opportunities appears to play a major role in leading to this problem. In a human study, almost 40% of respondents not only had no didactic training, but also did not have an opportunity to gain experience from observing other clinicians breaking bad news.
Delivering bad news is a complex communication task that you may have to do thousands of times. It is stressful for clinicians to carry the burden of responsibility for communicating bad news. Complicating factors include our experience (or inexperience) giving bad news, the relationship with the client and their pet, and often the limited treatment options.

For the client, the response is affected by their relationship to their pet, severity of the diagnosis, past experiences with other pets or human family members, other stressors in their life, and their support system.

When saying “Your pet has cancer”, be aware of where and when you deliver the bad news. Too many clients hear those fateful words with less than an appropriate manner in less than an appropriate setting. Common mistakes include having the conversation in a space with no privacy, having a conversation that is too short, and having no treatment plan to discuss. Poor communication can lead to general dissatisfaction and a loss of trust.

Instead, when saying, “Your pet has cancer”, think empathy and respect, go to a private location, have sufficient time and attention, and be sensitive.

SIX STEP STRATEGY: SPIKES

1. **SETTING UP** the Interview
   - Mental rehearsal
   - Privacy
   - Involve significant others
   - Sit down
   - Make connection
   - Eye contact
   - Touch

2. Assessing the client’s **PERCEPTION**
   - Before you tell, ask
   - Open ended questions
   - “What have you been told about…?”

3. Obtaining the client’s **INVITATION**
   - Some want full information for diagnosis, prognosis and details of the illness
   - Some do not
   - Some may want more information later

4. Giving **KNOWLEDGE** and Information to the Client
   - Warning that bad news is coming “Unfortunately I have some bad news”
   - Use non-technical terms: Spread vs metastasis, Sample of tissue vs biopsy
   - Chunk and check

5. Addressing the Client’s **EMOTIONS** with **empathetic** responses
   - Observe: silence, disbelief, crying, denial, anger
   - Let client express feelings
   - Make a connecting statement
   - Examples of empathic, exploratory, and validating responses (Baile, 2000)

6. **STRATEGY and SUMMARY**
   - Make a clear plan
   - Consider a referral
   - Address pain control and symptom relief
DISCUSSING PROGNOSIS
There are a few approaches to discussing prognosis. The first is realism. Interestingly, in people, 20% of patients do not want full prognosis information. Second is optimism. If overly optimistic, clients may lose opportunities to fulfill last wishes, prepare themselves and family, and spend quality time with their pet. With avoidance, you may appear evasive or dishonest. In addition, the veterinarian risks the trust and relationship with client, and the client could compromise pet’s care.

Do not make assumptions about what the client wants to know. Ask. “How much would you like to know about course of Myles’ lymphoma?” Some like details or the big picture. This is a good time to use chunk and check.

Balance hope and reality. The median survival time can be helpful. Acknowledge the client’s emotional reaction and remember to compose yourself, pace yourself, and allow time to reflect.

FIRST REACTION
Remember the client’s initial reaction may be to not treat at all. It is okay not to treat after we provide information about the diagnosis, treatment options and prognosis. We must provide accurate information about cancer, and a range of treatment options. We can replace misperceptions and fear with knowledge and hope and educate the client that pets with cancer can live longer, and live well – not only after treatment, but during treatment too.
GLAUCOMA – NEW INSIGHTS INTO AN OLD PROBLEM

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INTRODUCTION

If you ask any ophthalmologist to list the most frustrating problem that we face, invariably the answer will be glaucoma. It is the leading cause of blindness in dogs but is a nemesis in any species in which it presents. The frustrations stem not just from our frustrations at eliminating patient (and owner) suffering but also from the different causes and ways it may present, the potential for insidious onset and inexorable progression despite our best efforts, the number of ocular diseases and surgeries in which it may be a secondary complication, the time and economic commitment required for glaucoma monitoring, the myriads of treatments without one primary treatment that is uniformly successful, and the horrendous expense of medications, surgeries, and just lost time in man hours for both the owner and the practitioner. It is even frustrating for us to stand before you talking about the same disease year in and year out. However, we do have some new discoveries, new medicines, and new surgical techniques upon which we can rely. In the brief time we have with you today, I hope to bring you up to date on our newest discoveries and advances with the hope that we can through early detection and treatment prolong vision in affected animals.

NEW INSIGHTS ON ANATOMY – PRIMARY AND SECONDARY

Iridocorneal angle

We have long understood aqueous humor flows anteriorly from where it is produced in the ciliary processes into the anterior chamber where it exits through the iridocorneal angle. Primary glaucoma has been described as open or closed angle depending upon the appearance of the iridocorneal angle. Examination of the angle is possible using a gonioscopy lens provided the cornea and aqueous are clear. Narrowing or other abnormalities of the angle can thus impair the flow of fluid from the eye although the there appears to be a great deal of reserve capacity in that prominent obstructions can be visibly present in the presence of a normal intraocular pressure. At the same time, inflammation can result in swelling of outflow channels and/or clogging of those same channels with inflammatory cells, fibrin, and protein in the aqueous humor. (See Figures 1-4.)

Ciliary Cleft

In recent years, through the use of high-resolution ultrasound (HRUS), we have been able to image the ciliary cleft where aqueous passes after it moves through the iridocorneal angle, and the results have revealed another factor that can contribute to increased intraocular pressure whether the iridocorneal angle is normal or not. In the following figures, note the differences in the ciliary cleft between the right and left eyes of a 10 year-old spayed female Boston terrier. The dog presented for cataracts and had a mature cataract in the right eye and an early immature in the left eye. At the initial examination gonioscopy revealed the iridocorneal angles were normal in both eyes. An electroretinogram was scheduled to evaluate retinal function (since the retina in OD could not be visualized) and prior to dark adaption the pupils were dilated with 1% tropicamide (a short acting anti-cholinergic mydriatic). The intraocular pressures (IOPs) were measured prior to and following dilation of the pupils. Prior to and following dilation the IOP in OD was 18 mmHg whereas the IOP in the left eye was 21 mmHg prior to dilation and 26 mmHg following dilation. HRUS revealed a normal ciliary cleft in the right eye with the mature
cataract) and a markedly narrow ciliary cleft in the left eye with the immature cataract. (See Figures 5 and 6.) Such occurrences illustrate the potential for problems with dilation of affected eyes as well as for an increased susceptibility for complications following cataract surgery.

PREVENTION OF GLAUCOMA

Primary vs. Secondary

The prevention of glaucoma centers on the recognition and, when possible, elimination of the risk factors. With secondary glaucoma this would center on the early detection and control if inflammation and secondary scarring. This may be easier said than done, but it is much more likely than eliminating the anatomic and genetic factors at play with primary glaucoma. In the latter, the best we may achieve may be to delay the onset of the elevated intraocular pressure as long as possible.

The application of twice-daily topical 0.5% timolol with 0.1% dexamethasone or the use of 0.125% demecarium bromide once or twice daily with the 0.1% dexamethasone have been shown to delay the onset of angle closure glaucoma and may be administered to the eye with normal intraocular pressure. The exact mechanism remains to be fully elucidated, but prevention of inflammation and increasing facility of outflow are thought to be key factors. Some ophthalmologists favor immediately starting topical prostaglandin F₂-alpha analogues or carbonic anhydrase inhibitors in the normal eye, but such action would eliminate the potential for gradually increasing medications as pressure rises or for countering an acute glaucoma attack while vision is preserved long enough to allow surgical intervention.

MOST EFFECTIVE CURRENT MEDICAL THERAPIES

Carbonic Anhydrase Inhibitors

Acetazolamide: 3-5 mg/lb b.i.d.-t.i.d. (greatest systemic side effects)
Methazolamide: 1-2 mg/lb b.i.d.-t.i.d. (metabolic side effects still can occur)
Dorzolamide (Trusopt®) 2% ophth. soln.: 1 drop t.i.d. (Generics available)
Dorzolamide 2% with Timolol 0.5% (Cosopt®) : 1 drop t.i.d. (Generics available)
Brinzolamide (Azopt®) 1% ophth. soln.: 1 drop t.i.d.

Prostaglandin F₂-alpha Analogs - once or twice daily dosing

Latanoprost 0.005% (Xalatan® - available as generic)
Travoprost 0.004% (Travatan Z®)
Bimatoprost 0.01 and 0.03% (Lumigan®)

Act to increase aqueous outflow + ? (uveoscleral flow, others?)

Notes and precautions on prostaglandin F₂-alpha Analogs:

Increase iridal, eyelid and eyelash pigmentation in man and primates. Use with caution in uveitis & lens luxation because they are potent miotics
Hyper-osmotic Agents

While Mannitol (0.5-2 g/kg slow IV) is classically recognized, it is cumbersome to administer and not without significant risk of systemic side effects. Further it must be administered in the hospital. Oral U.S.P. pure glycerin is administered orally at 1/3 cc/lb with equal volume of water, milk, or melted ice cream. After dosing, withhold water for 1.5 hrs. May be repeated q. 8 h. and may be kept at home by the owner for emergency use in acute glaucoma.

CURRENT SURGICAL TREATMENTS

For Visual Eyes

Diode laser cyclophotocoagulation is perhaps the most successful long term surgery and is usually performed using an endoprobe to allow visualization and ablation of individual ciliary processes. It is usually performed with or without lens extraction, and an Ahmed valve or other shunt may be implanted prior to performing this procedure to allow a means of ameliorating postoperative pressure spikes associated with inflammation from the procedure. Alternatively repeated anterior chamber paracentesis may be performed postoperatively but that may need to be done several times per day for 2-4 days. Trans-scleral cyclophotocoagulation preceded this method but cause so much inflammation that shunts were/are usually tried first. Shunts are indicated to provide immediate lowering of IOP with less potential for postoperative inflammation. Repeated procedures may be needed and costs are significant.

For Non-visual Eyes

Enucleation, intraocular prosthesis, or intravitreal injections of gentamicin should be considered in cases where vision is irreversibly lost. These procedures along with their pros and cons will be discussed in lecture.

Figure 1 (Above left): Normal canine iridocorneal angle with well-differentiated pectinate ligament. Figure 2 (Above right): Normal feline iridocorneal angle
Figure 1 (Above left): A very narrow angle in cocker spaniel. The intraocular pressure in this eye was normal, but the fellow eye had markedly elevated intraocular pressure.

Figure 2 (Above right): This is the iridocorneal angle of an Australian shepherd with mesodermal goniodysgenesis in which the pectinate ligament did not differentiate completely resulting in areas where a broad membrane bridges the iridocorneal angle. While the IOP was normal in this eye, it was elevated in the fellow eye where hyphema was present after the dog was kicked in the head by a horse.

Figure 3 (Above left): OD - HRUS of the anterior segment of the right eye revealing a normal iridocorneal angle (solid angle) and ciliary cleft (open arrows)

Figure 4 (Above right): OS - Note the normal iridocorneal angle (solid arrow) but narrow to nonexistent ciliary cleft (open arrows) accounting for the reason for the increased IOP in this eye after dilation.

“References available from the author on request”
PROBLEM CORNEAS: WHEN CORNEAL ULCERS WON’T HEAL OR "Outlaw Ulcers: Gunfight at the Corneal Corral"

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Introduction

Diseases of the ocular surface may well comprise the most common ocular problems that confront the veterinary practitioner. Corneal ulcers present in a variety of ways, and most may be easily managed and heal readily thanks to the durability and physiology of that amazing ocular structure. However, when ulcers do not heal, the integrity and function of the entire eye is at risk since infection and progression of the ulcers can result in corneal perforation and compromise of intraocular structures. Identification of infectious agents in progressive corneal ulcers and their management with medical and even surgical therapy can be straightforward. But what happens when an ulcer is stagnant and does not heal? At that point the risk of progression is always present, and we can find our clients and ourselves questioning our ability to manage what appeared to start as a fairly simple problem. It is then that we feel like we are hopelessly "outgunned" in a shootout. In those times it is critical to know what is happening. Just like in a gunfight, you have to know friend from foe.

Today we will concentrate on non-healing corneal ulcers in dogs, cats, and horses and some of the intricacies of dealing with the differences in these species. We will focus on recurrent erosions and indolent ulcers with clarification of the differences and specific measures to treat each different entity, but we will also look at some deep progressive ulcers and what to do if time allows.

As if the confusion over the types of ulcers and differences in the species we treat were not enough, the proliferation of the names that have been assigned to different clinical entities that affect the cornea (and their treatment) can leave the practitioner befuddled not only regarding what they are treating but how to manage the problem. Numerous questions abound as the client is in the exam room with their furry family member with the "squinty eye" waiting for the sheriff (a.k.a. veterinarian) to banish the outlaw eye problem. After all, “It’s just a scratch, isn’t it?” As the beads of sweat begin to form we ask ourselves things such as the following: Do I debride this? Culture it? What antimicrobial agent(s), if any, do I need? Will it progress? When do I see it back? Why won’t this heal? You get the point.

To start things off we’ll look at some cases of problem corneas and get your opinions on when multiple punctate keratotomy or grid keratotomy should be used

What’s the Point?

The point of this exercise is that multiple punctate keratotomy (MPK) or grid keratotomy has become one of the most overused treatments for corneal ulcers. Quite often this is to the detriment of the cornea, the patient, and the pocketbook of the client. These procedures should only be used for the treatment of true recurrent erosions and NEVER for other types of corneal ulcers.
While we are on the subject of over-utilized measures, repeated corneal debridement is also often misused. Debridement should be used to remove necrotic or devitalized tissue. It should not be used to remove regenerating epithelium just because it is taking longer than usual for a diseased or stressed cornea to heal. Remember that unlike a cornea with a fresh wound, healing of ulcers of longer duration will heal more slowly.

**General Principles and Goals**

When dealing with “outlaw” corneal ulcers your goals should be to understand what pathogens (if any) are present, understand the pathophysiology of the disease process, and develop a rational therapeutic plan with an understanding of species and breed variations that may exist.

Addressing the last point first, with dogs and cats it is important to evaluate facial conformation and differences in corneal sensitivity as well as potential for allergic disease (especially in dogs), facial hair, keratoconjunctivitis sicca (either primary or secondary), and face rubbing. For cats, herpetic disease is always the number one consideration while viral disease in dogs is rare. For horses we have to remember that most, if not all, live in an environment that is rich in fungal spores, but other problems including immune mediated keratitis and herpetic disease are also possible. It is important to remember that bacterial and fungal disease require pre-existing damage to the cornea so primary infections, with the exception of viral disease, are rare. Knowledge of the most common pathogens for each species is a key factor in selection initial medical therapies and cultures will allow timely adjustment of therapy for corneal infections.

When presented with a corneal ulcer the clinician must evaluate or “stage” the severity of the ulcer and gauge the pathophysiology of the ulcerative process. With that accomplished he or she must then institute therapy and adjust it based upon timely follow-up assessments.

**What’s In A Name?**

As a point of clarification, let’s begin with simplifying what we call things into 2 categories, recurrent erosions and indolent ulcers. I know these terms have been used interchangeably over the years by general practitioners and specialists alike, but it has resulted (for me at least) in a certain degree of confusion.

In *Stedman’s Medical Dictionary*, the term indolent means inactive, sluggish, and painless or nearly so. In medicine it has been classically used in conjunction with a disease causing little or no pain, especially an ulcer that is slow to develop, progress, or heal; one that is persistent. When we speak of corneal ulcers we might argue about the “pain” part of that definition and even the “inactive” part, but it fits a wide range of conditions. Therefore, in this lecture, we’ll use the term indolent for any slow-healing ulcers that are NOT recurrent erosions (recurrent epithelial erosions) regardless of the reason they are not healing (infections, degenerations, depositions of calcium or lipids, etc.).
Recurrent Erosions – Diagnosis and Treatment

We will use the term recurrent erosion (a.k.a. recurrent epithelial erosions, Boxer ulcers, spontaneous chronic corneal epithelial defects or SCCEDs) to refer specifically to ulcers that clinically are spontaneous erosions of the corneal epithelium that do not involve the corneal stroma and that have loose, non-adherent epithelial edges. These erosions occur in middle-aged dogs, are not infected, and have been present for greater than 1-2 weeks. Corneal edema is generally minimal and corneal vascularization may be mild to severe. Fluorescein will spread under the epithelial edges and the erosions may be very large when the loose epithelium is debrided.

The pathophysiology of recurrent erosions is not completely understood, but the basic problem is that the epithelium does not adhere to the underlying stroma. We will discuss the histopathological appearance in lecture. Once healing is finally achieved, it is rare for the condition to recur in that eye.

The treatment of recurrent corneal erosions requires the re-establishment of the normal progression of epithelial adhesion the underlying corneal stroma. As noted above, the corneas are not infected, but a history of repeated multiple changes in antibiotic therapy is common in many of the cases we see. As a result the economic resources of the owners are sometime stretched and they are very frustrated. The most common and successful treatment involves the removal of the loose and dysfunctional epithelium and performance of a grid or multiple punctate keratotomy to penetrate into healthy corneal stroma. Debridement can be performed with a cotton tipped swab to remove all loose epithelium. Healthy epithelium will remain tightly attached. The keratotomy should extend 1-2 mm into the tightly adhered epithelium. A topical antibiotic is commonly used 3 times daily to prevent infection, and a bandage soft contact lens may be placed to protect the migrating epithelium and to increase comfort. An Elizabethan collar may be placed as needed to prevent rubbing the eye. In most instances, with tractable patients, the procedure can be performed awake with topical local anesthesia. Sedation or brief anesthesia may be needed in more excitable patients.

Recently debridement with a rotary diamond burr (Alger Brush II) without additional keratotomy has been used effectively, and results to date compare favorably with that in cases treated with grid or multiple punctate keratotomies.

After performing one of the above procedures it is ideal to allow at least 2 weeks for the epithelium to cover the defect since these corneas are stressed and return to normal physiology can be longer than with simple traumatic erosions. In some cases, corneal vascularization can be marked and granulation tissue can develop to an extent that interferes with epithelial migration and adhesion. Thus, while topical steroids should usually be avoided in the initial treatment of recurrent erosions, the judicious use of topical steroids with close monitoring, may be indicated when heavy vascularization has occurred.

Indolent Ulcers – Refractory Erosions and Ulcers

Indolent ulcers that remain superficial may occur in a variety of corneal diseases. The fact that they remain superficial with little if any involvement of the corneal stroma is an indication that infection is not usually a factor in their pathogenesis. The glaring
exception is in herpetic keratitis that most commonly affects cats. It can be a problem in horses as well (EHV 2).

Figure 2 above is a photo of a cat cornea with dendritic erosions that are pathognomonic for herpetic keratitis. In cats, grid or multiple punctate keratotomies are contraindicated not only because they fail to correctly address the cause of the disease but also because corneal sequestrum formation has been well documented as a sequella.

**Refractory Non-infected Corneal Erosions**

As initially discussed above, non-infected secondary refractory corneal erosions generally do not have loose epithelial edges and should prompt a close examination for the cause of the erosion. Particularly in young dogs (1-4 years of age) perhaps the most common cause is ectopic cilia, and a close examination of the palpebral conjunctiva is in order. Other conditions include trichiasis, distichiasis, and foreign bodies. If lid surgery has been performed, examination for migrating suture may be fruitful. As long as no infection is present, removal of the offending agents should result in rapid healing.

**Refractory Erosions/Ulcers in Senile Corneas**

In older dogs (typically greater than 12 years of age) senescent corneal degeneration can be characterized by significant depositions of calcium and/or lipids that can interfere with epithelial adhesion resulting in recurrent sloughing of the overlying tissue. In most cases the ulcers are relatively superficial and may initially heal with topical application of antibiotics to prevent infection. However, the ulcers will recur and eventually the slough of tissue can be deep with secondary keratomalacia with or without secondary infection. A descemetocele and even corneal perforation can eventually occur if the problem is not resolved.

In cases where calcium deposition is present the topical application of 1% EDTA may elute the calcium from the cornea. With lipid depositions or a combination of the two, keratectomy alone or keratectomy with corneal grafts, conjunctival flaps, or corneo-conjunctival transposition (CCT) is usually required. In some cases, it may be possible to use a diamond burr to remove the depositions, but close monitoring for keratomalacia postoperatively is needed. The point here is that since this occurs in older dogs, clinicians may try to avoid surgery with unsuccessful medical therapy. With these corneas early surgical intervention under carefully planned anesthesia is preferable to emergency surgeries to try to save the eye or to enucleation after the cornea has perforated.

**Corneal Endothelial Degeneration/Dystrophy and Bullous Keratopathy**

Progressive dysfunction of the corneal endothelium, whether related to dystrophy or to degeneration secondary to inflammation, will result in corneal edema that can involve all levels of the corneal stroma. Such edema can progress to bullous keratopathy in which bullae of water migrate to the corneal surface and rupture resulting in painful, indolent or recurrent ulcers. If infection and/or keratomalacia occur, such corneas can develop descemetoceles and even perforate.

Predisposed breeds for endothelial dystrophy include Boston terriers, Chihuahuas, Dachshunds, and possibly Basset hounds. Corneal endothelial degeneration can occur secondary to inflammation in virtually any breed.
Medical therapy for these cases is generally disappointing, but early intervention with Gunderson flaps (keratoleptynsis) not only can arrest the occurrence of the ulcers, but often results in decreased corneal edema.

**Indolent Ulcers Secondary to Infections**

Bacterial and fungal infections of the cornea are generally preceded by a history of damage to the corneal epithelium. Production of toxins as well as proteolytic and collagenolytic enzymes from the organisms and degenerate corneal tissue can compromise corneal function and healing leading to delayed healing and progression to deep ulcerations. Corneal perforation can occur.

Topical corticosteroids and even non-steroidal anti-inflammatory agents can inhibit the immune response and potentiate the activity of proteolytic and collagenolytic enzymes. Topical cyclosporine and tacrolimus inhibit cellular immunity but not the acute inflammatory response. They are therefore contraindicated in mycotic and viral keratitis but may be used in ulcers occurring with keratoconjunctivitis sicca with bacterial infections.

Aggressive measures must be employed in diagnosing and treating deep or progressive corneal ulcers. Diagnostic measures include bacterial and fungal cultures, antimicrobial sensitivity testing (when appropriate), and cytology. Antimicrobial therapy (including topical and systemic agents) should be initiated and adjusted based upon the results of the diagnostic tests and not a trial and error approach with different antibiotics. Inhibition of proteolytic and collagenolytic enzymes should be pursued, and removal of necrotic debris via judicious debridement is indicated. Surgery may be needed in some cases to support the cornea until it is able to heal.

**References available from the author on request.**
Both uveitis and glaucoma share many clinical signs and early cases are often misdiagnosed and treated as conjunctivitis when the clinician is not attentive. In addition, glaucoma can occur secondary to uveitis or can occur with pupillary block if uveitis is mistaken for glaucoma and treated with miotic agents. Therefore, it is extremely important for the clinician to accurately diagnose the uveitis.

Important questions to answer include:

1) How long has it been present?
2) Is vision present?
3) Do the pupils respond normally?
4) What is the pupil size and shape?
5) What is the intraocular pressure?
6) What is the iris color?
7) Fibrin, cells, or proteins present in the AC? On the anterior lens capsule?

Uveitis quite simply refers to an inflammation of the uveal structures (iris, ciliary body, and choroid) inside the eye. We may refer to iritis as inflammation involving just the iris, iridocyclitis as inflammation of both the iris and ciliary body, choroiditis as involving just the choroid, and so on. We may even use the term panuveitis as an inflammation of all three structures. However, this may well be an oversimplification because we do not then draw distinction about what limits the involvement with the inflammation to just one structure or more, what limits the inflammation, and, indeed, we may or may not clinically be able to discern involvement of the choroid if the anterior segment is severely involved.

While the definition may be simple, the disease may be quite complex and the methods of treating the inflammation can vary from very simple to extremely complex. The factors that determine the treatment requirements vary with the cause or causes, the severity and rapidity with which it is controlled, and the degree to which immune responses are recruited to participate in the inflammation. When inflammation is acute, such as with trauma, the responses to treatment may be rapid and complete, but when the inflammation persists and enters a subacute phase the potential for initiation of immunologic reactions increases dramatically and the requirements for successful therapy become much more complex. If the inflammation is not controlled during this time, progression will occur with progressive and sometimes irreversible damage to the sensitive ocular structures can occur. Therefore, it is up to the clinician to recognize the condition, treat it promptly, and insure that treatment is not discontinued too early.

Causes
The causes of uveitis are varied depending on the species and time does not allow us to cover all the specific diseases in this lecture. Most cases of uveitis are immune mediated and the majority of cases are idiopathic. Nevertheless, infectious agents (viral, bacterial, and mycotic), neoplasia, and toxic processes must be ruled out.
In the interest of time for this lecture we will consider these facts and apply it to the management of just two types of uveitis: Lens Induced Uveitis (LIU) and Familial Uveitis of Golden Retrievers. In doing this we can then apply the principles whenever we see uveitis in a patient.

Before we consider these distinct diseases, let us consider that inflammations of the uvea that persist are indeed immune mediated, that is tied to the immune responses of the body regardless of cause. As such we need to understand that the cellular and humoral immunity at work in uveitis is not something that is easily turned off with a few days or weeks of treatment. There is a potential for long term effects that are not countered with a few days on a topical and/or systemic steroid or non-steroidal anti-inflammatory drug. Even when clinical signs of inflammation appear to have abated there is cellular immunity and alterations in tissue structures and compartments that may persist for long periods of time. Therefore as clinicians we should consider what is going on at the cellular level as we assess uveitis and not just the gross appearance of the eye. Just as we understand that immunization for common systemic diseases is an active but subclinical process, a similar understanding should be applied to our management of uveitis. Let’s consider what this means for the two types of uveitis mentioned above. One is a common clinical disorder that we all should recognize, lens induced uveitis with cataract formation. The second is familial uveitis of Golden retrievers.

Clinical Signs
Clinical signs of uveitis include aqueous flare +/- cells, changes in iris color and texture (yellow or blue to green, hyperpigmentation), pigment on the anterior lens capsule, posterior synechiae, and increased vascular congestion with or without neovascularization. Dyscoria and altered pupillary light reflex may also be present. The intraocular pressure is normally low due to decreased secretion of aqueous by the ciliary body. In the event the intraocular pressure (IOP) is normal but other signs confirm the existence of uveitis, secondary glaucoma must be anticipated. Secondary glaucoma is associated with decreased aqueous outflow that is associated with inflammatory cells, fibrin and debris in the iridocorneal angle. Peripheral anterior synechiae may develop or posterior synechiae may result in pupillary block that can result in anterior displacement of the iris base. Corneal edema may occur in due to corneal endothelial decomposition. Uveal neoplasms may present with uveitis that masks the identification of the tumor in the eye, especially with lymphoma and ocular ultrasound may be required to delineate the uveal structures if the inflammation obscures visualization of the intraocular structures.

Lens Induced Uveitis
Lens induced uveitis (LIU) is an immune response to the short chain proteins released from degenerate lens cells in cataracts. The antigenic nature of these proteins occurs because the lens develops inside the lens capsule in the embryo and the cells of the lens continue proliferate throughout life. The immune system does not develop until after birth so the result is the immune system is not exposed to the lens cells to recognize them as “self” antigens making the inside of the lens is an immune privileged site. It follows that LIU must be regarded as a potential problem in virtually all eyes with cataracts and it is especially a problem in rapidly progressing cataracts. Therefore diabetics have a very significant risk and lens capsule rupture can be devastating. In
addition the probability of complications following cataract extraction greatly increases when pre-existing LIU is present.

Therefore, as will be discussed in the lecture on cataracts, the lens is not an inactive structure and cataracts can result in significant, long-term uveitis. As a result, therapy should be instituted early and maintained even when cataract surgery is not anticipated. In many cases, this may be as simple as using topical NSAIDs or steroids once or twice daily, but the eyes should be monitored closely.

**Familial Uveitis of Golden Retrievers**

Uveitis has been observed in related Golden retrievers, can start insidiously, and exist sub-clinically for some time. As it persists, posterior synechiae can form, aqueous flare may be noted, and fibrin can proliferate in the anterior chamber. Ultimately these eyes may develop secondary glaucoma. Early clinical signs in these dogs include low IOP, streaks of pigment on the anterior lens capsule, and conjunctival hyperemia. As the disease progresses these signs increase in severity with aqueous flare becoming evident. Fibrin formation and posterior synechiae that are very resistant to therapy occur, cataracts develop, and intractable secondary glaucoma may occur resulting in the need for enucleation.

Three “facts” stand out. Treatment needs to be early, aggressive, and continuous. Histopathological findings can be confusing since enucleation is usually performed late in the disease and pathologists often report no inflammation is present. There is still a lot we do not know about this disease!

**Principles for Treatment of Uveitis**

Very simply stated, there are three principles for the management of uveitis. The first principle is to control inflammation. Topical and systemic steroids form the bulwark of this initial arm of treatment, especially in acute uveitis. In severe or persistent cases (subacute to chronic) immune suppression is indicated. Most commonly this is accomplished with initial use of systemic steroids, but other systemic treatment modalities include azathioprine and cyclosporine, mycophenolate mofetil, and possibly others. The second principle for managing uveitis is to eliminate infectious causes (bacterial, fungal, protozoal, viral, and parasitic), and the third is to prevent synechiae, retinal detachment, cataracts, and secondary glaucoma.

As an alternative to systemic corticosteroids, I have used systemic (oral) administration of azathioprine at 0.5 mg/lb (1.1 mg/kg) q.d. for 7 days; then every other day thereafter with monitoring of CBC and serum chemistries. May initiate while on systemic steroids with tapering of the steroids beginning after 2 weeks. If tolerated, after 1 year, it can often be gradually decreased and even discontinued provided there is no flare up of the uveitis.

Mycophenolate mofetil has been recommended by some veterinary ophthalmologists, but we have limited data on its safety with long-term use. As noted in Plumb’s Veterinary Drug Handbook, 2015, “Because of the limited experience with mycophenolate in veterinary patients, the adverse effect profile is not well established. At ‘usual doses’ (10 mg/kg PO twice daily) it is usually tolerated well in dogs. Dose-dependent diarrhea appears to be the most common adverse effect, but vomiting, anorexia, lethargy/reduced activity, lymphopenia, and increased rates of dermal infections can be seen. Mild hypersensitivity reactions after intravenous administration are possible. Because of the
drug’s immunosuppressive actions, increased systemic infection and malignancy rates are possible, especially with long-term use.

The problem is knowing how long to treat. As long as the inflammation remains active or is allowed to flare up before the immunologically active cells die out in the tissue, new immunologically active cells will be recruited and the potential for inflammation will persist.

In lecture we will discuss the above specifics in more detail.

References available from the author on request.
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CATARACTS: MORE THAN MEETS THE EYE

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Cataracts are quite simply any opacity of the lens, but the approach to managing cataracts in animals is anything but simple. We will explore what is available for the medical and surgical management of cataracts and discuss new advances for dealing with diabetic cataracts. We will also discuss how to make sense of the numerous products on the market that are presented as “non-surgical cures for cataracts.”

Truth or Dare - Which of the following statements are true?

1. PRA causes cataracts.
2. Cataracts are an inert opacity of the lens.
3. Cataracts should be removed even if the retina is non-functional.

We will discuss those and other statements in today’s lecture.

Anatomy of a Cataract
The lens is made up of an array of living cells suspended inside a lens capsule behind the iris so that it is perfectly positioned to bring light (images) into focus on the retina. The capsule encloses the lens fibers in the developing eye in the embryo. Thus lens cells are living cells that exist in an immunologically privileged site. The lens is thus anything but inert and when disease processes affect the lens, the lens cells can quickly deteriorate to a point where recovery of function is irreversibly lost.

Can Medical Therapy Prevent or Reverse Cataracts?
Except for the development of the topical aldose reductase inhibitor, Kinostat®, no medications have been effective against the development of cataracts and none have ever been shown to reverse significant cataracts.

In addition the degenerate cells in cataracts can release short-chained proteins that I can leak through the lens capsule resulting in a response from the immune system that results in lens induced uveitis. In the event of lens capsule rupture, dramatic phacoclastic uveitis can occur and have disastrous effects on the eye. Thus, the concept that a cataract is “no big deal” needs to be re-evaluated, and some form of therapy should be initiated, even if cataract surgery is not under consideration.

Complications From Cataracts

Abstract — Outcomes for 77 cataractous eyes were compared after each eye underwent no treatment, topical medical treatment only, or phacoemulsification with intraocular lens implantation. Median follow-up time for all dogs was 2.3 y. Failure occurred in all untreated eyes and the rate of failure was 65 and 255 times higher than in medically and surgically treated eyes, respectively. The failure rate was 4 times higher in dogs.

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receiving only medical treatment compared with dogs that received surgery. Across all groups, the success rate for mature and hypermature cataracts was lower than for immature cataracts. Regardless of cataract stage, the chance of success was higher for eyes undergoing phacoemulsification than for eyes that received medical management only. Results of this study support prompt referral for phacoemulsification when cataracts are diagnosed in dogs or, if referral is not possible, topical anti-inflammatory therapy should be instituted.

Complications from cataracts, with or without surgery, include the following:

1. Severe or persistent uveitis
2. Secondary glaucoma
3. Retinal detachment
4. Infection
5. Accidental injury to the eye

So, even if surgery is not contemplated, medical therapy should be used to prevent serious intraocular inflammation and complications secondary to that inflammation. This does not, however, suggest cataract surgery should be recommended if there is no chance for vision as can occur with retinal detachments, progressive retinal atrophy (PRA), etc. In addition, client education about what this means for their pets is a never-ending task for the veterinarian.

Many people have the mistaken impression that cataract surgery in dogs and adult humans is similar with respect to controlling the potential complications. In reality the inflammatory response has been reported to be as much as 10,000 times greater than in adult humans, and is one reason we employ such aggressive pre- and post-operative therapy to control inflammation. In addition, monitoring and treatment for inflammation to prevent complications should persist indefinitely once the initial postoperative period (6 months in our clinic) has passed. Reasons include the immune stimulus and response caused by the cataracts in our patients, the fact that in many cases we tend to delay surgery until vision is more severely impaired than in humans, and there is a delay in recognitions of impending problems by the owners or by the veterinarians caring for the dogs. This is particularly true in diabetic dogs where cataracts can progress rapidly, becoming swollen (intumescent) in some cases to the point of lens capsule rupture. The latter is not surprising considering the fact that insulin regulation is no small matter and can distract from other issues.

Management of Diabetic Cataracts – Kinostat®: A New Paradigm
The reason that diabetic cataracts develop earlier and progress so quickly and with greater frequency in dogs rather than cats or humans is due to in great degree to the fact that dogs have much higher levels of aldose reductase than do other species. We will discuss this with respect to the development of Kinostat® as a topical treatment to prevent cataracts in dogs.

The “proof of concept” for the use of Kinostat® in diabetic dogs in a double masked clinical trial was reported by Kador et al in Veterinary Ophthalmology 13: 363-368, 2010. In that study 40 dogs (28 receiving Kinostat® and 12 receiving the vehicle control) with naturally occurring diabetes were treated t.i.d. in O.U. for a year. We have now completed a 9-month multi-center clinical trial (double masked) confirming the effectiveness of this drug, and we are awaiting approval of the FDA. In the lecture we will
discuss these trials. The results are exciting because it would be far better to prevent cataracts altogether than to manage the potential complications with or without surgery.

With diabetic dogs, the preservation or, in the case of surgery, restoration of vision should have the added benefit of increased activity that may aid in the regulation of diabetes. As has always been the case, therapy for diabetic dogs (whether undergoing cataract extraction or medical therapy alone) requires a dedicated and high motivated, patient owner for ultimate success.

On the subject of PRA, the suggestion that PRA causes cataracts from the effects of degenerative byproducts from the deteriorating retina has surprisingly not been investigated thoroughly. The theory was proposed, apparently in the 1970’s or before and has been accepted as a “truth” since that time. As we learn more and more about genetic mutations we may find other explanations such as both cataracts and retinal degeneration being caused by genetic mutations inherited concurrently.

References


Other references available from the author on request. 
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A Rational Approach to Treating Keratoconjunctivitis Sicca

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It is too simple to think of keratoconjunctivitis sicca as just a lack of tears, so let’s consider the problem and approach it from specific points with specific, targeted actions.

The tear film consists of 3 layers: A superficial lipid (oily) layer which decreases evaporation, a middle aqueous layer which contributes 90% of the tear film, and a deep mucin layer which facilitates spreading of the tears over the cornea. Keratoconjunctivitis sicca (KCS) occurs whenever there is decreased production of tears or increased evaporation or break-up of the tear film. The problem is most common in dogs but occasionally occurs in cats and horses. Usually the problem arises as a decrease in aqueous tear production that may occur as a result of toxic or inflammatory degeneration of the lacrimal glands (certain drugs, viruses, etc. have been incriminated), damage to the innervation of the lacrimal glands, or chronic conjunctivitis (especially allergic conjunctivitis). In many cases no specific cause can be identified. When the aqueous tear production decreases, mixing of the oily and mucin layers occurs producing a thick, yellowish, ropy discharge that clings to the eye. Drying of the cornea produces irritation that results in vascularization, pigmentation, and scarring of the cornea.

Principles in Managing KCS
First – replace what is lost! The most commonly recognized symptom is loss of tear production so obviously you need to treat with artificial tears to provide the needed moisture and washing action of the normal tear production until such time as the problem is controlled. Note that cyclosporine or tacrolimus (whatever the formulation) are NOT tears and do not provide moisture. That means topical tear and lubrication replacement must be used, the more severe the problem, the more frequent that is needed. This is indeed a very inconvenient truth since few of us have the time to apply tears to ourselves, much less our pets, as often as would be ideal. Note that ointments lubricate, but that is not the same as providing moisture so we will discuss the importance of tear solutions AND lubricants in treating this disease.

Next you need to determine the underlying cause. Most commonly (at least in my practice) the cause is damage to the lacrimal glands and ocular surface associated with chronic inflammation that is most often immune mediated - a.k.a. allergic. So control allergy/atopy etc. symptoms and cause. If the dog has allergic dermatitis and you are not addressing that, you are working at a grave disadvantage! Remember, the ocular surface tissues and the skin derive from the same embryonic origin - surface ectoderm. Therefore, for the thinking veterinarian, it should come as no surprise that the ocular surface and lacrimal glands are affected in dogs that have allergic dermatitis, otitis externa, etc. Topical cyclosporine (whether the manufactured ointment, Optimmune®, or compounded topical forms of cyclosporine) and tacrolimus are t-cell suppressors and they also stimulate tear production, but the results of their use are not instantaneous. It may take as long as 16 weeks to see any improvement, especially in severe cases, if any is to be seen. Therefore, remember the first point. You must supply both moisture (artificial tears) and protection (lubricants) until the tear production has returned to normal. Most commonly cyclosporine is compounded as 1-2% solutions in oil, and we have found the medium chain triglycerides are the best tolerated. (We will discuss other...
options in lecture as time allows.) Tacrolimus is usually compounded in low percentages (0.02-0.03%) in either oil or aqueous forms, but we have less information the effects of this medication over time when compared to cyclosporine. This is especially true of some recent recommendations on the use of 1% tacrolimus in severe cases.

Finally, you must control inflammation (t-cell suppressors help do this, but are not as immediately effective as steroids) with topical and even systemic steroids as long as the use of such therapy is not constrained or contraindicated by other concurrent problems. You must also control any infection (skin or ocular). Remember, tears wash the ocular surface and have antibacterial properties.

One further consideration is that since tears are lacking, they do not dilute manufactured products with preservatives so it is important to monitor for adverse effects over time from those preservatives.

As far as how to start, use of manufactured medications are generally proscribed by regulations effective in your locale so that would tend to mean you should use manufactured products first unless there are reasons that dictate otherwise. Such reasons may include but not be limited to such things as the manufactured product is not effective thus requiring a compounded product of higher percentage, inability of the client to administer the medication (some clients, especially those with tremors of the hands may be better able to administer solutions rather than ointments), the behavior or demeanor of the patient is not suited to the administration of the manufactured form of the product, the manufactured form of the medication is irritating or causes other problems, etc. As for whether to use topical cyclosporine or tacrolimus, it is important to remember, as noted above, that our history of the topical use of tacrolimus is not as extensive as with cyclosporine (ointment or solutions) so we have a better understanding of safety with cyclosporine than with topical tacrolimus. There has, to date, been no clinical trial (or preclinical trial for that matter) that has completed for tacrolimus while in the 1990's compounded cyclosporine was used extensively and was followed by a well controlled and FDA-regulated clinical trial on topical 0.2% cyclosporine ointment (Optimmune®) that showed both safety and efficacy.

**Summary**

We can discuss nuances for a long time, but the basics are as follows:

1. Provide what is needed and not present, i.e. artificial tears and lubricants.
2. Identify and eliminate (if possible) the cause.
3. Suppress inflammation and dysfunctional t-cell mediated immunity
4. Control infection(s)
5. Preserve corneal clarity by whatever means available to you.

**Parotid Duct Transposition**

When all else fails this surgical alternative has stood the test of time and can be employed, with informed consent, to provide moisture for the eyes.
THE “JOYS” OF FELINE OCULAR SURFACE DISEASE

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Herpes! Herpes! Herpes!
The most common cause of feline conjunctivitis is feline herpesvirus1 (FHV-1) with or without Chlamyphila felis. If we were to specify keratoconjunctivitis, FHV-1 would top the chart since corneal involvement is not characteristic of feline calicivirus (FCV) or Chlamyphila felis. Calicivirus is an unlikely and minor primary conjunctival pathogen. Mycoplasma is also a minor pathogen, and bacteria rarely cause primary conjunctivitis.

Given the above, the initial treatment for virtually any feline conjunctivitis or keratoconjunctivitis should be antiviral therapy coupled with something effective against concomitant ocular pathogens. So………

Truth or Dare - Which of the following statements are true?

Triple antibiotic medications, containing bacitracin, neomycin, and polymyxin B (BNP) are ideal adjuncts to antiviral therapy for feline conjunctivitis or keratoconjunctivitis.

Topical steroids are ideal for the control of inflammation in conjunctivitis and keratoconjunctivitis in cats, especially in young kittens to reduce scarring of the ocular surface.

L-Lysine is not beneficial in managing herpetic disease in cats.

Chronic corneal erosions in cats should be treated with debridement of loose epithelium followed by multiple punctate keratotomy (MPK) or grid keratotomy.

Let’s discuss these and other statements.

Antibiotic Therapy
Medications containing neomycin or polymyxin B have been known, albeit rarely, to cause anaphylaxis and even death in cats when administered topically to the eyes. While many of us have used these medications in cats, the more critical question is can their use be justified. Since neither have activity against any common feline pathogens their use cannot be justified unless culture and sensitivity testing reveals they are the only antibiotics to which a secondary bacteria is sensitive, and that is highly unlikely. Therefore, the use of these antibiotics in cats with conjunctivitis is indefensible and borders on negligence.

Erythromycin or chloramphenicol would provide coverage against Chlamyphila felis and Mycoplasma as well as some common secondary bacteria and thus are not only accepted medical practice but a superior choice in most ocular disease in cats.

Corticosteroids, Cyclosporine, and Tacrolimus in Herpetic Keratitis.
Now let’s consider the use of topical steroids and, for that matter, topical cyclosporine or tacrolimus. While these medications will result in a short term benefit by decreasing
inflammation, in the long run they inhibit the development of normal ocular surface immunity resulting in prolongation of the initial herpetic infections, increase the likelihood of progression of herpetic conjunctivitis to involve the cornea, and increase the frequency and severity of recurrent herpetic disease. Therefore, in the event that topical steroids, cyclosporine, or tacrolimus are needed to reduce inflammation it is prudent to at least use concurrent antiviral therapy.

It is worth noting that it is common for tear production, as measured by a Schirmer tear test, to decrease when cats are stressed or their eyes are inflamed. Therefore, unless there are concurrent signs supportive of keratoconjunctivitis sicca it is wise to think twice before initiating therapy with cyclosporine or tacrolimus. All too often once the inflammation has subsided the tear production will increase.

**Antiviral Therapy**

Antiviral agents basically depend on providing an inhibition of viral replication until the cat’s immune system can bring the infection under control. The key is for the medication to be incorporated in the viral DNA creating a nonsense sequence that interrupts replication of virus. As a result most topical regimens recommend very frequent applications. Of course cats love this “attention.” **NOT!** Topical antiviral medications most commonly recommended for FHV-1 infections include compounded idoxuridine 0.1% and cidofovir 0.5%. Presently the safest and most effective oral antiviral is Famciclovir. The following dose regimens have proven beneficial in our clinic:

**Idoxuridine 0.1%** - A regimen that we have found effective is to apply 1 drop to the eyes every 5 minutes for 30 minutes for the initial treatment only to saturate the corneal tissues; then administer 1 drop t.i.d.

**Cidofovir 0.5%** - Cidofovir may be compounded to a 0.5% solution in artificial tears from the IV solution and has been shown to be efficacious when administered twice daily. It is more expensive than idoxuridine so we commonly save this option for more severe problem cases.

**Famciclovir (A pro-drug of Penciclovir)** - Oral Famciclovir is an antiviral agent that has proven safe with oral use, especially when compared to acyclovir. We generally reserve oral therapy for cases that do not respond to initial topical antiviral therapy. The dosage recommended varies considerably and the most recent research by Dr. David Maggs suggests the optimal oral dose is 90 mg/kg t.i.d. for 21 days. However that is a whopping dose and many of us have found that a dose of 125 mg b.i.d. has been effective.

**What about L-lysine?** - There is some disagreement about whether this is effective at reducing the frequency and severity of herpetic flare ups that occur with stress because FHV-1 becomes latent in the sensory nerves that supply the ocular surface. Studies that were negative tended to look at its use in large catteries or shelters, while studies in smaller numbers in more stable situations documented benefit from such supplementation in cats that have frequent or severe attacks. We know that stress plays a huge role in recurrence so it tends to make some sense that studies on large populations would have little measurable effect, and in our clinic we believe it has benefit. It certainly does no harm.
What about Indolent Erosions in Herpetic Keratitis?
It should be stressed that the persistent geographic erosions seen in herpetic keratitis are NOT the same as recurrent erosions in dogs, and grid or multiple punctate keratotomies should never be performed in cats (unless your goal is to drive virus particles deeper into the corneal stroma and cause a corneal sequestrum. When such erosions persist a better route of therapy is to perform a keratectomy to remove the diseased corneal tissue.

Diagnosing Herpetic Disease
Simply stated, considering the prevalence of herpetic disease, we most often treat it as herpetic disease with successful resolution as a positive diagnosis. I readily admit that this is not entirely satisfying intellectually, but consider the potential for false negatives and positives as well as the expense of testing it does have a certain degree of logic.

To quote Dr. Maggs of U. C. Davis, “Diagnosis of FHV-1 represents one of the greatest challenges in the management of chronic herpetic diseases. Diagnostic tests either detect the virus (or some part of it) or the host response to the virus. However, at least 3 problems confound the diagnostic value of these tests:

1. Virus can be detected in up to half of normal cats;
2. HSV-1 (and possibly FHV-1) can be stimulated to reactivate by irritation of the peripheral sensory neurons. Therefore, virus detected at a peripheral site in a diseased animal may be there as a result rather than the cause of disease
3. No test differentiates vaccine from wild-type virus.”

Dendritic Corneal Ulcers. The one pathognomonic clinical sign for herpetic disease is a dendritic corneal erosions/ulcer because such lesions occur as the virus is activated from dormancy in the sensory branches of the Trigeminal nerve supply. Dendritic lesions that stain positive with fluorescein or rose Bengal confirm activated virus is infecting the corneal cells in the regions of the nerve branches, and such lesions are only seen with herpetic infection.

This cat (above) has ongoing keratitis of 3 weeks duration and is developing corneal erosions. True or False? A multiple punctate keratotomy or grid keratotomy would be a good treatment for this ulcer.
So, to sum up, it is critically important for us as clinicians to remember that our best diagnostic tool in dealing with feline conjunctivitis and keratoconjunctivitis is our ability to assess and classify the clinical signs and responses to therapy.
Chronic Pain Management

Conny Mosley, Dr.med.vet., DACVAA, CVA

As veterinarians, we still have difficulties defining chronic pain in our patients. Understanding the pathophysiology of chronic pain, how to recognize, assess and treat chronic pain, remains a challenge. Information is limited and often anecdotal or based on studies involving small numbers of patients, it is only briefly covered in the curriculum of many veterinary schools (though improvements are being made) and the science is difficult to interpret due to the complexity of the disease and our inability to assess chronic pain objectively. Undoubtedly a deeper knowledge of the pathophysiology of chronic pain, the pharmacology of analgesic drugs, and objective pain assessment tools will greatly improve chronic pain management in dogs and cats.

Recognizing chronic pain in our patients can be challenging. A stoic dog or animals in an unfamiliar/different environment may not demonstrate obvious signs of pain. As dogs and cats get older, changes in behaviour happen slowly over time and are difficult for owners to detect as signs of pain. Commonly the owner thinks these signs are simply normal changes associated with aging. Owners and veterinarians both struggle to clearly identify the specific level of pain in a patient, because an ideal and objective assessment tool does not exist. Various reliable and validated chronic pain assessment tools and scoring systems have been published to help with the assessment of pain, but considering that animals can have many different types of chronic pain (chronic osteoarthritis pain, chronic cancer pain, chronic neuropathic pain, chronic dental pain, chronic visceral pain, etc), any assessment tool or scoring system should reflect the specific chronic pain condition. Coexisting disease further complicate the evaluation. Behaviour changes related to chronic pain are often assessed to identify the intensity of chronic pain and are considered an important part of the assessment. But as we know, a change in the environment (like an assessment in the clinic) will most likely change the behaviour of the dog due to new smells, noises and absence or presence of an owner. Alone the elevated stress level of the owner in the clinic will contribute to a different behaviour in a dog and signs of pain that are clearly identified at home (lETHARGY, DEPRESSED, DOESN'T LIKE GETTING UP) are not present at the visit and a happy dog that greets everyone with excitement, might be what the veterinarian will see. This is where the owner’s descriptions of behaviours and behaviour changes play an important role and should not be dismissed. Owner questionnaires are available and can be used and should be modified to reflect the specific pain of the patient. Owners, not uncommonly, need training to recognize specific signs characteristic of pain.

There is still not a great chronic pain assessment tool in the clinic that shows consistent and reliable results. Force-plate gait analysis isn’t readily available to all and scoring scales of locomotor systems haven’t been shown to be reliable. Diagnostic images can identify anatomic abnormalities, but it is well known that the pathological changes seen on images do not always correlate with the degree of discomfort, though it can be useful to help diagnose a potential source for chronic pain. Thermography is another technique suggested, but lacks conclusive evidence, for its ability to identify chronic pain. Traditional activity monitors may be useful for trending changes in activity, but are time consuming to evaluate. However, newer devices that link directly with computers may prove more useful. Inability to assess and quantify chronic pain and therefore also assess the efficacy of pain treatment is probably the main reason for the high percentage of chronic pain patients being undertreated and underdiagnosed in small animals.

Treatment options are also limited and incomplete knowledge of the effects of analgesic drugs used to treat chronic pain is common. Concerns about adverse effects may further reduce options for treatments. As signs for pain can be subtle, signs for effective treatment can
occasionally be even less obvious and not uncommonly it is only after the analgesic was withdrawn, that owners perceived that their pets had improved on the treatment agent. Treatment of pain depends on the type of chronic pain affecting the patient. The goal is to provide effective analgesia with the fewest possible side effects. Realistic expectations should be discussed with the owners. This discussion includes duration, quality of life, realistic level of improvement with treatment, expected financial investment to treatment, and so on. Diaries are useful for determining the efficacy of treatments in many of these patients.

Treatment options include multiple modalities: analgesic drugs (NSAIDs, tramadol, gabapentin, opioids, amantadine, etc), physical modalities, dietary changes and food therapy as well as other integrative modalities (acupuncture and traditional Chinese medicine).

There are different approaches for choosing therapy depending upon the severity and duration of the pain. Starting with a single drug/technique is ideal and associated with greater compliance but in chronic pain a more multi-model approach is required to effectively manage the discomfort. A multimodal approach, though often more complex, takes advantage of the synergistic or additive effects of various drugs/techniques and may be associated with fewer side effects. However there is also the risk of negative interactions resulting in more significant side effects although these tend to be rare and well described (i.e. NSAID’s and corticosteroids).

Conventional medications are often the basis for chronic pain management:

**NSAIDs** are excellent drugs for reducing inflammation and manage all stages of pain due to the anti-inflammatory property of the drugs. Unfortunately, not all patients are able to tolerate NSAIDs long term due to coexisting diseases and/or their effects on kidney, liver or GI system. Although most dogs with osteoarthritis tolerate NSAID’s without any significant side effects a sub-population (i.e. geriatric patients) fall into this category and alternative analgesic agents might have to be selected.

**Tramadol** is frequently prescribed for essentially any pain (acute or chronic) condition, but in most instances with little evidence supporting its efficacy. It is often described as “an oral opioid, that can be given when needed”, is safe and has minimal side effects. However, it does not function as an opioid in our canine patients. Dogs do not produce substantial amounts of the metabolite (M1 or O-desmethyltramadol), which binds to the opioid receptor. This is in contrast to the situation in humans and cats where sufficient M1 metabolite is produced to have an opioid effect. Its main mechanism of action in dogs is associated with its serotonin and norepinephrine reuptake inhibition, making its use more appropriate for chronic pain patients (due to its modulation of the descending pain pathway). Also as a result of its short half life in dogs, a frequency of q6-8h for administration is required, while in cats a q12 administration is recommended. Most dogs and cats tolerate tramadol well, but restlessness, difficulty walking, salivation, vomiting, tremors, and convulsions have been reported. Some side effects become more apparent when co-administered with other drugs that also have serotonin effects.

**Gabapentin** is believed to work primarily through binding to the alpha-2/delta subunit of the voltage-gated calcium channel, although its complete mechanism of action is not fully understood. Calcium channels play an important role in pain transmission and through its bind to them; gabapentin decreases the release of excitatory neurotransmitters and therefore decrease pain signaling. Gabapentin is also believed to increase the brain concentrations of GABA, an inhibitory neurotransmitter. Unfortunately it has a short terminal half-life (dogs: 3–4 hours, cats: 3 hours), which requires every 8 hour dosing to maintain minimum targeted concentrations. Pharmacokinetic studies suggest dosages of 10 to 20 mg/kg every 8 hours for dogs and cats, but evidence for its effects are limited, although clinical impression seems to show beneficial effects. Side effects of gabapentin include sedation and ataxia, especially in animals with hind-end weakness, and are more likely seen at higher doses. Abrupt discontinuation after long-term administration of gabapentin may cause withdrawal and seizures.
(though not well documented) and tapering the dose down over a week is generally recommended. **Amantadine** is classified as an antiviral drug, but has some useful analgesic properties, it has blocking effect at the NMDA receptor, which may antagonize central pain sensitization and therefore is beneficial in patients, whose pain does not seem to be well controlled, despite using appropriate analgesic drugs. Amantadine seems to be most effective when combined with other pain medications like NSAIDs, opioids and gabapentin, enhancing their effects. Dosing interval should be q12h for most patients.

In addition to conventional analgesic agents, other “less conventional” techniques can play an important role. Chronic pain is a multifactorial disease process and hence it is not surprising that it often requires a more multidisciplinary effort to control. There has been increasing emphasis placed on this feature of chronic pain management in humans, where multiple disciplines converge to address the multiple aspects that contribute to chronic pain. This includes traditional pharmaceutical management, nutrition, traditional Chinese medicine, acupuncture and physical therapies.

Nutritional recommendations are dependent on the type of chronic pain and the coexisting disease processes exhibited by the patient. There are commercially available joint mobility support foods available as well as many home cooked food options (there is a belief that highly processed foods may have some pro-inflammatory components). Good quality foods are important and in some instances a weight loss program or close monitoring of weight will be important in selected chronic pain patients (osteoarthritis pain), whereas weight gain and adequate access to nutrition might be important in some cancer pain patients. Muscle mass itself is another factor, which can be included in the evaluation in such as muscles “protect” joints. If an osteoarthritic joint is surrounded by strong muscles to support its stability, it’s less likely to cause problems, but if muscle wasting in the affected leg is present, a more instable joint will likely cause more pain. Maintaining muscle strength is an important aspect of physical therapy, which will be discussed in the integrative pain management part of the lectures.

Other discussion points with the owners are the need to modify the home environment. Hardwood floors or laminate can be very slippery and will make walking for the patients much more difficult. Putting rugs down to make walking easier and slipping less likely and might increase the patients quality of life and mobility. Ramps to go up into cars or to bypass stairs can also be helpful. Any aids like the “Help Me Up” harnesses can dramatically improve an owners ability to cope with a mobility challenge pet (especially larger dogs), allowing the owner to comfortably assist their pet when rising and walking. It also gives a pet greater confidence in negotiating obstacles such as stairs or surfaces with poor footing.

References and suggested reading:

Integrative Approach to Pain Management

Conny Mosley, Dr.med.vet., DACVAA, CVA

Integrative Medicine

- Treating the animal/person’s disease with a whole-animal/person approach -- designed to treat the person/animal, not just the disease
- In humans, combines conventional Western medicine with complementary treatments, such as herbal medicine, acupuncture, massage, biofeedback, yoga, and stress reduction techniques -- all in the effort to treat the whole individual
- Integrative (veterinary) medicine uses a multidisciplinary approach, some proponents prefer the term "complementary" to emphasize that such treatments are used with mainstream medicine, not as replacements or alternatives
- Designed to minimize adverse effects, maximize successful treatment outcomes and improve the quality of life
- Some therapies used may be nonconventional, however a guiding principle within integrative medicine is to use therapies that have some quality evidence to support them
- Pain is a multifactorial experience and hence well suited to an integrative management approach, in human medicine integrative pain clinics have been very successful in treating patients as “a whole” by adding diet/exercise and physical therapy, natural supplements (herbal therapy) and acupuncture, manual therapies like massages/and psychological therapy and cognitive behavior modifications to the internal, neurological or orthopedic treatment of a specific patient, treatment is individualized to the patient rather than the disease (i.e. patients with similar diseases may be managed very differently)
- Clinical implications in Veterinary Medicine, opening avenue to explore novel and creative approaches for managing pain in a more holistic and balanced manner to patient, may incorporate physical therapy, including hydrotherapy, aquatic treadmill therapy, massage therapy, laser therapy, magnetic resonance therapy, diet/nutrition and traditional Chinese veterinary medicine (TCVM) including acupuncture, herbal medicine and specific food therapy and others to conventional treatment plans
- Goal of integrating traditional with non-traditional approaches for pain management is to reduce present pain, prevent the development of hypersensitivity or chronic maladaptive pain, to reduce side effects of conventional drugs and to speed functional recovery
- Improved functional ability of the patient brings the additional benefit of keeping patient engaged, therefore improving motivation and positive mentation, clients are also engaged and play a role in recovery of their patients

Integrative modalities:

- Most modalities require specific training, but a basic understanding can help veterinarians to make an informed decision on referral options or investments and/or continuing education/training for their own practice
- **Rehabilitation**, the cornerstone of rehabilitation is the ability to simultaneously manage pain and restore function. Rehabilitation may employ many different modalities used alone or in combination
  - **Physical therapy**: Different modalities of physical therapy play an important role in the postoperative recovery phase, for regaining mobility and muscle strength, for treating chronic pain and for preventing the development of chronic pain. The restoration of optimal physical function is essential for improving quality of life of patients with chronic pain, especially in patients with surgical orthopedic and neurological injuries and diseases. Hydrotherapy, aquatic treadmill therapy, stretches
and controlled exercises can be an essential and very effective component of physical therapy.

- **Phototherapy** (laser therapy) is becoming a popular modality in veterinary medicine. LASER (Light Amplification by Stimulation Emission of Radiation) follows the concept that light is utilized for therapeutic purposes. Lasers used in physical therapy are ‘cold’ or ‘low-level lasers’ with low power properties, which penetrate through the surface of the skin without damaging effects. It can target a small area of the body at a specific wave-length for stimulation of cellular metabolism and growth. Laser therapy is used for wound healing and pain relief.

- **‘Transcutaneous electrical nerve stimulation’ (TENS)**: electrical currents are produced and applied for therapeutic purposes. The electrode patches can be positioned across painful joints, over tight muscles or in an area of discomfort, pain relief is believed to be mediated through the release of neurotransmitters and activation of opiate, serotonergic and muscarinic receptors. TENS can be useful in the postoperative period.

- **Manual therapy techniques** can be used in the immediate postoperative phase or for chronically painful cases. It includes massages, joint mobilization and range of motion exercises, which increase circulation and reduce swelling. Muscle pain is reduced, muscle tension loosened and muscle flexibility improved. Trigger point release massage techniques are an example of techniques used for chronic conditions.

- **Osteopathy** is another manual therapy modality that evaluates the tissue quality, tissue position and imbalances in motility via palpation, to identify problems that may cause pain. The principle of osteopathic manual therapy is to maintain, improve or restore the normal physiological function of interrelated body structures and systems. It uses various manual assessment and treatment techniques/modalities to help animals with chronic pain by reducing swelling, improving tissue mobility and promoting efficient healing.

- **Chiropractic** is considered a medical discipline based on spinal manipulation with the principle that the spine plays a core role in overall health, due to its close relationship to the nervous system. Any alterations in spinal movement adversely affects the nervous system’s ability to regulate function. By correcting or minimizing vertebral subluxation the patient’s health is considered to be optimized and pain may be reduced.

- **Traditional Chinese Veterinary Medicine (TCVM)** has a long history, dating back over 3000 years. Acupuncture is the most known and accepted modality of TCVM, but it is only one modality of the 4 main components of TCVM. Herbal medicine, food therapy and TuiNa (a specific medical and meridian based massage technique) are the other 3 components. TCVM is based on the balance of energy and its undisrupted flow through the body and aims to resolve any imbalances and stagnations and therefore promoting health and preventing disease.

  - **Acupuncture** is the stimulation of a specific point (acupuncture point) on the body that elicits a therapeutic homeostatic response. These points are located along or around specific pathways called meridians, but they are also located in an area with a high density of free nerve endings, mast cells, small arterioles and lymphatic vessels. The stimulation of the points releases beta-endorphins, serotonin and other neurotransmitters that contribute to pain relief. However, with a deeper understanding of the TCVM meridian and energy (Qi) system and its philosophy one can further help to improve the condition of a patient by finding the underlying cause of a problem and treating its root cause, rather than just the pain (i.e. treating the disease rather than just the symptom).
Chinese herbs have been used for many years in both human and veterinary medicine for pain management. The anti-inflammatory properties of some Chinese herbs have been popular worldwide for humans and are also used for animals. Some have shown to have properties similar to the ancient natural remedies that our Western culture is currently rediscovering (i.e. cranberry or currants to decrease inflammation, chamomile to soothe the stomach, lavender to calm etc). A variety of pain relieving herbal combinations are available and can be used in addition to conventional drugs or as an alternative for dogs and cats. They can be used to improve pain management overall, help to decrease dosing of drugs like NSAIDs or tramadol and therefore reduce potential side effects.

**Electromagnetic wave therapy:**
- **Pulsed electromagnetic field therapy (PEMF):** The principle of the pulsed electromagnetic field therapy is the penetration of this dynamic natural electromagnetic field (low frequency) through the body, creating a cascade of effects within the body by stimulating the ions in the tissue. The PEMF has shown promising results for a variety of pathologies and conditions, but is most commonly used for bone healing, wound healing, decreasing inflammation and pain relief. The mechanism of action is considered to be on a cellular level by restoring and maintaining optimal cellular function.

- **MBST®- Nuclear magnetic resonance therapy:** MBST stimulates the hydrogen protons similar to the MRI, but on a much lower energy level, treating osteoarthritis pain, back pain and other conditions. Specific veterinary products like the MBST are on the market, in particular in Europe, and its use has been increasing significantly for chronic and acute inflammatory pain conditions.

**Nutrition/food therapy:**
- Nutritional recommendations are dependent on the type of pain and the coexisting disease processes exhibited by the patient. Commercially available joint mobility support foods are available as well as many supplements. Home cooked food options to incorporate food therapy are also increasing in popularity. Good quality foods that lack pro-inflammatory ingredients are essential. Weight loss programs or close monitoring of weight is important in selected chronic pain patients (osteoarthritis pain), whereas weight gain and adequate access to nutrition might be important in some patients in the acute recovery phase or in some cancer pain patients.

**In summary:** Patient education, early diagnosis of symptoms and aggressive treatment of pain using an integrative approach, combining pharmacotherapy (described in chronic pain management lecture), surgical intervention (not described here) as well as complementary technique (described above), should serve veterinarians well in dealing with this complex disease process. Every patient and owner is different and time consuming conversations with the owners are often needed to fine tune and adjust a pain management approach. Expectations should be set, but need to be realistic. Small improvements can be huge for some owners and not enough for others. One of the most challenging factors we face in managing pain, is simply the ability to assess pain. Acute and chronic pain scales are available for owners, but owner compliance and a certain degree of objectivity (if possible) are important to make a scoring scale meaningful. Daily scoring or diary keeping can be helpful for owners to open up discussions for quality of life issues, making changes to a long-lived routines and eventually end of life decisions.

References and suggested reading:
ANESTHESIA FOR COMMON DENTAL PATIENTS

Craig Mosley DVM, MSc, DACVAA

Dental procedures are becoming an increasingly important part of veterinary care and these cases often present unique anesthetic challenges. Describing the “average” dental patient is nearly impossible with dental care spanning the life of an animal. Patients range from healthy young patients requiring a professional dental cleaning and assessment to elderly patients with comorbidities requiring multiple extractions. In general, a standardized anesthetic protocol for elective procedures in healthy patients can improve work efficiency and patient safety however a more individualized approach is often necessary for managing aging dental patients. The cases presented in this lecture will draw on concepts required for managing geriatric patients and those with stable cardiac or renal disease.

General principles

• The specific drugs selected will not necessarily ensure a safer anesthetic; it is ultimately the skill of the anesthetist; their understanding of physiology, pathophysiology and the effects of the anesthetic related drugs on various body systems that make anesthesia safer.

• It is preferable to use drugs and techniques one is very familiar with in more complex cases rather than attempting to use novel or unfamiliar drugs and techniques. New techniques and drugs are best used first in predictably stable patients.

Anesthetic Considerations for Geriatric Patients

Geriatric in veterinary medicine is usually defined as any animal exceeding 75-80% of their predicted lifespan, with predicted lifespan varying among breeds. However, it is important to note that there may be little correlation between chronological age and physiological age requiring careful individual patient assessment.

Cardiovascular changes

• The functional reserve of the cardiovascular system is considerably reduced with age, but significant patient to patient variation can exist. Maximal attainable heart rate and cardiac output are reduced. They may also be more prone to arrhythmias as a result of myocardial fibrosis involving the conduction pathways. The autonomic responses to changes in the cardiovascular system also tend to be blunted. Overall, the response to changes in blood pressure and/or volume may be insufficient and these patients and they will be more prone to perioperative hypotension.

Respiratory changes

• The respiratory system undergoes mechanical changes associated with aging leading to decreased compliance of the thorax, atrophy of intercostal muscles and diaphragm, and decreased alveolar elasticity. Despite normal oxygen diffusion in the lung, the mechanical changes associated with aging result in disruption of the precise matching of ventilation and perfusion required for optimal oxygenation. This results in a linear decline in arterial oxygen tension with age. The ventilatory response to hypoxia and hypercarbia are also markedly blunted. These changes make the geriatric patient far more susceptible to hypoxia and hypercarbia in the
peri-anesthetic period. There is also some evidence that protective laryngeal and pharyngeal reflexes are reduced.

Renal changes
- There is as much as a 10-20% decrease in renal mass with a disruption of the renal microvascular architecture and a significant decrease in functional nephrons. This leads to a decrease in active tubular secretion and reabsorption of drugs and solutes. There is also a virtually linear decline in glomerular filtration rate (GFR) with age and this may be due in part to reductions in renal blood flow, loss of glomeruli and glomerulofibrosis. These changes can have significant effects on drugs requiring renal excretion for elimination and more prone to nephrotoxic or ischemic insults. The renin-angiotensin system also becomes less responsive and geriatric animals may be less tolerant of hypovolemia and dehydration. Subclinical renal insufficiency may also be more common in geriatric patients and a small insult (hypotension) may predispose the animal to later develop signs of failure. Adequate diuresis should be instituted if any concerns exist.

Hepatic changes
- Liver changes associated with aging are primarily quantitative and include a decreased liver mass and decreased total hepatic blood flow, both may be may be decreased by up to 50%. Qualitatively hepatocellular, microsomal and nonmicrosomal, enzyme function remains normal. The overall effects of these changes on plasma drug concentrations are complex and require a thorough understanding of the metabolism and excretion of a specific drug. In general, it is probably best to assume that drug effects may be prolonged though this is rarely significant with the perianesthetic drugs as most are not associated with significant toxicity but anesthetic “hangovers” can be anticipated to be more common.

CNS changes
- Many of the age related central nervous system changes have been characterized in humans and are related to changes in sensory and cognitive function that are less easily identified in animals. As aging progresses, there is an apparent increased sensitivity to anesthetics and anesthetic adjuncts. There is progressive neuron loss, as well as a depletion of central neurotransmitters that may explain the increased sensitivity, but this has not been conclusively determined. Peripherally, there is a loss of motor, sensory and autonomic nerve fibers. This “denervation” can lead to diffuse neurogenic muscle atrophy.

Body composition (water, fat, muscle) & metabolic changes
- As animals age, there are changes in body composition that may impact the distribution and elimination of various drugs. Aging leads to a decrease in muscle mass and gain in body fat as a percent of total body weight. This change in body composition may lead to altered plasma drug levels. Additionally, it is not uncommon to see absolute gains of body fat. Older pets are often “spoiled with food” and participate in less physical activity. Obesity can then further alter drug disposition, particularly with respect to the lipid soluble drugs.

Protocol selection
- There are no specific contraindications for use of any of the anesthetic drugs in geriatric patients but a knowledge of normal age related changes can help in
selecting the most appropriate protocol. In general, it is reasonable to expect that most anesthetic drugs will exert a greater than anticipated effect as a result of greater initial plasma levels, resulting from a contracted blood volume, and due to the apparent increased sensitivity of the CNS to anesthetic drugs. The overall effects will depend on the specific drug but again the perianesthetic drugs are not associated with significant toxicity and the use of conservative doses and administration to effect will avoid most unwanted side effects.

Additional Protocol considerations for patients with concurrent cardiac disease
• Anesthesia of the patient with cardiac disease requires knowledge of the hemodynamic alterations associated with the specific cardiovascular disease (congenital or acquired), knowledge of the hemodynamic effects of the commonly used anesthetic drugs, knowledge of the procedure and an understanding of how all these factors might combine to ultimately impact cardiovascular function. Successful anesthetic management of the cardiac patient requires an ability to select the most appropriate anesthetic drugs and techniques and to monitor, assess and treat complications (i.e. arrhythmias) if they arise.

• The cardiac patient should also be stabilized as much as possible prior to administering anesthesia. Most patients with cardiovascular disease will have reduced cardiovascular reserve and may not tolerate further anesthetic induced cardiovascular depression. Arrhythmia’s, congestion/edema, low cardiac output should all be addressed and their effects minimized. In addition some anesthetics, analgesics and sedatives can precipitate arrhythmias in some patients (i.e. inhalants enhancing hyperkalemia induced bradycardia, opioids causing bradycardia). However, this should not be taken to suggest that analgesics should be withheld. In general the opioids have very minimal cardiovascular effects (i.e. bradycardia) and these are relatively easy to manage (i.e. anticholinergic administration) and the pain itself can further contribute to cardiovascular impairment. Respiratory and cardiovascular support (i.e. oxygen, fluids) is often beneficial prior to administering anesthesia.

Additional Protocol considerations for patients with concurrent renal disease
• Patient preparation Patients with renal disease will present from essentially normal to those that are obtunded and markedly dehydrated from their disease. An appropriate history (including knowledge of any concurrent medications) and a physical exam are the foundation for determining the required preoperative preparation required.

• Hydration Many patients with renal compromise are very susceptible to dehydration if access to water is restricted, the author never recommends withholding water from these patients and will allow patients access to water until 1-2 hours prior to anesthesia. Ideally the patient should be admitted to the hospital the night before surgery and placed on intravenous fluids at a rate suitable for the individual patient. Some patients with renal failure may require higher maintenance fluid rates owing to their inability to efficiently conserve water.

• Anemia Some patients with renal disease may have a low hematocrit as a result of reduced erythropoietin production by the kidney. There is much controversy regarding “when to transfuse” and there is no one correct value to base all decisions upon. In addition to the PCV, the overall condition of the patient and the planned procedure that necessitates the anesthesia need to be considered. Generally any
normovolemic patient with a PCV < 18-20% is a potential candidate for a blood transfusion prior to anesthesia to ensure adequate oxygen carrying capacity. However, in addition to addressing the factors responsible for adequate delivery of oxygen to the tissues (hematocrit, cardiac output, local vasomotor tone) it is also important to ensure that oxygen demand is also minimized (discussed in more detail under intraoperative monitoring).

- **Azotemia/uremia** If azotemia is detected prior to anesthesia reasonable attempts to reduce its impacts should be considered. Certainly if the azotemia is pre-renal this should be corrected prior to initiating anesthesia as the associated hypovolemia/dehydration will predispose the kidney to a hypoxic insult. Long-standing azotemia associated with chronic stable renal disease should be evaluated in the context of serial biochemical assessments to better appreciate the stability of the renal disease. Persistent uremia may also been associated with uremic pneumonitis, neuropathy and encephalopathy. Clinically these rarely cause anesthetic complications however the anesthetist should watch for signs associated with pulmonary edema when using aggressive fluid therapy and there may be a tendency for some drugs to exert more profound effects (although properly titrating anesthetics should minimize this).

- **Acid base and electrolyte imbalances** Many patients with severe renal disease will have acid-base abnormalities and most can be managed by addressing the underlying electrolyte and/or water imbalances. Hypokalemia and hyperphosphatemia are relatively common in patients with chronic renal disease and are normally managed as part of the disease treatment process. Mild hypokalemia and hyperphosphatemia in an otherwise stable patient pose no significant anesthetic challenges. Hyperkalemia and hypochloremia may be present in patients with urinary obstructions or a ruptured bladder. Patients with hyperkalemia ( > 6.0 mEq/L) should be treated to reduce potassium levels prior to anesthesia. High levels of potassium are cardiotoxic and anesthesia of hyperkalemic patients may excacerbate its effects leading to a marked bradycardia that can progress to asystole. The bradycardia is normally unresponsive to anticholinergics and epinephrine. Hyperkalemia can be managed using intravenous fluid therapy and dextrose ± insulin. Calcium gluconate is occasionally used to antagonize the effects of potassium on the myocardium should other treatments fail to adequately reduce potassium levels.

- **Stress response** One aspect often overlooked in veterinary medicine is the effect of “stress” and pain on renal function. It is generally recognized that stress and pain causes release of many regulatory substances that directly affect renal function such as aldosterone, vasopressin, renin, and catecholamines. The vasoconstrictive effects of vasopressin and catecholamines may shunt blood away from the kidneys resulting in reduced oxygen delivery to the renal medulla. Appropriate premedication and analgesia in the perioperative period may help to protect the kidney from the vasoconstrictive effects mediated by the sympathetic nervous system in response to stress and pain.
ANESTHETIC ERRORS: HOW THEY OCCUR, AND HOW TO PREVENT THEM

Craig Mosley DVM, MSc, DACVAA

Recently, both veterinarians and physicians have begun speaking more openly about medical errors. Historically the topic of medical errors was largely avoided probably the result of many factors including, but not limited to: feelings of failure, shame, embarrassment and fear of litigation and loss of livelihood. Fortunately, we are not only admitting errors happen but, more importantly, we’re looking at the factors that lead to errors and how they can best be mitigated.

Terminology matters

Error
- an act that through ignorance, deficiency, or accident departs from or fails to achieve what should be done
- a transgression of law or duty; a mistake in conduct; a moral fault

The definition of error is inherently associated with an extremely negative connotation. This at least in part, accounts for our unwillingness to be associated with events described as errors. Who of us would want to be associated with an event that is the result of ignorance or deficiency? More recently scholars and advocates for patient safety have suggested that we need a more neutral definition of error (i.e. circumstances in which planned actions fail to achieve the desired outcome) and need to move from our culture of “blame and shame” and move towards one of “acceptance and correction

Types of errors

There are generally three major types of errors described in medicine; human errors, diagnostic errors and systems/process errors. Although for convenience it may be easier to describe and discuss them individually in practice they are very interrelated and overlap with one another.

Human errors

It is often stated that to “Err is human” and cognitively this is certainly true. Our cognitive processes make us inherently prone to errors and as long as we want a human element involved in the delivery of veterinary care errors will be inevitable. That is not to say we should simply accept errors since it is part of being human, but rather we should be more watchful for them and develop systems to help minimize the likelihood of errors. Factors that contribute to human error include some of the following:
- cognitive biases
- communication failures
- lack of effective training
- memory lapses
- inattention
- poorly designed equipment
- exhaustion, fatigue
- ignorance (insufficient knowledge)
- noisy working conditions
- workload (too light, too heavy)
- emotional/personal factors
Diagnostic errors
Diagnostic errors are a specific type of error resulting in incorrect diagnosis. Estimates for the rates of diagnostic errors in human medicine range between 10-15% of all diagnosis. Although we don’t have any similar estimates in veterinary medicine there is no reason to expect our error rate would be lower and indeed there are plenty of reasons it may be higher (i.e. multiple species/breeds, limited peer review of diagnosis etc.). Diagnostic errors are most commonly the result of knowledge deficits or our cognitive biases or some combination of the two. Very simply defined cognitive biases represent systematic errors in our “logical though process” but these biases are also believed to be highly adaptive enabling us to quickly make complex decisions and conclusions. Cognitive biases are at least part of what allows experts, to become expert. However, being aware of some of the more common cognitive biases (i.e. anchoring, conformation, availability etc.) may help us avoid making systematic errors in our diagnostic processes.

Systems/process errors
The terms system or process is used in this context to describe a system or process designed to prevent predictable human errors resulting from lapses in human performance (i.e. vigilance, judgment, training etc…). The systems approach takes the view that many errors reflect predictable human failings in the context of poorly designed systems. In other words our systems should be designed to help prevent our human failings, especially in the complex environment of providing medical care. The Swiss Cheese model is often used to describe a the systems approach to error mitigation whereby the holes in the cheese represent flaws in the systems, the larger and more numerous the holes the more likely an error is to occur but if another system is placed below with smaller, less numerous holes or in locations such that the holes don’t line up the likelihood of an error is reduced. This approach is commonly used in operational, equipment and environmental design. A systems approach is unique in that it aims to catch human errors before they occur or block them from causing harm rather than seeking to create flawless medical providers, a near impossibility as long as humans are involved in the decision making process. Systems as simple as checklists are effective tools that can significantly minimize and prevent human errors, but like any system, in order for it to be useful it must be used consistently and routinely among all team members.

So what are some common anesthetic related errors and what can be done to avoid them? Most catastrophic anesthetic errors can be avoided by implementing a few key tools into your practice.

Checklists
Checklists are a powerful tools used in many high-risk environments, particularly in those where the consequence of even a single error might result in death. For example, all pilots commercial or private run through a routine checklist before flying a plane. Although anesthesia is not exactly the same as flying a plane it is a situation where a large number of complex factors interact (i.e. patient, drugs, anesthetic machine, breathing circuit, patient monitors, anesthetist) and all must be properly functioning in order for the patient to have an optimal outcome. Additionally, an unrecognized error could quickly lead to disastrous consequences (i.e. close pop-off valve). An excellent and well-designed checklist and an accompanying booklet is available on the Association of Veterinary Anesthetists resource page. www.ava.eu.com/resources/
Automated alarms
Most patient monitors come equipped with automated alarms yet often these alarms are inactivated or simply ignored. Alarms should be set to reflect appropriate alarm limits for various patients. Most will now allow several different (i.e. dog, cat, small dog etc) alarm presets to be saved reducing the number of “false” alarms. In addition equipment monitors, such as capnographs and airway pressure monitors are invaluable for ensuring there are no malfunctions in the anesthetic machine and/or breathing circuits.

Anesthetic records
Anesthetic records should be kept on all patients under going anesthesia and this does not mean simply recording the drugs used for anesthesia but rather should include the timely (every 5 minutes) collection of physiological patient data throughout anesthesia. This serves at least two purposes; first it ensures ongoing vigilance and patient assessment and provides trending information that may be an early indication of impending problems.

Syringe labels
The use of syringe labels can help minimize the risk of administering the wrong drug and can increase efficiency by allowing multiple drugs to be pulled from the pharmacy at one time for later use. For example, all the drugs for each patient undergoing anesthesia for the day could be pulled up first thing in the morning with each syringe quickly and clearly labeled and placed in individual small organizing baskets for each patient.

Drug calculators/libraries
Automated drug calculators are readily available online or can be custom designed and printed as part of the anesthetic sheet using a spreadsheet program such as MS Excel. These programs again improve efficiency and provide a double check against manually calculated drug doses and prove invaluable in emergency situations where rapid and immediate drug administration may be required. Additionally many newer syringe pumps and IV pumps allow users to enter custom libraries with dose limits that can help avoid inadvertent overdoses and facilitate rapid response when a less familiar drug is required (i.e. lidocaine, fentanyl or dopamine CRI).

There is no way to completely eliminate all anesthetic related errors but, by using available tools and ensuring continual vigilance most catastrophic anesthetic events can easily be avoided. Our goals should be to reduce the occurrence of errors, minimize their impact on our patients and when they do occur we should accept them and respond with constructive efforts to avoid repeating our errors in the future.
COMMON ANESTHETIC MYTHS AND MISCONCEPTIONS

Craig Mosley DVM, MSc, DACVAA

In addition to our ever more challenging caseload, there has been an exponential increase in anesthetic and analgesic related information coming to us from a variety of sources. It is often difficult to wade through this vast amount of information to determine what can reasonably, and soundly, be harnessed to improve anesthetic and analgesic care in our own practices. Anesthesia is a necessary tool for many diagnostic and therapeutic procedures and its application can sometimes be influenced too much by fear and myth rather than guided by evidence.

Myth: Several dog and cat breeds are “sensitive” to anesthetic drugs.

There are no well-identified general breed sensitivities to anesthetics in the veterinary literature although there persists a large amount of “information” available that would suggest breed sensitivities are not uncommon. However, many of these so-called “sensitivities” are predictable, manageable and can be explained by breed specific anatomic and genetic factors. Perceived “sensitivities” are often the result of inappropriate drug selection and/or through not recognizing underlying breed associated diseases. Some of the most common purported “sensitivities” are listed below.

- **Brachycephalic breeds** have high complications rates resulting from their anatomical malformations. Excessive sedation when not intubated (premedication and recovery) and/or airway irritation resulting from difficult intubations are contributing factors for the development of upper airway obstruction. In general, brachycephalic breeds should be under constant observation when sedated.

- **Giant breed dogs** generally require lower mg/kg doses than smaller breed dogs and metabolic scaling should be employed when selecting drug doses for these patients. Lean muscled breeds such as *Sighthounds* generally have a lower volume of distribution of lipophilic drugs (most anesthetic drugs) leading to higher plasma concentrations at a given dose compared to patient with greater adipose tissue. *Greyhounds* are probably the only breed with scientifically proven sensitivity to anesthetics as a result of reduced cytochrome p450 activity leading to reduced drug metabolism, however this is rarely significant unless multiple doses or a total intravenous anesthetic is being administered. There is anecdotal evidence that a line of *Boxer dogs* originating in the UK developed syncopal events following acepromazine, presumably the result of pronounced arrhythmias. It is possible that these events were precipitated by unrecognized arrhythmogenic right ventricular cardiomyopathy in these patients.

- **Herding dogs** have been shown to carry a mutation in the gene (MDR-1) responsible for the transcription and translation of P-glycoprotein, necessary for transport of drugs out of the brain. Its absence may lead to toxic levels of some drugs in the brain, most notably ivermectin. Although acepromazine and butorphanol are both listed as being substrates of P-glycoprotein there is very little evidence for completely avoiding these drugs in herding dog breeds. However it may be prudent to select lower doses and watch for evidence of prolonged effects. There is a blood test available to test for this mutation.

- **Toy breed** dogs represent the opposite extreme from giant breed dogs and are specifically at risk for the rapid development of hypothermia.

Misconception: Tramadol is an effective analgesic for pain in dogs

There is very little convincing evidence that oral tramadol produces a clinically significant analgesic effect in dogs (ref). In dogs tramadol has a very short half-life with minimal production of the opioid M1 metabolite (the metabolite believed to account for the
majority of its analgesic effect in humans (1). Despite its lack of clinical efficacy (2) oral tramadol remains one of the most widely prescribed drugs for pain management probably owing to its perceived safety and lack of alternative options. In contrast to dogs, cats do seem to produce the M1 metabolite and there is a reasonable amount of evidence supporting its use as an analgesic in this species. Although tramadol may have a role as an adjunct analgesic drug resulting from its inhibition of serotonin and norepinephrine reuptake it should probably not be relied upon as the sole or even a particularly efficacious analgesic drug.

**Misconception: Butorphanol is a good and lasting analgesic in dogs**

Butorphanol is a drug that has agonist effects at kappa opioid receptors and antagonist effects at mu opioid receptors (agonist-antagonist opioid). Due to its receptor activity you would anticipate that butorphanol would have limited analgesic efficacy. And indeed butorphanol has not been shown to be effective at controlling acute or post-operative pain in dogs and cats (3,4). Butorphanol is likely only effective for controlling mild pain in dogs, and may be slightly more effective in cats. Its duration of effect appears short, in some cases less than 1 hour, perhaps slightly longer (2-3 hours) in cats. However butorphanol is arguably a more reliable sedative compared to the other opioids, although none of the opioids are particularly good sedatives. It is probably most useful as an adjunct for reversible sedation, particularly in those cases where avoiding a “hang over” effect is desirable (i.e. patients going home following radiographs).

**Misconception: Acepromazine should not be used in patients with history of seizures.**

Acepromazine is a derivative of the phenothiazine family of drugs, which exert at least part of their clinical effect through antagonism at central dopamine receptors. In the 1950’s drugs related to acepromazine (i.e. chlorpromazine) where used as a treatment for psychosis in humans and it was reported that in some cases seizure activity was observed following administration of these drugs. This then led to the widespread recommendation that acepromazine should be avoided in veterinary patients prone to seizures. However, there is essentially no credible information supporting this historical assertion. In fact, there is an increasingly large amount of information refuting this suggestion (5,6). It is no longer generally believed that there is any good reason to avoid acepromazine in patients prone to seizures when the drug is indicated.

**Misconception: Lowering the dose of dexmedetomidine will reduce cardiovascular effects**

The side effects associated with the use of alpha-2 agonists are often dramatic and occasionally alarming but should be anticipated. These drugs initially cause pronounced vasoconstriction and marked hypertension followed by a reflex bradycardia with both changes contributing to a marked reduction in cardiac output. With time, systemic vascular resistance may trend towards normal, resulting in a return to normal or slightly decreased blood pressure. In some instances it may be desirable to minimize these dramatic cardiovascular changes and so a lower dose is selected. The effects and side-effects, of most drugs are dose dependent (i.e. the greater the dose, the greater the effect). However, within the clinically useful dose range of dexmedetomidine lowering the dose does not attenuate the cardiovascular effects (7). A study using medetomidine, a racemic mixture of dexmedetomidine (active) and levomedetomidine (inactive) showed there is no significant differences in the cardiovascular effects when giving a 1 mcg/kg versus a 20 mcg/kg dose of medetomidine IV (7), equivalent to 0.5 mcg/kg and 10 mcg/kg of dexmedetomidine. Though the duration of cardiovascular depression was
shorter with the lower dose compared to the higher dose. If one wishes to avoid the cardiovascular effects of dexmedetomidine it would be best to choose an alternative drug as lowering the dose will have little effect beyond producing less reliable sedation.

Myth: Propofol is the safest anesthetic induction agent
Propofol is the dominant induction drug in many practices. It is popular as a result of its desirable induction and recovery characteristics, both being rapid and generally smooth. However, it is probably its rapid and predictable metabolism in the liver and extrahepatic sites that have contributed to its reputation as the safest induction agent. However, there is no evidence to support this assertion in fact when compared with the other commonly available injectable anesthetics (ketamine, alfaxalone) propofol has the greatest cardiovascular and respiratory depressant effects and lowest therapeutic margin. It can produce marked apnea with subsequent hypoxia and profound hypotension secondary to myocardial depression and vasodilation. However, proper administration technique (titration to effect) owing to its smooth induction characteristics make it a very safe and effective induction drug and one well suited for total intravenous anesthesia. There are no “safest” anesthetic drugs, all anesthetic drugs are inherently safe when used and administered appropriately.

Myth: There is no “real” benefit of adding a local anesthetic technique if the patient is undergoing general anesthesia
Although it is difficult to demonstrate a significant beneficial postoperative effect from using local anesthetic techniques in combination with general anesthesia there is good evidence supporting improved intraoperative stability, reduced stress response and improved recovery immediately following anesthesia when local techniques are combined with general anesthesia. General anesthesia does little to block the physiological responses (activation of the CNS) associated with a surgical insult, it simply prevents the brain from perceiving the information. This activation of the CNS results in a systemic “stress" response associated with increased levels of circulating catecholamines and other stress hormones. This response may be recognized in the anesthetized patient through changes in cardiovascular and/or respiratory variables and often indicates a need to alter anesthetic depth. Local anesthetics will help eliminate CNS activation leading to greater intraoperative patient stability and allow lower doses of inhaled anesthetics to be used reducing cardiovascular and respiratory depression. Also recovery from general anesthesia is significantly improved when local anesthetic techniques are used.

Myth: Anesthetic induction with gas is safer than with an injectable drug
It is true that anesthetic induction with a gas is arguably easier than using an injectable drug (no IV catheter) but there is no evidence to support it being safer. The pharmacokinetic profiles of the inhaled anesthetics are unique, as they do not require metabolism for elimination. They are predictably eliminated via the lungs through alveolar ventilation (breathing). It has thus been suggested that induction using an inhaled anesthetic is safer in the compromised patient. However, during mask inductions many patients will struggle and go through an excitement phase that is stressful for the patient. Increases catecholamines that may lead to arrhythmias and increased myocardial work. It is also slower and patients typically experience more cardiovascular and respiratory depression compared to appropriately delivered injectable anesthetic drugs. The dose of inhaled anesthetic required for endotracheal intubation is often greater than that required for surgery. The techniques also involve unnecessary personnel exposure to the inhaled anesthetics.
Myth: Longer fasting times will reduce the incidence of gastroesophageal reflux and vomiting
It has long been believed that 12 hrs or overnight fasting is required prior to elective surgery in healthy patients to prevent gastroesophageal reflux and vomiting. Interestingly, prolonged fasting may actually increase the risk of gastroesophageal reflux. In one study the incidence of GER was significantly lower in dogs fed a small meal within 2-4 hrs of the induction of anesthesia (8) and is similar to findings for humans (9). Another study showed that gastric pH was higher in dogs fed a small meal of canned food 3 hours prior to anesthesia (10) and adds further support that current recommendations for prolonged fasting may not be necessary and in fact a small meal within 4 hours prior to anesthesia may actually decrease the incidence of GER and the accompanying acidity of the reflux. Interestingly vomiting is an extremely rare occurrence during the induction of anesthesia (though relatively common following some premedications) in veterinary patients. In general, a small meal followed by 4-6 hours of fasting prior to anesthesia is suitable for most patients and may be associated with a reduction in the incidence of GER.

Myth: Premedication complicates anesthesia by adding drugs and prolonging recovery
Although premedication does add drugs to the protocol it is not generally associated with prolonged recoveries when anesthesia is appropriately delivered. Pre-anesthetic medications are used to reduce anxiety/stress, provide pre-emptive analgesia and reduce side-effects/complications. In general, premedications dramatically improve the overall quality, stability and recovery of patients undergoing general anesthesia. Those patients who do not receive premedications can be expected to experience a more stressful perianesthetic period.

References


DEVELOPMENTAL BONE DISORDERS
Anthony Pease, DVM, MS, DACVR

Neonatal imaging is riddled with problems, not the least of which is that it is not performed routinely. When you have an acutely lame puppy, the growth plates and the lack of ossification of the bones can cause confusion. The purpose of this lecture is to show various congenital disorders including osteochondritis dessicans, panosteitis, elbow dysplasia, hip dysplasia and nutritional abnormalities.

Osteochondritis dessicans is likely the most commonly diagnosed congenital disorder. This disorder is a failure of endochondral ossification that occurs in young growing animals. The key to this disorder is that to have failure of endochondral ossification, it must finish and therefore the diagnosis should only be made after the dog is 5-6 months of age. This disorder can occur in the tarsus, elbow, shoulder and stifle. For the tarsus, since it can occur in the talus, a flexed dorsoplantar projection of the tarsus and a flexed lateral can help to remove the superimposition of the distal tibia to identify the lesion.

Panosteitis is a self-limiting disorder that is hard to diagnosis because of the subtlety of the radiographic changes. It ranges from increased opacity of the medullary cavity to a decreased opacity or smooth periosteal proliferation. The ulna, radius and distal humerus are the most commonly affected areas and the contra-lateral limb should be obtained for comparison. Generally, lateral radiographs are all that is needed to make the diagnosis.

Elbow dysplasia is a current hot-topic in the breeding world with the Orthopedic Foundation for Animals (OFA) creating a 0-3 grading scale as part of the routine screening test for dogs. Small ridges on the anconeal process causes an elbow to go from a grade 0 to a grade 1, but when evaluated with CT, this irregularity is a normal variant in most cases and not associated with osteophyte formation. In fact, a current study at Michigan State University being performed by Dr. Chelsea Kunst is finding that grade 0 elbows can sometimes have osteoarthritic changes on CT whereas grade 1 elbows sometimes do not. The bottom line is that radiographic assessment of the elbow is mainly to determine the severity of disease rather than the presence. Such is the case with an united anconeal process. This is a normal finding in dogs < 5 months of age, but the apophysis should be fused by the age of 6 months. It is the cut point when an anconeal process is considered united. Fragmented medial coronoid processes are the most commonly suspected elbow disorder, but with the superimposition of the radius, are difficult to evaluate on standard radiographs. Computed tomography is the modality of choice for evaluating the elbow to minimize the superimposition and detect small fragments or lucencies within the bone.

Canine hip dysplasia is an orthopedic disorder that causes widespread confusion among breeders mainly due to the difficulty of predicting the likelihood of young animals developing osteoarthritis later in life due to hip laxity. Currently, two methods to evaluate the hips are used. The first is a standard ventrodorsal projection. This view is accepted by OFA in animals greater than 2 years old as a good predictor of hip health. The idea that is osteoarthritis or incongruity is present, the degree of the laxity can be
assessed and a grade of Excellent, Good, Fair, Borderline and Dysplastic can be made. Three board certified radiologists score the radiographs independently and then the assessment is average. The main limitation to this is that it is a subjective measure and does not take into account the breed of the animal. The other method is PennHip, which uses a distractor to assess the amount of passive joint laxity. This measure is then compared to all the dogs in the database of the same breed and a percentile is given. Greater than 50% is considered a pass and hips with a distraction index of < 0.3 are considered unlikely to develop osteoarthritis later in life. The benefits of this method is that it is an objective measure that is breed specific and that all evaluations must be submitted to PennHip to provide a general database of the breeds. In addition, this method can be used to accurately predict hip laxity after 6 months of age.

Nutrition in animals is also something that in these economic times can manifest as growth plate disorders or delayed ossification. Understanding the timelines when growth plates close and having normal radiographs or textbooks that show bone development of puppies helps with this assessment. Careful history and physical examination as well as thorough blood work can also aid in making this determination.

Neonatal radiographs can be difficult, but due to more concerned owners and the better resolution of radiographic examinations, care must be taken to adequately obtain and evaluate these images. Although not routinely performed, radiographs of the immature skeleton can show far more than just a fracture, but also can show growth plate disorders, nutritional deficiencies as well as developmental disorders. If detected early enough, these developmental disorders can be corrected and treated appropriately prior to irreparable damage.
DKA IN A NUTSHELL
Jennifer Kyes, DVM DACVECC

Diabetes mellitus (DM) is a non-emergent condition characterized by hyperglycemia due to a lack of insulin production or reduced functional insulin receptors. If DM goes unrecognized and untreated then the body utilizes fat reserves as the primary energy source instead of glucose. Using fat for energy results in dramatic weight loss for the patient and the formation of ketones the byproduct of fat metabolism. Many patients suffer from polyuria and polydipsia because of the osmotic effect glucose has on water. When glucose levels exceed the renal threshold glucose is then filtered by the kidney into urine and water follows.

Diabetes ketoacidosis is a medical emergency characterized by persistent hyperglycemia, the formation of ketonemia, metabolic acidosis, electrolyte derangements, and significant dehydration.

Hyperosmolar non-ketotic diabetics are considered the most severe form of diabetes. It is characterized by hypernatremia, marked azotemia, hyperglycemia and are non-ketotic. These are extremely difficult cases to manage and should be referred. There is a 60% mortality rate in treating these patients.

Clinical signs
- Vomiting
- Diarrhea
- Lethargy
- Anorexia
- Polyuria, polydipsia
- Plantigrade stance (cats)
- Weakness or recumbent

Diagnostics abnormalities
- Hyperglycemia
- Glucosuria
- Ketonuria
- Leukocytosis +/- left shift
- Elevated liver values
- Pre-renal azotemia
- Hypernatremia, hypochloremia, hypokalemia
- Metabolic acidosis pH <7.2, HCO3 <15

Since many of these patients have the same clinical signs and diagnostics abnormalities.
- Dehydration
- Metabolic acidosis
- Electrolyte imbalances
- Hyperglycemia and ketonuria
- Infection
- GI signs; anorexia, vomiting, diarrhea
Treatment

1. Dehydration
Correcting dehydration should be done using balanced electrolyte solutions such as LRS, Plasmalyte and Normosol. Acidifying solutions such as 0.9% NaCl should never be used as the replacement fluid especially when the Na is low.

Calculating the patient’s fluid replacement volume (mL) should be done and given in small boluses until the patient is stable. Stable means improved blood pressures, improved pulses, improved heart rate, moist mucous membranes and CRT <2 sec. The remaining fluid volume can be administered over a 24 hour period. I keep my DKA’s on 2x maintenance fluid rates. This actually works out to correct a patient with 7% dehydration over 24 hours.

| Estimated % dehydration x BW (kg) X 1000mL/L |

2. Metabolic acidosis
Metabolic acidosis is caused by the presence of ketones, which are acidic. iStat machines provide a pH, PCO2, and HCO3 which can assess severity of the metabolic acidosis. Fluids will help improve a metabolic acidosis but if the presence of ketones is the primary reason for an acidosis and this cannot be corrected quickly sometimes bicarbonate therapy is indicated. If you don’t have an iStat machine to assess blood gases but you have a moribund patient with excessive ketones the pet may benefit from bicarbonate therapy. If you do have access to blood gases then you should consider treating a patient with pH <7.2 but you absolutely should treat if <7.0. There are numerous equations for calculating bicarbonate. To make it easy, I use 1ml/kg over 10-30 minutes. It sounds aggressive but it works in increase the pH to >7.2 in 1-2 doses.

| Bicarbonate therapy 1mL/kg over 30 minutes for pH <7.2 |

3. Electrolyte imbalance
Sodium and chloride will be replaced with a balanced electrolyte solution. Potassium is typically low and becomes even lower with fluid boluses and insulin administration. It is important to supplement potassium in the early phases of treatment and monitor it closely throughout treatment. We supplement according to a slightly more aggressive form of the typical potassium charts in textbooks. Magnesium and phosphorus can be low in these patients as well so consider supplementing them if you have a refractory hypokalemia.
Serum K (mEq/L) | KCL/L (mEq) | Max rate (ml/kg/hr)
---|---|---
3.6-5.0 | 30 | 24
3.1-3.5 | 40 | 16
2.6-3.0 | 50 | 11
3.1-2.5 | 60 | 8
<2.4 | 80 | 6

You can also do a continuous rate infusion (CRI) of potassium using a syringe attached to IV extension set and a fluid pump.

Equation below (*) assumes KCL concentration is standard 2meq/mL.

\[
\text{KCL (mL/hr)} = \frac{0.5 \text{ meq/kg/hr} \times \text{BW(kg)}}{2\text{meq/mL}} \text{ and continue for 4 hours.}
\]

4. Hyperglycemia and ketonuria

Insulin administration is the only way to change fat metabolism and ketone formation back to carbohydrate (glucose) metabolism. Ironically, insulin is not a priority in our treatment protocol. Start insulin therapy when the patient is considered reasonably hydrated and ideally a K+ > 3.5. There are 2 main ways to administer insulin to a DKA. I strongly prefer the CRI method over the IM/SQ injections from a patient comfort standpoint and it is less time consuming.

Insulin CRI technique

Place 1 unit/kg (cat) and 2 unit/kg (dog) in a 250mL NaCl bag. Every 4 hours check the patient's blood sugar and adjust the insulin fluid rate according to the chart below.

<table>
<thead>
<tr>
<th>BLOOD GLUCOSE</th>
<th>REGULAR INSULIN:</th>
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| | Cat: 1u/kg in 250ml NaCl  
Dog: 2unit/kg in 250mL NaCl |
| >14 | 10 ml/hr |
| 11-13 | 7 ml/hr |
| 8-10 | 5 ml/hr |
| 5-7 | Run insulin at 5 ml/hr  
Add 2.5% dextrose in a buretrol to maintenance fluids* |
| <5 | Stop insulin infusion until the next recheck  
Add + 5% dextrose in a buretrol to maintenance fluids* |

(*) You can add 5% dextrose into the maintenance fluid bag and never to take it out continue using the chart as is
Insulin intermittent IM technique
  - Initial dehydrated dose 0.2U/Kg IM then 0.1u/kg IM hourly until blood glucose <14 then switch to regular insulin.

5. Infection
Diabetics are considered immunocompromised and often have concurrent infections. A diabetic’s urine is the ideal place for opportunistic bacterial colonization because of the availability of nutritional glucose for them. The most common is a urinary tract infection and the most common bacteria isolated is E. coli. The current standard of practice according to International Society for Companion Animal Infectious Diseases (ISCAID) is to use a fluroquinolone such as Enrofloxacin at 5-10mg/kg IV/PO every 24 hours. Remember that fluroquinolones are concentration-dependent antimicrobials and have more effective killing at high doses (generally 10x the MIC) and require once daily dosing.

6. GI signs
Many of these patients present with vomiting, diarrhea and anorexia. It is important to provide additional support to them using anti-emetics and antacids. Consider the following:
  - Famotidine 0.5mg/kg IV q12hr
  - Maropitant 1mg/kg IV/SQ q24hr
  - Metclopramide 0.2-0.4mg/kg SQ q6hr

Diet and maintenance insulin dosing
  - Complex carbohydrates
  - High fiber
  - Low fat
  - Once the patient is eating you can transition to injectable insulin. In dogs and cats, I generally start using 0.25 units/kg SQ every 12 hours after a meal and monitor their blood sugars every 4 hours. Random, nadir, or spot blood sugars are not appropriate for monitoring under these circumstances. Many animals will need to have their insulin dose adjusted further before discharge. It is not uncommon for them to end up on closer to 0.5 unit/kg SQ every 12 hours at discharge.

Follow up:
Recheck blood glucose curve should be done in the first 7-10 days after discharge. Blood sugar should be done every 4 hours until the dinner insulin is due. We do not recommend spot blood sugars as they can be misleading. For diabetics with other conditions routine blood work and urine should be monitored. For those with UTI’s routine urine and cultures should be performed. A fructosamine can also be done to assess diabetes regulation of the 2 weeks prior.
THE GAIT EXAM: UNDERUTILIZED BUT CRITICALLY IMPORTANT
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Gait examination is arguably the most important part of the neurologic examination but is often not performed by veterinary practitioners. This may be due to limited appointment times, restricted exam room space or a variety of other factors. However, with observation of an animal walking (or attempting to walk), an astute practitioner can frequently localize a neurologic lesion (or get very close) based on this information alone.

Performance of the Gait Examination

The animal can be initially observed within the exam room as historical information is gathered from the client. In fact, for cats and some dogs, this constitutes the entire exam. Choosing a room without areas to escape or hide is particularly important for cats. For most dogs, it is important to have the owner or an assistant walk them on a lead for more detailed observation.

The patient should be evaluated while walking towards and away from the examiner (best way to identify ataxia), and should also be observed from the side (best way to assess paresis and stride length). If the animal is unable to stand or bear weight, adequate support of the limbs in question should be provided while assessing the ability of the animal to voluntarily advance its limbs, bear weight, and move in a coordinated manner.

Some abnormalities may be accentuated by having the animal climb or descend stairs or a hill if available, although the author does not employ this routinely. Occasionally it is useful to have cats jump on or off an elevated service such as a chair or stool, which can also accentuate subtle abnormalities. Finally, it is useful to invest some time in watching many clinically normal dogs & cats ambulate, which helps one to appreciate subtle abnormal gaits.

Abnormalities

Several abnormalities may be detected with the gait examination. These include:

Ataxia: incoordination characterized by a failure to walk or move the limbs in a straight line, crossing of the limbs over the body midline, and possibly stumbling and falling. Ataxia always indicates neurologic dysfunction, and may be caused by involvement of several areas of the nervous system.

Sensory (proprioceptive) ataxia: Lesions of the peripheral sensory nerve, spinal cord or brainstem commonly cause incoordination. With spinal cord and brainstem lesions, ataxia is typically accompanied by paresis (see below). Peripheral sensory nerve lesions are rare in veterinary patients.

Cerebellar ataxia: Cerebellar lesions can cause a profound ataxia characterized by dysmetria (hypermetria and/or hypometria), and intention tremors. Animals with pure cerebellar lesions maintain good strength without obvious paresis.

Vestibular ataxia: Characteristic incoordination typified by leaning, drifting, stumbling, falling, and occasionally rolling to one side. Usually accompanied by a head tilt,
nystagmus, and possibly positional ventral strabismus. Bilateral involvement of the vestibular system can lead to ataxia and bizarre, wide head excursions but without an obvious head tilt or ataxia, although loss of a normal physiologic nystagmus can often be appreciated.

**Paresis:** muscular weakness or incomplete voluntary movement. On the gait exam, this is characterized by scuffing of the nails, dragging of one or more limbs, a short-strided gait, or rapid tiring with activity/exercise. Paresis denotes dysfunction of the nervous (motor) or muscular systems.

**Lameness:** Inability or reluctance to bear weight on one or more limbs. Lameness often indicates a lesion in the long bones, joints, tendons, or musculature (i.e., orthopedic diseases), although entrapment or compression of a nerve or nerve root can also lead to lameness (known as a "root signature").

**Short-strided Gait:** This gait may occur secondary to paresis as described above. A short-strided gait in all four limbs (particularly in the absence of ataxia) is suggestive of a neuromuscular condition (i.e., disease affecting the peripheral nerves, muscles or neuromuscular junctions). However, such gaits may also occur secondary to orthopaedic conditions affecting multiple limbs (e.g., polyarthritis).

**Disconnected Gait:** Also known as a “two-engine” gait, this describes an animal with different stride lengths between the thoracic and pelvic limbs. Most commonly, the thoracic limbs have the shorter stride although the opposite can also be seen.

**Compulsive Pacing and Circling:** This is typical of a forebrain lesion and such animals typically also show alterations of mentation and consciousness.

**Dysmetria:** Dysmetria refers to both hypermetria and hypometria and is typically seen with cerebellar ataxia as mentioned above. In rare cases, dysmetria may be seen in one or more limbs without ataxia.

**Postural Abnormalities:** These may be observed during ambulation or when the animal comes to a stop and include keeping the head and neck low or ventroflexed, standing with a plantigrade or palmigrade stance, maintaining a wide-based stance and holding the pelvic limbs in a rostral position (i.e., flexed at the hip).

**Examples of Gait Abnormalities and their Causes**

**Compulsive pacing and/or circling:** forebrain lesion (e.g., neoplasia, encephalitis, stroke)

**Ataxia in all 4, paresis in all 4 limbs:** cervical spinal cord or brainstem lesion. Best differentiated by cranial nerve examination or identifying a vestibular quality to the ataxia

**Ataxia in all 4 limbs with a tendency to drift, stumble fall or roll to one side:** vestibular lesion

**Ataxia (usually profound) in all 4 limbs without paresis:** cerebellar lesion (usually accompanied by intention tremors & hypermetria)
Disconnected (two-engine) gait characterized by short-strided thoracic limbs and ataxia with paresis in the pelvic limbs: C6-T2 myelopathy (particularly cervical spondylomyelopathy or “Wobbler’s syndrome”)

Ataxia and paresis in the pelvic limbs: T3-L3 myelopathy; L4-S2 myelopathy is also possible but less likely

Paresis in the pelvic limbs characterized by a short-strided gait: L4-S2 myelopathy or neuromuscular lesion

Paresis in all 4 limbs characterized by a short-strided gait; often worsens with activity or exertion; notable lack of ataxia: neuromuscular lesion; polyarthritis also possible

References


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Fracture management can be challenging because of the emotional, temporal and financial obligations on the part of the owner, the veterinarian and the hospital staff. In a perfect world, all fractures that should be treated surgically would be, no questions asked. But we don’t live in a perfect world so we need to be flexible, understanding, accommodating and at times, creative. A major portion of the veterinarian’s role is to ensure that the client really understands the considerations involved, the short and long term results while maintaining focus on the love and bond they have with their pet and not becoming intimidated by the price tag or the commitment. Sometimes there are very viable alternatives to internal fixation of a fracture, and at times, there are not. Some clients are willing to do ‘whatever it takes' and spend ‘whatever it takes'; some clients are not willing to do anything, no matter how attainable, affordable and palatable the veterinarian ‘makes it'; and then, there is every one in between. Hopefully this information will help the veterinarian with ‘every one in between’ make the most appropriate decision for themselves and their pet.

Fractures that are ideal for splinting include: green stick fractures; non displaced fractures; a two, or more, bone system with at least one bone not fractured (eg. radius and ulna with the ulna intact); transverse fracture with a minimum of 25%, but preferably 50%, overlap of the fragments; the pes and the manus. So, does this mean that none of the other fracture configurations should be treated via splinting? The “perfect world” answer to that question is “yes” but in this world, splinting less than ideal fractures is common. What is important is to understand the potential outcomes and costs with these less than ideal situations.

When splinting a fracture, the joint below and above the fracture should be totally immobilized.

Pes/manus - so easy to splint, include the carpus/tarsus but no need to go much higher. Success rate very high with splinting alone, even when all 4 MC/MT bones are fractured. Splinting till healed is still required with internal fixation. Complication rate high with internal fixation (compared to that of other bones). The splint itself tends to keep adequate alignment of the fragments. The are no significant muscles in this area thus shortening/contraction is not usually a problem. Overall, there is a higher rate of non-union with these bones when compared to other long bones, whether splinted on repaired. Fractured or dislocated phalanges are also best treated with external coaptation. Amputation of a digit may be an ‘easier’ solution than 8 weeks of splinting. Just something to think about.

Scapula: If the shoulder joint itself is not involved then this bone heals very well with a spica splint. The thoracic cavity on the medial surface offers a lot of stabilizing support and the numerous large muscles aid in vascularization as well as stabilization.

Radius-Ulna - Although surgery is best for fractures of the radius and ulna; it is an easy area to splint. If the fracture is in the distal 1/3rd of the bone (which it is most of the time), then adequate immobilization of the fragments can still be achieved without including the elbow in the splint. The elbow must be incorporated into the splint in order to immobilize mid to proximal fractures of the radius. Place the splint in a manner to allow the patient to walk on the foot (i.e. include the foot int he bandage but not the splint). Proximal ulnar fractures not to do well with splinting because of the pull of the triceps which naturally distract the fracture line.

Tibia-Fibula - this is not an easy bone to splint. The angulations and funnel-shape of the hind limb make it difficult to properly contour and fit bandage and splinting material. Fractures in the proximal third of the tibia are not adequately immobilized with a splint. Mid to distal third shaft of the tibia can do well with a splint. It is important to maintain flexion at the hock. Check the splint regularly as decubital ulcers often from. Place the splint in
a manner to allow the patient to walk on the foot (i.e. include the foot int he bandage but not the splint).

Humerus - the humerus can be immobilized with a spica splint. However, the pull from the strong muscles that surround this bone tend to distract the fragments and therefore, a malunion will result. The upside is that the position of the muscles tend maintain the shoulder and elbow joints in alignment which may allow for a limb that functions adequately despite a malunion.

Femur: this bone can not be immobilized with a splint/bandage so it is best to leave the patient within a crate. The strong muscles surrounding this bone will help to keep it immobilized. Without surgery, a malunion will occur without a doubt. Just like the humerus, the directionality of the muscles tends to maintain the hip and stifle within reasonable alignment which may allow for a limb that functions adequately despite a malunion.

Prognosis for fracture repair is dependent on configuration and location. Almost all fracture types can be repaired by a trained/experienced surgeon, however, the more complex configurations (multiple fragments especially when involving the extremities of the bones) can have a higher complication rate. For many clients, understanding that the prognosis is very good and complication rate low is often all they need to make the decision to proceed with a repair rather than an amputation or settling for a splint.

**Diaphyseal fractures:** Overall these fractures carry an excellent prognosis. The simple (2 piece) fractures also tend to have a very low complication rate. Do not hesitate to recommend surgical repair for these fractures, assure the clients that failure and complication rates are low in the hands of an experienced surgeon. These are also the better fractures for someone who has just taken a fracture repair course to tackle and gain confidence with; the exceptions to this rule are the humeral fractures as the approach to this bone is very difficult. Fortunately, the humerus is the least commonly fractured long bone. The multi-fragmented fractures tend to be much more difficult to repair, often requiring more implants and therefore the surgery may cost a bit more and the risk of complications may be higher; however, once healed, these also carry and excellent prognosis. The complex fractures are best left in the hands of a trained, experienced surgeon.

**Metaphyseal fractures** overall tend to be a bit more difficult to repair than diaphyseal fractures; especially when involving the humerus or femur. The approach to these areas is more difficult because the muscles attach to the metaphyses. Also the metaphyseal fragments will be small, therefore difficult to manoeuvre into place and may not accommodate an adequate number of screws or pins; the plates often need to be significantly contoured, something that can be difficult to accurately achieve. These fractures are best left for the trained surgeon.

**Articular fractures** involve the epiphysis. Congruency is important for comfort when weight bearing. Accurate surgical reduction of the fragments is difficult but important to achieve; these
Fractures are best served being repaired by a trained surgeon. Patients with articular fractures will develop osteoarthritis (OA) whether they have surgery or not. However, with surgery, the OA will tend to be less severe; and the animal will be able to ambulate much more comfortably after surgery. The most common long term complications with articular fractures is loss of range of motion and OA. Rehabilitation will help with both these factors. OA is best managed with weight control, appropriate activities as well as chondroprotectives. NSAIDs are often necessary sporadically and perhaps continuously as the animal ages and the OA progresses. Overall, OA is manageable and owners should not be discouraged from pursuing repair because of it. Fractures of the coxofemoral joint can be treated with a femoral head osteotomy (FHO). This surgery is one that does not require a lot of specialized equipment and can readily be learnt through a workshop.

**Physeal fractures:** Although there is always a concern of potential growth abnormalities with physeal trauma, in reality, that does not occur commonly. The exception to this is when a young animal fractures the radius (often a diaphyseal fracture) but not the ulna; typically the ulnar physis is damaged in the original trauma. Overall, physeal fractures have an *excellent* prognosis with surgical repair, so do not hesitate to recommend surgery! Without surgery the fracture will heal, perhaps with an insignificant to marked malunion depending on the fracture displacement. Even with a mild displacement, a residual mild to moderate lameness tends to remain. Of course, physeal fractures occur in young animals, so, that is potentially a lot of years with a lameness. Physeal fractures are generally repaired with k-wires, keeping the cost of implants low. These fractures require careful handling of the physeal surface and are best left in the hands of the trained surgeon. Because of their proximity to the joint, physeal fractures *may* result in the formation of OA.

**Splint/bandage application and care:**
Creating a stable, comfortable and suitable splint takes some practice. Use stirrups, not to hold the bandage up, but to ensure that the two central toes remain visible. Create and place donuts over the bony protuberances. Apply the cast padding - use protouch™ or another such material that comes in a manageable roll. Avoid using the heavy rolls of cotton, this should be reserved for a Robert Jones bandage. Start at the toes if possible and ensure you have 50% overlap of the cotton as you unroll it up the limb. Make sure the cotton padding lies flat and smooth. Next apply the kling, again starting at the toes with 50% overlap and ensuring that the kling is smooth. The more padding you have placed, the tighter you can pull the kling. Now tape the stirrups to the kling and place the splint. Tape the splint in place with non-stretchy adhesive tape. If you moulded the splint from fibreglass material, consider placing labels on it (proximal, caudal, etc) so that it is readily recognized how it should fit at the time of a bandage change. Now apply the Vetwrap™.

For a demonstration on how to apply a spica bandage/splint visit [www.focusandflourish.com](http://www.focusandflourish.com) and select “teaching videos”.

The owners should be given specific bandage care instructions (keep bandage dry, examine toes 3-4 times daily for swelling, call if the bandage slips, if the patient starts to chew at the bandage, becomes lame or if it is malodorous) and they should be made to understand why these instructions are important (infection, pain, bandage falls off, foot/legs falls off). Change the bandage/splint every week, especially if sores start to form under the bandage. Take follow up radiographs every 4 weeks.
Diagnosis and Treatment of Canine Oral Osteosarcoma
Fernanda Mantovani, DVM, DVSc, Diplomate ACVIM (Oncology)

Oral osteosarcoma, affecting the mandible or maxilla, represent up to 12% of all forms of canine osteosarcoma, and are the 4th most common non-odontogenic tumor of the oral cavity in dogs. It typically affects dogs 9-10 years of age, and weighing more than 20 kg.

Clinical signs of oral osteosarcoma include oral bleeding, oral pain - manifested when opening or closing the mouth, chewing toys or food, or when the face is touched - actual oral mass noted by the owners, draining tract or oral fistula, and halitosis.

At the time of presentation a mass is identified on oral examination in most cases. It is important to perform an oral examination in dogs presenting with any of the above clinical signs, and most dogs tolerate at least a brief oral examination without sedation, which can allow the veterinarian to at least identify a mass, even if the exact size and extension can not be determined with the patient awake. Oral osteosarcomas can be red and fleshy, and not necessarily hard on palpation. Maxillary tumors can cause facial asymmetry or bulges on the muzzle. It is very important to retropulse both eyes and palpate the mandibular lymph nodes in all dogs with oral tumors, as a thorough physical examination can give valuable information regarding the extent of the tumor.

Dental radiographs can provide helpful information on the extent of bone lysis - often more informative than skull radiographs. Osteosarcoma presents as lytic or proliferative bone patterns, or a mixture of both. Bone lysis is a common feature for oral osteosarcomas. CT scan of the head provides the most information regarding extent of the tumor, specially in cases of maxillary masses, which can invade into the nasal cavity or retrobulbar space, as this soft tissue involvement is not typically seen on radiographs.

Biopsy and histopathology is required for a definitive diagnosis of oral osteosarcoma. In some cases fine needle aspirates of the tumor can be with the animal awake, depending on the location of the mass and temperament of the patient - fine needle aspirates of maxillary masses can be performed from the haired skin aspect of the muzzle, and masses located on the rostral mandible or maxilla may be accessed via the oral cavity without major restraint of the patient. Cytology can be very helpful in ruling out an abscess or other inflammatory lesion, and can confirm the presence of neoplasia or specifically mesenchymal neoplasia; however cytology would not differentiate between osteosarcoma and other sarcomas.

Important considerations for biopsy of oral tumors:

1) Always retrieve the sample through the oral cavity/ oral mucosa and do not approach through the haired skin of the muzzle - the biopsy tract must be removed at the time of definitive surgery and the skin is often used for the reconstructive aspect of surgery, and should therefore be left intact.
2) If the mass is larger than 1 to 1.5 cm, an incisional biopsy is preferable over excisional biopsy, to avoid excision of mucosal tissue required for definitive surgery and large biopsy tract.
3) If an excisional biopsy is performed, taking pictures and documenting the exact size and location of the mass will be very helpful to plan definitive surgery after - it can be difficult to find small scars from excisional biopsies, and sometimes it is important to known clearly where the tumor was located when patients are referred to oncologists and surgeons.
Oral osteosarcomas are locally aggressive tumors. If treatment of the local tumor is not performed or not successful, typically local tumor growth and associated pain, reluctance to eat, oral bleeding and halitosis lead to death or euthanasia of affected dogs.

Wide resection with excision of the underlying affected bone is the local treatment of choice for oral osteosarcoma. Surgery typically intends to achieve 1 cm tumor free margins based on clinical and CT evaluation. Cases that are often more amenable for surgery are tumors < 2 cm and located in the rostral oral cavity. Mandibular tumors are often more amenable for surgery than maxillary tumors.

Oral osteosarcoma has a reported metastatic rate of 45-60%. The most common site of metastasis are lungs, and uncommonly can occur to other bones and draining lymph node. In dogs treated with surgery and chemotherapy, the median time until metastasis is 18 months.

### Summary of treatment options and associated survival time

<table>
<thead>
<tr>
<th>Treatment modality</th>
<th>Prognosis and comments</th>
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| **Pain medications**               | If used as single treatment, 2-3 months survival depending on tumor size and growth rate  
If no signs of pain, NSAID only  
With pain progression multimodal treatment required: NSAID, tramadol, amantadine, gabapentin  
Soft food, soft chew toys           |
| **Pamidronate**                    | Inhibit osteoclast activity - important role in bone pain from primary and metastatic bone tumors  
Used in conjunction with oral pain medications  
Given IV over 2-4 hours with crystalloid fluids, once monthly  
Can aid in comfort of dogs with oral osteosarcoma that do not undergo surgery, may or may not improve overall survival compared to oral pain medications only, but can improve quality of life |
| **Surgery and chemotherapy**       | Up to 60% metastasis rate  
Local recurrence after surgery for incompletely excised tumors - median time of 4-6 months  
If complete margins, dogs that develop metastasis have a 1.5 to 2 year survival, and long term survival is possible in dogs that do not develop metastasis |
| **Palladia**                       | Tyrosine kinase inhibitor, given orally at home. Reported biological activity against metastatic osteosarcoma, no reports on activity against oral osteosarcoma.  
50% response rate, mostly stable disease (slowing down tumor growth with no actual shrinkage), for a median duration of 4-6 months. |
| **Radiation therapy**              | Main role in palliative treatment rather than curative intent treatment, as osteosarcomas typically do not shrink with radiation.  
Survival of 3-4 months with palliative radiation protocols |
References:


Cognitive dysfunction syndrome (CDS) is a neurodegenerative disorder of senior dogs characterized by gradual and progressive cognitive decline. Advancing brain pathology is expressed behaviorally by signs related to learning, memory, perception, awareness, social interactions, sleep and activity. Research indicates that symptoms and pathology may resemble the early stages of human Alzheimer’s disease (AD). 1-5

The diagnosis is based on clinical signs described by the acronym DISH, including disorientation, altered social interactions and sleep-wake cycles, and loss of housetraining and other learned behaviors. 6-10 In addition, while activity may initially decline, an increase in spontaneous activity with greater severity of cognitive dysfunction. 11 Increased anxiety and agitation are also associated with CDS. In one study the most common signs in dogs with marked CDS were sleeping more during the day and restlessness at night (57%), altered social interactions (51%), disorientation (49%) and anxiety (46%). For dogs with mild CDS, the principal sign was increased daytime sleep (70%) with anxiety at 11%. Anxiety was seen in only 4% of unaffected dogs. 12 In another study, anxiety in senior dogs was associated with a more passive response compared to dogs <7 years, and may therefore be less easy to recognize. 13 Therefore the acronym DISHAA offers more extensive screening.

While a decline in learning and memory may be the most important indicator of cognitive decline, the average pet may appear minimally challenged. In fact, other than knowing its name, a few behaviors trained on cue, and housetraining there are few changes that are likely to be noticed until the dysfunction becomes severe. However, with the development and validation of neuropsychological tests, learning and memory impairments have been objectively quantified in the laboratory in senior dogs. These tests also provide a mechanism by which the effect of therapeutic agents can be assessed. As these tests require trained personnel, cognitive assessment apparatus and validated methodology, they are a not a practical option in the clinical setting. While pet owners may not notice behavior changes of CDS until 11 years or older, deficits in learning and memory have been identified early as 6 years of age. 3,4,14

Prevalence
Overall prevalence of CDS in dogs over 8 years of age has been estimated to range from 14% to over 60%. 7,14,15 In addition, both prevalence and severity progress with increasing age. 7,8,12,14,15 In one study, 28% of dogs 11-12 years of age were reported to have at least 1 category of DISHA and 10% were positive for 2 or more categories, while in dogs 15-16 years old, 68% were positive for at least 1 category and 36% for 2 or more categories.16 Katina et al identified CDS in 13-16% of dogs aged 8-11, and 87%-100% in dogs > 13.15 In another study, over 6 months, 42% of dogs with no impairment progressed to mild impairment and 24% of dogs with mild impairment progressed to moderate. After 1 year, this increased to 71% and 50% respectively.7 Therefore, to insure the earliest possible identification and diagnosis, pets over 8 should be scheduled for twice yearly visits and screened with a cognitive assessment questionnaire at each visit.1,6-9,14,17-19 As initial signs of cognitive decline may be subtle, most cases go undiagnosed until signs become sufficiently problematic for the pet or the owner.7 In one study, 85% of cases had not been diagnosed. 14

Age related pathology
In dogs, with increasing age frontal lobe and temporal lobe volume decrease, ventricular size increases and there is meningeal calcification, a reduction in neurons and an increase in toxic
Circulatory changes including microhemorrhage and infarcts may also be responsible for signs of CDS. As in humans, a decline in the cholinergic system has also been identified which may contribute to declining cognitive and motor function.

In dogs, cats, and humans there is an accumulation of diffuse beta amyloid plaques and perivascular infiltrates with increased Aβ correlating with cognitive impairment. Most recently cognitive decline has been shown to be related to neuro-inflammation and tau hyperphosphorylation in synapses in dogs. Taken together, the pathological changes, together with the clinical signs, learning and memory impairment and progression with increasing age, dogs have been categorized as a model for early Alzheimer’s disease (AD). On the other hand in contrast to human AD, senile plaques remain diffuse and neurofibrillary tangles are rare. In addition, in pets CDS seldom leads to mortality.

Diagnosis

When signs are identified, a diagnostic workup is necessary to rule out medical, physical and motor dysfunction as a cause of the signs. Next to neurological disease, sensory decline, endocrine and metabolic disorders and musculoskeletal disease are the primary rule-outs. Determining what needs to be assessed and with what diagnostic tools, should be based on the presenting signs and physical exam.

Treatment

a) Environmental enrichment

Both mental and physical enrichment can play an important role in treatment. In fact, the combination of nutrition and behavioral enrichment have been demonstrated to slow the progression and improve the clinical signs of cognitive dysfunction. In addition, nutrition has been found to be a risk factors with dogs fed high quality commercial diets designed for age, size, or health, less likely to develop CDS than dogs fed low quality commercial food or table scraps.

When considering the type and level of enrichment for senior pets, don’t just limit to what the pet seems to want; identify, determine and provide for what the pet needs within the limitations of its physical and mental health. This includes eating, sleeping, grooming, elimination, social interaction, and physical and mental enrichment including social and object play, exercise and training. Shorter, slower, more limited or less frequent physical activities might need to be provided. Continuing to practice learned cues, teaching new cues or adding in new forms of enrichment (e.g. scent work, hide and seek, food, activity puzzles) can provide ongoing mental and social enrichment even if mobility and stamina are reduced.

b) Medical and nutritional therapy

Selegiline is a monoamine oxidase B inhibitor which has demonstrated efficacy in improving cognitive signs. It has been shown to increase 2-phenylethylamine in the dog brain, a neuromodulator that enhances dopamine and other catecholamines in the cortex and hippocampus. It may also contribute to a decrease in free radical load through decreased production and increased clearance. Dose is 0.5 – 1.0 mg/kg daily.

Since the elderly are particularly susceptible to the effects of anticholinergic drugs, it is prudent to avoid drugs with anticholinergic effects. In fact, use of anticholinergic drugs might potentially contribute to further cognitive impairment. For anticholinergic effects of drugs see http://www.agingbraincare.org/uploads/products/ACB_scale_-_legal_size.pdf. Drugs or natural products that enhance cholinergic transmission might have benefit for improving signs of CDS.
In addition two drugs available in Europe but not North America are propentofylline, a xanthine derivative that might increase blood flow and inhibit platelet aggregation and thrombus formation and nicergoline an alpha 1 and alpha 2 agonist.

A senior diet (Canine b/d, Hills Pet Nutrition) supplemented with fatty acids, antioxidants (vitamins C and E, beta carotene, selenium, flavonoids, carotenoids), and dl-alpha-lipoic acid and l-carnitine to enhance mitochondrial function has been shown to improve signs and slow the progress of cognitive decline.4,25,26 The highest cognitive scores were seen in the dogs that received both the antioxidant diet and added enrichment.

Purina Veterinary Diet supplemented with botanic oils containing medium chain triglycerides provides ketone bodies as an alternate source of energy for aging neurons, has also been shown to significantly improve CDS in dogs.27 Studies in dogs have demonstrated a significant reduction in cerebral glucose metabolism in 6 year old dogs compared to one year of age.28 Over an 8 month trial the group supplemented with 5.5% MCT showed significantly better performance over a placebo diet in a variety of neuropsychological test protocols. In addition the group given MCT supplement showed significantly elevated levels of the ketone body, β-hydroxybutyrate (BHB).27

Senilife® (CEVA Animal Health), has demonstrated efficacy in improving cognition in both a laboratory model and clinical studies in dogs. It contains phosphatidylserine, a membrane phospholipid as well as Gingko biloba, vitamins E and B6 and resveratrol.9,29 Royal Canin Canine Mature Consult diet and Royal Canin Feline Senior Consult also contain phosphatidylserine, l-tryptophan and an antioxidant blend. S-adenosyl-l-methionine (Novifit®, Virbac) may help to maintain cell membrane fluidity and receptor function, regulate neurotransmitter levels and increase production of glutathione. Improvement has been demonstrated in both dogs and cats in laboratory studies and in a clinical trial in dogs.30,31 Apoaequorin (Neutricks™) is a protein found in jellyfish that in laboratory trials improved learning and attention in dogs. It is a calcium buffering protein that may provide neuroprotection against aging.32

References/Suggested Reading (full references available from author on request)
APPRAOCHES TO THE ICTERIC ANIMAL
Dr. Shannon Westgarth, BSc, DVM, DVSc, DACVIM

Formation of bile
Within the liver there are canaliculi, which are responsible for bile formation. The bile canaliculi unite to form lobar ducts, which then form the hepatic ducts and eventually terminate in the common bile duct and gallbladder. The gallbladder is where water and electrolytes are absorbed and the bile salts are concentrated (up to 10 fold), mucin is added as a lubricant, and bile is acidified. Mucin production here is also stimulated by inflammatory cytokines and prostaglandins, which can lead to disease processes. From the gallbladder, the bile empties in response to several hormones (CCK) into the cystic duct which eventually meets up with the common bile duct (CBD). In the cat, the CBD fuses with the pancreatic duct before entering the duodenal papilla, whereas in the dog, the CBD opens near the minor pancreatic duct which results in the differences in their disease processes.

Evaluation of patient with biliary tract disease
Biochemical profile – Increased ALP, GGT, hyperbilirubinemia, less substantial increases in ALT and AST
Complete blood count – Neutrophilic leukocytosis and possibly a left shift
Radiography
- May see mineral densities in the biliary tree reflecting choledolith formation or stasis
- Poor serosal detail with pancreatitis or biliary rupture
- Gas may represent an emphysematous process
- Sternal lymphadenopathy with cats with cholangiohepatitis
Ultrasonography
- Insufficient to assess for diffuse parenchymal disorders
- Assess size, masses, wall thickness of gallbladder (GB) and biliary tree, echogenicity of liver, pancreas, lymph nodes, presence of fluid
- Engorged GB and dilated cystic ducts within 24 hours of complete extrahepatic biliary duct obstruction (EHBDO), intrahepatic bile duct distention within 5-7 days

Disorders causing icterus in animals

Cholecystitis
This is an inflammatory condition of the GB due to many different causes. Patients may present with abdominal pain and fever. Bloodwork reveals elevated ALP, GGT, hyperbilirubinemia and possible other elevated hepatic enzyme activity. On ultrasound, thickened GB or CBD wall noted, and with necrotizing cholecystitis, there may be free fluid or gas surrounding the GB due to rupture requiring surgical intervention. Cholecystitis can be caused by a thromboembolism to the artery of the GB, bacterial infection, duct obstruction, or GB mucocele causing wall ischemia. Having emphysematous cholecystitis is associated with diabetes mellitus, GB mucocele or neoplasia. Treatment consists of fluids, antibiotics (metronidazole, ampicillin, and enrofloxacin), analgesia (Cerenia?), and possible surgery. Vitamin K1 may be needed if chronic EHBDO due to deficiency in vitamin K from altered hepatobiliary excretion of bile acids required for absorption. Cultures should be submitted of bile, wall, and liver.

Gallbladder mucocele
Gallbladder mucoceles occur due to the hyperplasia of the mucous-secreting glands of the GB resulting in an accumulation of tenacious mucin-laden bile. If they are not attended to, they can lead to necrosis of the GB, peritonitis, and infections. The median age of occurrence is 10 years, and they are more common in cocker spaniels and miniature schnauzers which is
thought to be secondary to hyperlipidemia and hypercholesterolemia. They can also occur secondary to GB dysmotility resulting in stasis and mucous hypersecretion. Finally, many dogs with GB mucoceles also concurrently with have vacuolar hepatopathy and many have hyperadrenocorticism or hypothyroidism. Medical management may be attempted with ursodeoxycholic acid (UCDA) at 15-25 mg/kg/day and s-adenosyl methionine (SAMe) at 20-40 mg/kg/day. Progression should be monitored with serial biochemical profiles and ultrasounds every 6 weeks. Surgery should be performed if there is concern about bilirubin elevations, if the animal is clinically ill, or if there is concern for rupture. It also can be considered even for asymptomatic ones, as stable mucoceles have a better outcome than those that have ruptured. Following cholecystectomy, chronic therapy with choleretic therapy is recommended.

**Extrahepatic bile duct obstruction**

Obstruction of the bile duct leads to free radical damage to the cell membranes and organelles. Obstruction of greater than 6 weeks can lead to biliary cirrhosis, portal hypertension, and acquired portosystemic shunts (PSS). This can also lead to fat malabsorption secondary to lack of bile acid delivery. With acute obstruction patients can have jaundice, lethargy, fever, and vomiting. As it becomes more chronic they can develop hepatomegaly, acholic feces, coagulopathies, and hypotension. ALT and AST increase from damaged hepatocytes and necrosis. In 8-12 hours ALP and GGT increase. In the beginning, cholesterol increases due to obstruction and lack of elimination. With fulminant liver failure, the cholesterol will decline, and coagulopathies can develop from vitamin K deficiency due to lack of absorption. Also, the absence of bile in the intestine deprives the gut of IgA, which normally prevents bacterial adherence to the intestinal mucosa. If there is concern for complete obstruction, relief can immediately be achieved with ultrasound guided cholecystocentesis to relieve the pressure, but this has risks of rupture. Surgical intervention is generally required if there is complete obstruction. Choledochal biliary stenting through the duodenal papilla can be attempted. Otherwise, cholecystoenteromy may need to be considered, but has a poor prognosis in cats.

**Cholelithiasis**

In small animals most of the time choleliths are silent. Small breed dogs (primarily miniature schnauzers and poodles) are predisposed to the development of choleliths. Most stones contain calcium carbonate and bilirubin pigments, less commonly cholesterol unlike in people. Stasis of bile flow can lead to cholelithiasis formation. Clinical signs include vomiting, anorexia, fever, pain, jaundice. The stones may not necessarily cause hyperbilirubinemia but can induce infection. Abdominal ultrasound is helpful to see stones 2 mm or greater in the GB. Treatment can be medical (broad spectrum antibiotics, choleretic therapy), or may require surgery.

**Feline cholangitis/cholangiohepatitis**

Feline cholangiohepatitis is the most common acquired inflammatory liver disease in the cat. It also can occur concurrently with inflammation of the duodenum, pancreas, and kidneys. This condition is thought to occur more commonly in cats than in dogs because of the difference in their anatomy. There are different types that occur in the cat. Neutrophilic cholangitis causes more overt clinical illness, and has the shortest duration of clinical signs. It more commonly occurs in young and middle aged cats. Cats with this condition have moderate increases in ALT and AST as well as modest increases in ALP and GGT. Cytology of the liver can also be performed which may reveal bacteria. Also, enlarged sternal lymph nodes can be seen on thoracic radiographs due to drainage of the cranial abdomen. The findings on abdominal ultrasound tend to be variable depending on if there are any other concurrent disorders going on. Treatment includes antibiotic therapy (ampicillin, metronidazole, enrofloxacin), UDCA, SAMe, enteral nutrition. With lymphoplasmacytic or lymphocytic cholangitis they tend to be middle aged to older, and have a more chronic history. They can present with episodic vomiting.
and diarrhea, as well as jaundice. These cats can have elevated ALT and AST, as well as hyperglobulinemia and poikilocytes. The initial treatment for these cats is very similar to neutrophilic. Additional therapy to be considered following aspirates or biopsy (to determine if bacteria are present) includes prednisolone (2-4 mg/kg/day). Metronidazole (7.5 mg/kg twice daily) can also be helpful at reducing inflammation.

References used throughout:
MANAGEMENT OPTIONS FOR HYPERCALCEMIA IN THE DOG AND CAT
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Introduction

Hypercalcemia is defined as an elevation in serum total or ionized calcium. The presence of hypercalcemia should warrant an immediate investigation. Hypercalcemia can have consequences to organ systems, and also may indicate the presence of significant disease, such as neoplasia.

Causes of Hypercalcemia

There are several possible causes of hypercalcemia. The two most common causes in the dog are primary parathyroidism (PHP), and hypercalcemia of malignancy. In cats, the most common causes include idiopathic hypercalcemia, renal disease and hypercalcemia of malignancy. Many students will recall the mnemonic for causes of hypercalcemia (HARD IONS, see Table 1).

Table 1: HARD IONS mnemonic for causes of hypercalcemia in the dog and cat.

| H | Hyperparathyroidism, Houseplants |
| A | Addison’s disease, Aluminum or vitamin A toxicity |
| R | Renal disease, Raisins and grapes |
| D | Vitamin D toxicosis |
| I | Idiopathic (common in cats, rare in dogs) |
| O | Osteolytic |
| N | Neoplasia (Humoral hypercalcemia of malignancy) |
| S | Spurious, Skeletal growth |

Nonpathologic increases can be due to laboratory error, skeletal growth, and lipemic samples causing spurious results. Rechecking a calcium level, and ensuring the absence of lipemia by submitting fasting serum, should be the first step when hypercalcemia is detected. Transient increases in total serum calcium can be seen with hyperalbuminemia secondary to dehydration.

Pathologic causes of hypercalcemia can be divided into PTH (parathyroid hormone)-dependent processes, and PTH-independent processes. The PTH-dependent cause of hypercalcemia is primary hyperparathyroidism. PTH-independent processes include hypoadrenocorticism, renal disease, neoplasia, toxicosis (houseplants, vitamin A, vitamin D, grapes, raisins, aluminum), and idiopathic hypercalcemia. The most common causes of hypercalcemia of malignancy include lymphoma (most commonly T cell) and anal sac adenocarcinoma.

Clinical Signs

Many pets with hypercalcemia will not have clinical signs, and the abnormality is noted on screening blood work. Clinical signs are most commonly seen when the hypercalcemia develops rapidly, when there is severe hypercalcemia, or when both of these occur. Clinical signs can include polyuria and polydipsia, lethargy, weakness, inappetence or anorexia, vomiting, and less commonly seizures, muscular twitching, diarrhea or constipation, collapse, and symptoms related to calcium oxalate urolithiasis. In addition, clinical signs may be related to the cause (such as tenesmus in a pet with anal gland adenocarcinoma, vomiting and diarrhea in a pet with hypoadrenocorticism).
Physical Examination Findings

The abnormalities noted on physical examination will vary based on the underlying cause. A pet with lymphoma will often present with enlarged peripheral lymph nodes. Anal sac adenocarcinoma can usually be detected on rectal palpation, and enlarged sublumbar lymph nodes may be present. Arrhythmia may be present but is rare with hypercalcemia. Physical examination in a pet with PHP is usually normal, unless uroliths can be palpated in the urinary bladder. There are no consistent physical examination findings in cats with idiopathic hypercalcemia. Pets with hypercalcemia secondary to toxicosis are usually symptomatic, but there are no classic physical examination findings. Pets with renal disease and hypoadrenocorticism may have some degree of cachexia present, and/or a poor hair coat.

Diagnosis

Once hypercalcemia has been noted on a routine serum biochemical profile, it should be confirmed either with a repeat, fasted total serum calcium level ensuring a normal albumin level, or through an ionized calcium (iCa) level if available in house. If the elevated serum calcium level is repeatable on a fasted sample with a normal serum albumin level, or an in house iCa level is elevated, then additional calcium homeostasis testing is indicated. A paired iCa and PTH level should be performed at minimum, and ideally a PTH-rp (PTH-related protein) is measured as well. In the presence of an elevated iCa, PTH should be suppressed and therefore the PTH level should be low.

The presence of an elevated iCa along with a high normal or elevated PTH confirms a diagnosis of primary hyperparathyroidism (PHP). Cervical ultrasound with an experienced ultrasonographer is then recommended, to identify one or more enlarged parathyroid glands. In the presence of an elevated iCa, a low PTH rules out PHP. A low normal PTH in the presence of an elevated iCa is considered grey zone.

In the presence of an elevated iCa, if the PTH is low, then the differential diagnoses include hypoadrenocorticism, neoplasia, toxicosis, and idiopathic hypercalcemia. Renal disease usually results in an elevated total serum calcium but a normal iCa. If a PTH-rp is performed and is elevated, neoplasia should be suspected, although there are rare cases of infectious disease leading to a positive PTH-rp result.

The next steps after finding an elevated iCa with a low PTH will be dependent on the signalment. Peripheral lymph node and rectal palpation should be performed in all cases, however additional screening for neoplasia is indicated (thoracic radiography, abdominal ultrasonography, possible bone marrow sampling, protein electrophoresis). A young pet should have an ACTH stimulation test performed, especially if adrenal glands are small on ultrasonography. Questioning on possible toxin exposure is indicated. Long bone pain should be evaluated. In cats, if renal disease is ruled out, neoplasia is not detected, and there is no evidence for hypoadrenocorticism or toxin exposure, then a diagnosis of idiopathic hypercalcemia is made. However, it is sometimes difficult to detect neoplasia, as some cases are occult and may become evident in weeks or months.

Treatment

The most effective treatment for hypercalcemia is eliminating the underlying cause. For PHP, removal of the enlarged parathyroid gland(s) will usually resolve the hypercalcemia. Pets should
be monitored closely for post-operative hypocalcemia, which is usually transient but can be marked. For hypercalcemia of malignancy, therapy for the neoplasia will typically resolve the hypercalcemia. Monitoring for recurrence of hypercalcemia may indicate relapse of the disease. As hypercalcemia associated with renal disease is usually an elevation in total serum calcium but not iCa, therapy for the hypercalcemia is not indicated.

Hypercalcemia associated with toxin exposure should resolve with supportive care. This should include intravenous fluid therapy, typically with isotonic saline solution. Once rehydration is attained, a loop diuretic (usually furosemide) can be initiated to enhance renal calcium excretion. Sodium bicarbonate will rapidly reduce the iCa levels, but is reserved for critical patients. Pamidronate infusion, although more commonly used for hypercalcemia of malignancy, can be used to treat hypercalcemia secondary to toxin ingestion via inhibition of osteoclasts. As it can be toxic to the kidneys, rehydration is essential prior to use. Calcitonin can also be used to reduce calcium levels in toxicosis cases via inhibition of osteoclasts and inhibition of renal tubular reabsorption of calcium. Glucocorticoids are usually reserved for cases with confirmed neoplasia, confirmed hypoadrenocorticism, or for longer term control of idiopathic hypercalcemia. They can be used in the critical patient if deemed appropriate, once diagnostic samples have been obtained.

For cases with either idiopathic hypercalcemia, or with a disease process that cannot be addressed (refractory neoplasia, PHP in a case where surgery is not possible), long term medical management of the hypercalcemia can be implemented. Options include dietary management, glucocorticoids, bisphosphonates, and calcimimetics.

High fibre diets have been advocated for the treatment of hypercalcemia, although studies are lacking to assess their efficacy. Glucocorticoids reduce hypercalcemia by reducing bone resorption, reducing intestinal resorption, and increasing renal excretion. A typical dosage for prednisone or prednisolone to reduce hypercalcemia would range from 0.5-1 mg/kg once daily per os with food. Dexamethasone, if used in place of prednisone/prednisolone, can be initiated at around 0.1 mg/kg once daily per os with food.

Bisphosphonates reduce hypercalcemia by inhibiting osteoclastogenesis and osteoclast activity. Pamidronate is administered as an intravenous infusion with sodium chloride. Alendronate sodium is an oral bisphosphonate with little information in the veterinary literature, although it seems to be less effective than IV pamidronate. In humans, it can cause gastrointestinal irritation and erosions, and gastrointestinal upset has been reported sporadically in cats and dogs. Esophageal erosion and stricture is reported, and therefore caution must be used when administering alendronate sodium to pets. As the medication should be given on an empty stomach, it is recommended to syringe in water orally after administration of pills, and/or butter the lips. Recommended dosing in dogs is 0.5-1 mg/kg once daily on an empty stomach per os. In cats, a dose of 10 mg PER CAT ONCE WEEKLY on an empty stomach is recommended. If this is not effective, then the dose can be increased to 10 mg PER CAT once every 3-4 days on an empty stomach, or up to 30 mg PER CAT once weekly on an empty stomach.

Calcimimetics are a new class of drugs to combat hypercalcemia. They suppress PTH secretion via inhibition of calcium-sensing receptors, therefore they are not indicated for use in hypercalcemia of malignancy or vitamin D toxicosis. However, they have been shown to be effective in some cases of hypercalcemia (idiopathic, PHP). An initial dose of 7.5-30 mg PER DOG once daily is recommended.

References available from the author on request.
AN OVERVIEW OF PROTEIN-LOSING NEPHROPATHY IN THE DOG
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Introduction

In the healthy kidney, protein is retained in the blood stream via both filtration in the glomerulus and resorption of smaller filtered proteins in the proximal tubule. Pathologic proteinuria can be present due to three mechanisms: pre-glomerular proteinuria, glomerular proteinuria, and post-glomerular proteinuria. Glomerular proteinuria is caused by protein-losing nephropathy (PLN), which are a collection of renal diseases that result in persistent pathologic proteinuria due to glomerular disease. There are a number of different causes of PLN, under the broad categories of familial conditions and acquired diseases.

Causes of Protein-Losing Nephropathy

Familial PLN are recognized in several canine breeds. The most well known breeds that are predisposed to familial PLN are the Soft Coated Wheaton Terrier due to a glomerulopathy, along with familial renal amyloidosis in the Chinese Shar Pei dogs. Other breeds that are known to have a predisposition to PLN include the Airedale Terrier, Beagle, Bernese Mountain Dog, Bullmastiff, Bull Terrier, English Cocker Spaniel, Rottweiler, Samoyed, and English Foxhound. There are DNA tests available for some of these breeds.

Acquired PLN may be primary with no known underlying cause (idiopathic), or secondary to an underlying infectious, inflammatory or neoplastic condition. Examples of secondary conditions include Lyme borreliosis and other tick borne diseases, Leishmaniasis, heartworm disease, parasitism, chronic bacterial infections, systemic lupus erythematosus, hyperadrenocorticism, and diabetes mellitus. Some drugs can cause a secondary proteinuria, such as the non-inflammatory glomerulopathy seen with Palladia, an anti-neoplastic tyrosine kinase inhibitor.

Causes of non-infectious acquired PLN include immune complex glomerulonephritis (ICGN), non-immune complex-mediated glomerulonephropathies (non-ICGN), and reactive amyloidosis. Immune complex glomerulonephritis (ICGN) can be subdivided into membranoproliferative glomerulonephritis and membranous glomerulonephropathy. Non-immune complex-mediated glomerulonephropathies (non-ICGN) can be subdivided into glomerulosclerosis, minimal change disease, and focal segmental glomerulosclerosis.

In many cases, a renal biopsy is not performed, and therefore the underlying mechanism is not known. Recent studies have indicated that renal biopsy may be quite important for specific cases of PLN, as results may alter both treatment and prognosis. However, cases may be misclassified if transmission electron microscopy and immunofluorescence are not included in evaluation of biopsy samples. Studies suggest that about half of dogs with PLN will have ICGN, which is the deposition of immune complexes in the glomerulus. These cases may benefit from immunosuppressive therapy, which is currently not widely used in PLN cases.

Consequences of Progressive Protein-Losing Nephropathy

As excessive proteinuria persists over time, there are several potential consequences. Hypertension is common in pets with PLN, and this often goes undiagnosed. Hypoalbuminemia develops once significant amounts of albumin are lost in the urine, and can eventually lead to a reduced osmotic pressure in the vasculature, resulting in peripheral edema, ascites, and pleural effusion.
Loss of coagulation factors such as antithrombin-III result in a state of hypercoagulability, with some cases developing catastrophic thromboembolic events, most commonly in the lungs. The number of cases that develop asymptomatic thromboembolism is unknown; it is reported that 25% of dogs with PLN have thromboembolism, however this number may be lower than the true incidence. Many, but not all, late-stage cases will develop chronic renal insufficiency and then chronic renal failure.

A small percentage of dogs will develop nephrotic syndrome, defined as the concurrent presence of hypoalbuminemia, proteinuria, hyperlipidemia, and fluid accumulation in interstitial spaces and/or body cavities. This term is becoming used less commonly, as many animals present with some of these criteria, but not all. In addition, the prognosis is also quite closely linked to the development of azotemia, which is not a component of the nephrotic syndrome.

Clinical Signs and Diagnostic Testing

Many pets with PLN do not have any symptoms at all. In fact, clinical signs do not usually develop until very late in the course of disease. This is typically seen when severe hypoalbuminemia results in peripheral edema and intracavitary fluid accumulation, such as ascites and/or pleural effusion. In some cases, the disease is detected when thromboembolism occurs. In some cases, only edema is noted, and pets have no other clinical signs. Other cases will present late in the course of disease with poor appetite, weight loss, poor hair coat and lethargy. Once the ability to concentrate urine is lost, then polyuria and polydipsia are noted. However, animals can present with severe proteinuria, intracavitary fluid accumulation, and loss of appetite without renal failure. Once moderate to severe azotemia develops, pets may develop vomiting, dehydration and a uremic odour along with polyuria and polydipsia.

For cases where proteinuria is noted on a routine urinalysis or on a microalbuminuria test, a urine protein:creatinine (UPC) ratio should be performed. This should not be performed on a urine sample with active sediment or bacterium present, although these conditions will not result in severe proteinuria. As recommended by the IRIS (http://www.iris-kidney.com), a UPC of $> 0.2$ in urine with a quiet sediment is considered abnormal. The range of a UPC of $0.2-0.5$ is considered borderline proteinuric in dogs. Above these ranges, a patient is considered to have proteinuria. Previously, there were recommendations made for the requirement to repeat the UPC several times over a period of weeks to ensure persistent proteinuria prior to recommending additional diagnostic testing and therapy. However, this does depend on the presentation and degree of proteinuria. In an at risk breed with a severe elevation in UPC in a urine sample with no active sediment, then repeated measurement of the UPC is less necessary or may be considered unnecessary. Ideally, a urine culture should be performed in every case to rule out urinary tract infection.

Diagnostic work up should include a thorough history, including travel history, medication administration, and the possibility of exposure to infectious diseases and ticks. A thorough physical examination, complete blood cell count, biochemical profile and blood pressure measurement should be performed. Infectious disease screening appropriate for the geography should be performed. For dogs with no travel history outside of Ontario, a 4Dx SNAP test is reasonable, along with leptospirosis testing appropriate for the specific animal. In a dog with a positive qualitative C6 Lyme test (ie 4Dx SNAP test), it is recommended to obtain a quantitative C6 titre to be able to follow reduction in titre with treatment. Recent recommendations state that an abdominal ultrasound and thoracic radiographs should be performed in all cases with a UPC $>3.5$, progressive proteinuria, hypertension, hypoalbuminemia, and/or azotemia. Testing for
hyperadrenocorticism is warranted in cases with appropriate clinical signs, physical examination findings, and ultrasound findings.

**Treatment – Primary Protein-Losing Nephropathy**

The standard therapy for primary (idiopathic) PLN is use of an angiotensin converting enzyme inhibitor (ACEi). An ACEi may reduce the degree of proteinuria, and may also address the hypertension. The goal of this therapy is to maintain a UPC of <0.5, or reduce the UPC by at least 50%. In many cases, this will not be attained, but stabilization of the increase of UPC may occur. Close monitoring of renal parameters is important while using this medication, especially if significant azotemia is present as an ACEi can exacerbate end stage renal failure. If use of an ACEi does not resolve the hypertension, then another anti-hypertensive agent can be used. Amlodipine, a calcium channel blocker, is typically used.

Angiotensin-receptor blockers (ARB) are a type of medication recently labelled for use in cats with chronic renal disease and proteinuria, and have a theoretical benefit over ACEi in PLN. In cats with chronic kidney disease, a recent study revealed that telmisartan resulted in an improvement in UPC whereas benazepril did not. There is currently little data on the use of telmisartan in dogs. However, there is a case report of the use of telmisartan in a dog with refractory PLN, and it could be considered in cases of severe or refractory PLN. Concurrent use of ACEi and ARB are not recommended due to the potential for severe hyperkalemia, and a finding in humans that there was an increased risk of kidney failure and death. However, as more data becomes available for the use of ARB in dogs with PLN, these recommendations may change.

The benefit of immunosuppressive therapy in PLN is not completely understood. Immune complex-based (ICGN) forms of acquired PLN may benefit from immunosuppressive therapy. Drugs that have been suggested are glucocorticoids, mycophenolate, cyclosporine, azathioprine, chlorambucil, and cyclophosphamide, although no clinical trials are available to guide clinicians in the choice. In cases without a renal biopsy, the recommendation is to consider the use of immunosuppressive therapy in cases that are resistant to traditional therapy, and have progressive azotemia, severe hypoalbuminemia, or moderate to severe increase in creatinine. Patients will require very close monitoring if immunosuppressive therapy is used.

Dietary therapy plays an important role in management of PLN, and also chronic kidney disease if present. The benefit of a diet consisting of a high quality protein in reduced amounts has been proven, along with a degree of salt restriction. Renal diets also have restricted phosphorus levels, and are supplemented with appropriate fatty acids and often anti-oxidants. In order to reduce the risk of thromboembolic disease, use of an anti-thrombotic agent is often recommended. Low dose aspirin or clopidogrel (Plavix) can be used. Doxycycline should be used to treat cases diagnosed with Lyme disease.

Monitoring is recommended in all cases, and the frequency of rechecking blood pressure, renal parameters and UPC will be case dependent. The prognosis is also quite variable, as some cases can be managed long term, while others progress rapidly to end stage renal failure and/or severe hypoalbuminemia.

References available from the author on request.
SURGICAL MANAGEMENT OF CANINE CALCULI
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If you would like a copy of the illustrated version of these notes on CD and a video of this surgical procedure on DVD go to www.videovet.org.

Key Points
• Patients with urethral calculi present with stranguria
• Retropulsion of urethral calculi into the urinary bladder simplifies management of urethral calculi
• Aggressive lavage of the urethra and bladder should be performed during cystotomy
• Permanent urethrostomy is an acceptable method of managing chronic stone formers

Definition: Cystic and urethral calculi have various compositions (i.e., oxalate, struvite, urate, uric acid, cystine, silicate) and may be present in the urinary bladder or lodged in the urethra, respectively. They may be multiple or single, may cause partial or complete obstruction (i.e., urethral), and may require surgical manipulation for removal.

Synonyms: Bladder stones, urethral stones

Diagnosis
Clinical presentation:
  Signalment: There is no age predisposition. Dalmations are more likely to present with uric acid calculi and commonly present with calculi lodged in the urethra. Schnauzers are more likely to present with struvite calculi and Daschunds are more likely to present with cystine stones.
  History: Patients generally present with a history of urinary obstruction and/or signs of urinary tract infection. Common complaints include difficulty urinating, straining to urinate, hematuria, dripping blood tinged urine from the prepuce, and/or a distended abdomen. Patients that present several days after complete obstruction may have a distended and painful abdomen and a history of anuria. These patients may be so compromised that they present in shock.
  Clinical signs: The most frequently reported clinical signs in patients with cystic and urethral calculi include unproductive straining to urinate, blood tinged urine dripping from the prepuce, hematuria, and/or polakiuria. Severity of clinical signs may vary with the degree of urethral obstruction and duration of obstruction prior to presentation. Patients with complete obstruction for several days may show signs of post-renal azotemia (i.e., severe depression, recumbant, shocky).

Physical examination: Observation in the examination room may reveal multiple unsuccessful attempts to urinate. Abdominal palpation may reveal a full urinary bladder; occasionally, calculi within the bladder may be palpable.
  Patients with severe clinical signs (i.e., presented several days after complete obstruction) may show azotemia, shock, and/or severe depression. Abdominal
palpation generally reveals a large, turgid urinary bladder and may result in discomfort to the patient.

**Laboratory findings:** Results of a complete blood count and serum chemistry profile are generally normal in patients presenting acutely; urinalysis may show evidence of urinary tract infection and and/or crystalluria.

Patients presenting after several days of complete obstruction may have significant changes in their biochemical profile including increased BUN, increased creatine, metabolic acidosis, and severe electrolyte abnormalities. Urine is generally grossly hemorrhagic and urinalysis may show signs of urinary tract infection and crystaluria.

**Radiography:** Survey radiographs may show presence of radiodense calculi in the urethra and/or urinary bladder as well as a distended urinary bladder. Occasionally, radiolucent calculi occur and can only be visualized using retrograde contrast cystourethrography. The most common location of urethral calculi in male dogs is immediately caudal to the os penis. Careful evaluation of the kidneys and ureters should be done to rule out renal and ureteral calculi.

**Ultrasound:** Ultrasonographic examination of the bladder, ureters, and kidneys may be helpful in diagnosis of cystic, ureteral, or renal calculi.

**Differential diagnosis:** Any disorder causing urinary obstruction, including urethral neoplasia, granulomatous urethritis, urethral stricture, and urethral trauma. Definitive diagnosis is based on clinical signs, inability to pass a catheter, and evidence of calculi on survey or contrast radiographs.

**Medical management:** Immediate care: In animals with complete obstruction of a duration long enough to cause azotemia, temporary urinary diversion is provided by either passing a small urinary catheter (e.g., 5 French) alongside the calculus, performing a prepubic cystostomy (see technique described below), or frequent cystocentesis (i.e, tid to qid). Azotemia is treated with crystalloid IV therapy prior to calculus removal.

**Retrograde hydropulsion:** See the DVD for a detailed video of this technique. This technique should result in a 90-95% success rate of retropulsing urethral calculi into the urinary bladder!

**Technique**
1. Select the largest diameter sterile high density polypropylene urinary catheter that will fit past your patients os penis (generally 6, 8, or 10 French diameter)

2. If the selected catheter turns out to be a 6 French diameter then mix 30cc of Sterile KY Jelly with 70cc of sterile physiologic saline solution.

3. If the selected catheter turns out to be an 8 or 10 French diameter then mix 40cc of Sterile KY Jelly with 60cc of sterile physiologic saline solution.
4. Thoroughly mix the sterile saline and KY Jelly in a 35 or 60 cc syringe and attach the syringe to the urinary catheter.

5. Anesthetize the animal, extrude the penis and pass the lubricated urinary catheter in the urethra up to and against the calculus. Place a dry gauze sponge around the extruded tip of the penis and occlude the penis around the catheter by squeezing it with thumb and finger.

6. Using a back and forth action on the catheter, simultaneously inject the saline/lubricant mix under extreme pressure. Be certain the catheter tip hits the calculus like a battering ram to help dislodge it and encourage the saline-lubricant mix to surround the calculus and coat the urethral wall. During injection the calculi and urethra are lubricated by the saline/lubricant mix while the viscosity of the mixture (i.e., KY jelly and saline) encourages the calculus to dislodge and become retropulsed into the urinary bladder.

This technique is successful regardless of how many stones are in the urethra and no matter where the calculi are lodged.

If the above technique fails, place a finger in the rectum, palpate the urethra and occlude its lumen (this dilates the urethra); repeat the above maneuvers and when maximum pressure is exerted on the urethra by the saline/lubricant mix (i.e., the urethral is maximally dilated), suddenly release digital urethral occlusion allowing lodged calculi to flush into the urinary bladder.

**Surgical treatment:** The objective of surgical treatment is to remove all retropulsed calculi from the urinary bladder and any remaining urethral calculi that were unable to be retropulsed. Bladder calculi are removed via cystotomy, urethral calculi are removed via urethrotomy, and patients that are frequent stone formers may benefit from a permanent urethrostomy to allow continual passage of small urethral calculi.

**Preoperative management:** Patients that present acutely can be anesthetized immediately and retropulsion attempted (see above described technique). If urinary tract infection is suspected, preoperative treatment with antibiotics may be instituted.

Patients that present after several days of complete obstruction should be treated medically until the azotemia resolves, blood gas abnormalities resolve, and electrolytes return to normal. The patients electrocardiogram should be monitored if hyperkalemia is present preoperatively. Medical treatment may consist of intravenous fluids, systemic antibiotics, continuous ECG monitoring, and bladder decompression. Bladder decompression may be accomplished via passing a small gauge urinary catheter (e.g., 5 French) past the calculus, multiple cystocentesis (i.e., tid or qid), or placement of a antepubic cystostomy tube (described in detail below).

**Anesthesia:** Routine general anesthesia is performed in patients that present acutely without signs of azotemia.

Azotemic, shocky patients with moderate to severe biochemical abnormalities should be treated as described above until these abnormalities return to normal.
**Surgical anatomy:** The male canine penile urethra consists of urethral mucosa (i.e., urothelium) surrounded by corpus cavernosum urethra, which is in turn surrounded by tunica albuginea. Because of the fluid filled corpus cavernosum urethra (blood) and the tough fibrous connective tissue tunica albuginea, the urethra can withstand tremendous pressure (e.g., as with aggressive retropulsion) without the fear of urethral rupture.

The urinary bladder consists of the following layers; serosa, muscular, submucosa and mucosa. The bladder is lined with transitional epithelium.

**Positioning:** Patients are positioned in dorsal recumbancy for retropulsion, urethrotomy, urethrostomy, cystostomy tube placement and cystotomy.

**Surgical technique:** The surgical techniques vary depending upon the procedure chosen, and are described in detail below:

**Retropulsion:** The technique for retropulsion of urethral calculi is described above in medical management.

**Percutaneous cystostomy tube placement:** Occasionally, it may be necessary to perform a percutaneous antepubic cystostomy to decompress the urinary bladder whilst treating a severely azotemic patient until they become a better anesthetic and surgical risk.

The patient is sedated and placed in dorsal recumbancy. A 3-4cm incision is centered between the umbilicus and pubis. Subcutaneous tissues are dissected to expose the ventral midline (i.e., linea alba). A 2-3cm incision is made in the linea alba and the bladder wall located. A 12–14 French Foley catheter is advanced through a skin incision 2-3 cm lateral to the abdominal incision, tunneled in the subcutaneous tissue and brought into the abdominal cavity at a location just lateral to the midline abdominal incision. A pursestring suture is placed in the bladder wall at the proposed site of Foley catheter placement with 3-0 monofilament absorbable suture. A 1cm incision is made into the bladder lumen and the Foley catheter advanced. The pursestring suture is carefully tightened to create a water-tight seal, but not to tight as to create bladder wall necrosis. The bladder wall is pexied to the abdominal wall at the point of entry of the Foley catheter with 3-0 monofilament absorbable suture in a simple interrupted pattern. The abdominal wall is closed in a routine fashion.

The cystostomy catheter is held in place with a Chinese finger trap friction suture technique using #1 monofilament nonabsorbable suture and attached to a closed collection system to avoid urinary tract infection. The cystostomy tube remains in place until the patient is ready for definitive surgical treatment.

**Urethrotomy:** See DVD for detailed video of this technique.

The urethral calculus to be removed is located by evaluation of radiographs, palpation of the os penis and its relationship to the calculus, and/or passing a catheter in the urethra until it contacts the stone, removing the catheter and using it as a measure to locate the calculus.
A 2–3 cm skin incision is made directly over the calculus. Subcutaneous tissues are dissected until the retractor penis muscle is exposed. The cream colored retractor penis muscle (smooth muscle) is dissected off the corpus cavernosum penis (the corpus cavernosum penis has a bluish tint from venous blood) and retracted laterally. A sharpe #15 BP scalpel blade is used to incise the urethra directly over the calculus being careful to incise the urethra directly on its midline to help decrease cavernous sinus bleeding. No attempt is made to control cavernous sinus hemorrhage with cautery or hemostats as this creates excessive urethral trauma and is generally unsuccessful at controlling hemorrhage. Rather, hemorrhage is controlled via digital pressure and suction until suturing can commence. The calculus is grasped with forceps and removed from the urethra.

The urethral incision can be left open to heal by second intention; if this method is chosen moderate to severe hemorrhage can be expected for several days postoperatively.

Alternately, the urethral incision can be closed using 5-0 multifilament or monofilament absorbable suture in a simple interrupted or continuous pattern. Subcutaneous tissues are closed with 3-0 monofilament absorbable suture in a simple continuous pattern and skin with 3-0 or 4-0 nonabsorbable monofilament suture. If this method is preferred by the author over healing by second intention as postoperative hemorrhage is significantly reduced.

Both urethrotomy techniques (i.e., sutureless or sutured) result in predictible urethral healing without evidence of urethral stenosis or stricture.

Urethrostomy: See DVD for detailed video of this technique.
Urethrostomy is generally performed in patients that are recurrent stone formers. It provides a permanent opening caudal to the os penis that is large enough to accommodate passage of most urethral calculi. This technique is often performed in Dalmations for treatment of recurrent uric acid calculi.

Scrotal urethrostomy is the location of choice for urethrostomy in dogs. It is a convienient location for surgical manipulation, this area of the urethra generally bleeds the least, the urethral diameter will accommodate passage of most urethral calculi, and there is less urine scald postoperatively. Other locations for urethrostomy include prescrotal and perineal.

Prior to surgery a urethral catheter (the largest size that will fit past the os penis) is passed, if possible. After a routine castration and scrotal ablation have been performed, the subcutaneous tissues are dissected to expose the retractor penis muscle. The retractor penis muscle is smooth muscle and appears light grey to cream colored. The retractor penis muscle is dissected from its attachment to the corpus cavernosum urethra. The blood filled cavernous tissue gives the urethra a bluish color. The urethral catheter is palpated and used as a firm surface to cut against when incising the urethra. Every attempt is made to incise the urethra exactly on the midline to help decrease hemorrhage. A 3–4 cm incision is made in the urethra. The caudal
aspect of the urethral incision is positioned directly over the ishial arch. As this is the new point of urine flow it is most efficent to have urine exit before it makes a sharp turn ventrally. No attempt is made to control cavernous tissue hemorrhage with cautery or hemostatic forceps; only pressure, suction, and suture pressure should be used.

After incision of the urethra, the glistening urethral mucosa is identified, 4-0 or 5-0 nonabsorbable monofilament suture with a swaged on cutting or taper-cut needle is recommended by the author to suture urethral mucosa to skin. The first urethrostomy suture is placed at the midpoint of either side of the urethral incision to include urethral mucosa, tunica albuginea, and skin (suture split thickness of skin). The suture is tied leaving the end without the needle 3-4 cm long to act as a stay suture. The second suture is placed directly across from the first suture and tied as described for the first. The urinary catheter can now be removed. After the first two sutures are placed, the needle end of one suture is used to begin suturing the cranial portion of the urethrostomy using a simple continuous suture pattern. When the opposite suture is encountered, the stay suture is used to tie off the first continuous suture line. The opposite suture is then used to suture the caudal portion of the urethrostomy in a similar fashion tying the final suture to the remaining stay suture. Fine ophthalmic instruments make tissue handling and suturing easier. Use of a magnifying loupe (about 2x) and head lamp light source enhances visualization of the urethral mucosa and facilitates accurate suturing. It is critical that the surgeon recognize glistening urethral mucosa and suture it to skin. This will decrease (or eliminate) the chance of urethral stricture. It has been shown that a continuous suture pattern incorporating the urethral mucosa and tunica albuginea (i.e., squeezes the cavernous tissue) results in less postoperative hemorrhage.

Cystotomy: See DVD for detailed video of this technique. After successful retropulsion of urethral calculi into the bladder, the catheter used to retropulse calculi is passed into the urethra and bladder and left in place. A portion of the catheter can be left exiting the penis. Leaving a catheter indwelled in the urethra ensures that remaining cystic calculi will not roll back into the urethra during patient transfer to the surgery suite and during patient prep.

Just prior to aseptic preparation of the abdomen a soft, 10-12 French red rubber catheter or feeding tube is placed into the prepuce and a prepucal lavage is performed with 180cc of a 1:50 dilution of saline and 1% betadine solution. This aseptically prepares the penis and prepuce so they can remain in the surgical field throughout the cystotomy procedure. In female patients the vulva and vaginal vault are similarly aseptically prepared.

A paraperpucial incision is made from just caudal to the umbilicus to pubis. The prepuce is retracted and a midline celiotomy is performed. The bladder is exteriorized and examined. Stay sutures of 3-0 suture are placed in the apex and neck of the bladder. A scalpel blade is used to penetrate the ventral aspect of the bladder and enter the lumen. The ventral cystotomy incision is extended with metzenbaum scissors. The bladder should be opened from apex to neck to allow proper visualization of bladder mucosa and calculi. Stay sutures are placed on each side of
the incision at its midpoint to facilitate visualization of the bladder interior. Large hemostats are placed on the stay sutures to help retract the bladder margins to maintain visualization of the bladder interior. A cystotomy spoon is used to scoop the bladder neck for calculi. This is performed several times. When no more calculi can be removed with the spoon, digital palpation of the bladder neck is performed to identify presence of further calculi. If further calculi are palpated further attempts are made to retrieve the calculi. Once no more calculi can be spooned or palpated the previously placed indwelling urethral catheter is removed.

Next, the largest urinary catheter or feeding tube that can be passed through the os penis is passed in the penile urethra to the level of the os penis (i.e., retrograde). A dry sponge is used to grasp the extruded penis to create a water tight seal around the catheter. A 60cc syringe filled with sterile saline is injected through the catheter under moderate pressure. The stay sutures on the bladder incision are retracted to enable visualization of the bladder lumen during lavage. Suction or intermittent spooning is performed during lavage in an attempt to identify and remove any remaining stones. After several lavages and negative results in obtaining stones, the catheter is placed from the bladder to the bladder neck and pelvic urethra (i.e., normograde). Lavage is once again performed in an attempt to identify and remove any remaining stones. After several lavages and negative results the catheter is advanced until it can be seen coming out of the penile urethra. The catheter is run back and forth in the urethra several times to ensure that there are no remaining calculi (i.e., gritty feeling while passing the catheter).

Finally, a piece of bladder mucosa is excised from the cut edge of the cystotomy incision for culture and susceptibility testing. The interior of the bladder is examined for urachael diverticulm, masses, etc. and biopsied as necessary. The bladder wall is closed with 3-0 or 4-0 absorbable monofilament suture material using a swaged on taper or taper-cut needle in a simple continuous or simple interrupted appositional suture pattern. Only one layer closure is necessary. Abdominal closure is routine.

**Suture material/special instruments:**
Urinary catheters of various sizes, Foley catheter, head lamp light source, 2X loupes, ophthalmic instruments, 4-0 and 5-0 monofilament absorbable suture material.

**Postoperative care and assessment:**
Postoperative care varies depending upon procedure performed:

**Percutaneous cystostomy tube:** It is important to keep the percutaneous cystostomy tube attached to a closed collection device. The tube can be connected to a sterile collection bag via a sterile intravenous catheter connection set. An elizabethan collar may be necessary in some patients to prevent iatrogenic removal of the cystostomy catheter. Careful management is important to control catheter related urinary tract infection.

**Sutureless Urethrotomy:** If urethrotomy without suturing is performed, patients must be monitored for blood loss from the urethrostomy site. Blood loss can be severe
enough to lower the PCV by 2 – 3%. An Elizabethan collar may be necessary in some patients to prevent self-mutilation. Patients should be kept quiet and away from other animals (especially bitches in heat!). Tranquilization is occasionally necessary to control hyperactive or overly excitable patients. Clients should be warned that drops of blood may be present from the urethrotomy site as long as 2 weeks postoperatively.

Sutured Urethrotomy: If a sutured urethrotomy is performed, patients will exhibit very little blood loss. However, an Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals (especially bitches in heat!). Tranquilization is occasionally necessary to control hyperactive or overly excitable patients.

Scrotal Urethrostomy: The most common postoperative complication of scrotal urethrostomy is bleeding from the urethrostomy site. Utilization of a simple continuous suture pattern incorporating the urethral mucosa and tunica albuginea (i.e., squeezing the cavernous tissue and creating a air-tight/water-tight seal) has significantly decreased the incidence of postoperative hemorrhage in the authors opinion. An Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals (especially bitches in heat!). Over excitement immediately postoperatively can result in frank hemorrhage or subcutaneous hemorrhage. Tranquilization is occasionally necessary to control hyperactive or overly excitable patients.

Cystotomy: An indwelling urethral catheter is not recommended after an uncomplicated cystotomy for removal of cystic calculi. An Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals.

Prognosis: The prognosis for surgical management of urethral and cystic calculi is dependant upon preoperative management of azotemic patients prior to anesthesia, success of retropulsion of urethral stones into the urinary bladder, care in removing all stones via cystotomy, and care of ensuring urethral mucosa to skin apposition during urethrostomy.

Patients that have successful retropulsion of urethral calculi and do not require urethotomy or urethrostomy have a excellent prognosis. If careful attention is paid during cystotomy to ensure that no calculi are left behind (see discussion on cystotomy technique), the prognosis for cure is excellent. Long term prognosis is dependant on evaluation of calculus composition, dietary management, management of urinary tract infection, and attention to urine pH.

Patients that require sutured or sutureless urethrotomy have a favorable prognosis if all of the remaining calculi are removed from the urinary bladder via cystotomy to ensure that no calculi are left behind (see discussion on cystotomy technique). Attention must be paid to careful lavage during cystotomy to ensure removal of all cystic calculi.
Patients that have an elective urethrostomy have a favorable prognosis if attention is paid to proper surgical technique (i.e., urethral mucosa is sutured to skin). Occasionally, chronic stone forming patients will form a calculus that is too large to pass through the urethrostomy stoma.
SURGICAL MANAGEMENT OF FELINE CALCULI
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Key Points
- Patients with cystic and urethral calculi present with stranguria
- Retropulsion of urethral calculi into the urinary bladder simplifies management of urethral calculi
- Aggressive lavage of the urethra and bladder should be performed during cystotomy
- Permanent urethrostomy is an acceptable method of managing chronic stone formers

If you would like a video of this surgical procedure on DVD go to www.videovet.org or contact videovet@me.com. You may click on the ‘Seminar Price’ for any DVD you would like to purchase.

Definition: Cystic and urethral calculi have various compositions (i.e., oxalate, struvite, urate) and may be present in the urinary bladder or lodged in the urethra, respectively. They may be multiple or single, may cause partial or complete obstruction (i.e., urethral), and may require surgical manipulation for removal.

DIAGNOSIS
Clinical presentation:
- Signalment: There is no age, sex or breed predisposition.
- History: Patients generally present with a history of urinary obstruction and/or signs of urinary tract infection. Common complaints include difficulty urinating, straining to urinate, hematuria, blood tinged urine in the litter pan, and/or a distended abdomen. Patients that present several days after complete obstruction may have a distended and painful abdomen and a history of anuria. These patients may be so compromised that they present in shock.
- Clinical signs: The most frequently reported clinical signs in patients with cystic and urethral calculi include unproductive straining to urinate, blood tinged urine seen in the litter pan, hematuria, and/or polakiuria. Severity of clinical signs may vary with the degree of urethral obstruction and duration of obstruction prior to presentation. Patients with complete obstruction for several days may show signs of post-renal azotemia (i.e., severe depression, recumbant, shocky).
- Physical examination: Abdominal palpation may reveal a full urinary bladder; occasionally, calculi within the bladder may be palpable. Patients with severe clinical signs (i.e., presented several days after complete obstruction) may show azotemia, shock, and/or severe depression. Abdominal palpation generally reveals a large, turgid urinary bladder and may result in discomfort to the patient.
- Laboratory findings: Results of a complete blood count and serum chemistry profile are generally normal in patients presenting acutely; urinalysis may show evidence of urinary tract infection and and/or crystalluria. Patients presenting after several days of complete obstruction may have significant changes in their
biochemical profile including increased BUN, increased creatinine, metabolic acidosis, and severe electrolyte abnormalities. Urine is generally grossly hemorrhagic and urinalysis may show signs of urinary tract infection and crystaluria.

**Radiography:** Survey radiographs may show presence of radiodense calculi in the urethra and/or urinary bladder as well as a distended urinary bladder. Occasionally, radiolucent calculi occur and can only be visualized using retrograde contrast cystourethrography. Careful radiographic evaluation of the kidneys and ureters should be done to rule out renal and ureteral calculi.

**Ultrasoundographic examination** of the bladder, ureters, and kidneys may be helpful in diagnosis of cystic, ureteral, or renal calculi.

**Differential diagnosis:** Any disorder causing urinary obstruction, including urethral neoplasia, granulomatous urethritis, urethral stricture, and urethral trauma. Definitive diagnosis is based on clinical signs, inability to pass a catheter, and evidence of calculi on survey or contrast radiographs.

**MEDICAL MANAGEMENT:**

**Immediate care:** In animals with complete obstruction long enough to cause azotemia, temporary urinary diversion is provided by performing a prepubic cystostomy (see technique described below) or frequent cystocentesis (i.e, tid to qid). Azotemia is treated with crystalloid IV therapy prior to calculus removal.

**Urethral catheterization of a female cat:**

See the DVD for a detailed video description of this technique (www.videovet.org).

- Female urethral catheterization is easier than male
- Use a closed ended tom cat catheter
- Ventral recumbancy is recommended
- Pass the catheter with no evidence of resistance

**Urethral Catheterization – Female**

**Indications:** Urethral catheterization is indicated in patients with urethral calculi (aids in retropulsion), measuring urinary output, chronic decompression of the urinary bladder, performing contrast cystography and preoperative placement to prevent cystic calculi from lodging in the urethra during cystotomy.

**Applied Anatomy:** The urethra leaves the bladder at the neck and courses caudally. The female urethra is short, straight, and wide, passing directly to the vestibule. Urinary catheterization of female cats is relatively easy because of the anatomic characteristics mentioned above.

**Anesthesia:** Heavy sedation or preferably, general anesthesia, is recommended for predictably successful catheterization of the female urethra. Occasionally, unsedated, unanesthetized cats will tolerate the procedure if they are slightly depressed.

**Technique:**

- **Positioning:** The cat is placed in either lateral recumbency or ventral recumbency with the hindquarters elevated on a rolled fleece. Regardless of position chosen, it is important to maintain positional symmetry during the procedure. This author prefers ventral recumbency. The patient is placed on the rolled fleece with the
hind legs hanging over the fleece, abducted slightly, and the tail held or tied directly over the back.

**Patient preparation:** The long hairs around the vulva can be clipped to enhance visualization of the vulvar lips. Alcohol preparation of the vulvar lips is performed prior to catheterization. The vaginal vault can be lavaged with a 1:50 dilution of 1% betadine solution and saline.

**Catheters:** A closed ended polyethylene tomcat catheter or a 3-1/2 French diameter feeding tube is recommended for urethral catheterization of female cats. Open-ended tomcat catheters may be used but may be more traumatic to the urethra during placement.

**Catheter placement:** The catheter is removed from the sterile packaging taking special care to maintain sterility during placement. Sterile K-Y jelly lubricant is generously placed on the tip and shaft of the catheter. Closed ended polyethylene tomcat catheters have a gentle curve when they are removed from their original sterile package. This curve is used to help ‘aim’ the catheter into the urethral papilla during placement.

With the catheter in the right hand, use the left index and middle finger to gently spread the vulvar lips. With the curve of the catheter pointing toward the floor, pass the tip of the catheter along the ventral midline of the vaginal vault and vestibule, taking care not to allow the catheter tip to enter the clitorin fossa. Gently pass the catheter in a cranial direction until the catheter can be felt to ‘fall’ into the urethral papilla. If any resistance is met during attempted placement, pull the catheter caudally into the vaginal vault, re-direct the catheter to the ventral midline of the vagina and re-insert the catheter. Once the catheter is felt to ‘fall’ into the urethra, pass the catheter into the urinary bladder until urine begins to drip from the catheter, ensuring proper placement.

**Securing the catheter:** If the catheter is to be maintained for an extended period of time select a soft 3.5 French diameter catheter and secure it to the vulva using a Chinese finger-trap friction suture technique. Attach the catheter to a closed collection device to maintain asepsis.

**Catheter removal:** Cut the Chinese finger-trap friction suture and gently pull the catheter. Hematuria may be seen for 12 – 24 hours after catheter removal.

**RETROGRADE HYDROPULSION OF LODGED URETHRAL CALCULI**

**Calcium removal:** Retrograde hydropulsion: See the DVD for a detailed description of this technique. This technique should result in an 80-85% success rate for retropulsing urethral calculi into the urinary bladder!

Thoroughly mix 20 cc of sterile saline and 5 cc of Surgilube or K-Y Jelly in a 35 cc syringe and attach the syringe to a 3.5 - 5.0 French soft rubber catheter/feeding tube.

Anesthetize the animal, extrude the penis and pass the lubricated urinary catheter in the urethra up to and against the calculus. Place a dry gauze sponge around the extruded tip of the penis and occlude the penis around the catheter by squeezing it with thumb and finger.
Using a back and forth action on the catheter, simultaneously inject the saline/lubricant mix under extreme pressure.

a) During injection, the calculi and urethra are lubricated by the saline/lubricant mix while the viscosity of the mixture (i.e., KY jelly and saline) encourages the calculus to dislodge and become retropulsed into the urinary bladder.

b) This technique is attempted, and generally successful, regardless of how many stones are in the urethra and no matter where they are lodged.

If the above technique fails, use a stiffer catheter (i.e., open or closed ended tomcat catheter) and repeat the above maneuvers. Use care when manipulating these stiffer catheters against the calculus.

SURGICAL TREATMENT:

The objective of surgical treatment is to remove all retropulsed calculi from the urinary bladder and any remaining urethral calculi that were unable to be retropulsed. Bladder calculi are removed via cystotomy, urethral calculi are removed via urethrotomy, and patients that are frequent stone formers may benefit form a permanent urethrostomy to allow continual passage of small urethral calculi.

Preoperative management: Patients that present acutely can be anesthetized immediately and retropulsion attempted (see above described technique). If urinary tract infection is suspected, preoperative treatment with antibiotics may be instituted.

Patients that present after several days of complete obstruction should be treated medically until the azotemia resolves, blood gas abnormalities resolve, and electrolytes return to normal. The patients’ electrocardiogram should be monitored if hyperkalemia is present preoperatively. Medical treatment may consist of intravenous fluids, systemic antibiotics, continuous ECG monitoring, and bladder decompression. Bladder decompression may be accomplished via multiple cystocentesis (i.e., tid or qid), or placement of an antepubic cystostomy tube (described in detail below).

Anesthesia: Routine general anesthesia is performed in patients that present acutely without signs of azotemia. Azotemic, shocky patients with moderate to severe biochemical abnormalities should be treated as described above until these abnormalities return to normal.

Surgical anatomy: The male feline penile urethra consists of urethral mucosa (i.e., urothelium) surrounded by corpus cavernosum urethra, which is in turn surrounded by tunica albuginea. Because of the blood filled corpus cavernosum urethra and the tough fibrous connective tissue tunica albuginea, the urethra can withstand tremendous pressure (e.g., as with aggressive retropulsion) without the fear of urethral rupture.

The urinary bladder consists of the following layers; serosa, muscular, submucosa and mucosa. The bladder is lined with transitional epithelium.

Positioning: Patients are positioned in dorsal recumbancy for retropulsion, cystostomy tube placement and routine cystotomy.

Surgical technique: The surgical technique varies depending upon the procedure chosen and are described in detail below:

Retropulsion: The technique for retropulsion of urethral calculi is described above in medical management.
**Percutaneous cystostomy tube placement:** Occasionally, it may be necessary to perform a percutaneous antepubic cystostomy to decompress the urinary bladder whilst treating a severely azotemic and metabolically derranged patient until they become a better anesthetic and surgical risk.

**Surgical technique:** The patient is sedated and placed in dorsal recumbancy. A 3-4cm incision is centered between the umbilicus and pubis. Subcutaneous tissues are dissected to expose the ventral midline (i.e., linea alba). A 2-3 cm incision is made in the linea alba and the bladder wall located. A 12–14 French Foley catheter is advanced through a skin incision 2-3 cm lateral to the abdominal incision, tunneled in the subcutaneous tissue and brought into the abdominal cavity at a location just lateral to the midline abdominal incision. A purse string suture is placed in the bladder wall at the proposed site of Foley catheter placement with 3-0 monofilament absorbable suture. A 1cm incision is made into the bladder lumen and the Foley catheter advanced. The purse string suture is carefully tightened to create a water tight seal but not too tight as to create bladder wall necrosis. The bladder wall is pexied to the abdominal wall at the point of entry of the Foley catheter with 3-0 monofilament absorbable suture in a simple interrupted pattern. The abdominal wall is closed in a routine fashion. The cystostomy catheter is held in place with a Chinese finger trap friction suture technique using #1 monofilament nonabsorbable suture and attached to a closed collection system to avoid urinary tract infection. The cystostomy tube remains in place until the patient is ready for definitive surgical treatment.

**Urethrostomy:** Urethrostomy is generally performed in patients that are recurrent stone formers. It provides a permanent opening that is large enough to accommodate passage of most urethral calculi, crystals and mucoid debris.

**Perineal urethrostomy; perineal approach:** is the location of choice for urethrostomy in cats. It is a convenient location for surgical manipulation, the urethral diameter will accommodate passage of most urethral calculi and there is less urine scald postoperatively.

See the DVD for a detailed video description of the surgical procedure. Prior to surgery a urethral catheter is passed, if possible. After a routine castration, an elliptical incision is made around the scrotum and penis. Then the subcutaneous tissues are dissected to expose penile urethra. The penile urethra is dissected free from surrounding connective tissue. The ventral attachment of the pelvic urethra to the pubis (i.e., ishiocavernosus m.) is identified and transected. The penile urethra is freed from its connective tissue attachments to the pelvic floor using blunt digital dissection.
The retractor penis muscle is identified on the dorsal aspect of the penis and is dissected from its attachment on the penis. The dissected retractor penis muscle is then used to develop the dorsal plane of dissection to separate the pelvic urethra from its dorsal connective tissue attachments. Once the urethra is dissected enough to visualize the dorsolaterally located bulbourethral glands penile dissection can stop. The penis is catheterized and the urethral orifice identified. An incision is made from the penile urethra to the pelvic urethral to the level of the bulbourethral glands using a Stevens tenotomy scissor or Iris scissor. The urethral orifice at the level of the bulbourethral glands is generally of large enough diameter to accept the flange of a tomcat catheter.

After incision of the urethra, the glistening urethral mucosa is identified. 5-0 nonabsorbable monofilament suture with a swaged on cutting or taper-cut needle is recommended by the author. The first urethrostomy suture is placed at the dorsal aspect of the urethrotomy incision on the right or left side at a 45o angle to include urethral mucosa and skin (suture split thickness of skin). The suture is tied and cut leaving the ends 3-4 cm long to act as a stay suture. A mosquito hemostate is placed on this suture to provide traction and countertraction to enhance visualization of the urethral mucosa. The second suture is placed opposite the first suture and tied as described for the first. A stay suture is also placed here. A third urethrostomy suture is placed directly on the dorsal midline to hold the dorsal margin of urethral mucosa to the dorsal margin of the skin incision. Alternating sutures from dorsal to ventral are placed until approximately one half of the penile urethra has been sutured to skin. The remainder of the penis is amputated and the subcutaneous tissue and skin are closed routinely.

Perineal urethrostomy; dorsal approach: See the DVD for a detailed video description of this surgical procedure. Perineal urethrostomy can be performed with the patient placed in dorsal recumbency. This positioning is more ergonomic for the surgeon and allows easy access of the urinary bladder for concurrent cystotomy. When positioning the cat on the operating table tie the hind limbs cranially until the pelvis is slightly elevated off the surgery table. Place a folded towel under the pelvis to support this slightly elevated position. The surgical technique is as described above for the perineal urethrostomy performed using a perineal approach.
**Cystotomy:** See the DVD for a detailed video description of this procedure. After successful retropulsion of urethral calculi into the bladder the catheter used to retropulse calculi is passed into the urethra and bladder and left in place. Leaving a catheter indwelled in the urethra ensures that remaining cystic calculi will not roll back into the urethra during patient transfer to the surgery suite and during patient prep. The patient is place in dorsal recumbancy with the hind legs tied gently cranially to slightly elevate the pelvis. A folded towel is placed under the pelvis to help support it in this position. This positioning will greatly facilitate exteriorizing the penis during surgery.

Just prior to aseptic preparation of the abdomen a soft, 5-8 French red rubber catheter or feeding tube is placed into the prepuce and a prepucal lavage is performed using 20 cc of a 1:50 dilution of 1% betadine solution and sterile saline. This aseptically prepares the penis and prepuce so they can remain in the surgical field throughout the cystotomy procedure.

A caudal midline incision is made from umbilicus to pubis. The bladder is exteriorized and examined. Stay sutures of 3-0 suture are placed in the apex and neck of the bladder. A scalpel blade is used to penetrate the ventral aspect of the bladder and enter the lumen. The ventral cystotomy incision is extended with Metzenbaum scissors. The bladder should be opened from apex to neck to allow proper visualization of bladder mucosa and easy retrieval of all calculi. Stay sutures are placed on each side of the incision at its midpoint to facilitate visualization of the bladder interior. Large hemostats are placed on the stay sutures to help retract the bladder margins. A cystotomy spoon is used to scoop the bladder neck for calculi. This is performed several times. When no more calculi can be removed with the spoon, digital palpation of the bladder neck is performed to identify presence of further calculi. If calculi are palpated further attempts are made to retrieve them. Once no more calculi can be spooned or palpated the indwelling urethral catheter placed after retropulsion is removed.

Next, a 3.5 - 5 French urethral catheter is placed in the penile urethra (i.e., retrograde). A dry sponge is used to grasp the extruded penis to create a water tight seal around the catheter. A 35cc syringe filled with sterile saline is injected through the catheter under moderate pressure. The stay sutures on the bladder incision are retracted to enable visualization of the bladder lumen during lavage. Suction or intermittent spooning is performed during lavage in an attempt to identify and remove any remaining stones. After several high pressure lavages and negative results in obtaining stones, the catheter is placed from the bladder lumen into the bladder neck and pelvic urethra (i.e., normograde). Lavage is once again performed in an attempt to identify and remove any remaining stones. After several lavages and negative results, the catheter is advanced until it can be seen coming out of the penile urethra. The catheter is run back and forth in the urethra several times (‘urogenital floss’) to ensure there are no remaining calculi (i.e., gritty feeling while passing the catheter).

Finally, a piece of bladder mucosa is excised from the cystotomy incision for culture and susceptibility testing. The interior of the bladder is examined for urachal diverticulum, masses, etc. and biopsied as necessary. The bladder wall is closed with 3-0 or 4-0 absorbable monofilament suture material using a swaged on taper or taper-
cut needle in a simple continuous or simple interrupted appositional suture pattern. Only one layer closure is necessary. Abdominal closure is routine.

**Suture material/special instruments:** Urinary catheters of various sizes, Foley catheter, head lamp light source, 2X loupes, ophthalmic instruments, 4-0 or 5-0 monofilament nonabsorbable suture material.

**POSTOPERATIVE CARE AND ASSESSMENT:**
Postoperative care varies depending upon procedure performed:

- Percutaneous cystostomy tube: It is important to keep the percutaneous cystostomy tube attached to a closed collection device. The tube can be connected to a sterile collection bag via a sterile intravenous catheter connection set. An elizabethan collar may be necessary in some patients to prevent iatrogenic removal of the cystostomy catheter. Careful management is important to control catheter related urinary tract infection.

- Cystotomy: An indwelling urethral catheter is not recommended after an uncomplicated cystotomy for removal of cystic calculi. An Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals.

- Perineal Urethrostomy: An Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals. An indwelling urinary catheter placed routinely postoperatively is NOT necessary following an uncomplicated urethrostomy.

**PROGNOSIS**

The prognosis for surgical management of urethral and cystic calculi is dependant upon preoperative management of azotemic patients prior to anesthesia, success of retropulsion of urethral stones into the urinary bladder, care in removing all stones via cystotomy, and care of ensuring urethral mucosa to skin apposition during urethrostomy.

Patients that have successful retropulsion of urethral calculi and do not require urethrostomy have a excellent prognosis. If careful attention is paid during cystotomy to ensure that no calculi are left behind (see discussion on cystotomy technique), the prognosis for cure is excellent. Long term prognosis is dependant on evaluation of calculus composition, dietary management, management of urinary tract infection, and attention to urine pH.

Patients that have an elective perineal urethrostomy have a favorable prognosis if attention is paid to proper surgical technique (i.e., urethral mucosa is sutured to skin). Occasionally, chronic stone forming patients will form a calculus that is to large to pass through the urethrostomy stoma.
CHRONIC VAGINITIS AND INCONTINENCE: CAN VULVARPLASTY HELP?
Perivulvar Pyoderma – Episioplasty (Vulvarplasty)

Any plastic operation upon the pubic region or vulva can be classified as an episioplasty. The term usually refers to a specific technique used in the treatment of perivulvar pyoderma. Perivulvar pyoderma occurs most frequently in obese, usually spayed bitches (Figure 1).

The objective of surgical treatment is to remove the redundant fold of skin and adipose tissue around the vulva which predispose the area to accumulation of moisture, irritation, and infection (tissue pinched in the surgeon's fingers).

The affected area is treated with cleansing and drying agents preoperatively to reduce inflammation.

Surgical Technique

The patient is placed in perineal position (Figure 2) and a purse string suture placed in the anus.

perivulvar dermatitis
Forceps are used to grasp the redundant fold of skin and retract it dorsally to help determine the amount of skin to be removed. Once this determination has been made, two inverted U shape incisions are made dorsal to the vulva (Figure 2).
The island of excised skin is resected along with any underlying adipose tissue (Figure 4).
The wound is closed in two layers; simple interrupted 2-0 or 3-0 synthetic absorbable suture (PDS, Maxon, Vicryl or Dexon) with a swaged on taper needle in the subcutaneous tissue and the skin with simple interrupted monofilament nonabsorbable suture (Nylon or Prolene) with a swaged on cutting needle.

The first suture is placed on the dorsal most point of the ventral incision and sutured to the dorsal most point of the dorsal incision Figure 3.
The remainder of sutures are place to complete the closure. Care is taken to limit puckering of the incision between sutures by gradually taking larger bites on the dorsal incision relative to bites closer together on the ventral incision.

Figure 5.
After care consists of an Elizabethan collar, anagesics, systemic antibiotics and local cleansing. The long term prognosis is excellent. Recurrence of the pyoderma is uncommon.
Wound Management Secrets
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Split-shot Wound Management
Key Points
- Skin has the ability to stretch when placed under mild tension
- Normal wound contraction often stops before wound edges appose.
- Split-shot wound management can be used to encourage skin edges to contract.

If you would like a copy of the illustrated version of these notes on CD and a video of this surgical procedure on DVD, go to www.ivseminars.com and click VideoVet or contact videovet@me.com.

Indications: Use of various appliances to create tension on the local skin of non-contracting open wounds is not new. Subcutaneously buried silastic balloons (i.e., skin expanders) injected every 24 hours with varying amounts of saline will stretch local skin and have been used extensively in human plastic and reconstructive surgery. Skin expanders have also been described for use in veterinary patients. Skin expansion may be indicated in wounds that have undergone normal wound contraction without successful wound margin apposition. The most common locations for inappropriate wound contraction in small animals are extremities, head, and tail.

Applied Anatomy: Skin is made up of several layers that collectively form a complex organ system. Skin is not capable of regeneration. One method of getting ‘more’ skin for wound coverage is encouraging local skin to undergo intussusceptive growth. This can be accomplished by applying tension to local skin around the wound. If tension is constant, skin layers will accommodate the increase tension by becoming thinner thus allowing the skin to ‘stretch’.

Anesthesia: Patients undergoing split-shot wound management should be placed under general anesthesia.

Technique: Positioning: Patients are positioned with the wounded area uppermost.

- **Patient preparation**: Wounds identified for split-shot wound management should be treated as an open wound until there is evidence of a healthy granulation tissue bed. Routine aseptic preparation of the local skin is performed.

- **Special instruments and suture**: Metallic split-shot (i.e., other than lead) can be purchased at any local sporting goods or fishing store. Split-shots are placed in a cold sterilization media for an appropriate time period and thoroughly rinsed prior to use. Monofilament non-absorbable suture with a swaged-on taper needle, size 00 to #1 depending upon location and size of wound is recommended. A sterile rubber bumper is fashioned from a feeding tube or catheter.
Split-shot technique: The wound and surrounding skin are prepared for aseptic surgery. Two bumpers are created by cutting one 1/2 inch piece off the flanged end of a 20 French feeding tube or catheter. This segment of tube is then split in two.

An appropriate size monofilament nonabsorbable suture is selected. The skin edges are gently undermined being careful not to trim the wound edge. The swaged-on needle is placed through the rubber bumper and enters the wound at the commissure. The wound edges are then sutured using a simple continuous pattern. Care is taken to engage the needle in the tough collagen laden subcutaneous tissue. Patients with thin subcutaneous tissue (i.e., cats, small dogs, areas of thin skin) may require penetration of skin instead of subcutaneous tissue. Once the entire length of the wound has been sutured, the suture is passed out through the skin of the remaining commissure of the wound. Knots are not tied in either end of the suture.

Gentle traction is placed on the exiting ends of the suture until mild tension is placed on the wound edges and local skin. A split-shot is placed on each end of the exiting suture against the bumper. The split-shot is then gently but firmly clamped against the suture; this maintains tension on the skin edges and local skin. The wound is bandaged, an Elizabethan collar placed, and the patient confined to a cage. Each day the bandage is removed, the ends of the suture gently pulled and a split-shot is placed between the bumper and the original split-shot. Daily tension is performed without the need for general anesthesia or sedation. Skin may be responsive to tension for 7 to 10 days. When the wound is closed to your satisfaction, the suture and bumpers are removed. The remaining wound is bandaged only if it requires further protection.

Tie-over Bandage Technique
If you would like a copy of the illustrated version of these notes on CD and a video of this surgical procedure on DVD, go to www.ivseminars.com and click VideoVet or contact videovet@me.com.

Key Points
• The most important aspects of wound management are debridement, debridement, debridement.
• The solution to pollution is dilution.
• A tie-over bandage can cover the most difficult to bandage wounds.
• A tie-over bandage can help 'stretch' local skin.

If you would like an instructive DVD of this topic, go to www.ivseminars.net and click on Video Vet.

WOUND MANAGEMENT: The area should be clipped and cleaned as soon as possible to provide a clean environment beneath the bandages that will eventually be applied. Sterile, water soluble gel placed on the wound is a convenient means of temporary wound protection. Dried blood and debris should be removed from the surrounding skin with antiseptic soap, using care to avoid contact between the soap and exposed tissues which can result in lipolysis and tissue damage. The primary goal of wound management is to decrease bacterial numbers and debris and enhance the animal's defense mechanisms (i.e., debridement). Gross particulate matter, hair, etc. should be
removed manually from the wound. Lavage is beneficial in further decreasing infection-promoting debris and bacteria. Saline is indisputably the ideal lavage solution, although dilute chlorhexidine (0.05 to 0.005%), or povidone-iodine (0.01%) may be used. The effectiveness of lavage is dependent upon volume and pressure. Studies have shown that high pressure (25-60 psi) is superior to low pressure (0.5-5.0 psi) when wounds are only lavaged one time. Medium pressure, which has also been shown to be beneficial can be generated using an 18 gauge needle and large syringe (35-60 ml). Surgical debridement of necrotic-appearing tissue and embedded foreign material limits nutrients for bacterial growth and enhances the animal's local defense mechanisms.

OPEN WOUND MANAGEMENT: Open wound management allows optimal drainage and daily inspection, debridement and lavage of tissues. Following surgical excision of necrotic tissue, etc., continued mechanical debridement can be performed using an adherent dressing (wet-to-dry, dry-to-dry, or wet-to-wet). Wide-mesh gauze sponges are ideal for adherent bandages. The type of dressing used depends on wound conditions. Wet-to-dry dressings can be used for wounds with necrotic tissue, foreign matter and viscous exudate. The wet dressing dilutes the exudate and allows absorption. As the dressing dries, necrotic tissues adhere to the gauze and are removed with the bandage. Dry-to-dry dressings have similar indications as wet-to-dry except without the presence of viscous exudate. Wet-to-wet dressings are indicated when viscous exudate is present without necrotic tissues. The contact adherent layer should be covered by an absorbent outer layer. Once necrotic tissues have been removed and granulation tissue begins to form, adherent gauze should be replaced with nonadherent pads (telfa).

SECOND INTENTION HEALING: Second intention healing occurs by formation of granulation tissue, wound contraction and epithelialization. The advantages of this process are drainage remains optimal, wound infections are rare and the time and expense of surgery is avoided. However, second intention healing may cause disfigurement or loss of function due to wound contracture, and the epithelium formed may be easily disrupted.

TIE-OVER BANDAGE: Indications: Large surface area wounds (i.e., abdomen, thorax, back, neck) or wounds in ‘difficult-to-bandage’ areas (i.e., tail, perineum, head, paraprepuccial,proximal extremeties) may not be amenable to routine bandaging techniques. These areas generally lend themselves nicely to placement of a tie-over-bandage.

Technique: The wound bed is prepared as described above. Several # 0 or #1 monofilament non-absorbable suture loops are placed in the skin on the periphery of the wound. Loop sutures are generally placed 360° around the wound and spaced 2 or 3 cm apart. Appropriate wound covering materials are placed in the wound bed (i.e., wet to dry, gauze, telfa, etc) and a sterile laparotomy pad placed on top to provide protection to the wound. Several lengths of 1/4 inch or 1/2 inch umbilical tape are passed through the loops of suture, over the laparotomy pad and through the suture loop on the opposite side of the wound. The umbilical tape passes over the wound mutiple times to
hold the laparotomy pad in place (an therefore the wound covering materials). Enough traction is placed on the suture loops to place mild tension on the skin edges of the healing wound. This bandage is easily removed and replaced for ease of bandage change.

Once the granulation bed is healthy and the wound is considered surgically clean it can be closed primarily (i.e., delayed primary closure). If there are small defects at the time of suture removal these can generally heal by second intention.
Key Points
- Pay attention to basic surgical principles
- Submucosa is the layer of strength
- Use synthetic absorbable suture materials
- Appositional techniques are best
- Identify breakdown via abdominal tap

General principles of small intestinal surgery
The small intestine is a very forgiving organ system when considering surgical intervention. It has an excellent blood supply with many collateral mesenteric vessels. If the blood supply can be preserved during surgical manipulations, small intestinal incisions can be expected to heal rapidly, gaining almost 70% of their original unwounded tensile strength in 14 - 21 days. It should be remembered that the small intestine is a low pressure conduit system that contains primarily liquid contents and has a relatively low bacterial content proximal to the ileocecal valve (compared to the colon). These properties make surgical manipulations of the small intestine predictably successful if basic principles of tissue handling, preservation of blood supply, and hemostasis are followed.

Principles of intestinal surgery include:
1) Incorporation of the collagen laden submucosal layer in the surgical closure.
2) Minimize trauma and contamination.
3) Maintain good blood supply to the surgical site.
4) Avoid tension across the suture line as this may increase the possibility of leak and/or breakdown.
5) Pay attention to your established criteria when suturing intestinal defects.

Preoperative preparation
Preoperative assessment should include a thorough historical and physical examination. This can often be helpful in localizing the problem to an upper versus lower GI obstruction, complete or partial obstruction, or strangulating versus nonstrangulating obstruction. Minimum data base should include complete blood count, electrolytes (i.e., sodium, potassium, chloride), glucose, and BUN. If available, blood gas analysis should be done on patients with severe vomiting, dehydration, or possible sepsis.

Criteria for Use of Prophylactic or Therapeutic Antibiotics
Prophylactic antibiotics should be considered in the following situations:
1. Old age (e.g., >7 years old), debilitated patient
2. Likely to open devitalized bowel
3. Estimated surgical time greater than 90 minutes
4. “Break” in aseptic surgical technique

Therapeutic antibiotics should be considered in the following situations:
1. Non GI associated infection that cannot be treated prior to surgery (e.g., severe dental disease, pyoderma)
2. Gross peritoneal contamination at surgery
3. Operation involving strangulation obstruction
4. Operation in patient with existing peritonitis (e.g., gun shot wound to abdomen with bowel perforation)
5. Reoperation of intestinal surgical breakdown with peritonitis

Operative Considerations
1) Proper “packing off” of the surgical field using moistened laparotomy pads should be performed around the exteriorized bowel to prevent accidental abdominal contamination from intestinal contents.
2) Keep abdominal contents warm and moist throughout surgery with a warm, balanced electrolyte solution.
3) Handling abdominal viscera should be kept to a minimum. Gentle manipulation of intestine with moistened gloves or stay sutures is helpful in preventing unnecessary tissue trauma. DeBakey forceps are the most atraumatic forceps for handling abdominal visceral organs.
4) The collagen laden, tough submucosa is the layer of strength in the small intestine; this layer must be incorporated into any small intestinal closure (i.e., enterotomy, anastomosis).
5) It may be difficult to visualize the submucosal layer due to mucosal eversion. Visualization of submucosa may be enhanced if everted mucosa is trimmed away.
6) Intestinal contents should be "milked" away from the anastomosis or enterotomy site. Intestinal clamps (e.g., Doyen intestinal clamps, Alice tissue forceps with a rubber feeding tube interposed, hair clips, or Penrose drains) may be used to prevent intestinal contents from contaminating the surgical site whilst manipulating intestine during anastomosis or enterotomy.
7) The enterotomy or anastomosis should be irrigated prior to its return to the abdominal cavity and instruments and gloves changed prior to abdominal closure.
8) Abdominal lavage with 2-3 liters of body temperature, sterile, physiologic saline solution should be accomplished prior to closure. The objectives of repeated abdominal lavage include dilution of bacteria and endotoxin and mechanical removal of fibrin and necrotic debris. The fluid of choice is body temperature, sterile, physiologic saline solution with no additives (i.e. betadine solution, chlorhexidine, antibiotics, etc). Lavage solution is poured into the abdominal cavity using a sterile stainless steel bowl, the abdominal viscera gently aggitated, and fluid and debris suctioned out with a suction device and a sump suction tip.

Suture Material
Absorbable suture
Catgut. Catgut is NOT recommended in contaminated, infected, hypoproteinemic, or debilitated cachexic patients. Its unpredictable absorption and rapid loss of tensile strength in such situations may result in an unacceptably high number of anastomotic leaks and /or breakdowns. Use of catgut suture in gastrointestinal surgery is not recommended.

Dexon, Polysorb, and Vicryl. Synthetic absorbable braided suture (i.e., polyglactin, polyglycolic acid) have become very popular. The braided nature however does result in increased tissue drag and difficult knotting ability.
Biosyn and Monocryl. These sutures have similar properties to Dexon, Polysorb and Vicryl however they are monofilament. They were developed to overcome the problem of tissue drag and knot slipping found in the braided synthetic absorbables. Their predictable hydrolytic absorption is unaffected by their immediate environment (i.e., infection, contamination, hypoproteinemia). They retain high tensile strength for a long period of time (2-3 weeks) and have very good handling characteristics. These suture materials are ideal for use in gastrointestinal surgery. These sutures are the authors choice for gastrointestinal surgery.

PDS and Maxon. PDS and Maxon, are synthetic absorbable monofilament suture materials with similar properties to that of Dexon and Vicryl. They have been shown to retain approximately 70% of their tensile strength at 3-4 weeks, and are absorbed by hydrolysis (unaffected by infection, contamination, hypoproteinemia). These suture materials are ideal for use in gastrointestinal surgery. Possible disadvantages include stiffness, a tendency to kink and prolonged absorption time.

Nonabsorbable suture
Nylon, Polypropylene, Polybutester. Monofilament, nonabsorbables are excellent suture materials for use in contaminated or infected surgical sites. They have a high tensile strength, are relatively inert in tissue, noncapillary, and do not act as a nidus for infection. These materials pass through tissue with essentially no tissue drag and have excellent knot tying security at sizes 3-0 to 5-0. For their properties, effectiveness, and cost, these are the author's nonabsorbable sutures of choice for intestinal anastomosis and enterotomy closure. Possible disadvantage of these materials is their memory.

Silk, Mersilene, Bronamid, Vetafil. In general, stay away from burying multifilament or braided nonabsorbable suture material. These sutures may harbor infection for years and may result in suture related abdominal abscesses or draining tracts. They should never be used in gastrointestinal surgery.

Suture size
For the majority of small intestinal surgical procedures in dogs, 3-0 or 4-0 size suture material is adequate; in cats, size 4-0 or 5-0 is recommended. The tensile strength of this size suture is greater than the tensile strength of the tissues that are being sutured (i.e., intestinal wall). Larger size suture may contribute to anastomotic failure by increased trauma to tissues and its effect on the blood supply of tissue margins.

Needles
Swaged-on "atraumatic" reversed cutting, narrow taper point, or fine taper-cut needles can all be used for gastrointestinal surgery. The author prefers a narrow taper point needle.

Suture Placement
When suturing intestine, sutures should be placed 3 - 4 mm from the cut edge of the intestine and no more than 2 - 3 mm apart. It is important to recognize everted mucosa and be sure the 3 - 4 mm bite in the intestinal wall is not just in mucosa but engages all layers of the intestinal wall. Measure your intestinal wall bite from the cut edge of the serosa.

Suture Patterns
There is considerable controversy regarding specific suture pattern for use in small intestinal surgery. Everting, inverting, and appositional suture patterns have been used experimentally
and clinically for suturing enterotomies and anastomoses. Appositional patterns are recommended as they cause little lumen compromise postoperatively.

**Everting:** Everting patterns (i.e., horizontal mattress) have been shown to encourage adhesions and result in lumen stenosis. This technique is NOT recommended. The evertting technique is not to be confused with the mild eversion of mucosa that occurs in the appositional techniques described below.

**Inverting:** In small animals adequate lumen diameter is an important consideration with any technique. Inverting patterns result in substantial lumen compromise of the small intestine and are NOT recommended in dogs and cats.

**Apposition:** Anatomic apposition of individual layers of the bowel wall (i.e., mucosa, submucosa, muscularis, and serosa) result in primary intestinal healing. This technique is superior to inverting or evertting techniques because apposition of intestinal margins eliminates lumen compromise. This is the authors preferred technique for suturing all hollow viscus organs in the abdominal cavity. Suture patterns of choice include:

1) Simple interrupted apposing. This technique involves suturing all layers of the intestinal wall and tying the knots on top of the serosa to approximate cut edges. The sutures should be tied tight enough to effect a watertight seal, yet not so tight as to blanch the tissue and cause ischemia of intestinal margins. This technique is simple, fast, reliable, and does not result in lumen compromise.

2) Simple continuous apposing. This technique is similar to the simple interrupted appositional technique however, a continuous suture pattern is used rather than an interrupted pattern. Advantages include faster anastomosis, equal suture tension over the entire anastomosis, airtight-watertight seal, and mucosal eversion is minimized.

**Commonly performed small intestinal surgical procedures**

**Key Points**
- intestinal sutures should engage at least 3 - 4 mm of submucosa and be no further apart than 2 - 3 mm
- always handle bowel wall with atraumatic technique
- examine the integrity of your anastomosis visually
- 50 - 60% of the small intestine of dogs and cats can be resected

**ENTEROTOMY:**
See the DVD for a detailed video description of this technique (www.videovet.org).
An enterotomy incision may be necessary for removal of intraluminal intestinal foreign bodies (e.g., balls, rocks, toys, linear foreign bodies), intestinal biopsy, exploration of the bile duct papilla or intestinal lumen, or rarely intestinal decompression. The segment of bowel to be incised should be removed from the abdominal cavity and packed off with moistened laparotomy pads. An incision parallel to the long axis of the bowel (i.e., longitudinal) or perpendicular to the long axis of the bowel (i.e., transverse) may be made on the antimesenteric border, preferably in healthy bowel (i.e., the aboral side of the foreign body). Closure is performed using the appositional techniques previously described (i.e., simple
continuous or simple interrupted). Omentum can be placed over the enterotomy, but need not be sutured.

**Transverse closure:** If a large full thickness piece of intestine must be excised (i.e., mural mass) longitudinal closure may result in stenosis. To prevent this, transverse closure of the linear incision is recommended. This ensures adequate lumen diameter without the need for intestinal anastomosis.

**INTESTINAL ANASTOMOSIS:** Intestinal anastomosis is indicated for resection of nonreducible intussusception, necrotic bowel wall secondary to complete intestinal obstruction, intestinal volvulus, stricture secondary to trauma, linear foreign body with multiple perforations, and intestinal neoplasia (e.g., leiomyoma, leiomyosarcoma, adenocarcinoma).

After a complete abdominal exploration, the affected length of bowel is delivered from the peritoneal cavity and isolated with the use of moistened laparotomy pads and crib towels. If possible, the intestinal anastomosis should be performed on a water resistant surface (e.g., plastic drape, crib towel) to prevent ‘strike‘ through contamination.

Once the level of resection has been determined, the appropriate mesenteric vessels are identified and ligated, and the portion of intestine to be resected is isolated by clamping the bowel at a 60° angle away from the mesenteric border. This angle ensures adequate blood supply to the antimesenteric border.

**Everted mucosa:** Occasionally when the segment of intestine to be removed is amputated mucosa ‘everts‘ from the cut edge of the intestinal wall making it difficult to visualize the cut edge of the serosa. If this occurs it is ‘highly‘ recommended to excise the everted mucosa to enable the surgeon to easily visualize the cut edge of the intestinal serosa. It is vital that the surgeon engage at least 3 – 4 mm of intestinal wall with each suture to guarantee adequate bites in the collagen laden submucosa.

**Bowel lumen diameters:** In cases where the oral end of the bowel is dilated and the aboral end is normal size, several options exist to create intestinal lumens of equal diameter:

1) Increase the angle of resection on the smaller diameter segment of bowel (i.e., aboral segment). This will increase the orifice size by 5-10 mm depending upon bowel diameter (e.g., dog vs cat).

2) In larger lumen size discrepancies the antimesenteric border of the smaller diameter stoma can be incised longitudinally to enlarge the lumen diameter.

3) An end-to-side anastomosis can be performed by closing the larger diameter stoma of the intestinal resection with a single layer continuous apposing suture pattern then anastomosing the smaller diameter segment of bowel to an appropriate size enterotomy made in the antimesenteric border of the larger diameter segment of bowel.

4) The larger diameter segment of bowel can be made smaller in diameter by suturing its cut edge until its lumen is equal in size to the smaller diameter intestine (this technique is often used for subtotal colectomy in cats).

**Intestinal Anastomosis Technique:**
See the DVD for a detailed video description of this technique (www.videovet.org).
When suturing an anastomosis, atraumatic handling of bowel wall and perfect anatomic apposition of incised margins is important. It is recommended to begin suturing at the mesenteric border as this allows adequate visualization of mesenteric vessels and helps prevent encircling these vessels when placing the first few sutures. Any of the appositional suture patterns previously described (i.e., simple continuous or interrupted) will result in a high success rate, both in the short-term (i.e., leakage, breakdown) and long-term (i.e., stricture, stenosis).

The following tips may prove helpful when performing an intestinal anastomosis (see the anastomosis video clip at www.videovet.org for detailed description of the surgery tips below:
1) First, place a stay suture to hold the mesenteric border of each segment of bowel in apposition. Tie this suture, leave the ends long, and place a hemostat on the suture end without the needle.
2) Place a second stay suture in the antimesenteric borders of each segment to be sutured to bring the ends of the intestinal segments into apposition. Place a hemostat on the ends of this suture.
3) Place gentle traction on the mesenteric and antimesenteric stay sutures to bring the two intestinal segments into apposition.
4) Using the needled segment of suture from the mesenteric stay suture, begin a simple continuous appositional anastomosis being careful to get a 3 - 4 mm bite in the submucosa and placing each suture no more than 2 - 3 mm apart (2 mm apart in cats). When the anastomosis is complete, tie the suture to the mesenteric stay suture.
5) If a simple interrupted apposing suture pattern is used, be careful to get a 3 - 4 mm bite in the submucosa and place each suture no more than 2 - 3 mm apart.
6) Evaluate the integrity of the anastomosis. The author’s preference for evaluating the integrity of anastomotic closure is to **visually** examine each suture to be certain that suture placement is no more than 2 - 3 mm apart and that each suture has a 3 mm bite in the submucosa.

**Postoperative care**
Intravenous fluids to maintain hydration and ensure renal function are continued postoperatively, until the patient begins to eat and drink. Intravenous fluids should then be tapered over a 24 to 48 hour period.
Systemic antibiotics are continued postoperatively for 5-7 days; 10 - 14 days in cases with peritonitis and/or sepsis.

**Feeding:** Early return to enteral feeding is best for the overall health of the intestine. Feeding the postoperative gastrointestinal surgical patient is generally based on the following criteria:
- a) preoperative condition of the patient
- b) the condition of the bowel at the time of surgery
- c) surgical procedure performed (i.e., enterotomy, anastomosis, pyloroplasty)
- d) presence or absence of peritonitis
- e) postoperative condition of the patient.
The earlier patients can be returned to oral alimentation the better.

**Complications**
The most common postoperative complication of small intestinal surgery is leakage; leak is either associated with breakdown of the anastomosis or improper surgical technique (i.e.,
improper suture placement, inappropriate suture material, knot failure, sutures to far apart, inappropriate bite in the collagen laden submucosal layer, suturing nonviable bowel).

A presumptive diagnosis may be accomplished by the following:
1) Body temperature (may be up if acute or down if moribund).
2) Abdominal palpation: periodic, gentle abdominal palpation for pain (gas or fluid?).
3) General attitude (depression-anorexia).
4) Incision: examination of the patients incision for drainage (look at cytology if drainage is present)
5) CBC: leukocytosis followed by leukopenia (sepsis), or a degenerative left shift may imply breakdown.
6) Glucose: low glucose generally implies sepsis (this occurs early in sepsis and may be used as a screening test).
7) Abdominal radiographs: generally not helpful, they are difficult to critically assess due to the presence of postoperative air and lavage fluid. It can take 1 - 3 weeks for peritoneal air to diffuse from the abdominal cavity after routine abdominal surgery. Time variation is dependant upon the amount of air remaining in the abdominal cavity postoperatively (i.e., large deep chested animal vs a small obese animal).
8) Abdominal tap (paracentesis): a four quadrant abdominal tap is accomplished by aspirating fluid using a 5cc syringe and 20 gauge needle or placing a plastic IV catheter into the peritoneal cavity and allowing fluid to drip onto a slide. This may be the most sensitive diagnostic test for determining the presence or absence of intestinal leak.
9) Peritoneal lavage (if paracentesis is not productive): infuse 10-20cc/kg of sterile physiologic saline solution into the abdominal cavity, then gently palpate the abdomen and repeat the four quadrant paracentesis. This technique increases the sensitivity of paracentesis to 90%.

Once fluid has been obtained, a smear should be stained and evaluated microscopically. Depending upon the cell types seen, a determination of the presence of leakage can be made.

Below are examples of expected cytology in patients with and without leak.
1) Healthy PMNs with few degenerate PMNs and a moderate number of red blood cells: This cytology may be expected in any postoperative abdominal procedure (e.g., OHE, abdominal exploratory, cystotomy). Your index of suspicion for anastomotic breakdown should be low. However, if clinical signs continue to deteriorate, repeat paracentesis (2 - 3 times daily, if necessary) to determine the “trend” of the abdominal fluid cytology is recommended.
2) Healthy polymorphonuclear leukocytes with bacteria located intra or extracellularly, degenerate PMNs with intracellular bacteria, free bacteria, or food particles--imply breakdown. Exploratory laparotomy is indicated.

In a recent morbidity/mortality study of patients undergoing intestinal surgery it was found that animals requiring a second abdominal surgery to treat intestinal disorders were less likely to survive than patients requiring only one laparotomy. Also, the longer it took to determine whether or not intestinal leakage had occured the less likely the patient would survive reoperation. The take home message is: pay attention to detail during the first surgery and if a leak occurs, diagnose it as soon as possible.

**Prognosis** The overall prognosis for uncomplicated GI surgery is excellent. The surgeon must pay attention to detail when suturing any hollow viscus organ with liquid contents.
VISCERAL ORGAN BIOPSY  
Howard B. Seim III, DVM, DACVS  
Colorado State University

If you would like a copy of a video of this surgical technique on DVD, go to www.videovet.org.

Key Points
- Open the abdomen from xyphoid to pubis
- Use the same method of exploration in each case
- When in doubt, biopsy, biopsy, biopsy
- Close the linea alba using a continuous suture pattern

General Considerations and Indications
The systematic, thorough observation and palpation of all abdominal structures is mandatory with any abdominal exploratory procedure. It is easy to miss a second intestinal foreign body, an area of metastasis or a ‘second’ tumor if one does not get in the habit of performing a complete exploratory.

An exploratory can be done in any order but it is best to establish a routine and follow it for every case. With experience the procedure can be completed in less than five minutes. The best way to recognize an abnormal finding is to know the normal. Take advantage of any laparotomy to observe normal structure, color, consistency and position of all abdominal organs.

Abdominal exploratory may be indicated in following situations:
- Abdominal mass
- Undiagnosed GI disorders
- Urogenital abnormalities unresponsive to medical management
- Abdominal disorders of unknown origin
- Penetrating trauma
- Acute abdomen
- Generalized peritonitis
- Diagnosis and treatment of portosystemic shunts
- Spleenic abnormalities
- Uncontrolled abdominal hemorrhage

Preoperative Considerations
A midline abdominal incision from xiphoid to pubis is the easiest and most versatile approach. Positioning the patient’s head toward the top of the table and tilting the table at a 30° to 40° angle will facilitate gravitation of abdominal viscera out of the thorax. Rarely is it necessary to extend the incision into the thorax via a median sternotomy however if your index of suspicion is high that this may be necessary (e.g., diaphragmatic hernia, chylothorax, porosystemic shunt) the animal should be properly and adequately prepared.

ABDOMINAL EXPLORATORY TECHNIQUE
Position, Preparation and Draping
The abdomen is always clipped and prepared wider and longer than you may anticipate for a “routine” procedure. This generally means from cranial to the xyphoid to a point 4-5 cm caudal to the brim of the pubis and laterally to include the ventral aspect of the abdomen. The animal is placed in dorsal recumbency in a V-trough with front and hind limbs secured with ropes to the table. If the penis or prepuce does not need to be accessible during surgery, the prepuce is clipped but not flushed. If the penis needs to be exposed and handled by the surgeon (lower urinary system exploratory), a prepuccal lavage with a1:50 dilution of betadine solution in sterile saline is performed prior to skin
preparation. An example is when the surgeon feels it is necessary to pass a urinary catheter during surgery (i.e., urethral and cystic calculi). In this case, the prepuce is clipped then flushed using dilute Betadine or chlorhexidine prior to the prep. Standard skin preparation is performed.

A ventral midline incision is made from xyphoid to pubis. After identifying the linea alba, a scalpel blade is used to open the abdominal cavity via a stab incision. Mayo scissors are used to complete the abdominal incision. Make sure you can see or feel the internal side of the linea so that you do not damage abdominal viscera during abdominal wall incision. The falciform ligament is excised with scissors from its attachment to the abdominal wall midline and xyphoid. This will allow better inspection of viscera, particularly in the cranial quadrant, and facilitate easier closure of the abdomen. Moisten laparotomy sponges may be placed on the incision if viscera are to be brought out of the abdomen. All viscera should be kept moistened with saline solution.

Examination of abdominal viscera can be done in any order but it is best to establish a routine and follow it every time the abdomen is explored. Generally, start with proximal GI tract and move distally, then liver and pancreas, then urinary tract. Be thorough, and always be gentle when handling tissue. It is easy to miss a second intestinal foreign body or an area of metastasis if one does not get in the habit of performing a complete exploratory. With experience, complete exploration can be performed in less than five minutes. The best way to recognize an abnormal finding is to know the normal. Take advantage of any laparotomy to observe normal structure, color, consistency and position all of abdominal organs. You should be able to identify the following structures:

1. Skin, subcutaneous tissue and linea alba
2. Falciform ligament and fat should be removed with scissors from both sides of the incision, no ligation is usually necessary
3. Abdominal aorta and its major branches
4. Caudal vena cava
5. Portal vein
6. Kidneys - artery - frequently multiple; vein - frequently multiple; ureter - note its location at bladder neck (dorsolateral) and its path along psoas muscles
7. Liver lobes
8. Gallbladder
9. Pancreas (always be gentle handling this organ because you can induce pancreatitis) - left and right limbs
10. Diaphragm - left and right crus; aortic hiatus; esophageal hiatus; caval foramen; costal arch
11. Abdominal esophagus
12. Stomach - cardia; fundus; body; pylorus (note close relationship of common bile duct and pancreatic ducts); arterial supply; gastrohepatic ligament
13. Small bowel - note blood supply to each area
14. Cecum - note size and consistency, feel ileoceccocolic junction
15. Colon - ascending, transverse and descending portion
16. Lymph nodes - mesenteric and sublumbar
17. Omentum and mesentary; greater omentum; lesser omentum; mesoduodenum - used to displace and pack off cranial portions of abdomen from the right to the left; mesocolon - used to displace and pack off caudal aspect of abdominal contents from the left to the right
18. Urinary bladder; ureter entrance on dorsal trigone area; apex and trigone; lateral ligaments
19. Female; ovary, ovarian bursa, proper ovarian ligament; uterine horn; uterine body; cervix; round lig; broad lig; suspensory lig (broken down to mobilize ovary during spay)
20. Male; prostate; ductus deferens (and relation to ureters)
21. Adrenal glands; phrenicoabdominal arteries and their position in relation to ureters
22. Spleen - usually will be very large and turgid as a result of barbiturate anesthesia
**Closure**

The linea alba is closed with monofilament absorbable or nonabsorbable suture using a simple continuous pattern. If the abdominal incision was made directly on the midline (i.e., linea alba) closure requires full thickness bites of the linea alba. If the abdominal incision was slightly off the midline, suture the rectus sheath only (do not include rectus abdominus muscle or peritoneum). The most important tissue in the abdominal closure is the collagen dense external rectus sheath. Incorporation of the internal sheath (i.e., peritoneum) is unnecessary as the peritoneum has very little holding power. Sutures should engage approximately 5-7 mm of rectus sheath on each side of the incision line.

Subcutaneous tissues are closed separately with a simple continuous pattern using monofilament or multifilament synthetic absorbable suture. Tissues should be approximated and not strangulated. In cats, the subcutaneous closure is not recommended.

Skin is closed with simple interrupted monofilament nonabsorbable skin sutures or a continuous intradermal suture (i.e., subcuticular).

**Postoperative Care**

Postsurgical care may include systemic antibiotics, appropriate pain medication, careful monitoring of the patient's breathing, temperature, and color. Hypothermic patients should be kept in a warming cage or on a warm water circulating blanket for at least 24 hours. Analgesics may be used to relieve patient discomfort, however care should be taken to monitor the effects of various analgesic drugs on respiratory effort.

**BIOPSY TECHNIQUES FOR ABDOMINAL ORGANS**

A variety of visceral organ biopsy techniques are illustrated on a video DVD available through VideoVet at [www.videovet.org](http://www.videovet.org).

**Liver Biopsy**

Liver biopsy is indicated whenever an abdominal exploratory is being performed in patients thought to have liver disease or in cases that liver disease was not the primary reason for exploratory but the liver appears grossly normal.

Liver biopsy is one of the most important diagnostic aids available for evaluation of liver disease. Samples for cytologic examination may be obtained via percutaneous needle biopsy, laparoscopy, or exploratory laparotomy. Percutaneous needle biopsy techniques are the most efficient in terms of time and expense.

Several techniques are available for obtaining liver specimens during exploratory laparotomy. The simplest method is performed by cutting a strip of liver parenchyma 5 to 6 mm thick along the border of the liver lobe. Excessive bleeding is rarely a problem with this technique; hemorrhage is controlled via cautery or direct pressure. Diffuse liver disease must be present if this method is to be diagnostic.

A second technique involves placing an encircling ligature around a pedicle of liver tissue. As the ligature is tightened, it cuts through the hepatic parenchyma, ligating hepatic vessels and bile ducts. This technique is widely known as the Guillotine technique. This method requires the presence of diffuse liver disease to obtain a diagnostic biopsy unless the lesion is present in the distal aspect of the liver lobe.

A technique that can be used in ‘bulbous’ liver lobes with no convenient edges to biopsy is performed by penetrating the central portion of the proposed biopsy site with 2-0 or 3-0 suture on a swaged-on
curved taper needle. The suture ends are left long and a mosquito hemostat attached. A second pass of the suture is made through the same location as the first needle pass. A second stay suture is made. Each stay suture is tied individually to “cut” through the liver. A “V” wedge is cut through the liver when both strands of suture have been tied. A number 15 BP scalpel blade or fine Metzenbaum scissors is used to cut the V-shaped liver biopsy wedge from the sutures.

Another technique for use in patients with diffuse fibrotic liver disorders is performed by penetrating the affected liver lobe with a straight mosquito hemostat. The hemostat tip is placed on the surface of the liver lobe to be biopsied and gently plunged through the liver lobe until the tip of the hemostat is seen penetrating through the opposite side of the liver. The jaws of the hemostat are opened just wide enough to accept a piece of 2-0 or 3-0 Maxon or Biosyn suture. The suture is doubled on itself, the loop is passed into the jaws of the hemostats, and the loop pulled through the liver lobe. The exiting loop is cut leaving two strands of suture coursing through the liver lobe. Each strand is tied individually to “cut” through the liver. A “V” wedge is cut through the liver when both strands of suture have been tied. A number 15 BP scalpel blade is used to cut the V-shaped liver biopsy wedge from the sutures.

Occasionally it may be required to biopsy a small lesion located distant from the liver lobe margin. This can be accomplished using a 4mm or 6mm ‘sharp’ skin punch biopsy instrument. The skin punch is placed directly over the lesion on the surface of the liver. With mild downward pressure and a gentle back and forth twisting motion the punch biopsy instrument cuts a circular hole in the liver thus engaging a piece of the liver. The punch is then angled to attempt to cut the base of the biopsy specimen. The punch is removed and either the biopsy specimen comes out with the punch or a DeBakey forcep may be needed to remove the specimen from its remaining attachment to the liver. This technique results in a very small biopsy specimen and thus an additional marginal liver biopsy is performed.

Pancreatic biopsy
The old wives tale stating “don’t touch the pancreas” needs to be put to rest. Gentle manipulation and biopsy of the pancreas is a predictably successful procedure with almost no incidence of postoperative pancreatitis.

Biopsy of the pancreas is performed in a similar manner as biopsy of the liver. In patients that have diffuse pancreatic disease, a segment of the right or left limb of the pancreas is identified. An encircling ligature of 3-0 Biosyn is placed around the pedicle. As the ligature is tightened, it cuts through the pancreatic parenchyma, ligating vessels and pancreatic ducts. The distal pedicle of pancreas is carefully removed with a number 15 BP scalpel blade or metzenbaum scissors. Care is taken to avoid cutting the suture. If a relatively large portion of pancreas is to be removed (e.g., removal of insulinoma), a similar technique is used. In this situation, 2-0 or 3-0 monofilament nonabsorbable suture should be used.

Stomach and Small Intestine
Patients with chronic vomiting or chronic diarrhea of unknown origin often require gastric and intestinal biopsies for definitive diagnosis. In many cases, the surgeon will examine the gastrointestinal tract carefully and conclude that there are no apparent abnormalities. In this situation, ALWAYS perform gastric and multiple intestinal biopsies (i.e., duodenum, jejunum, ileum). Remember these words of wisdom when concluding that you have a negative exploratory laparotomy “your eyes are NOT microscopes”.

Gastric biopsy
The stomach should be visually examined for any obvious abnormalities on the serosal surface. In addition, the stomach should be carefully palpated to determine if there are mural or mucosal abnormalities present. In the case of an observed or palpated abnormality, the surgeon should plan the gastric biopsy to include a portion of the abnormal stomach, the margin of normal and abnormal stomach, and normal stomach. Full thickness biopsies should always be taken. In the case of diffuse disease or if an abnormality cannot be located, a 3-4 cm incision should be made in the ventral aspect of the stomach equidistant from the greater and lesser curvature. Stay sutures are placed at the midpoint of the incisied edges and the interior of the stomach visually and digitally examined. If a mucosal abnormality is detected, the area should be biopsied either from inside the stomach or from the serosal surface directly over the lesion.

Gastric wall incisions (e.g., biopsy, gastrotomy, partial gastrectomy) should be closed with a single layer, simple continuous or simple interrupted suture pattern being careful to get full thickness bites. Sutures should be placed no further apart than 3 mm and at least a 4 mm bite of gastric wall is recommended. Monofilament absorbable suture with a sharp taper or taper-cut (penetrating point) needle is the authors’ preference.

Small intestinal biopsy
Several techniques can be used to successfully biopsy the intestine. Always remember; FULL THICKNESS biopsy is mandatory for the pathologist to give you the most accurate diagnosis.

When taking an intestinal biopsy, the easiest way to guarantee you will get an adequate size, full thickness piece of intestine is to use a brand new 4mm skin punch biopsy instrument. The skin punch is placed on the antimesenteric border of the proposed segment of intestine and ‘drilled’ through all layers of intestine until the biopsy punch can be felt to enter the lumen of the intestine. The skin punch is removed and the biopsy retrieved from the shaft of the skin punch biopsy. This technique is particularly useful for ileal biopsy as it is easy to biopsy between the mesenteric and antimesenteric vessels. Transverse closure of the biopsy site is recommended to eliminate the possibility of lumen compromise. Suture technique is as described above for enterotomy closure. This is the authors’ preferred technique for intestinal biopsy.

An alternate technique for intestinal biopsy is to make a 2-3 mm long incision on the antimesenteric border of the intestinal segment. A #11 or #15 BP scalpel blade is used to penetrate the intestinal wall. The blade is withdrawn to create a 2-3 mm long incision. A second parallel incision is made 1 – 2 mm from the original incision. A DeBakey forcep is used to grasp one end of the parallel incisions, a Metzenbaum scissor is used to cut out the piece of intestine. The surgeon should be careful not to crush the specimen with forceps. Only handle one end of the specimen whilst excising the biopsy specimen. If excessive trauma is created during biopsy, the pathologist may not be able to determine if the pathology is real or surgically created. The excised piece of intestine is examined closely to ensure that all layers have been included in the specimen. The biopsy site is closed using a simple interrupted or simple continuous suture pattern. 3-0 or 4-0 monofilament absorbable suture with a swaged-on sharp taper or taper-cut (penetrating point) needle is recommended. Care is taken to ensure that at least 3 mm bites are taken into the intestine and the sutures are no more that 2-3 mm apart.

Biopsy of the duodenum, jejunum, and ileum is recommended whenever a chronic vomiting/diarrhea patient is explored.

Complications associated with multiple intestinal biopsies are rare. Even patients that present with protein loosing enteropathy. One study looking at the complication rate of intestinal surgical procedures in patients with normal protein levels and patients that were hypoproteinemic found no
difference. Complications in patients undergoing intestinal surgical procedures are generally related to the surgeons technical ability not the patients preoperative status.
Surgical Management of Abdominal Trauma
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OVERVIEW
Patients that sustain a traumatic injury severe enough to cause an abdominal hernia or patients that sustain penetrating abdominal wounds (i.e., gunshot, bite wounds) should have an exploratory laparotomy. After a review of general principles and techniques of abdominal surgery in cats and dogs, including the surgical techniques for ventral midline celiotomy, abdominal exploration, abdominal wall closure, and management of complications such as wound dehiscence, these proceedings outline the surgical management of traumatic abdominal wall hernias and peritonitis. Step by step descriptions of the surgical technique for repair of abdominal hernias are provided. For peritonitis, we will cover the indications and techniques for abdominocentesis, exploratory surgery, diagnostic abdominal lavage, and open abdominal drainage. In the lecture, case examples admitted to the author’s critical care service will be used to illustrate the surgeons’ decision making techniques. Video of clinical case material will be used to illustrate all techniques.

If you would like a copy of the video of this surgical procedure on DVD go to www.videovet.org.

KEY FACTS
• The abdomen generally is explored by means of a ventral midline incision from xyphoid to pubis. In most animals the entire abdomen, including the inguinal areas and the caudal thorax, should be prepared for aseptic surgery to allow extension of the incision into the thoracic or pelvic cavities if necessary.
• Various techniques can be used to systematically explore the entire abdomen; every surgeon should develop a consistent pattern to ensure that the entire abdominal cavity and all structures are visualized and/or palpated in each animal.
• Complications of abdominal surgery, including dehiscence (incisional hernias), may occur if improper surgical technique is used. The most common cause of wound dehiscence in the early postoperative period results from the surgeon’s inability to recognize the rectus sheath or not getting adequate bites in the collagen dense rectus sheath.
• Patients that sustain a traumatic injury severe enough to cause an abdominal hernia or patients that sustain penetrating abdominal wounds (i.e., gunshot, bite wounds) should have a xyphoid to pubis abdominal exploratory laparotomy.
• For most abdominal hernias, perform a ventral midline abdominal incision to allow the entire abdomen to be explored. Assess the extent of visceral herniation. Reduce the herniated contents and amputate or excise necrotic or devitalized tissue around the hernia. Close the muscle layers of the hernia with simple interrupted or simple continuous sutures.
• Abdominocentesis— the percutaneous removal of fluid from the abdominal cavity— usually is done for diagnostic purposes although it may occasionally be therapeutic. Indications include shock without apparent cause, undiagnosed disease with signs involving the abdominal cavity, suspicion of postoperative gastrointestinal dehiscence, blunt or penetrating abdominal injuries (i.e., gunshot wounds, dog bites, vehicular injury), and undiagnosed abdominal pain.
• Exploratory surgery is indicated when the cause of peritonitis cannot be determined or when bowel rupture, intestinal obstruction (e.g., bowel incarceration, neoplasia), or mesenteric avulsion is suspected.
• Although the practice of routinely lavaging the abdominal cavity of animals is controversial, lavage is always indicated with diffuse peritonitis. Lavage should be done with care in animals with localized peritonitis to avoid dissemination of infection.

• Open abdominal drainage is a useful technique for managing animals with peritonitis. Reported advantages include improvement in the patient’s metabolic condition secondary to improved drainage, less formation of abdominal adhesions and abscesses, and access for repeated inspection and exploration of the abdomen. With this technique the abdomen is left open, and sterile wraps are placed around the wound.

• The prognosis for animals with generalized peritonitis is guarded; however, with proper and aggressive therapy, many survive. Some authors have suggested that the mortality rate approaches 50%. The mortality rates reported in animals with generalized peritonitis treated with open abdominal drainage have varied from 20% to 48%.

GENERAL PRINCIPLES AND TECHNIQUES
Definitions
Celiotomy is a surgical incision into the abdominal cavity. The term laparotomy often is used synonymously, although it technically refers to a flank incision. A sudden onset of clinical signs referable to the abdominal cavity (e.g., abdominal distention, pain, vomiting) is called an acute abdomen.

Surgical Techniques
The abdomen generally is explored by means of a ventral midline incision from xyphoid to pubis. In most animals the entire abdomen, including the inguinal areas, and the caudal thorax should be prepared for aseptic surgery to allow extension of the incision into the thoracic or pelvic cavities if necessary.

Ventral Midline Celiotomy in Cats and Female Dogs
With the patient in dorsal recumbency, make a ventral midline skin incision beginning near the xyphoid process and extending caudally to the pubis. Sharply incise the subcutaneous tissues until the external fascia of the rectus abdominis muscle is exposed. Ligate or cauterize small subcutaneous bleeders and identify the linea alba. Tent the abdominal wall and make a sharp incision into the linea alba with a scalpel blade. Palpate the interior surface of the linea for adhesions. Use scissors to extend the incision cranially or caudally (or both) to near the extent of the skin incision. Digitally break down the attachments of one side of the falciform ligament to the body wall or excise it and remove it entirely if it interferes with visualization of cranial abdominal structures. Clamp the cranial end of the falciform ligament and ligate or cauterize bleeders before removing it.

Ventral Midline Celiotomy in Male Dogs
Patients undergoing exploratory laparotomy for abdominal trauma should have the prepuce and penis aseptically prepared for surgery and they should remain in the surgical field; particularly if lower urinary trauma is suspected. Make a ventral midline skin incision beginning at the xyphoid process and continuing caudally to the prepuce. Curve the incision to the left or right of the penis and prepuce and extend it to the level of the pubis. Incise the subcutaneous tissues and fibers of the preputialis muscle to the level of the rectus fascia in the same plane as the skin incision. Ligate or cauterize large branches of the caudal superficial epigastric artery and vein at the cranial aspect of the prepuce. Retract incised skin, prepuce and penis and subcutaneous tissues laterally to locate the linea alba and external fascia of the rectus abdominis muscle. Do not attempt to locate the caudal linea alba until subcutaneous tissues have been incised and the
abdominal musculature fascia identified. Tent the abdominal wall and make a sharp incision into the linea alba with a scalpel blade. Palpate the interior surface of the linea for adhesions. Use scissors to extend the incision cranially or caudally (or both) to near the extent of the skin incision.

**Abdominal Exploration**

Systematically explore the entire abdomen. Various techniques may be used; however, every surgeon should develop a consistent pattern to ensure that the entire abdominal cavity and all structures are visualized and/or palpated in each animal.

Use moistened laparotomy sponges to protect tissues from drying during the procedure. If generalized infection is present or if diffuse intraoperative contamination has occurred, flush the abdomen with copious amounts of warmed, sterile saline solution with no additives (i.e., antiseptics or antibiotics). Remove the lavage fluid and blood and inspect the abdominal cavity before closure to ensure that all foreign material and surgical equipment have been removed. Perform a sponge count and compare it with the preoperative count to ensure that surgical sponges have not been left in the abdominal cavity.

**Abdominal Wall Closure**

The linea alba may be closed with a simple continuous (author’s preference) or a simple interrupted suture pattern. The simple continuous technique does not increase the risk of dehiscence when properly performed (i.e., secure knots, appropriate suture material, adequate bites in the rectus sheath), and it allows for a rapid and more secure closure. Synthetic monofilament absorbable suture (Maxon, PDS) should be used for continuous suture patterns, and six to eight knots should be placed at each end of the incision line.

On each side of the incision, engage a 5 to 7 mm bite of white rectus sheath with each suture. Place sutures no further apart than 3 to 4 mm, depending on the animal’s size. Tighten sutures sufficiently to appose but not enough to strangulate tissue, because sutures that strangulate tissue negatively affect wound healing. Incorporate full thickness bites of the abdominal wall in the sutures if the incision is midline (i.e., through the linea alba). If the incision is lateral to the linea alba and muscular tissue is exposed (i.e., paramedian incision), close the external rectus sheath without including muscle or peritoneum in the sutures. Close subcutaneous tissues with a simple continuous pattern of absorbable suture material and reappose the preputialis muscle fibers in the male dog. Use nonabsorbable sutures (simple interrupted or continuous appositional pattern) or stainless steel staples to close skin. Place skin sutures without tension.

**Complications**

Dehiscence (incisional hernias) may occur if improper surgical technique is used (see the above discussion). The most common cause of wound dehiscence in the early postoperative period results from the surgeon’s inability to recognize the rectus sheath or not getting adequate bites in the rectus sheath. Bites should engage at least 5 to 7 mm or more depending upon patient size.

**TRAUMATIC ABDOMINAL WALL HERNIAS**

**Definitions**

External abdominal hernias are defects in the external wall of the abdomen that allow protrusion of abdominal contents; internal abdominal hernias are those that occur through a ring of tissue confined within the abdomen or thorax (i.e., diaphragmatic hernia, hiatal hernia). External abdominal hernias may involve the abdominal wall anywhere other than the umbilicus, inguinal ring, femoral canal, or scrotum.
Surgical Treatment
Patients that sustain a traumatic injury severe enough to cause an abdominal hernia or patients that sustain penetrating abdominal wounds (i.e., gunshot, bite wounds) should have a xyphoid to pubis abdominal exploratory laparotomy. All visceral structures should be carefully examined to signs of trauma (e.g., mesenteric rents, ruptured hollow viscous organs, avulsed kidney, ureteral damage). In addition, abdominal celiotomy approach facilitates abdominal hernia closure. Most abdominal hernias can be repaired by suturing torn muscle edges or apposing the disrupted abdominal wall edge to the pubis, ribs, or adjacent fascia. In rare cases synthetic mesh must be used to repair the defect. Some hernias (i.e., intestinal strangulation, urinary obstruction, concurrent organ trauma) require emergency surgical correction. The extent of devitalized muscle may not be apparent initially, however, for patients in stable condition, delaying surgery until muscle damage can be accurately assessed facilitates surgical correction. The most common complications of surgery are hernia recurrence and wound infection. Abdominal hernias that occur secondary to bite wounds usually are contaminated; wound infection and dehiscence of the skin or hernial repair (or both) may occur. Mesh should not be placed in these hernias, hernial closure is performed during exploratory laparotomy, and the skin wounds should be left open to drain. Treatment of infected wounds includes cultures, drainage, antibiotics, and/or flushing.

Positioning
For ventral hernias the animal is placed in dorsal recumbency and the area around the hernia is prepared for aseptic surgery. Repair of ruptures of the cranial pubic ligament may be facilitated by placing the animal in dorsal recumbency with the rear limbs flexed and pulled cranially.

Surgical Techniques
Abdominal Hernias
For most abdominal hernias, perform a ventral midline abdominal incision to allow the entire abdomen to be explored. Assess the extent of visceral herniation. Reduce the herniated contents and amputate or excise necrotic or devitalized tissue around the hernia. Close the muscle layers of the hernia with simple interrupted or simple continuous sutures.

Cranial Pubic Ligament Hernias
Make a ventral midline skin incision and identify the ruptured tendon and its pubic insertion. Evaluate the inguinal rings and vascular lacuna; these hernias may extend into the femoral region as a result of rupture of the inguinal ligament. Reattach the free edge of the abdominal wall to the cranial pubic ligament with simple interrupted sutures. As an alternative, suture the tendon remnant to the muscle fascia and periosteum covering the pubis or anchor it to the pubis by drilling holes in the pubic bone through which sutures can be placed. If the hernia extends into the femoral region, it may be necessary to suture the body wall to the medial fascia of the adductor muscles. When doing so, take care to avoid damaging the femoral vessels or nerves.

Prognosis
The prognosis generally is good, and recurrence is uncommon. When recurrence occurs, it generally is noted within a few days of surgery. Most animals have excellent long-term results when appropriate techniques are used.

PERITONITIS
Definition
Primary generalized peritonitis refers to spontaneous inflammation of the peritoneum without any pre-existing intra-abdominal pathologic condition. Secondary generalized peritonitis
occurs in conjunction with an intra-abdominal pathologic condition and may be further classified as infectious or noninfectious.

**Surgical Treatment**

**Abdominocentesis** (see below) is the percutaneous removal of fluid from the abdominal cavity, usually for diagnostic purposes, although it may occasionally be therapeutic. Indications include shock without apparent cause, undiagnosed disease with signs involving the abdominal cavity, suspicion of postoperative gastrointestinal dehiscence, blunt or penetrating abdominal injuries (i.e., gunshot wounds, dog bites, vehicular injury), and undiagnosed abdominal pain. A multifenestrated catheter should be used to enhance fluid collection. Physical and radiographic examinations should precede abdominocentesis to rule out instances in which it may not be safe and to guide needle placement. Four-quadrant paracentesis may be performed if simple abdominocentesis is not successful in retrieving fluid. It is similar to simple abdominocentesis except that multiple abdominal sites are assessed by dividing the abdomen into four quadrants through the umbilicus and tapping each of these four areas. Diagnostic peritoneal lavage should be performed in animals suspected of having peritonitis if the above methods are unsuccessful in obtaining fluid for analysis.

Exploratory surgery is indicated when the cause of peritonitis cannot be determined or when bowel rupture, intestinal obstruction (e.g., bowel incarceration, neoplasia), or mesenteric avulsion is suspected. Serosal patching and plication reduce the incidence of intestinal leakage, dehiscence, or repeated intussusception. Animals that require surgery and that have peritonitis secondary to intestinal trauma (disruption of mesenteric blood supply, bowel perforation, chronic intussusception, foreign body) often are hypoproteinemic. The role that protein levels play in healing intestinal incisions is not well understood. However, most surgeons are concerned that hypoproteinemic patients may not heal as quickly as patients with normal protein levels, despite one study that showed similar complication rates among animals with normal protein levels and those that were hypoproteinemic and undergoing intestinal surgery. Most experimental evidence has shown that retardation of wound healing is not seen with moderate protein depletion but only with severe deficiencies (<1.5 to 2 g/dL).

Although the practice of lavaging the abdominal cavity of animals with peritonitis is controversial, lavage generally is indicated with diffuse peritonitis. Lavage should be done with care in animals with localized peritonitis to avoid dissemination of infection. When lavage is performed, as much of the fluid as possible should be removed because fluid inhibits the body’s ability to fight off infection, probably by inhibiting neutrophil function. Historically, many different agents have been added to lavage fluids, especially antiseptics and antibiotics. Povidone-iodine is the most widely added antiseptic; however, its use may be contraindicated with established peritonitis. Furthermore, no beneficial effect of this agent has been shown in repeated experimental and clinical trials in animals. Although a great many antibiotics have been added to lavage fluids over the years, there is no substantial evidence that their addition is of any benefit to patients who are being treated with appropriate systemic antibiotics. Warmed sterile physiologic saline is the most appropriate lavage fluid.

**Open abdominal drainage** (OAD) is a useful technique for managing animals with peritonitis. Reported advantages include improvement in the patient’s metabolic condition secondary to improved drainage, less formation of abdominal adhesions and abscesses, and access for repeated inspection and exploration of the abdomen. With this technique the abdomen is left open and sterile wraps are placed around the wound. The frequency of wrap changes depends on the amount of fluid drained and the amount of external soiling. Complications of open abdominal drainage include persistent fluid loss, hypoalbuminemia, weight loss, adhesion of
abdominal viscera to the bandage, and contamination of the peritoneal cavity with cutaneous organisms.

There is evidence to suggest the use of Jackson Pratt drains are an efficient means of draining the peritoneal cavity for 2 to 4 days postoperatively. This technique allows the surgeon to perform a primary abdominal closure yet still provide abdominal drainage.

**Positioning**
For abdominocentesis and diagnostic lavage, the abdomen should be clipped and prepared aseptically. These procedures may be performed with the animal in lateral recumbency or standing.

**Abdominocentesis**
Insert an 18- or 20-gauge, 1½-inch plastic over-the-needle catheter (with added side holes) into the abdominal cavity at the most dependent part of the abdomen. Do not attach a syringe; instead allow the fluid to drip from the needle and collect in a sterile tube. If sufficient fluid is obtained, place it in a clot tube and an ethylenediamine tetraacetic acid (EDTA) tube, submit samples for aerobic and anaerobic culture, and make four to six smears for analysis. If fluid is not obtained, apply gentle suction using a 3-mL syringe.

It is difficult to puncture bowel by this method because mobile loops of bowel move away from the tip of the needle as it strikes them. Perforations created by a needle this size usually heal without complications. The major disadvantage of needle paracentesis is that it is insensitive to the presence of the small volumes of intraperitoneal fluid and thus a negative result can be meaningless. At least 5 to 6 mL of fluid per kilogram of body weight must be present in the abdominal cavity of dogs to obtain positive results in most cases using this technique.

**Diagnostic Peritoneal Lavage**
Make a 2-cm skin incision just caudal to the umbilicus and ligate any bleeders to avoid false-positive results. Spread loose subcutaneous tissues and make a small incision in the linea alba. Hold the edges of the incision with forceps while the peritoneal lavage catheter (Stylocath) without the trocar is inserted into the abdominal cavity. Direct the catheter caudally into the pelvis. With the catheter in place, apply gentle suction. If blood or fluid cannot be aspirated, connect the catheter to a bottle of warm sterile saline and infuse 20 mL/kg of fluid into the abdominal cavity. When the calculated volume of fluid has been delivered, roll the patient gently from side to side, place the bottle on the floor, vent it, and collect the fluid by gravity drainage. Do not be surprised if you do not retrieve all of the fluid, particularly in dehydrated animals.

**Exploratory Laparotomy**
Perform a ventral midline incision from the xiphoid process to the pubis. Obtain a sample of fluid for culture and analysis. Explore and inspect the entire abdomen. Find the source of infection and correct it. Break down adhesions that may hinder drainage. Lavage the abdomen with copious amounts of warm, sterile saline if the infection is generalized. Remove as much necrotic debris and fluid as possible. Close the abdomen routinely, place an abdominal drain, or perform open abdominal drainage.

**Prognosis**
The prognosis for animals with generalized peritonitis is guarded; however, with proper and aggressive therapy, many survive. Some authors have suggested that the mortality rate approaches 50%. The mortality rates reported in animals with generalized peritonitis treated with open abdominal drainage have varied from 20% to 48%.
SPLENECTOMY
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INDICATIONS
Splenectomy is indicated for removal of splenic neoplasm, rupture, torsion, infarct, abscess and hypersplenism.

PATIENT POSITIONING
The patient is placed in dorsal recumbency for routine celiotomy.

RECOMMENDED INSTRUMENTS
A Balfour self-retaining abdominal retractor is essential to maintain adequate exposure allowing complete exploration of the abdominal cavity as well as visualization of the splenic blood supply. When large amounts of blood or fluid are present in the abdominal cavity suction is helpful. It is best to have a variety of sizes of hemostats available. The author recommends a minimum of 4 medium to large hemostatic forceps (Crile, Kelly or Carmalt) and 4 – 5 small hemostatic forceps (mosquito).

Ligation of individual blood vessels or clusters of vessels is performed using 3-0 or 4-0 synthetic absorbable suture material. Common sutures include Biosyn, Monocryl, Dexon, Vicryl, Polysorb, PDS or Maxon.

SURGICAL TECHNIQUE
A ventral midline incision from xyphoid to pubis is made to allow adequate exposure of all abdomen organs. The spleen is located in the cranial left quadrant of the abdominal cavity just caudal to the greater curvature and fundus of the stomach. A Balfour self-retaining retractor is positioned to provide exposure of the abdominal cavity.

The spleen is identified, and gently elevated through the abdominal incision. If the surgeon is dealing with a bleeding spleen (e.g., hemangiosarcoma) the exteriorized spleen is placed across the body wall to help place pressure on the splenic blood vessels. In addition, a dry laparotomy pad can be placed directly on the point of hemorrhage and gentle pressure applied.

Several structures should be identified. The greater curvature of the stomach, dorsal and ventral layers of the greater omentum, the gastroplenic ligament and the left limb of the pancreas. Trace the splenic artery and vein as they course from the dorsal layer of the greater omentum into the gastroplenic ligament. Identify the left gastroepiploic artery and vein, the many splenic arterial and venous branches into the hilus of the spleen, the short gastric vessels and the vessels continuing into the greater omentum.

The spleen receives its blood supply from 3 major sources. Three to five short gastric vessels supply the cranial aspect of the spleen. The central portion of the spleen is supplied by the major splenic artery and vein and the caudal pole of the spleen by 4-5 small omental tributaries.
The spleen can safely be removed using a technique requiring only 3 to 4 ligatures. Visualization of these vessels is accomplished by first elevating the spleen from the abdominal cavity. When attempting to exteriorize the spleen it is noted that the cranial pole is tethered by the 3 to 4 short gastric vessels. These vessels are identified and cluster ligated with two encircling ligatures. The vessels are transected between ligatures thus releasing the tethering effect. The spleen can now be further mobilized from the abdominal cavity allowing easy exposure of all remaining vessels.

Next the major splenic artery and vein is located and ligated prior to its bifurcation. Care should be taken to visualize the left limb of the pancreas and make certain it is a safe distance from the proposed ligature site. This splenic artery and vein are generally double ligated and depending upon size the artery can be transfixed. Finally the remaining vessels supplying the caudal pole of the spleen are cluster ligated using one or two ligatures.

During the procedure, several points should be remembered:
1) identify the location of the pancreas and do not occlude its blood supply
2) double ligate all major vessels
3) carefully inspect all ligated vessels for evidence of hemorrhage

CLOSURE
The Balfour retractor is removed and the abdominal incision is closed in a routine fashion.

POSTOPERATIVE CONSIDERATIONS
Postoperative care involves monitoring the patient for blood loss that may be encountered should a ligature slip from the ligated vessels.
FOCUS ON INFECTION PROGRAM
GUIDELINES FOR THE DIAGNOSIS OF URINARY TRACT INFECTIONS IN DOGS AND CATS
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INTRODUCTION
Urinary tract disease is commonly in dogs and cats, and a leading reason for antimicrobial use. Proper diagnostic and treatment plans are critical for optimal patient care and prudent (and effective) antimicrobial use. In human medicine, detailed guidelines are available and provide excellent guidance to physicians on management of various infectious diseases, including urinary tract infection (UTIs). Practice guideline development is a relatively new phenomenon in veterinary medicine and is hampered by a relative lack of adequate research. However, a combination of available data, general principles of infectious diseases and antimicrobial therapy and expert opinion have been used to develop preliminary guidelines for urinary tract infections.¹

All urinary tract infections are not alike, and the approach to diagnosis and management can be different. Current guidelines have addressed lower urinary tract disease under three main areas, simple uncomplicated UTI, complicated UTI and subclinical bacteriuria. While these may provide a reasonable overview, there is more complexity to disease and there may be important subgroups within these categories. As a result, guidelines are being broadened to cover a range of more distinct entities, including first instance UTI in female and neutered male dogs (comparable to simple uncomplicated UTI), first episode UTI in the cat, first episode UTI in the intact male dog, recurrent UTI, UTI in the compromised dog or cat, pyelonephritis, bacterial prostatitis, UTI in the pregnant bitch, subclinical bacteriuria, urinary catheters, urologic impact and medical management of infection-induced uroliths. Some of these are highlighted below.

SPORADIC BACTERIAL CYSTITIS IN FEMALE AND NEUTERED MALE DOGS
These are cases of cystitis that occur in an otherwise healthy individual with normal urinary tract anatomy and function, and no relevant comorbidities. The presence of relevant comorbidities (e.g. diabetes mellitus, urinary or reproductive tract conformational abnormalities) or 3 or more episodes per year indicates a different category of infection and the need for other considerations.

A key aspect for this, and other categories, is the presence of infection (cystitis). Infection implies disease, not solely the presence of bacteria in the urine (bacteriuria, discussed below). Thus, UTI implies the presence of a clinical abnormality, and is characterized by dysuria, pollakiuria and/or increased urgency of urination along with the presence of bacteria in urine.² These clinical signs are not pathognomonic for infection and can also be caused by non-infectious conditions, and the likelihood that dysuria relates to an infectious cause varies between species and circumstances.

Clinical signs are non-specific and should not be used alone for diagnosis of UTI. Rather, the presence of clinical abnormalities should indicate the need for further testing.

Sediment analysis alone is inadequate for diagnosis of UTIs because of problems regarding the variable quality of interpretation, stain contamination and false positive results from bacteriuria in the absence of clinical infection, but identification of cytological abnormalities is an important step in the diagnostic process, particularly when clinical signs are vague. Complete urinalysis, including urine specific gravity, urine glucose level determination and examination of the sediment for crystalluria is considered a minimum database for evaluation of suspected UTI and
may be helpful to investigate underlying causes of infection, if present. Pyuria and hematuria are not, themselves, indications of a need for antimicrobials.

Urine samples should be collected in all cases, and this should be done by cystocentesis whenever possible. Culture should be performed and samples should be submitted to the laboratory as quickly as possible. Results of samples that take 24 hours or more to reach the laboratory should be interpreted with caution because of the potential for both false positive and false negative results, particularly if a urine preservative was not used.

Urinary tract infections tend to be caused by a limited range of bacteria, particularly *E. coli*. The ability to achieve high concentration of some antimicrobials in urine is of great benefit and can simplify treatment of UTIs, even with resistant microorganisms. However, emergence and dissemination of some highly drug resistant bacteria have created challenges.

Empirical treatment is reasonable while awaiting culture results or in situations where owners will not consent to culture. A range of antimicrobials can be considered but guidelines from the International Society for Companion Animal Infectious Diseases (ISCAID) recommend amoxicillin (11-22 mg/kg PO q8-12h) or trimethoprim-sulfonamide (15 mg/kg PO q12h) (Tables 1 and 2) as first line options. Amoxicillin/clavulanic acid (12.5-25 mg/kg PO q8-12h) could also be considered but there is a lack of evidence regarding the need for clavulanic acid since it is ideal to use the narrowest spectrum that is possible, amoxicillin is preferred initially. While these are reasonable empirical options, pathogen and resistance patterns vary regionally and veterinarians should be aware of the pathogen and antimicrobial resistance trends among urinary pathogens isolated from patients in their clinic.

A key concept in infectious diseases is ‘treat the patient, not the lab result’, and this applies for UTIs. If culture and susceptibility testing indicates the presence of an isolate that is resistant *in vitro* to initial therapy but there has been apparent clinical response, maintaining the current treatment is acceptable provided a follow-up urinalysis, including culture, is performed after treatment has been completed to ensure resolution of infection. If culture and susceptibility data indicate that the isolate is not susceptible to the chosen antimicrobial and there is a lack of clinical response, then therapy with the original drug should be discontinued and treatment with an alternative drug begun.

In humans, treatment of UTIs is typically of short duration (e.g. 3 days). In animals, longer durations have typically been used, with no clear reason. Inadequate research is available to guide duration recommendations for dogs and cats; however recent studies have provided evidence that short term treatment (e.g. 3 days) may also be effective in dogs. Thus, 3-5 days of treatment may be adequate for uncomplicated infections, and probably increase compliance and decrease the risk of adverse antimicrobial effects (including resistance).

There is no indication for measures beyond monitoring of clinical signs. Provided the full course of antimicrobials is administered correctly, there is no evidence that intra- or post-treatment urinalysis or urine culture is indicated in the absence of ongoing clinical signs of UTI.

**SUBCLINICAL BACTERIURIA**

As noted above, the goal of management is to treat disease. This is not necessarily synonymous with the presence of bacteria in urine. Subclinical bacteriuria is defined as the presence of bacteria in urine as determined by positive bacterial culture from a properly collected (i.e. cystocentesis) urine sample, in the absence of clinical evidence of signs of lower urinary tract disease. Subclinical bacteriuria is not uncommon, even in individuals with no known
predisposing factors. Rates of 2.1-8.9% have been reported in healthy dogs, with higher rates (15-31%) in groups such as dogs with diabetes mellitus, morbidly obese dogs, puppies with parvoviral enteritis and dogs treated with cyclosporine or glucocorticoids. Study of subclinical bacteriuria has been limited in cats and the prevalence may be lower than reported in dogs, as one study identified bacteriuria in only 0.9% of healthy cats. No evidence of an association between subclinical bacteriuria and risk of development of UTI or other infectious complications has been reported in dogs or cats. A study of healthy female dogs identified bacteriuria in 8.9% of dogs and found no association with subsequent UTI development over a 3 month follow-up period.6 In humans, there is abundant support for not treating asymptomatic bacteriuria (the human analogue of subclinical bacteriuria), even in most compromised patients. While bacteriuria rates are high in various populations (e.g. diabetics, the elderly, patients with paralysis), treatment guidelines such as Infectious Diseases Society of America Guidelines for the Diagnosis and Treatment of Asymptomatic Bacteriuria in Adults do not recommend treating asymptomatic bacteriuria in almost all patient groups.7 Exceptions are patients undergoing transurethral resection of the prostate and patients that will be undergoing urologic procedures that result in mucosal bleeding. Screening and treatment of pregnant women is recommended; however, this has recently been questioned because while an association between untreated bacteriuria and pyelonephritis was identified, the low burden of pyelonephritis and potential adverse effects of antimicrobials may not justify universal treatment. Treatment is specifically not recommended for pre-menopausal, non-pregnant women, those with diabetes, older individuals in the community, elderly institutionalized individuals or individuals with spinal cord injuries. Thus, even in what would be considered high-risk populations, treatment of asymptomatic bacteriuria is discouraged and intensive measures are used to reduce the treatment of asymptomatic bacteriuria. These efforts are typically focused around antimicrobial stewardship from an antimicrobial resistance standpoint, but reduction in unnecessary treatment is also desirable because of cost, adverse effects of antimicrobials, and lack of evidence that treatment improves outcome in almost all patient groups. While treatment might eliminate the current bacteriuria event, recolonization often follows. A systematic review in humans concluded that while bacteriuria may be eliminated in the short-term, the effect is not sustained and re-colonization is common, leading to no impact on overall morbidity or mortality.8 Further, two studies have reported significantly higher bacteriuria recurrence rates in women treated for asymptomatic bacteriuria compared to untreated controls.9,10 Treated women also had higher rates of antimicrobial resistance relaxation.

UPPER URINARY TRACT INFECTIONS (PYELONEPHRITIS)
Given the potential severity, accurate and prompt diagnosis is required to institute effective treatment as soon as possible. Whenever pyelonephritis is suspected, culture and susceptibility testing should always be performed. As with lower UTIs, cystocentesis samples should be used for culture whenever possible. Parallel blood culture may also be useful. Imaging is particularly important to determine whether pyelonephritis may be present. Interpretation of susceptibility data should be based on antimicrobial breakpoints for serum rather than urine concentrations, since renal tissue levels are the key, not urine levels.

Immediate treatment is indicated, using an antimicrobial with good activity against Gram negative Enterobacteriaceae. If ascending infection is suspected, urine culture results obtained for diagnosis of lower UTI might be the basis of initial therapy. If the upper UTI results from hematogenous spread, initial therapy should be based on cultures of blood or the infected site, whenever available. Otherwise, empirical therapy with a drug typically effective against Gram negatives should be chosen. Knowing resistance trends in E. coli from UTIs in the practice can be helpful to guide initial therapy. Combination therapy can be considered initially, with changes potentially made based on culture results. If combination therapy was initiated and the isolate is susceptible to both drugs, one might be discontinued if supported by evidence of clinical
response. If resistance is reported to one of the drugs, that antimicrobial should be discontinued. A second drug to which the isolate is susceptible should be substituted if the patient has not responded sufficiently; substitution is not necessary if patient response has been sufficient. There is little evidence to guide duration of treatment. Treatment of 4-6 weeks is often recommended and that is reasonable, although a shorter duration of therapy might be effective.

**Table 1**: Selected antimicrobial treatment options for urinary tract infections in the dog and cat (draft revision of 1).

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amoxicillin</td>
<td>11-15 mg/kg PO q12h</td>
<td>Good first-line option for bacterial cystitis in the absence significant tissue involvement (e.g. prostatitis, pyelonephritis). Excreted in urine predominantly in active form if normal kidney function is present.</td>
</tr>
<tr>
<td>Amoxicillin / clavulanate</td>
<td>12.5-25 mg/kg PO q12h</td>
<td>First-line option for bacterial cystitis in the absence significant tissue involvement (e.g. prostatitis, pyelonephritis).</td>
</tr>
<tr>
<td>Cefovecin</td>
<td>8 mg/kg single SC injection.</td>
<td>Duration is longer than is typically needed. Should only be used in situations where oral treatment is problematic. Enterococci are resistant. Limited efficacy against Enterobacteriaceae apart from cystitis. Lack of clinical breakpoints hampers assessment of susceptibility, especially in tissue-associated infections.</td>
</tr>
<tr>
<td>Cefpodoxime proxetil</td>
<td>5 to 10 mg/kg q24h PO</td>
<td>More active than cephalexin or cefadroxil against Enterobacteriaceae. Enterococci are resistant.</td>
</tr>
<tr>
<td>Doxycycline</td>
<td>5 mg/kg PO q12h</td>
<td>Reserved for infections caused by pathogens that are resistant to drugs that are actively excreted in urine in active form.</td>
</tr>
<tr>
<td>Fluoroquinolones</td>
<td>various</td>
<td>Excreted in urine predominantly in active form. Reserve for documented resistant cystitis but good first line choice for pyelonephritis in dogs at the higher end of the dosing range. Considered first-line choice for infections that involve the prostate. Not recommended for enterococci. Enrofloxacin not recommended for cats because of the risk of retinopathy at high doses and resistance emergence at lower doses.</td>
</tr>
<tr>
<td>Nitrofurantoin</td>
<td>4.4-5 mg/kg PO q8</td>
<td>An option for some cases of cystitis, particularly when multidrug resistant pathogens are involved. Not appropriate for tissue-associated infections.</td>
</tr>
<tr>
<td>Trimethoprim-sulfonamide</td>
<td>15-30 mg/kg PO q12h</td>
<td>Concerns regarding idiosyncratic and immune-mediated adverse effects in some patients, especially dogs and with prolonged therapy. Avoid in dogs that may be sensitive to potential adverse effects such as KCS, hepatopathy, hypersensitivity and skin eruptions.</td>
</tr>
<tr>
<td>Fosfomycin</td>
<td>40 mg/kg PO (with food) q12h</td>
<td>Should be reserved for multidrug resistant infections. Not appropriate for tissue-associated infections. Efficacy in dogs or cats is undetermined.</td>
</tr>
</tbody>
</table>
References
BACTERIAL TRANSLOCATION – FACT OR FICTION?

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Introduction
Translocation is the passage of viable microbes - bacteria or yeast - across an intact epithelium from the lumen of gastrointestinal tract into the mesenteric lymph nodes, lung, liver, spleen, blood, peritoneum and lymph. Many human patients dying of multiple organ system failure have been found to have no septic focus either clinically or at autopsy. As a result the gut has been focused on as a potential source of pathogens. It is also felt that the passage of endotoxins across this barrier may be as important as the passage of microorganisms. Although it is well recognized that bacteria can translocate, at this time there is no direct proof that sepsis of gut origin is a cause of multiple organ failure.

There appear to be 3 general mechanisms which promote translocation: 1) disruption of indigenous enteric flora resulting in bacterial overgrowth with enteric bacilli, 2) impaired host immune defenses, 3) physical damage of the intestinal mucosa. Translocation has been shown to occur following malnutrition, starvation, burns, trauma, total parenteral nutrition, immune suppression, abdominal radiation and exposure to endotoxin. Gut atrophy and bacterial translocation can occur within hours of acute illness, injury or burn.

The Gut as a Source of Pathogens
Physical disruption of the gastrointestinal tract characteristically yields growth of enterococci, E. coli and B. fragilis. Pseudomonas, Candida and S. epidermidis have been found to be common isolates in ICU infections in patients with multiple organ failure. They have also been found to be common isolates in gastric and jejunal fluid. Systemic Candidiasis has been shown to be significant in post surgical patients, especially those with burns. This is thought to be due to antibiotic therapy which causes overgrowth of this yeast.

Defenses
The defense against bacterial translocation depends on a healthy gut, physical barriers such as the presence of gastric acid, mucus and tight junctions between cells, normal motility, phagocytic cells and gut associated lymphoid tissue.

Overgrowth of bacteria in the stomach appears to depend on the level of acidity and the lack of motility. These bacteria are a source of infection for the respiratory tract leading to nosocomial pneumonia and may also lead to overgrowth in the small intestine. In cimetidine treated patients bacterial overgrowth of predominantly intestinal coliforms has been shown to be rapid when the pH is kept above 4.

Circulation
In people approximately 30% of the cardiac output is directed to the gut through the celiac, cranial and caudal mesenteric arteries and seventy-five percent of this is distributed to the mucosa. Microcirculatory mechanisms act to sustain the flow of blood in the gut despite up to a 70% decrease in arterial flow, although sympathetic stimulation will direct blood toward the submucosa away from the mucosa potentially leading to a severely compromised mucosal blood supply. Due to the close proximity of the blood vessels in the intestinal villus a
countercurrent mechanism exists causing oxygen to diffuse rapidly between the arterioles and venules. Under normal circumstances as much as 80% of the blood oxygen diffuses directly out of the arterioles into the venules without getting near the tips of the villi. During episodes of vascular compromise oxygen delivery to the villus tip can be severely compromised leading to necrosis. Vasodilation of the gut circulation occurs though stimulation of motility as well as hormonal secretion which can be triggered by parasympathetic stimulation, enteral feeding and some glands secrete kinins with potent vasodilatory effects. Decreased oxygen can also lead to vasodilation possibly through the release of adenosine. Under normal circumstances the increased metabolic rate during gut activity may be sufficient to decrease the oxygen levels to cause this vasodilation.

Hypovolemia has been shown in dogs to result in sloughing of intestinal mucosal cells, cessation of mucus production and an increase in opening of tight junctions. The presence of pancreatic elastin, trypsin and chymotrypsin appear to contribute significantly to the loss of the physical barrier in the ischemic gut.

**Reperfusion**
Hypoxia has been thought to be the key factor in the development of intestinal mucosal lesions; however, there is substantial evidence to show that reperfusion after ischemia with the release of reactive oxygen species significantly worsens the lesions.

Translocation of endotoxin may be more important than the translocation of bacteria. Endotoxin increases the permeability of the gut to bacteria, impairs host immune defenses and is associated with conditions resulting in multiple organ failure. It is common after thermal or mechanical trauma. Absorption of endotoxin could lead to monokine and macrophage activation leading to the hypermetabolism of the sepsis syndrome. Endotoxin promotes bacterial translocation in a dose-dependent fashion from gut to the mesenteric lymph nodes, and has been shown to be present after 2 hours of shock in rats. Endotoxin only absorbs in the first 24 hours after hemorrhagic shock; bacteria were present up to 48 hours after administration of endotoxin unless the host immune system was otherwise affected. Endotoxin also promotes overgrowth of bacteria.

**Immune System**
Compromise of the host defenses may cause spread of the bacteria to body organs. Secretory IgA (S-IgA) is responsible for preventing the uptake of enteric antigens and for preventing the binding of enterotoxins and microorganisms to the intestinal microvilli. Intestinal antigenic stimulation is the primary mechanism by which a secretory immune response is generated. Stimulated gut B cells from Peyer's patches produce S-IgA which enters the thoracic duct via the mesenteric lymph nodes and is released into the circulation. From there the cells travel to distant secretory sites and produce S-IgA. The exact relationship between S-IgA and translocation is uncertain. Impaired T-cell immunity appears to lead to bacterial translocation and increases the survival of bacteria that reach the mesenteric lymph nodes, especially under conditions of other stresses such as burns; however, since endotoxin can promote translocation in macrophage-deficient mice it appears it can occur without activating the reticuloendothelial system. Animals with genetic resistance to the systemic effects of endotoxemia were not resistant to the endotoxin-mediated bacterial translocation suggesting translocation is not mediated by activation of the immune system. Immunosuppression causes translocation to the mesenteric lymph nodes, spleen, liver and kidneys; however, with antibiotics the bacteria spread systemically. Antibiotics allow overgrowth of indigenous gram negative facultative enteric
bacilli with subsequent translocation to the mesenteric lymph nodes which has been shown to resolve on discontinuation of the antibiotic.

**Shock**
Bacterial translocation occurs within 2-4 hours of severe hemorrhagic shock. The longer the shock the worse the ileal necrosis and the higher the bacterial counts. After hemorrhagic shock and resuscitation in animals, bacterial translocation no longer occurs after 24 hours. Similar results have been shown in people experiencing severe shock with no evidence of hollow viscus injuries.

**Intraabdominal Pathology**
Gentle manipulation of the bowel causes bacteremia and as a result it has been speculated that transient translocation may occur fairly commonly even with an intact immune system; however, in a series of patients undergoing abdominal surgery and with confirmed translocation none developed postoperative infections.

**Mechanism of Translocation**
Bacterial translocation was first suggested by Fine in 1950 following experiments in which gut-derived E. coli were recovered from the peritoneum. Translocation probably occurs across the ileal and cecal mucosa. Bacteria translocate to the mediastinal lymph nodes at which point they either continued to spread to other organs in the body or are contained in the lymph nodes. In a hemorrhagic shock model the bacteria appeared to travel from the mediastinal lymph nodes via the lymphatics to the thoracic duct and from there to the lungs. From the lungs the bacteria are transported to the liver, kidney and spleen.

**Nutrition**
The gastric mucosa has extremely low glycogen stores and is dependent on a continuous supply of energy substrates. Intraluminal glucose has been shown to provide significant protection to ischemic cells of the small intestine. Glucose has been shown to protect gastric cells but this has been speculated to occur from systemic absorption rather than local protection. Enteral nutrition was compared with cimetidine in the prevention of gastrointestinal bleeding and was found to be far more effective. Malnutrition causes a decrease in the height and density of intestinal villi (most pronounced at 21 days) but does not disturb the integrity of the barrier and prolonged malnutrition actually decreases the ability of the bacteria to colonize the gut. Intravenous nutrition leads to villus hypoplasia, and TPN has been shown to promote bacterial translocation. A significant increase in the cecal bacterial count, decrease in the S-IgA levels and increase in the translocation of bacteria to the mesenteric lymph nodes (despite weight gain) has been shown following 2 weeks of TPN. The gut exhibits accelerated activity in critical illness despite lack of enteral feeding. Immediate enteral nutrition can inhibit the hypermetabolic response by 80%.

References available on request.
Canine Lyme Consensus (and lack of) Update

Michelle Evason, BSc, DVM, DACVIM (SAIM)

In 2006 the American College of Veterinary Internal Medicine (ACVIM) published a Consensus statement on Lyme disease in Dogs (1). In 2016, this resource was updated and presented at the annual ACVIM conference.

Background:

Due to the continued emergence of Lyme disease (Borreliosis), this medical concern has become an increasingly common Canadian veterinary clinic and pet-owner discussion (and dilemma). This is the direct result of the increasing number of *Ixodes scapularis* ticks that continue to ignore the border, immigrate and thrive on Canadian soil. Hot-spots for these ticks appear to be south-eastern and south-central Canada; specifically certain regions of Ontario, Manitoba, and parts of the Maritimes. Within Ontario, *Ixodes scapularis* is endemic to hyperendemic (i.e. occurring with high regularity) in some parts of eastern Ontario (e.g. northeastern shore of Lake Ontario and the St. Lawrence river) along with some focal regions in the southwest (e.g. Long Point, Turkey Point). Sero-positivity to *Borrelia burgdorferi* is increasingly documented in other parts of Canada, in parallel with *Ixodes* sp. tick movement and habitat expansion, along with people travelling with their pets to endemic Lyme areas.

Context:

Before plunging into Lyme disease, it’s important to remove the ‘fear factor’ element and put things into context for pet-owners and us. To begin, while Lyme (*Borrelia* sp. seropositivity) has become more common in Canada; Lyme disease remains uncommon. One example that might aid perspective would be comparing Lyme disease to canine obesity. We think that of 100 dogs that are seropositive for *Borrelia burgdorferi* about 5 of them will develop clinical signs of Lyme disease (2). And we know that 40-60 dogs out of 100 that walk into a veterinary clinic are either obese or overweight (3-5). So…context is important. Risk assessment for likely outcome of clinical disease is similarly important. Again, using the above example, few dogs that are seropositive for *Borrelia burgdorferi* will develop clinical illness and IF they do, response to therapy is excellent (rapid and complete) in the vast majority of cases (1). In the canine obesity example, many dogs have concurrent disease (e.g. osteo-arthritis, risk of cardiac and endocrine concerns), therapy is difficult and relapse highly likely- thus treatment failures with obesity are common. Perspective on Lyme disease (i.e. what is the actual risk and outcome?) can be helpful for balanced client and clinic discussions.

Since much of this talk will focus on the ACVIM Consensus guidelines, it is also important to recognize and address why the consensus statement is helpful (or not) as a guide for practitioners- and in which geographic areas.

**Consensus Statement Q & A:**

The following is a brief review (and summary) of the 2006 and 2016 Lyme disease consensus guidelines, and the common questions that arise during ‘Lyme disease’ related discussions.

**First let’s define what Lyme disease is.** This is considered true clinical disease and associated illness. This is NOT seroprevalence, or a positive test result on one of the available testing methods for *Borrelia* sp. Two very different things, and this must be understood and put into context for pet-owners and clinic staff. Next, it is important to understand that there are a series of ‘IF’s’ that have to occur before disease or even the...
question of whether Lyme disease truly is (or more often isn’t) occurring in a given dog arises. Considering criteria for making a clinical diagnosis of Lyme disease is REALLY important. The following algorithm can be used to determine whether Lyme disease is likely in a given dog:

1. Review of Clinical History:
   a. Has this dog been to (travelled to) or currently lives in a known Lyme endemic area?
   b. Has there been tick exposure?
2. Physical exam- Are there consistent clinical signs (e.g. polyarthropathy, fever)?
3. Were other differentials that might cause similar clinical signs excluded? This means make a ‘problem list’ and cross stuff off or not.
4. Diagnosis: Is there documented *Borrelia* sp. seropositivity on a test? Is this result new (i.e. If the dog was tested previously as part of wellness screening, was it negative at that time)?
5. Therapeutic response: If the above criteria were met and this led you treat, was there a rapid response/resolution of illness with doxycycline therapy? If it’s really *Borreliosis/Lyme disease the dog should improve within 48 h, usually sooner (1).

A positive test result for anti-*Borrelia* sp. antibodies does NOT equal a clinical diagnosis of Lyme disease. Despite this, dismissal of a positive test result can be challenging, for both clinicians and concerned pet-owners, even while observing a clinically normal patient or using the above criteria.

*Consensus: Use a Lyme algorithm or template in order to aid diagnoses of Lyme disease.*

**What clinical syndromes are associated with Lyme disease in dogs?**

Clinical signs of Lyme disease fall into two groups, and these are considered common and uncommon (with the above caveat and obesity example for context). Common consists of: acute fever (39.5-40.5°C, 103.1-104.9 °F), polyarthropathy (shifting limb lameness), non-specific signs (lethargy, decrease in appetite) and sometimes mild lymphadenopathy.

A highly uncommon (and never reproduced experimentally) Lyme consequence is ‘Lyme nephritis’, a potentially severe form of protein losing nephropathy (PLN). This is a presentation of severe and rapidly progressive azotemia, polyuria and polydipsia, uremia, proteinuria, which has been described in dogs in Lyme endemic regions.

*Consensus on the common and uncommon clinical signs of Lyme disease.*

**How is Lyme diagnosed?**

There are two categories of diagnostics:

1. Standard diagnostics to work-up a dog that presents with fever and polyarthropathy. These include: CBC, serum biochemistry, urinalyses and potentially joint taps or radiographs, etc. and dependent on clinical judgment, specific tests for *Borrelia* sp. among others.
2. Specific tests to document *Borrelia* sp. seropositivity. These tests include: the Snap 4DX, Accuplex, Multiplex and the VetScan.

*Consensus on diagnosis- all tests are the same in efficacy and work well.*

**How to interpret specific *Borrelia* antibody (sero-positive or negative status) tests?**
This is one reason Lyme disease is so confusing; because these tests aren’t typically used when one is concerned about making a diagnosis of clinical Lyme disease. These tests are being performed as part of a general health screen or for Heartworm screening in healthy dogs. As such, despite potentially high specificities of available tests, when there is a low likelihood that the patient has the disease, the positive predictive value (the likelihood that a positive test truly indicates disease) will be low.

Additionally, while a positive test result is great for discussions on tick prevention improvement, and to emphasize the importance of urinalyses for proteinuria (and as part of a wellness screen in a pet), along with raised human health awareness… it’s not as clear-cut when it comes to making therapy recommendations.

Context is important, and interpretation of diagnostics should reflect which dog group is being tested:
1. Healthy dogs being screened for other things and unlikely to have Lyme disease.
2. A dog with consistent clinical signs like fever and polyarthritis.

Antibody positive doesn’t automatically equal clinical disease. This is particularly true in endemic areas where many dogs are going to be positive, but few have clinical illness related to Lyme disease (2,6,7,8).

To answer the question about test choice, in dogs with consistent clinical signs and history the above tests all work well to detect antibodies (seropositive status) to *Borrelia burgdorferi*. The Quant C6 and Multiplex may be useful to help establish an antibody baseline with a numerical (quantified) result.

*There is no consensus on the use of these quantitative tests as a monitoring tool (1), since changes in antibody titres have not been shown to be predictive of either response or potential for complications.*

To expand on test interpretation, if the test is positive:

1. The dog has antibodies to *Borrelia* sp. as the result of being bitten by an *Ixodes* sp. tick that has attached long enough to infect the dog, typically over 48 h.
2. A positive test provides little context for ‘how long’ or ‘when’ the dog was infected, since antibodies can remain for a long period of time, months to years.
3. Sero-positive status cannot be used to guide therapy choice, or length of therapy required.
4. A positive test can be a great way to start (or repeat) a discussion about tick prevention…since the current program of tick intervention is not working if the dog is positive, ie. a new positive.

*Consensus on test interpretation as above.*

**When to treat for Lyme disease?**

*Consensus: Dogs who meet the above clinical criteria should be treated (i.e. those with clinical abnormalities potentially due to Lyme disease).*

Dogs who do NOT have clinical signs should not be treated; however, they should have their urine checked for proteinuria, consider assessment for other tick borne disease, AND a discussion with their owners regarding tick prevention and public health should occur (1). *No consensus, some panel members treat all non-proteinuric sero-positive dogs with doxycycline. Some advocate for pro/cons discussion with owners.*
But... what do I do with the healthy dog who is sero-positive for *Borrelia* sp?

A swirl of controversy and debate surrounds the topic of therapy of seropositive dogs, and centres on the concern of Lyme nephritis or PLN. As mentioned above, *there is no consensus on this topic*, and much of this issue is on the ‘what if’ and nobody wants to be the ‘what if’ or be the vet who didn’t give the doxycycline and the dog developed PLN and died and the owners are super sad- and so are you. On the other hand doxycycline therapy isn’t completely benign, and we are in an age of antimicrobial stewardship where pros and (perhaps more importantly) cons of antimicrobial use have to be carefully weighed (9).

There is agreement (*consensus*) that all dogs who are seropositive for *Borrelia* sp. should have their urine checked for protein; however, no-one seems to know when to check the urine for protein, how long to monitor this and if there is a decision to treat with doxycycline how long to do this for (i.e. *no consensus*). Not all that helpful for most of us… and there are vocal advocates of treating all seropositive dogs with doxycycline, and others who suggest talking to the client about pros and cons and making a decision together.

What I recommend at present (for Canadian DVMs) is as follows: I would not treat an otherwise healthy, seropositive dog with no proteinuria (again, getting that urine sample is really important here)…but it’s useful to know that there are other specialists who would.

What is therapy for Lyme disease?

*Consensus:* At present treatment *is* to advise doxycycline at 10 mg/kg q 12h or q 24h for 30 days. *There is no consensus on using this dose q 12 h or q 24h.* Early therapy has been shown to result in lowered antibody titres, quicker resolution of clinical signs, and reduction in *Borrelia* sp. numbers. Additional therapy could include analgesia for lameness-associated pain, in addition to reduced activity (rest).

Should monitoring (repeat serology) occur?

It’s important to note that many/most dogs will remain seropositive (for months to years) despite antimicrobial therapy and resolution of clinical signs. *As such seropositive status should not be used to guide further (or ongoing) therapy,* consensus.

Therapy of Lyme nephritis dogs with PLN varies markedly with renal IRIS stage and illness severity, and is beyond the scope of this talk (10). Briefly, there is *no consensus on therapy for Lyme nephritis dogs,* for how long to monitor, or how to monitor.

*Consensus-* Ensure rule-out and test for all (any) cause of PLN. If elect to treat for Lyme nephritis therapy is doxycycline, additional therapy for PLN (based on IRIS stage), and potentially consider addition of mycophenolate based on lack of therapy response (1,10). Specialist consultation is advised for these cases (or suspects).

What is prevention?

Lyme disease IS preventable, and tick attachment prevention recommendations for dogs are strongly encouraged. There are many excellent (and effective) products available. These include collars, oral products, and topicals that stop tick attachment or kill shortly after tick attachment.

Tick intervention is the most important recommendation a vet clinic can make, follow up with, and re-discuss when (or ideally before) that test lights up positive. It’s also good to review appropriate tick removal with clients.
Consensus: Advise tick prevention that works in < 24 h of tick attachment.

Vaccines: Another hot Lyme topic is vaccines, as in “Do I (or should I) recommend them?” This also includes ‘Do they work? For how long?’ and ‘For what geographic area are they appropriate (Lyme endemic, emerging or all)?’

No consensus on ‘to or to not’ vaccinate, but consensus on the below:

Vaccine products these days are safe, effective and have fewer side effects than before- along with a longer duration of immunity. The counter point would be, why vaccinate if the dog is unlikely to be exposed based on geography, or has low risk of becoming ill. It’s also important that vaccines are not considered an ‘instead of tick prevention’ option. There is also a concern about vaccines causing or leading to protein losing nephropathy- so again a discussion about pro/cons.

In the updated (2016) ACVIM consensus statement, there is supposed to be a pro/cons vaccine table to help with decision-making and owner discussions.

Research needed:

It is clear that further research (and studies in Lyme disease emerging areas) is needed to fill existing knowledge gaps. Ideally, additional research will resolve current lack of consensus, lead to improved understanding and optimize recommendations and care for dogs- and their owners.

References:

THE PERFECT STORM? REDUCING INFECTIOUS DISEASE IN K-9 GROUP SETTINGS

Jason W. Stull, VMD, MPVM, PhD, DACVPM

Introduction
Settings and events where groups of dogs come together, such as shows, sporting events, dog parks, dog daycare, and boarding, are an excellent opportunity for canine and human socialization, bonding and benefit. However, these settings also provide a high risk for spread of infectious diseases. These shared environments are generally temporary, and include many dogs, some from other regions of the country or different countries. These events have the potential to spark disease outbreaks that can move into the local community and then spread rapidly to other locations. Dog group activities and events are very common, yet the occurrence of outbreaks and recommendations to control and prevent disease risks are not well known. Veterinary staff has an important role to play in educating participants and organizers and serving as a critical resource in developing and implementing infection control plans and policies.

Beginning in 2015, a working group encompassing veterinary infectious disease experts from the USA and Canada researched and drafted evidence-based guidelines to inform and reduce the risk of infectious disease transmission for dogs in group settings. Draft guidelines were developed following an extensive literature review. Draft guidelines were later finalized following a survey and focus group of constituents directly involved with a variety of canine group settings. A number of resulting products from the working group are available and aimed at veterinary professionals and the public (http://go.osu.edu/IDk9risk).

Level of Risk
Over 20 published reports of outbreaks involving canine infectious diseases in group settings were identified. These outbreaks included shelters, kennels, veterinary facilities, pet stores, and training centers. The number of dogs affected ranged from a few dogs to over 1,000 dogs. Common themes included high dog density/dog-dog contact, inadequate quarantine of new/returning dogs, poor dog confinement/wildlife exclusion, inadequate vaccination, poor vector control, and inadequate disinfection practices. Given underdeveloped surveillance systems for companion animal diseases, these reports likely far underestimate the occurrence of such outbreak events. Robust companion animal surveillance programs are strongly needed at the facility/program, local and national levels in order to further gauge need and direct implementation of recommendations. An existing local effort (e.g., Worms and Germs Map http://www.wormsandgermsmap.com) is an excellent step and veterinary facility participation is encouraged.

Additionally, some canine infectious diseases (especially many of the tick-borne diseases) are zoonotic. Many of these diseases in people are reportable to human public health groups - human disease occurrence (locations, time of year) can help inform risks for dogs.

Recommendations
Based on summarized disease characteristics and modes of transmission, prevention and control recommendations were developed and grouped into related categories. In total, 64 recommendations were developed; each was assigned an evidence-based score (ranging from "strongly recommended and supported by well-designed studies" to "suggested for implementation and supported by limited clinical or epidemiologic studies").
Nine areas were identified for recommendations. Below is a subset of the recommendations for each area. The complete list of recommendations, accompanying level of evidence, and supporting documentation is available as an open-assess peer-reviewed journal article. It is important to recognize that not all recommendations are applicable or able to be incorporated into all canine group settings – participants, setting organizers and veterinarians need to carefully evaluate setting risks and to choose and adapt recommendations to best address these risks.

1. General Recommendations
   - Develop prevention and control protocols based on a risk assessment (e.g., geography, ages of dogs involved)
   - Attending/consulting licensed veterinarian familiar with the event on or off-site to provide expertise and develop protocols for the specific event/activity
   - Only dogs without evidence of infectious disease able to participate or be present at the event

2. Vaccination Recommendations
   - Core for group settings: Distemper, Adenovirus, Parvovirus, Rabies, Bordetella, Parainfluenza
   - Verify vaccination status and time for immunity
   - Noncore based on risk (Lyme, leptospirosis, canine influenza virus)
   - Weigh benefits vs. risks of dogs with incomplete or inadequate vaccine protective status being present at event (e.g. puppy classes)

3. Insect and Wildlife Control Recommendations
   - Limit insects, rodents, and other wildlife
   - Immediately remove feces, unnecessary organic debris, garbage
   - Pest control program in/around buildings, parks, and kennels

4. Vector Control and Vector-borne Disease Recommendations
   - Environment: based on risks, alter to make less hospitable to fleas, ticks, mosquitos
   - Individual dog: use prevention products prior and during event based on risk (location, season, event)
   - Group: monitor dogs for fleas and ticks; do not allow infested dogs to participate

5. Enteric Disease Recommendations
   - Prompt dog feces removal
   - Endoparasite prevention
   - Fed diet processed to reduce foodborne bacteria

6. Environmental Disinfection and Hygiene Recommendations
   - Develop and use a cleaning & disinfection program
   - Use materials in the setting that are able to be disinfected
   - Staff & owners perform hand hygiene (wash hands with soap and water or use alcohol-based hand sanitizer)
   - Bathe dog before entry/regularly
• Reduce multi-dog contact to items: bring (and use) own items, single dog use items provided by setting

7. Additional Exclusionary Recommendations
• Exclude dogs not formally part of the event (especially young puppies)
• If from outside USA or Canada, exclude dogs from events for 2 weeks upon return

8. Facility Design and Traffic Control Recommendations
• Reduce unnecessary dog-to-dog and person-to-dog contact; use semi-permanent groups when able
• Avoid high density kennel situations (especially if young puppies)
• Have a dedicated isolation area available to temporarily house dogs suspected to be infectious

9. Disease Recognition and Response Recommendations
• Surveillance program in-place to identify and record dogs that develop disease at event and encourage reporting of dogs that develop disease within 2 weeks of event
• Record keeping (location, dog, owner)
• Visually monitor dog health and report dogs with signs of disease
• Immediately remove ill/infectious dogs
• Outbreak management plan highlighting how sick dogs will be identified and how officials will respond

Gauging and Communicating Risk
One of the largest hurdles in promoting change in dog group settings is helping participants, setting owners and organizers recognize disease risks and take the necessary actions to reduce risks. In an effort to help communicate disease risks, an on-line risk calculator was developed (available at http://go.osu.edu/IDk9risk). Through a series of short multiple choice questions, the calculator will help users identify higher risk practices and provide recommendations for reducing these risks. The calculator was developed to take than less than 10 minutes to complete.

Additionally, a White Paper on the topic was written for those who have dogs involved in dog group settings, organize an event, or own/work at a group setting. This document provides an overview of the infectious disease risks in dog group settings with practical, specific recommendations for reducing risks. Tables of key infectious diseases of concern, checklists for event/facility participants and organizers and vaccine and disinfectant recommendations are included to assist in implementation of the recommendations. The white paper can be downloaded at http://go.osu.edu/IDk9risk. This site also contains several fact sheets aimed at dog owners that address key canine pathogens important in the group setting.

Summary
Canine infectious disease risks vary between animals and settings, with many factors being modifiable. Group settings are at high risk for pathogen transmission and potentially outbreaks, where both individual and group decisions alter disease risk. Changes are needed to reduce infectious disease risks with buy-in and desire for change by participants. Enforcement (top-down approach) is unlikely to be solely successful. Rather a collective participant and event staff enforcement model, where all are genuinely invested in success, is likely to make
important improvements in reducing disease risk at group dog events. Veterinary staff provides a key role in educating participants and organizers on disease risks and recommendations to reduce risks. Settings are in need of knowledgeable veterinarians and technicians to assist in infection control plans and policies.

References
CROSS-BORDER CANINES AND IMPORTED INFECTIONS

Maureen E.C. Anderson DVM, DVSc, PhD, Dip ACVIM

A large but unquantified number of companion animals (particularly dogs) are imported into Canada every year. Some of these animals carry pathogens that are uncommon or rare in Canada, and some of which are zoonotic. There is currently no monitoring and minimal control of companion animal importation into Canada. Inter-jurisdictional movement of companion animals within Canada (including movement from remote Northern regions) is associated with similar issues and risk of disease transmission and introduction, and essentially no monitoring or control. Although there is increasing pressure for regulatory changes to be made, the process can be slow and is not without its costs. In the interim, veterinarians have a very important role to play in raising awareness of and detecting these diseases to help prevent them from being imported to new areas, and minimize their impact on local domestic animal, human and wildlife populations.

Number of canine imports to Canada

The number and origin of dogs that are imported into Canada every year are unknown. Recently, an independent group of concerned citizens identified 197 Canadian “rescue” organizations (including SPCAs and Humane Societies) that imported dogs into the country in 2013-2014, and an additional 21 foreign rescues that exported dogs to Canada. In total, 6189 imported dogs from at least 29 different countries were identified through these rescue groups, but this is likely a significant underestimation of the number of animals imported into the country in this timeframe. This number does not include animals that were imported by private individuals (e.g. those who adopted an animal while travelling or working abroad, pets belonging to individuals who immigrated to Canada), nor the frequent movement of pets across the Canada-US border accompanying short-term visitors (e.g. vacationers). There is no registration requirement for rescue organizations, so there are likely others that were not identified by this group. Most of the dogs were imported into Ontario, Alberta and British Columbia.

Current Canadian import requirements

Owned dogs over 3 months of age require a current rabies vaccination certificate, or a veterinary certificate declaring the animal to be from a country recognized by Canada as rabies-free. The veterinary certificate is not required to make any additional statement regarding the health of the animal. There is no waiting period between the time of rabies vaccination and importation, even for primary vaccination. Evidence of a rabies neutralizing antibody titre of at least 0.5 IU/mL is also considered acceptable. Animals that do not meet these requirements are still allowed to enter Canada, but must be vaccinated at the owner’s expense within two weeks, and the vaccination record provided to the Canadian Food Inspection Agency (CFIA). Import permits, health certificates, microchipping or quarantine are not required. There are no specific import restrictions at all for dogs less than 3 months of age, or for assistance/service dogs when the importer is the user of the dog and accompanies the dog into Canada.

The ability to import dogs from the US under the “rescue dog” category, except in the event of a documented natural disaster, was removed in November 2013. Dogs under 8 months of age that were previously imported under this category now have to follow the requirements for commercial entry, which requires an import permit, microchip or tattoo, a veterinary health certificate, and a rabies vaccination certificate. Commercial dogs include dogs for sale, adoption, breeding, show or exhibition, scientific research, etc. It has been suggested that rescue groups may attempt to avoid these requirements by having the dogs “adopted” by
someone in Canada prior to importation, and thus the dogs are imported as owned rather than commercial/rescue animals. Commercial dogs over 8 months of age only require a current rabies vaccination certificate.

The Canadian Border Services Agency (CBSA) has the authority to deny entry to any animal presented for importation, including animals that appear sick with a communicable disease. There is currently no information regarding how often this authority is exercised. In these cases, confinement of the animal and further examination by a CFIA veterinarian at the owner’s expense may be required prior to the animal being returned to its place of origin.

Within Canada, there are no health requirements for movement of companion animals except for those pertaining to humane transportation.

**Falsified documents**

There is growing concern related to the use of fraudulent documentation (both rabies vaccination certificates and rabies antibody titre result reports) to import dogs across international borders. For example, a dog recently imported into the US from Egypt by a rescue organization developed clinical rabies once in the US; on further investigation it was discovered the dog had been imported into the US on the basis of a forged rabies vaccination certificate.

**Welfare concerns**

In some cases there are also significant animal welfare concerns with transportation of dogs, particularly with long-distance movement of animals that are clinically ill. These animals may be confined to transportation kennels in cargo areas for hours with little to no monitoring (e.g. on long-haul trans-oceanic flights), potentially leading to significant physiological and psychological stress.

**Disease risks**

A significant number of imported dogs may subsequently be presented to veterinarians because they are infected with pathogens that are considered “exotic” to Canada, but are not necessarily reportable or notifiable in animals (e.g. *Leishmania* spp., *Brucella canis*). Anecdotal information indicates that imported dogs also have a high frequency of diseases that are present in Canada but are relatively uncommonly seen in the local canine population (e.g. canine distemper). There is potential for some of these pathogens to spread and become established/endemic within the Canadian pet population, and/or within local wildlife populations. In some cases ongoing climate change may also play a role, as insect vector ranges change and increase the potential that competent insect vectors could be present locally. Imported animals have also raised public health concerns because some of the diseases they carry are zoonotic, but animal owners, veterinarians and physicians are often unfamiliar with or unaware of the risks.

The Canadian National Canine Importation Working Group identified the top diseases of concern with regard to canine importation into Canada. Some of the risks and control challenges are briefly summarized here:

**Brucellosis:** There is relatively low risk to public health and the local pet population associated with importation of dogs carrying *B. canis*, unless they are involved in breeding activities. Disease in humans can be severe / chronic in a small percentage of cases. Spayed / neutered dogs are minimal risk for transmission.
Canine Influenza: Although highly infectious amongst dogs, most disease is mild and there is no evidence of increase risk of human infection from currently circulating strains. The virus is unlikely to persist in stable community groups once immunity develops, but can be more problematic in shelter populations. Testing for influenza virus prior to import is impractical based on timing requirements and current cost of testing. A vaccination requirement prior to import could be problematic due to limited availability of vaccine in some areas/countries, and existing vaccines may not provide protection against new/emerging subtypes (i.e. H3N2). Quarantine of imported dogs for 48-96 hours and testing to detect canine influenza would potentially be effective based on the short incubation period of the virus, but is impractical and excessively costly given the limited risk posed to public and animal health at this time.

Leishmaniasis: There is currently limited risk of spread from infected imported dogs to humans or other animals via bites or blood exposure, as long as there is no competent vector present in Canada. However, if a local insect species is found to be a competent vector and comes in contact with a positive animal, the disease could spread to wildlife hosts making it extremely difficult to eradicate thereafter. Northward spread of known competent vectors from the US is also possible, particularly with climate change; however, as the vectors spread the disease will likely spread with them, at which point imported animals would not be significant contributors to the disease issue. High-risk breeds (Foxhounds, Corsicas, Spinones and Neapolitan Mastiffs) from any jurisdiction (especially the US) may pose the highest risk to Canadian dogs, particularly within these breeds where there is evidence of dog-dog transmission. Rescue or commercial dogs from high risk countries can be screened for exposure to Leishmania prior to import.

Rabies: There is significant risk of importation of rabies with severe consequences for exposed animals and people. Some control measures are already in place, but additional measures are required. The current vaccination requirement ensures that each animal has been examined by a veterinarian at some point, but does not necessarily reduce the risk of a rabid animal being imported as there is no waiting period following vaccination.

Alveolar echinococcosis: Infected imported animals are a reservoir capable of causing significant environmental contamination, potentially resulting in infection of people, the consequences of which are very severe. Infection of wildlife intermediate hosts (small mammals, rodents) also increases the local wildlife reservoir, leading to increased risk of spread to both wild canids and other domestic dogs. Treatment of dogs with intestinal infection from endemic regions is relatively simple and non-invasive, and can be done immediately before or immediately after importation. Based on the occurrence of cases to date in Ontario dogs and other species, it appears that E. multilocularis is likely already established in the provincial wildlife population. This makes it difficult to impose border controls if the disease is considered endemic.

Canine lungworm: Negligible public health risk, low to moderate risk to domestic and wild canids. Already endemic in the Maritimes, with increasing evidence of westward spread as far as Ontario. Sensitivity of Baerman fecal evaluation or fecal float is unknown. However, treatment is straightforward and easily accessible (benzimidazoles or avermectins), and is also effective against a number of other endoparasites and ectoparasites (including fleas).

Canine heartworm: Infected imported animals are a reservoir capable of infecting mosquitoes and pose a genuine threat to other canids during mosquito season. Sensitive, specific, relatively non-invasive, readily available testing is available, but must be repeated 6 months after importation due to the long incubation period. Given that the parasite is already present in
Canada (albeit at a much lower level than many other regions) with no control program, it would be difficult to impose national import requirements.

**Tick-borne diseases:** “Adventitial” ticks on imported dogs are of limited concern as they will generally not result in establishment of new tick populations (possible exception of *R. sanguineus*). Dogs are dead-end hosts for most tick-borne diseases of concern and therefore low risk of spreading infection to local tick populations. Examination of all dogs for tick infestations on arrival would be very time consuming, insensitive and impractical based on this risk. Testing of imported dogs for tick-borne diseases is problematic due to reliance on serology which does not differentiate infection from exposure.

**Proposed actions**

Action is needed to help mitigate disease risks from animals being imported into Canada or moved from high-risk areas within Canada, as well as to address concerns regarding the welfare of companion animals during transportation, particularly if they are clinically ill. Key industries involved include transportation companies (particularly airlines), the veterinary profession, and Canadian animal shelters. The pet rescue “industry” plays one of the largest roles, but unfortunately is not regulated or even sufficiently organized or defined at this time to be a useful conduit for action in and of itself.

Action could ultimately be regulatory (either federal or provincial) or non-regulatory (voluntary) in nature, and in either case could be government or industry led, or a combination thereof. Each of these routes has its own advantages and limitations to consider. In any case, measures must be practical and (if applicable) enforceable.

Education of stakeholders, including the public, canine rescue organizations, transportation companies, veterinarians and animal shelters is considered a priority. Education alone will not be sufficient to achieve the necessary behaviour change, but other interventions for management and surveillance are likely to be far more successful if the reasoning behind these measures is better understood. There are no regulatory barriers to educational measures, so these options can essentially be actioned immediately.

Initially, the most feasible and effective option for applying some degree of monitoring and/or control to canine importation is expansion of the existing permitting system for importation of commercial dogs less than 8 months of age to include all dogs (commercial and non-commercial, regardless of age). This would facilitate collection of more detailed information on canine importation in order to better target future interventions, provide a flexible means of applying additional import restrictions as policies are developed, and may help discourage international importation of dogs overall.

Some disease-specific import requirements could significantly help reduce the further spread of certain diseases to and within Canadian domestic animal and wildlife populations. However, due to international trade rules most of these cannot implemented without first establishing control programs for these diseases within Canada.

Ideally, from a disease and risk management standpoint, dog importation would simply be halted altogether. The majority of dog importers are trying to “do the right thing” but are often simply unaware of the problems these animals can have and cause going forward. Realistically, dog importation (both international and domestic) will not be stopped, but the goal is to manage the process without encouraging it. Any system employed also needs to be flexible enough to adjust to changing disease patterns and risks.
Portions of these proceedings are drawn from:


Other References


Import reference documents (as referenced in the Health of Animals regulations):

Importing or travelling with domestic dogs: http://www.inspection.gc.ca/animals/terrestrial-animals/imports/policies/live-animals/pets/dogs/eng/1331876172009/1331876307796


Equine Injectable Anesthesia 2.0

Eric J. Abrahamsen, DVM, DACVA

Injectable anesthesia, sometimes referred to as Total Intravenous Anesthesia (TIVA), can be accomplished with bolus administration or bolus administration followed by a "constant" rate infusion (CRI). The equine injectable anesthesia techniques covered in this lecture can also be utilized in a clinic setting where additional trained help is available to accomplish longer and more complex procedures.

IV Catheter - necessary?

In normal healthy elective patients intravenous (IV) access immediately following anesthetic induction using contemporary drugs is typically not critical. So, is a catheter necessary in cases where you are "reasonably positive" the procedure will be completed without additional anesthetic administration?

Catheter Advantages

Placing a 14g 5.25" (BD Angiocath, Anicath L/A IV, EQUIVET HiFlow) catheter prior to induction minimizes stimulation during administration of the induction bolus. Any anxiety or apprehension will "decentralize" cardiac output to some extent and send an increased portion of the anesthetic induction bolus to the muscles, though good needle technique is generally sufficient (see Pre-medication section). A catheter also provides ready access for administration of additional anesthetic or emergency drugs, should they be required. Hitting a jugular in a struggling horse is not much fun and the likelihood of accidentally administering drug into the carotid artery is increased. A 14g 5.25" catheter is required for induction protocols where guaifenesin is rapidly infused to effect. Guaifenesin is generally only used now for induction of compromised patients to minimize alpha-2 administration.

Heparinized saline (1-2 IU/ml is sufficient for short-term applications) can be made by filling the hub of a 35 ml syringe with 1000 IU/ml heparin and then carefully drawing up 35 ml of saline.

Catheter Disadvantages

Time? It only requires a few minutes to place and secure a catheter. Expense. The cost of a 14g 5.25" catheter, extension set, stopcock (or injection cap), a 35 ml syringe of heparinized saline, and suture to secure catheter (20 g needle & segment of fine spool suture) adds up. An 18g 2" catheter can be placed following needle induction to reduce cost slightly, though at the expense of valuable peak analgesia time. Due to differential movement of skin and underlying structures during transition from standing to recumbency pre-induction standing placement of an 18g 2" catheter is not recommended. Patient discomfort? Moderate sedation? Moderate sedation (perhaps half of the pre-induction xylazine dose) and good technique (rapid insertion of catheter & suture needle) minimize patient response. Catheter placement in foals is a greater "insult" and mishaps more common. I typically needle them down and place a catheter (14g 5.25" or 18g 2") and extension set capped with a stopcock as soon as they are recumbent. Concerns about standing placement of 18g 2" catheters apply to foals as well.
Oxygen Supplementation

Despite well-documented pulmonary dysfunction in recumbent and anesthetized horses, oxygen supplementation is generally not necessary in short-term equine IV anesthesia. Due in large part to their unique oxygen-hemoglobin dissociation curve and the level of cardiac output, arterial oxygen content and oxygen delivery to tissues are generally adequately maintained in normal healthy patients despite even dramatic reductions in $P_aO_2$. Cardiac output (tissue flow) is the more important of these variables in ensuring adequate tissue oxygenation. Tissues are able to cope with reductions in arterial oxygenation by using locally controlled vasodilation, but only a modest increase in oxygen extraction is available to cope with reductions in flow.

Arterial blood pressure, an imperfect surrogate of cardiac output, has been traditionally monitored during anesthesia to evaluate tissue perfusion. As with all monitoring modalities an understanding of its limitations is important in evaluating the information provided. Like us, the brain monitors central pressure and uses adjustments in peripheral resistance to maintain blood pressures at the expense of tissue flow, allowing large changes in cardiac output to occur with little change in arterial blood pressure.

Monitoring arterial blood pressure is still important and easily accomplished in the field setting by assessing turgidity of peripheral arteries. The facial and dorsal metatarsal arteries are the best sites for making this evaluation due to their superficial location between skin and underlying bone. Developing good digital evaluation skills requires practice and “known” values for comparison, which for individuals working in strictly ambulatory practices can simply be the turgidity of normal standing patients, post-ketamine induction, compromised colic, etc. Battery powered patient monitors are becoming more cost effective and a unit equipped with indirect (oscillometric) blood pressure capability can be attached to the tail when procedure allows. Inotropes such as dobutamine can be infused in the field setting to augment cardiac output to help maintain adequate tissue oxygen delivery.

Supplemental oxygen does improve oxygenation, but its effectiveness diminishes as the degree of pulmonary shunting increases. Supplemental oxygen has been traditionally recommended for injectable anesthesia cases exceeding 1 hour in duration. Whether it makes a difference clinically or not, using nasal insufflation or with a demand valve attached to an endotracheal tube may be a good idea from a liability standpoint when duration of IV anesthesia is expected to be long, especially cases involving dorsal recumbency. Using a battery-powered portable pulse oximeter to follow oxygen saturation ($S_\text{a}O_2$) levels can help determine when supplemental oxygen may be warranted (e.g. $S_\text{a}O_2$ values < 92).

Fasting Prior to Anesthesia

I no longer recommend the routine complete fasting of equine patients prior to anesthesia. Extensive experience has convinced me there are no problems associated with allowing horses to nibble on hay up until the time of surgery. The stress of muzzling and the potential for upsetting an already temperamental gastrointestinal system do not justify this dogmatic ritual any longer. An additional benefit of not fasting is the flexibility it affords. There is less urgency to get the case done when the patient is not being fasted. I still recommend grain be withheld the day of surgery. If the horse is a pig about eating, periodically feeding light amounts of hay can be used to help prevent an engorged stomach during anesthesia. There are a few "exceptions" to my no fasting rule. When you are anticipating a longer duration of dorsal recumbency in a field anesthesia case it may be somewhat beneficial to withhold hay for 2-3 hours prior to anesthesia to allow the stomach to "empty". This may reduce the degree of
pulmonary gas exchange dysfunction that results from compression of the dorsocaudal region of the lungs in that position. Patients with compromised respiratory systems, though not ideal candidates for field anesthesia would probably benefit from fasting to reduce compression of the already compromised lungs. There are abdominal procedures that benefit from a reduction of abdominal contents and require an extended period of fasting.

Pre-medications

The importance of pre-medications in anesthesia cannot be emphasized enough. Pre-medications are administered for three basic reasons: 1) to achieve a smoother induction and recovery, 2) to intensify analgesia or the effects of the primary anesthetic agent, 3) to counter adverse effects produced by the primary anesthetic agents. Adequate sedation or tranquilization can reduce the dose of certain induction agents (thiopental) by up to fifty percent. It is also very important for achieving the full duration of effect from the induction bolus administered.

Unfortunately, most pre-medications also produce some degree of undesirable side effects. Xylazine is the predominant pre-medication used in equine anesthesia. Xylazine, along with its close relatives detomidine and romifidine, are from the drug family known as alpha2-adrenergic agonists (often referred to as alpha 2's). Alpha 2's possess potent sedative and analgesic effects. They are commonly used alone or in conjunction with an opioid to provide standing chemical restraint in equine practice. Due to their increased potency, duration and expense, detomidine and romifidine are not generally utilized as pre-medications for equine anesthesia, though there are certain situations where their use is justified. I will focus this discussion on xylazine, though most of the background information applies to all of the members of this drug family.

Xylazine's potent sedative effects produce a dose dependent calming effect. This not only greatly aids in behavioral management of the equine patient, but also produces a centralization of the cardiac output. The reduction in apprehension and muscle activity produced by the sedative effects of xylazine allows the cardiac output to be redistributed to favor the vital organs. Therefore a greater portion of the anesthetic agent administered will be directed to the target sites in the central nervous system. The importance of achieving the proper degree and duration of pre-induction sedation cannot be overemphasized! Insufficient sedation will leave a degree of apprehension while excessive sedation will result in ataxia. Either condition will prevent to some degree the desired level of centralization of the cardiac output and send an increased portion of the anesthetic induction bolus to the muscles. When lipid soluble drugs such as anesthetics are given as an IV bolus, it is redistribution from the vital organs to skeletal muscle via continued circulation that ends the clinical effects of the drug. Any increase in the amount of drug sent directly to muscle will decrease the impact of the anesthetic bolus (decreasing duration or, in rare cases, resulting in a "failed" induction). It is also important that an appropriate duration of this sedated state occur prior to the administration of the anesthetic bolus to allow this redistribution process to progress sufficiently. Five minutes of sedation prior to administration of the anesthetic bolus is the traditionally recommended interval, though time required for adequate centralization depends on patient's initial demeanor prior to sedative administration. The patient should exhibit moderate to profound sedation (depending on whether a benzodiazepine is administered with the ketamine bolus). "Head drop" (halfway down) and lack of interest or response to surroundings indicate a proper degree of sedation. Xylazine produces muscle relaxation and mild swaying may occur. Adjusting the horse's stance as sedation occurs to aid stability and applying counter balancing input via the halter should be used to minimize movement. In instances where excessive muscle relaxation occurs a second person can apply steadying input to the horse’s hip. Applying tension to the tail prior to the “oblivious phase” (10-15 second period preceding drop) during the induction process should generally be avoided.
since it usually creates some degree of apprehension in the patient. Minimizing noise and other
distractions will greatly assist in achieving the desired level of pre-anesthetic sedation with the
recommended initial dose of xylazine. I have found a soothing voice and reassuring physical
interaction helps many horses soften up. Despite all these techniques there will be the
occasional patient that requires a supplemental dose of xylazine to achieve to the desired level
of sedation. Achieving the proper degree of sedation is important not only for ensuring a smooth
induction, but also for obtaining the full duration afforded by the technique. The calming
influences of xylazine's sedative effects are also very important in achieving a smooth recovery.

The sedative effects of xylazine also counter the neuroexcitatory effects of ketamine, preventing
extensor rigidity, excitement and perhaps seizure like activity during and immediately following
induction. For this reason xylazine administration has been considered essential prior to
induction with ketamine. However, my experience with using smaller and smaller doses of
xylazine to minimize it's bradycardic effects in hyperkalemic foals eventually resulted in
inductions with straight ketamine-diazepam (Ket-Val), which, at least administered “slowly” (4-5
seconds), results in a smooth uneventful induction. I can only assume the onset of diazepam’s
sedative effect is quicker than ketamine's neuroexcitatory effect when administered together. I
have expanded this experience to weanlings with the same result, but have not tried it in an
adult horse to date. It should be noted that without the use of prior xylazine, the duration of
straight ketamine-diazepam is not nearly as long as when proper xylazine sedation is achieved
before induction. In normal healthy unsedated foals catheter placement is a greater "insult" and
mishaps are more common. I frequently needle (22g) down foals with only Ket-Val when
maintenance anesthesia is planned (the shortened duration has not been an issue in the
transition to the chosen maintenance technique). Using a single needle stick minimizes the
unpleasant aspects of anesthetic induction is these capricious patients. Crazy may be heritable,
but needle shyness is created.

Administering xylazine IV produces well-documented dose related side effects. Cardiovascular
side effects include 30% reductions in heart rate and cardiac output. First or second degree
heart block occurs at least transiently in all horses and persists longer in some patients. IV
xylazine produces a biphasic change in blood pressure (initial increase in blood pressure
produced by peripheral vasoconstriction followed by a gradual decrease to below base-line
values due to reductions in sympathetic nervous system tone). Detomidine's cardiovascular
effects are even more potent, producing larger decreases in cardiac output and heart rate. In
contrast to xylazine, detomidine sedation results in prolonged elevation of blood pressure.
Persistent peripheral vasoconstriction in the face of decreased cardiac output does not promote
good tissue blood flow. The cardiovascular changes produced by alpha 2's are generally well
tolerated in the normal healthy horses, but may be life threatening in patients suffering from
hypovolemia or certain rhythm disturbances. In compromised patients a small dose of xylazine
followed by an infusion of guaifenesin can be used to achieve the desired level of sedation and
centralization prior to induction. Xylazine sedation decreases respiratory rate 30% while tidal
volume seems to increase resulting in only minor alterations in arterial blood gas values.
Respiratory depression is greater with detomidine and extremely large doses have resulted in
respiratory compromise. The respiratory effects of alpha 2's are generally well tolerated in
normal healthy horses. The use of detomidine should be avoided in horses with compromised
respiratory function.

Xylazine sedation is also important in the recovery phase. Arousal occurs abruptly when
ketamine (or tiletamine) blood level reaches a critical value due to redistribution. Patients are
generally not ready to stand at this point. The residual sedative effects of xylazine delivered in
the induction process and any supplemental anesthetic administration are critical in preventing
attempts to stand until blood levels of these intravenous anesthetic agents have decreased sufficiently to assure a coordinated effort (braking effect). Patients often want to roll up once arousal occurs. Patients ideally remain sternal until sedation has resolved enough so head is consistently up and patient seems mentally engaged. Gentle physical and/or verbal restraint is often effective in slowing these transitions and improving recovery quality. External stimulation during this period can result in premature attempts, so a calm environment is still important.

**Anesthetic Agents**

Ketamine, tiletamine and the ultra-short barbiturate thiopental are the injectable anesthetic agents used in equine practice. Ketamine is by far the most common injectable anesthetic agent used in equine practice. Thiopental is no longer generally used in equine practice for induction or maintenance of anesthesia, but remains the fastest option for restoring an anesthetized state when equine patients get too light during inhalation maintenance anesthesia. Thiopental (1.1 mg/kg IV, or 500 mg/450kg) is also very useful in preventing swallowing during surgery of the upper airway done under IV ketamine anesthesia. Tiletamine, a more potent and longer lasting relative of ketamine, is available only in combination with the benzodiazepine zolazepam as Telazol. Many factors, including expense, lack of familiarity, and the tendency to produce a rougher recovery when compared to ketamine based techniques has limited the use of Telazol in equine practice. The poor recovery quality associated with Telazol anesthesia is easily explained and corrected. Telazol provides twice the anesthetic duration of ketamine, outlasting the calming effects of the xylazine administered prior to induction. Additional xylazine (0.34-0.44 mg/kg IV, or 150-200 mg/450kg) administered approximately 25 minutes post-induction dramatically improves the recovery quality of Telazol anesthesia, but does extend the duration of the recovery phase somewhat. The longer duration of effective analgesia provided by Telazol (20-30 minutes) can be useful for procedures expected to take more time than typically provided by a single administration of diazepam-ketamine (Ket-Val). The cost of this simplicity is substantial, but it does eliminate the potential of accidentally overdosing triple drip or the hassle of administering supplemental boluses of xylazine-ketamine. Reconstituted Telazol can be frozen for up to six months. Recumbency can last up to 50-60 minutes when xylazine is supplemented in the recovery phase; so busy practitioners must plan accordingly.

Both ketamine and tiletamine draw upon sympathetic nervous system reserve to augment cardiac output and blood pressure, which helps counter their direct negative inotropic and vasodilatory effects (and those produced by xylazine). I can't emphasize enough the need for caution in dosing these seemingly safe drugs in compromised patients where sympathetic reserve may be severely limited. A short period of apnea and/or irregular (apneustic) breathing is commonly observed immediately following induction when using ketamine or Telazol. The duration is generally short and does not seem to cause a problem in normal healthy patients. Cardiovascular function in normal healthy patients anesthetized with ketamine-based protocols is good to excellent.

**Extending Anesthetic Duration**

Things do not always go as planned. I feel it is always important to be prepared to extend anesthesia. Having the drugs and supplies required at the procedure site is ideal, but at the very least they should be stocked in the vehicle. There are two basic approaches to extending injectable anesthesia in equine patients. The first is intermittent bolus administration of xylazine and ketamine. The second involves a CRI of a drug mixture. Drug levels and their clinical effects decay over time following bolus administration. A CRI technique provides a more consistent level of drug effect.
Bolus Techniques

**Classic Technique:** 1/3 to 1/2 of the initial dose of both xylazine and ketamine is administered IV at predetermined intervals or when signs of a lightened plane of anesthesia are observed. Anesthetic duration is extended 7-12 minutes depending on the size of the supplemental doses administered. The lighter the patient becomes prior to adminstering the supplemental dose, the shorter the extension achieved due to increased muscle blood flow (decentralization). Timed administration (7 minutes intervals for 1/3 and 10 minute intervals for 1/2 of the initial doses) is intended to avoid patient arousal, smoothing the process.

Interval from the last supplemental dose to patient arousal is similar regardless of the number of supplemental doses administered. Interval from last supplemental dose to standing increases with the number of supplemental doses administered. This is due to the difference between the clearance rates of ketamine and xylazine. The blood level of ketamine, which is cleared faster than xylazine, regulates anesthetic effect. Xylazine accumulates with repeated administration of this combination, slowing the recovery process.

**Modified Technique:** In situations requiring more than a couple supplemental doses of this combination gradually reducing the xylazine component may reduce the impact on recovery time. Quality of recovery is generally good when extending anesthesia using this method.

**Advantages:** Simple and handy. Cardiorespiratory function is adequately maintained in normal healthy horses using this technique to extend anesthetic duration. Research has verified the safety of administering up to seven supplemental doses providing nearly two hours of duration in normal healthy horses. The research paper did not explore a greater number of supplemental doses.

**Disadvantages:** Anesthetic plane is not as even as with a constant infusion technique. Anesthetic depth must be "monitored" more closely, requiring a trained second person or some of the surgeon's attention. Catheter is recommended.

**Summary:** Extending anesthesia with supplemental doses of xylazine-ketamine is a good choice when only a short period of additional time is required to complete the procedure OR in an emergency situation where xylazine and ketamine are the only anesthetic agents available.

CRI Techniques

CRI techniques often utilize a combination of drugs to produce their anesthesia effect. Xylazine-guaifenesin or midazolam-ketamine combinations are the most popular in equine practice due in large part to their proven safety. Midazolam is water soluble, so preferable to diazepam for CRI mixtures. Each drug contributes to the overall effect:

**Xylazine**
- Sedative effect
- Systemic analgesia
- Muscle relaxation
- "Braking effect" during recovery (sedative & perhaps analgesia effects)

**Guaifenesin or Midazolam**
- Sedative effect (milder than xylazine’s)
- Muscle relaxation (significant when combined with xylazine’s muscle relaxation)
Ketamine

- Anesthetic effect
- Systemic analgesia (at sub-anesthetic levels) may be useful in recovery phase

Drug volumes added are small enough to not require compensatory volume adjustments to prevent dilution.

Drug Accumulation

The blood level of ketamine regulates the anesthetic effect. Xylazine and guaifenesin tend to accumulate as duration progresses, which increases the risk of prolonged and/or rough recoveries, especially at higher delivery rates. Limiting guaifenesin delivery to 100 mg/kg/hr (or 900 mg/450kg/hr) has been recommended to minimize weakness during recovery. When delivery surpasses 1 hour even that rate may prove somewhat problematic. Increasing the ketamine concentration in the mixture allows delivery rate to be decreased, reducing guaifenesin (and xylazine, if desired) accumulation.

CRI Delivery Control

Note: Constant Rate Infusion notes provide more detailed information on delivery methods.

CRI delivery can be controlled in several ways. Infusion pumps provide the most accurate control (+/- 5%) and make setting or adjusting delivery easy. They are fairly common in equine hospitals and, if their rechargeable battery is still functional, can be used in the field setting. The Heska Vet/IV 2.2 is an excellent infusion pump (compact, easy to use, reliable, operates on AC or rechargeable battery), Last time I checked they were approximately $1200 new ($1000 “show special” pricing), which is likely difficult to justify for ambulatory equine practices given the limited need and effective alternatives.

In-line IV flow control devices (Dial-A-Flo, numerous manufacturers produce similar devices) are another method of controlling delivery rate, but solution concentration must allow target delivery rate to fall within the confines of the devices control limits. Setting and adjusting infusion rates with an IV flow control device is easier and faster than using the roller or screw clamp of a solution administration set, though studies have demonstrated similar levels of accuracy (+/- 10%). Using an IV flow control device adds approximately $7 to cost. The Dial-A-Flo has "settings" (ml/hr) of OFF, 20, 30, 40, 50, 60, 70, 80, 100, 125, 165, 210, 250, and OPEN, but intermediate delivery rates (e.g. not indicated on dial) can be set. The distance between the level of fluid in the IV solution container and the patient catheter can be thought of as the pressure head responsible for flow. IV flow control devices are designed to work within a certain specified height differential. IV flow control devices provide steady delivery outside of this range, but rate will vary from setting. Viscosity of the solution and alterations in venous backpressure also impact the accuracy of IV flow control devices. Delivery rate should be verified initially and periodically evaluated when using an IV flow control device. Periodic inspection is also required to detect occlusive problems in delivery system. IV flow control devices have direct contact with the infusion solution. Like the solution administration sets they replace or supplement, they are disposable items intended for short-term use.

Drip rate is a common method used for controlling delivery rate of a CRI, especially in the field setting. For decades there were basically two solution administration sets available (10 drop/ml
& 60 drop/ml). In recent years 15 and 20 drop/ml solution administration sets have been added to the mix. The 15 drop/ml set are actually more common now than 10’s. When using drip rate to control CRI delivery it is critical to know the conversion factor for the solution administration set used (Table 1-2). Inadvertently using a 15 drop/ml set instead of a 10 drop/ml set decreases delivery by 33% (360 – 240 = 120/360 = 33%). Inadvertently using a 10 drop/ml set instead of a 15 drop/ml set increases delivery by 50% (360 – 240 = 120/240 = 50%). Counting the number of drops per 10 seconds provides a more accurate assessment of delivery rate. As drip rate deviates farther from 1 drop/s this approach is critical when using a solution administration set.

Drip Assist (Veterinary DripAssist Infusion Rate Monitor, 000A2773, Hallowell EMC, $225) is a simple battery powered (1 AA) device easily placed around the drip chamber of a solution administration set to continuously monitor delivery rate and volume. Display can be set to read drops/min, ml/hr, or total volume delivered (ml). It does not control delivery rate, but makes setting and adjusting it easier (display responds to new setting within 3-4 drops). Unit includes an audible alarm functions that warns user if delivery deviates +/- 13% of selected rate. If solution concentration allows DripAssist can be used in series with an IV flow control device (e.g. Dial-A-Flo).

Table 1: Delivery volumes for select drip rates.

<table>
<thead>
<tr>
<th>Drops/s</th>
<th>10 drop/ml solution administration set</th>
<th>15 drop/ml solution administration set</th>
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<tbody>
<tr>
<td></td>
<td>ml/min/ml/hr</td>
<td>ml/min/ml/hr</td>
</tr>
<tr>
<td>1</td>
<td>6/360</td>
<td>1/240</td>
</tr>
<tr>
<td>2</td>
<td>12/720</td>
<td>2/480</td>
</tr>
<tr>
<td>3</td>
<td>18/1080</td>
<td>3/720</td>
</tr>
</tbody>
</table>

Table 2: Drip rates for select delivery volumes.

<table>
<thead>
<tr>
<th>Dose</th>
<th>10 drop/ml solution admin. set</th>
<th>15 drop/ml solution admin. set</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 ml/kg/hr</td>
<td>1.25 drops/s</td>
<td>1.9 drops/s</td>
</tr>
<tr>
<td>(7.5 ml/450kg/min)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 ml/kg/hr</td>
<td>2.5 drops/s</td>
<td>3.75 drops/s</td>
</tr>
<tr>
<td>(15 ml/450kg/min)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 ml/kg/hr</td>
<td>3.75 drops/s</td>
<td>5.6 drops/s</td>
</tr>
<tr>
<td>(22.5 ml/450kg/min)</td>
<td></td>
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</table>

Guaifenesin-Ketamine-Xylazine (“Equine Triple Drip” or “GKX”)

**Technique:** Patient in anesthetized with xylazine-ketamine or xylazine-Ket-Val. CRI delivery of an anesthetic cocktail is then used to maintain anesthesia. **Traditional Equine Triple Drip** is created by adding ketamine (1 mg/ml) and xylazine (0.5 mg/ml) to a 5% guaifenesin solution. **Equine Triple Drip 2X** (ketamine 2 mg/ml) reduces delivery of xylazine and guaifenesin, making it a better initial choice when longer duration is anticipated or situations requiring a second bag of solution (don’t forget to adjust delivery rate). Reducing xylazine and guaifenesin delivery may eventually prove beneficial in many cases. Delivery rate recommendations vary: 1-3 drops/450kg/s (x), to effect up to 1 ml/kg/hr (1) and 2-3 ml/kg/hr (2). The1-3 drops/450kg/s delivery rates commonly used were developed using 10 drop/ml solution administration sets. Drip rates need to be increased (1.34, 2.7, and 4 drops/450kg/s, respectively) to maintain equivalent delivery using a 15 drop/ml solution administration set. An easier alternative might be the use of **Corrected** Equine Triple Drip, which contains a 50% increase in xylazine (0.75 mg/ml) and ketamine (1.5 mg/ml) to compensate for the decreased delivery rate of the 15
drop/ml set. This approach reduces guaifenesin delivery by 33%, which is unlikely to adversely impact muscle relaxation for most procedures and could prove advantageous in recovery.

For best results the “Equine Triple Drip” infusion should be instituted as soon as possible after horse becomes recumbent. Ideally the initial infusion rate selected provides the desired steady plane of anesthesia, but delivery may need to be adjusted based on patient response (e.g. to effect). “Equine Triple Drip” infusion can also be instituted later in a procedure when signs of a lightened plane of anesthesia occur to restore and extend anesthetic duration. A bolus (amount depends on how light the patient has gotten) is typically necessary prior to instituting the maintenance infusion rate. Bolus required depends on the degree of muscular activity present (de-centralization of cardiac output). If muscular activity is limited a “rapid” bolus of 0.22-0.55 ml/kg (or 100-250 ml/450kg) is often effective. In extreme cases induction level or greater doses may be necessary. A bolus can also be used if anesthetic plane needs to be deepened quickly. Onset of Equine Triple Drip is somewhat slow - be careful not to administer too much when responding to a lightened plane of anesthesia. If untrained assistance is used make sure transient increases in delivery are not inadvertently maintained.

Advantages: Provides a more stable anesthetic plane over time with less intervention by the surgeon. Cardiopulmonary function is adequately maintained in healthy patients over a reasonable period of time. Anesthetic durations of 60-120 minutes have been safely accomplished using “Equine Triple Drip”. Longer periods of anesthetic duration have not been adequately evaluated, but have proven safe so far. Recovery is generally very smooth.

Disadvantages: Requires catheter placement. Recovery to standing will typically take 15-30 minutes. Prolonged administration of Triple Drip will slow recovery as xylazine accumulates. Excessive guaifenesin administration may produce some weakness at recovery. Guaifenesin may not be routinely stocked in vehicle or practice.

Summary: CRI techniques provide a more consistent plane of anesthesia and are the preferred method for extending duration of injectable anesthesia whether in a field or hospital setting.

Improving CRI Technique

Efficacy of “Equine Triple Drip” depends on the drug combination, delivery rate, and stimulation involved (level, timing). Lower delivery rates of Traditional Equine Triple Drip generally extend duration of anesthesia produced by the induction drugs, but a bolus and/or rate increase is eventually required to cover significant stimulation. The closer the delivery rate is to the “effective rate”, the longer the extension. Intermediate delivery rates tend to provide prolonged coverage for a wider array of procedures, though not all. The highest delivery rates typically provide effective coverage for most procedures, but also increase likelihood of longer and/or rougher recoveries. Comparing the theoretical amount of supplemental drug administered to "maintain" 1 hour of anesthesia with the various “Equine Triple Drip” formulations and delivery rates to intermittent bolus administration is instructive (Table 3-7).

The ketamine totals for both intermittent bolus examples are greater than all but Equine Triple Drip 2X (18 ml/450kg/min), though Equine Triple Drip 2X (12 ml/450kg/min) and Traditional Equine Triple Drip (3ml/kg/hr) were close. When you consider anesthetic plane is waning at the 60-minute end point with the bolus techniques (which are not overly aggressive) while the anesthetic plane is maintained at steady state until the 60-minute point with the CRI techniques the results become even more interesting. Variation in anesthetic plane with bolus administration likely increases the amount of ketamine sent directly to skeletal muscle
(decentralization of cardiac output), which explains part of the increased drug amount required. Does the mild sedative effect of guaifenesin reduce the ketamine requirement of the CRI techniques?

**Table 3a**: Drug delivery (mg) for **Traditional Equine Triple Drip** (ketamine 1 mg/ml) using a 10 drop/ml solution administration set (450 kg).

<table>
<thead>
<tr>
<th>Rate</th>
<th>6 ml/min</th>
<th>12 ml/min</th>
<th>18 ml/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
<td>X</td>
<td>K</td>
<td>D</td>
</tr>
<tr>
<td>Induction</td>
<td>500</td>
<td>1100</td>
<td>50</td>
</tr>
<tr>
<td>1 hr</td>
<td>180</td>
<td>360</td>
<td>18000</td>
</tr>
<tr>
<td>Total</td>
<td>680</td>
<td>1460</td>
<td>50</td>
</tr>
</tbody>
</table>

X = xylazine, K = ketamine, D = diazepam, GG = guaifenesin

**Table 3b**: Drug delivery (mg) for **Traditional Equine Triple Drip** (ketamine 1 mg/ml) using a 15 drop/ml solution administration set (450 kg).

<table>
<thead>
<tr>
<th>Rate</th>
<th>4 ml/min</th>
<th>8 ml/min</th>
<th>12 ml/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
<td>X</td>
<td>K</td>
<td>D</td>
</tr>
<tr>
<td>Induction</td>
<td>500</td>
<td>1100</td>
<td>50</td>
</tr>
<tr>
<td>1 hr</td>
<td>120</td>
<td>240</td>
<td>12000</td>
</tr>
<tr>
<td>Total</td>
<td>620</td>
<td>1340</td>
<td>50</td>
</tr>
</tbody>
</table>

X = xylazine, K = ketamine, D = diazepam, GG = guaifenesin

**Table 4**: Drug delivery (mg) for **“Corrected” Equine Triple Drip** (xylazine 0.75 mg/ml & ketamine 1.5 mg/ml) using a 15 drop/ml solution administration set (450 kg).

<table>
<thead>
<tr>
<th>Rate</th>
<th>4 ml/min</th>
<th>8 ml/min</th>
<th>12 ml/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
<td>X</td>
<td>K</td>
<td>D</td>
</tr>
<tr>
<td>Induction</td>
<td>500</td>
<td>1100</td>
<td>50</td>
</tr>
<tr>
<td>1 hr</td>
<td>180</td>
<td>360</td>
<td>12000</td>
</tr>
<tr>
<td>Total</td>
<td>680</td>
<td>1460</td>
<td>50</td>
</tr>
</tbody>
</table>

X = xylazine, K = ketamine, D = diazepam, GG = guaifenesin

**Table 5a**: Drug delivery **Equine Triple Drip 2X** (ketamine 2 mg/ml) using a 10 drop/ml solution administration set (450 kg).

<table>
<thead>
<tr>
<th>Rate</th>
<th>6 ml/min</th>
<th>12 ml/min</th>
<th>18 ml/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
<td>X</td>
<td>K</td>
<td>D</td>
</tr>
<tr>
<td>Induction</td>
<td>500</td>
<td>1100</td>
<td>50</td>
</tr>
<tr>
<td>1 hr</td>
<td>180</td>
<td>720</td>
<td>18000</td>
</tr>
<tr>
<td>Total</td>
<td>680</td>
<td>1820</td>
<td>50</td>
</tr>
</tbody>
</table>

X = xylazine, K = ketamine, D = diazepam, GG = guaifenesin
Table 5b: Drug delivery **Equine Triple Drip 2X** (ketamine 2 mg/ml) using a 15 drop/ml solution administration set (450 kg).

<table>
<thead>
<tr>
<th>Rate</th>
<th>4 ml/min</th>
<th>8 ml/min</th>
<th>12 ml/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
<td>X K D GG</td>
<td>X K D GG</td>
<td>X K D GG</td>
</tr>
<tr>
<td>Induction</td>
<td>500 1100 50</td>
<td>500 1100 50</td>
<td>500 1100 50</td>
</tr>
<tr>
<td>1 hr infusion</td>
<td>120 480</td>
<td>240 960</td>
<td>360 1440</td>
</tr>
<tr>
<td>Total</td>
<td>620 1580 50</td>
<td>12000 24000</td>
<td>36000</td>
</tr>
</tbody>
</table>

X = xylazine, K = ketamine, D = diazepam, GG = guaifenesin

Table 6: Drug delivery **Traditional Equine Triple Drip** (ketamine 1 mg/ml) at 1, 2, & 3 ml/kg/hr (450 kg).

<table>
<thead>
<tr>
<th>Rate</th>
<th>7.5 ml/min (1 ml/kg/hr)</th>
<th>15 ml/min (2 ml/kg/hr)</th>
<th>22.5 ml/min (3 ml/kg/hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
<td>X K D GG</td>
<td>X K D GG</td>
<td>X K D GG</td>
</tr>
<tr>
<td>Induction</td>
<td>500 1100 50</td>
<td>500 1100 50</td>
<td>500 1100 50</td>
</tr>
<tr>
<td>1 hr infusion</td>
<td>225 450</td>
<td>22500 45000</td>
<td>675 1350</td>
</tr>
<tr>
<td>Total</td>
<td>725 1550 50</td>
<td>22500 45000</td>
<td>1175 2450</td>
</tr>
</tbody>
</table>

X = xylazine, K = ketamine, D = diazepam, GG = guaifenesin

Table 7: Drug amounts administered when using xylazine-ketamine boluses to maintain anesthesia in 450 kg patient.

<table>
<thead>
<tr>
<th>Frequency</th>
<th>10 minute intervals</th>
<th>15 minute intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
<td>X K D</td>
<td>X K D</td>
</tr>
<tr>
<td>Induction</td>
<td>500 1100 50</td>
<td>500 1100 50</td>
</tr>
<tr>
<td>10 minutes</td>
<td>250 500</td>
<td>500 1000</td>
</tr>
<tr>
<td>15 minutes</td>
<td>250 500</td>
<td>500 1000</td>
</tr>
<tr>
<td>20 minutes</td>
<td>250 500</td>
<td>500 1000</td>
</tr>
<tr>
<td>30 minutes</td>
<td>250 500</td>
<td>500 1000</td>
</tr>
<tr>
<td>40 minutes</td>
<td>250 500</td>
<td>500 1000</td>
</tr>
<tr>
<td>45 minutes</td>
<td>250 500</td>
<td>500 1000</td>
</tr>
<tr>
<td>50 minutes</td>
<td>250 500</td>
<td>500 1000</td>
</tr>
<tr>
<td>Supplemental Drug</td>
<td>1250 2500 50</td>
<td>750 1500 50</td>
</tr>
<tr>
<td>Total Drug Administered</td>
<td>1750 3600 50</td>
<td>1250 2600 50</td>
</tr>
</tbody>
</table>

X = xylazine, K = ketamine, D = diazepam

Efficacy experience and the above delivery analysis indicate a higher level of ketamine delivery (somewhere in the range of 720-1440 mg/kg/hr, or 1.6-3.2 mg/kg/hr) is needed to provide prolonged coverage for procedures involving major stimulation. When experimenting with new techniques it generally wise to start conservatively. An intermediate value of 2.1 mg/kg/hr was selected for the initial reformulation effort (3).

\[
2.1 \text{ mg/kg/hr} \times 450\text{kg} = 945 \text{ mg/450kg/hr} \\
1 \text{ drop/s (@ 15 drops/ml)} = 4 \text{ ml/min} = 240 \text{ ml/hr} \\
945 \text{ mg} ÷ 240 \text{ ml} = 3.94 \text{ mg/ml}, or 4 \text{ mg/ml (ketamine concentration in TIVA solution)}
\]
Increasing the ketamine concentration allows guaifenesin delivery to be decreased, reducing accumulation and the risk of weakness at recovery. Xylazine delivery can also be reduced to improve recovery times.

**Guaifenesin Issues**

Guaifenesin 5% solution is no longer available from a pharmaceutical manufacturer, but is available in from several compounding pharmacies, though price has increased fourfold ($44 for 1 liter bag). **Traditional Equine Triple Drip** costs approximately $60 per liter. At 4 ml/min a liter lasts 250 minutes ($15/hr). At 8 ml/min a liter lasts 125 minutes ($30/hr). Delivery of Equine Triple Drip is dictated by ketamine kinetics. Increasing the ketamine concentration (e.g. **Equine Triple Drip 2X**) will decrease the amount of guaifenesin delivered, reducing cost. This approach is only cost effective if leftover mixture doesn’t have to be discarded due to age (one week in “less conditioned” storage).

Drug accumulation (xylazine, muscle relaxant component) is an issue with all CRI anesthetic maintenance technique discussed, but the fixed 5% concentration of guaifenesin makes this issue more difficult to manage. **Equine Triple Drip 2X** decreases xylazine and guaifenesin delivery, reducing their impact on recovery as administration duration increases, but also reduces the level of muscle relaxation. Creating concentrated solutions for Dial-A-Flo control would further limit muscle relaxation.

Guaifenesin comes in 500 and 1000 ml bags, which limits volume flexibility. The 500 ml volume is preferred because it reduces waste potential in shorter cases and when reformulation is desired to reduce recovery impact with prolonged use (similar to approach outlined for the modified xylazine-ketamine bolus technique).

Titrated administration of guaifenesin is useful for reducing pain associated with excessive muscle tension / spasm, but its limited uses make it more likely to reach expiration in many practices.

**Midazolam-Ketamine-Xylazine (“MKX”)**

**MKX** is basically a modification of the “Equine Triple Drip” technique. Midazolam (an aqueous benzodiazepine) is used in place of guaifenesin to provide muscle relaxation and some sedation. Saline (or LRS) is used as the carrier solution. Midazolam-based TIVA solutions offer several advantages over their guaifenesin-based counterparts. Volume and concentration flexibility is nearly unlimited, which expands delivery control options while also reducing cost and waste. It also makes adjustments to limit drug accumulation much easier.

50mg midazolam ($5.80) + 1 liter saline ($7.20) = $13 vs. $44 for 1 liter of 5% guaifenesin
100mg midazolam ($11.60) + 1 liter saline ($7.20) = $18.80 vs. $44 for 1 liter of 5% guaifenesin

Midazolam purchased for **MKX** use can be substituted for diazepam (same dose) in Ket-Val for anesthetic induction if expiration date is approaching.

**MKX Base Recipe**

**Ketamine target dose (from efficacy & delivery analysis):**

2.1 mg/kg/hr x 450kg = 945 mg/450kg/hr
Target delivery rate:

1 drop/450kg/s @ 15 drops/ml = 4 ml/450kg/min = 240 ml/450kg/hr

945 mg ÷ 240 ml = 3.94 mg/ml, or 4 mg/ml (ketamine concentration in TIVA solution)

Xylazine (0.5 mg/ml) and midazolam (0.05-0.1 mg/ml) concentrations were selected to provide a level of muscle relaxation and sedation equivalent to Traditional Equine Triple Drip administered a 240 ml/450kg/hr (or 1 drop/s/450kg @ 15 drop/ml) to retain its reasonable recovery time and quality.

**MKX Base Recipe** Doses:

Midazolam (low) 240 ml/450kg/hr x 0.05 mg/ml = 12 mg/450kg/hr = 0.027 mg/kg/hr
Midazolam (mid) 240 ml/450kg/hr x 0.1 mg/ml = 24 mg/450kg/hr = 0.053 mg/kg/hr
Ketamine 240 ml/450kg/hr x 4 mg/ml = 960 mg/450kg/hr = 2.13 mg/kg/hr
Xylazine 240 ml/450kg/hr x 0.5 mg/ml = 120 mg/450kg/hr = 0.27 mg/kg/hr

Cost approximately $30-36 per liter. At 4 ml/min would last 250 minutes ($7.50-9/hr). At 8 ml/min would last 125 minutes ($15-18/hr).

The **MKX Base Recipe** is designed to provide good balance between coverage and recovery time / quality. The **MKX Base Recipe** was developed for the broad middle of the population to reduce risk in more sensitive patients, though carefully titrated delivery is required in severely compromised patients with limited sympathetic reserve. For patients that prove to be less sensitive an increase in delivery (with or without a bolus dose) can be used to provide effective coverage.

Adjustments in base delivery rate are based on patient monitoring (“trained assistant” or the peripheral attention of the clinician). Arterial blood pressure is generally good to excellent with ketamine-based anesthetic protocols in normal healthy patients. Turgidity and size of the facial (or other superficial large arteries) can be used to estimate blood pressure and volume, respectively, prior to induction and during maintenance phase as needed. The palpebral reflex is typically very brisk with ketamine-based anesthetic protocols and changes associated with depth of anesthesia generally too subtle to be really useful. Ventilation (tidal volume more than rate) generally provides the most useful estimate of changes in anesthetic depth when using ketamine-based anesthetic protocols. A marked increase in tidal volume (deep sigh) is generally the best indicator of “impending lightness”. Purposeful movement is obviously an indication of an inappropriately light plane of anesthesia. If you don’t have an occasional patient “get light” (deep breath is close enough), you are keeping some too deep.

Delivering a bolus of MKX can be used to quickly deepen patient, allowing reduced delivery rates to be explored if “trained” assistance is available. Size of the bolus required depends on the impending or actual deficit. Simply think of the ketamine dose you would bolus and administered the requisite volume (e.g. 400 mg = 100 ml of **MKX Base Recipe**). Muscular activity decentralizes cardiac output, increasing the bolus dose required.

The difference in muscle relaxation between horses anesthetized with xylazine-ketamine and xylazine-ketamine-diazepam is very evident. Many procedure performed using injectable
anesthesia do not require marked muscle relaxation. The “mid” concentration of midazolam may be more appropriate for procedures requiring a higher level of muscle relaxation. The “mid” concentration may also be more appropriate when Ket-Val is not used for induction.

The blood level of ketamine regulates the anesthetic effect. Arousal occurs abruptly when ketamine (or tiletamine) blood level reaches a critical value due to redistribution. Patients are generally not ready to stand at this point. The residual sedative effects of xylazine delivered in the induction process and any supplemental anesthetic administration are critical in preventing attempts to stand until blood levels of these intravenous anesthetic agents have decreased sufficiently to assure a coordinated effort (braking effect). Patients often want to roll up once arousal occurs. Patients ideally remain sternal until sedation has resolved enough so head is consistently up and patient seems mentally engaged. Gentle physical and/or verbal restraint is often effective in slowing these transitions and improving recovery quality. Temperament impacts many aspects of anesthesia, but perhaps most importantly recovery.

Braking effect varies over time. The initial peak produced by the large premed dose of xylazine is gradually reduced by metabolism. Xylazine is cleared slower than ketamine, so it accumulates over time when a fixed combination is infused. CRI delivery of xylazine slows the initial decline in braking effect and continued delivery eventually produces a gradual increase, creating a valley.

The time required for ketamine to redistribute is relatively constant for a given delivery level. The duration and quality of recovery largely depends on the differential between the braking effect and ketamine clearance time. When perfectly matched patient temperament and level of environmental stimulation become the determinant factors. That is generally the case with xylazine-ketamine anesthesia (single bolus of each), which is why it is considered the “gold standard” of equine recovery. Excess braking effect slows recovery, while a deficit results in a more abrupt transition, increasing the likelihood of a rougher recovery.

A TIVA mixture delivering xylazine (1 mg/kg/hr), midazolam (0.12 mg/kg/hr), and ketamine (1.8 mg/kg/hr) was evaluated using a group of horses undergoing experimental surgical manipulation of the digital nerves (4). Supplemental boluses of ketamine were required in 2 of 6 subjects during the procedure, and in 3 of 6 subjects to facilitate hoisting into the recovery stall at one hour post-induction. Infusions were started 10 minutes after induction and the effective loss of the loading doses (especially ketamine) administered at induction may have factored into the breakthroughs reported. Recovery length reported (time to sternal 49 ± 31 minutes) may be due to the level of xylazine delivery used in the study. At 240-360 ml/450kg/hr Traditional Equine Triple Drip’s xylazine delivery rate (0.25-0.4 mg/kg/hr) is markedly lower. MKX Base Recipe’s xylazine delivery rate (0.27 mg/kg/hr) is also markedly lower, while its ketamine delivery rate (2.1 mg/kg/hr) is slightly higher than level used in the study.

Longer recovery times, especially with extended delivery, are an issue with Traditional Equine Triple Drip and an early MKX combination. The xylazine-ketamine ratios of these techniques (1:2 & 1:1.8, respectively) are fairly high. The xylazine-ketamine ratio of the MKX Base Recipe (1:8) was designed to minimize recovery time, even with extended delivery. Experience is with MKX anesthesia is still limited. There could be a brief anesthetic interval corresponding with the valley floor where the MKX Base Recipe does not provide sufficient braking effect. A small supplemental dose of xylazine administered at the end of anesthesia (I would start with 0.11-0.22 mg/kg IV, or 50-100 mg/450kg) will correct this issue and is preferable to increasing the xylazine concentration. The same approach can be used when the situation requires a little extra braking effect (quality recovery imperative, temperamental patients, etc.).
As with any new technique refinement may be necessary to obtain optimal results. Input from practitioners is critical to the refinement of the MKX technique (abrahamsen@earthlink.net).

**MKX OPTIONS**

CRI solutions are divided into 2 basic types. Stock solutions are created for a prototypical patient (e.g. 450kg horse) with adjustments in delivery (using simple ratios) to accommodate variations in patient size and/or alter the level of effect. Custom solutions are designed for a particular patient size, with delivery adjusted to alter level of effect. Drug concentrations can be altered individually to modify effect or in unison to suit a particular infusion control method.

When creating solutions the degree of rounding permissible is relative to the dilution of the solution, accuracy of the delivery control method used, and the safety margins of the drugs. The serial effect of rounding must also be considered. It likely violates significant numbers doctrine, but I’m cautious with rounding until nearing the end when doing multi-step calculations.

**MKX (dilute stock solution)**

Delivery Control: Infusion pump or solution administration set (15 drop/ml, with or without DripAssist).

Target Weight: 450kg

Target Delivery Rate: 1 drop/450kg/s @ 15 drops/ml = 4 ml/450kg/min = 240 ml/450kg/hr

Ratios are used to adjust delivery for patient size

\[
360kg + 450kg = 0.8 \times 240 \text{ ml/hr} = 190 \text{ ml/hr}, \text{ (subtle rounding is allowed)}
\]

\[
190 \text{ ml/hr} = 48 \text{ drop/min}, \text{ or 8 drops/10s}
\]

Apply **MKX Base Recipe** Dosages

- **Midazolam (low)**: \(0.027 \text{ mg/kg/hr} \times 450\text{kg} = 12 \text{ mg/hr}\)
- **Midazolam (mid)**: \(0.053 \text{ mg/kg/hr} \times 450\text{kg} = 24 \text{ mg/hr}\)
- **Ketamine**: \(2.13 \text{ mg/kg/hr} \times 450\text{kg} = 960 \text{ mg/450kg/hr} \)
  \(\text{(could be rounded to 950 or 1000 mg/hr)}\)
- **Xylazine**: \(0.27 \text{ mg/kg/hr} \times 450\text{kg} = 121.5 \text{ mg/hr} \)
  \(\text{(rounded to 120 mg/hr)}\)

Calculate Solution Concentrations

- **Midazolam (low)**: \(12 \text{ mg} \div 240 \text{ ml} = 0.05 \text{ mg/ml}\)
- **Midazolam (mid)**: \(24 \text{ mg} \div 240 \text{ ml} = 0.1 \text{ mg/ml}\)
- **Ketamine**: \(960 \text{ mg} \div 240 \text{ ml} = 4.0 \text{ mg/ml}\)
- **Xylazine**: \(120 \text{ mg} \div 240 \text{ ml} = 0.5 \text{ mg/ml}\)

Since this is a 450 kg stock solution we have ended up back at the **MKX Base Recipe** concentrations.
Select mixture volume: 250ml (approx. 1 hour duration)
500 ml bags of saline may be cheaper than 250ml. If so, discard 250 ml via solution administration set prior to adding drugs. Flush line with drug solution prior to use!

Apply Solution Concentrations to Determine Drug Amounts Added to Fluid Bag

Midazolam (low) 0.05 mg/ml x 250 ml = 12.5 (can be rounded to 15 mg)
Midazolam (mid) 0.1 mg/ml x 250 ml = 25
Ketamine 4 mg/ml x 250 ml = 1000 mg
Xylazine 0.5 mg/ml x 250 ml = 125 mg

MKX (dilute custom solution, designed for patients weight)

Delivery Control: Infusion pump or solution administration set (15 drop/ml, with or without DripAssist).

Patient weight 250 kg

Target Delivery Rate: 1 drop/250kg/s @ 15 drops/ml = 4 ml/450kg/min = 240 ml//250kg/hr

Apply MKX Base Recipe dosages

Midazolam (low) 0.027 mg/kg/hr x 250 kg = 6.75 mg/hr
Midazolam (mid) 0.053 mg/kg/hr x 250 kg = 13.25 mg/hr
Ketamine 2.13 mg/kg/hr x 250 kg = 533 mg/hr
Xylazine 0.27 mg/kg/hr x 250 kg = 68 mg/hr

Calculate Solution Concentrations

Midazolam (low) 6.75 mg/hr + 240 ml/hr = 0.028 mg/ml
Midazolam (mid) 13.25 mg/hr + 240 ml/hr = 0.055 mg/ml
Ketamine 533 mg/hr + 240 ml/hr = 2.22 mg/ml
Xylazine 68 mg/hr + 240 ml/hr = 0.28 mg/ml

Select mixture volume (250ml)
500 ml bags of saline may be cheaper than 250ml. If so, discard 250 ml via solution administration set prior to adding drugs. Flush line with drug solution prior to use!

Apply Solution Concentrations to Determine Drug Amounts Added to Fluid Bag

Midazolam (low) 0.028 mg/ml x 250 ml = 7 mg (can round to 10)
Midazolam (mid) 0.055 mg/ml x 250 ml = 12.5 mg (can round to 15)
Ketamine 2.22 mg/ml x 250 ml = 555 mg (round to 550)
Xylazine 0.28 mg/ml x 250 ml = 70 mg

One could create a stock solution table for a variety of common patient weights.
MKX (concentrated stock solution) (Dial-A-Flo)

Delivery Control: designed for use with Dial-A-Flo or infusion pump. With a pediatric solution administration set (60 drops/ml, with or without DripAssist) the base drip rate of 13 drops/10s is easy to set and adjust. With a conventional solution administration set (15 drop/ml) the base delivery rate of 20 drops/min, or 3.3 drops/10s is very slow for accurate visual control and requires more time to set or adjust, even with a DripAssist.

Target Patient Weight: 450kg

Target Delivery Rate: 80ml/450kg/hr

The Dial-A-Flo has "settings" (ml/hr) of OFF, 20, 30, 40, 50, 60, 70, 80, 100, 125, 165, 210, 250, and OPEN (intermediate values can be set). To provide latitude for adjustments based on varying body size, it is best to tailor a stock solution when possible to a setting that accommodates that widest range of expected applications. Selecting 80 mg/hr for the base 450kg delivery rate provides 2 close upper settings and a range of lower settings to accommodate variations in patient size and/or alter the level of effect.

Apply MKX Base Recipe Dosages

Midazolam (low) 0.027 mg/kg/hr x 450kg = 12 mg/hr
Midazolam (mid) 0.053 mg/kg/hr x 450kg = 24 mg/hr
Ketamine 2.13 mg/kg/hr x 450kg = 960 mg/450kg/hr
   (could be rounded to 950 or 1000 mg/hr)
Xylazine 0.27 mg/kg/hr x 450kg = 121.5 mg/hr
   (rounded to 120 mg/hr)

Calculate Solution Concentrations

Midazolam (low) 12 mg/hr ÷ 80 ml/hr = 0.15 mg/ml
Midazolam (mid) 24 mg/hr ÷ 80 ml/hr = 0.3 mg/ml
Ketamine 960 mg/hr ÷ 80 ml/hr = 12 mg/ml
Xylazine 120 mg/hr ÷ 80 ml/hr = 1.5 mg/ml

Select mixture volume (100ml)

500 ml bags of saline may be cheaper than 250ml. If so, discard 400 ml via solution administration set prior to adding drugs. Flush line with drug solution prior to use!

Apply Solution Concentrations to Determine Drug Amounts Added to Fluid Bag

Midazolam (low) 0.15 mg/ml x 100 ml = 15 mg (can round to 2)
Midazolam (mid) 0.3 mg/ml x 100 ml = 30 mg (can round to 15)
Ketamine 12 mg/ml x 100 ml = 1200 mg
Xylazine 1.5 mg/ml x 100 ml = 150 mg
A table can be created for varying patient sizes (Table 8).

\[
70 \text{ ml/hr} \times 11.8 \text{ mg/ml (ketamine concentration)} = 826 \text{ mg of ketamine} \\
826 \text{ mg} \div 2.1 \text{ mg/kg/hr (ketamine target dose)} = 393 \text{ kg}
\]

\[
60 \text{ ml/hr} \times 11.8 \text{ mg/ml (ketamine concentration)} = 708 \text{ mg of ketamine} \\
708 \text{ mg} \div 2.1 \text{ mg/kg/hr (ketamine target dose)} = 337 \text{ kg}
\]

Repeat calculation for each setting.

A simple ratio approach can be used (e.g. \(80/450 \times 70/x\), \(80x = 31,500\), \(x = 393.75\)

**DO NOT use a generously rounded value as basis for ratio calculations when creating delivery rate tables!**

<table>
<thead>
<tr>
<th>Setting (ml/hr)</th>
<th>20</th>
<th>30</th>
<th>40</th>
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<td>225</td>
<td>280</td>
<td>340</td>
<td>390</td>
<td>450</td>
<td>560</td>
<td>700</td>
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</table>

Changes in delivery between Dial-A-Flo settings are not uniform (Table 9).

<table>
<thead>
<tr>
<th>Delivery Change</th>
<th>Change</th>
<th>Delivery Change</th>
<th>Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>125 ml/hr (\to) 100 ml/hr</td>
<td>-20%</td>
<td>100 ml/hr (\to) 125 ml/hr</td>
<td>+25%</td>
</tr>
<tr>
<td>100 ml/hr (\to) 80 ml/hr</td>
<td>-20%</td>
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<td>+25%</td>
</tr>
<tr>
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<td>-13%</td>
<td>70 ml/hr (\to) 80 ml/hr</td>
<td>+14%</td>
</tr>
<tr>
<td>70 ml/hr (\to) 60 ml/hr</td>
<td>-14%</td>
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<td>+17%</td>
</tr>
<tr>
<td>60 ml/hr (\to) 50 ml/hr</td>
<td>-17%</td>
<td>50 ml/hr (\to) 60 ml/hr</td>
<td>+20%</td>
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<tr>
<td>50 ml/hr (\to) 40 ml/hr</td>
<td>-20%</td>
<td>40 ml/hr (\to) 50 ml/hr</td>
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</tr>
<tr>
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<td>-25%</td>
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<td>+33%</td>
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<tr>
<td>30 ml/hr (\to) 20 ml/hr</td>
<td>-33%</td>
<td>20 ml/hr (\to) 30 ml/hr</td>
<td>+50%</td>
</tr>
</tbody>
</table>

**MKX (concentrated stock solution) (pediatric solution administration set)**

Delivery Control: Designed for use with pediatric solution administration set (60 drops/ml, with or without DripAssist) or infusion pump. With a conventional solution administration set (15 drop/ml) the base delivery rate of 15 drops/min, or 2.5 drops/10s is too slow for accurate visual control and requires more time to set or adjust, even with a DripAssist. The more concentrated the solution, the greater the risk due to error. Solution creation and delivery control must be more precise.

Target Patient Weight: 250 kg

Target Delivery Rate: 1 drop/250kg/s @ 60 drops/ml = 1 ml/250kg/min = 60 ml//250kg/hr

Apply **MKX Base Recipe** dosages
Midazolam (low) 0.027 mg/kg/hr x 250 kg = 6.75 mg/hr
Midazolam (mid) 0.053 mg/kg/hr x 250 kg = 13.25 mg/hr
Ketamine 2.13 mg/kg/hr x 250 kg = 533 mg/hr
Xylazine 0.27 mg/kg/hr x 250 kg = 68 mg/hr

Calculate Solution Concentrations
Midazolam (low) 6.75 mg/hr ÷ 60 ml/hr = 0.11 mg/ml
Midazolam (mid) 13.25 mg/hr ÷ 60 ml/hr = 0.22 mg/ml
Ketamine 533 mg/hr ÷ 60 ml/hr = 8.9 mg/ml
Xylazine 68 mg/hr ÷ 60 ml/hr = 1.13 mg/ml

Select mixture volume (100ml)
500 ml bags of saline may be cheaper than 250ml. If so, discard 400 ml via solution administration set prior to adding drugs. Flush line with drug solution prior to use!

Apply Solution Concentrations to Determine Drug Amounts Added to Fluid Bag
Midazolam (low) 0.11 mg/ml x 100 ml = 11 mg (can round to 10 mg)
Midazolam (mid) 0.22 mg/ml x 100 ml = 22 mg (can round to 25 mg)
Ketamine 8.9 mg/ml x 100 ml = 890 mg (round to 900 mg)
Xylazine 1.13 mg/ml x 100 ml = 113 mg (can round to 115 or 120 mg)

**MKX (concentrated custom solution) (Dial-A-Flo)**

Delivery Control: Designed for use with Dial-A-Flo or infusion pump. With a pediatric solution administration set (60 drops/ml, with or without DripAssist) the base drip rate of 13 drops/10s is easy to set and adjust. With a conventional solution administration set (15 drop/ml) the base delivery rate of 20 drops/min, or 3.3 drops/10s is very slow for accurate visual control and requires more time to set or adjust, even with a DripAssist.

Target Patient Weight: 250 kg
Target Delivery Rate: 80 ml/kg/hr

The Dial-A-Flo has "settings" (ml/hr) of OFF, 20, 30, 40, 50, 60, 70, 80, 100, 125, 165, 210, 250, and OPEN (intermediate values can be set). To provide latitude for adjustments based on varying body size, it is best to tailor a stock solution when possible to a setting that accommodates the widest range of expected applications. Selecting 80 mg/hr for the base 250kg delivery rate provides 2 upper settings and a range of lower settings to alter level of effect.

Apply **MKX Base Recipe** dosages
Midazolam (low) 0.027 mg/kg/hr x 250 kg = 6.75 mg/hr
Midazolam (mid) 0.053 mg/kg/hr x 250 kg = 13.25 mg/hr
Ketamine  2.13 mg/kg/hr x 250 kg = 533 mg/hr  
Xylazine  0.27 mg/kg/hr x 250 kg = 68 mg/hr

Calculate Solution Concentrations

Midazolam (low)  6.75 mg/hr ÷ 80 ml/hr = 0.08 mg/ml  
Midazolam (mid)  13.25 mg/hr ÷ 80 ml/hr = 0.17 mg/ml  
Ketamine  533 mg/hr ÷ 80 ml/hr = 6.66 mg/ml  
Xylazine  68 mg/hr ÷ 80 ml/hr = 0.85 mg/ml

Select mixture volume (100ml)

500 ml bags of saline may be cheaper than 250ml. If so, discard 400 ml via solution administration set prior to adding drugs. Flush line with drug solution prior to use!

Apply Solution Concentrations to Determine Drug Amounts Added to Fluid Bag

Midazolam (low)  0.08 mg/ml x 100 ml = 8 mg (can round to 10 mg)  
Midazolam (mid)  0.17 mg/ml x 100 ml = 17 mg (can round to 20 mg)  
Ketamine  6.66 mg/ml x 100 ml = 666 mg (round to 700 mg)  
Xylazine  0.85 mg/ml x 100 ml = 85 mg

MKX Storage

Once created solutions should probably be discarded after a week of “less conditioned” storage (ambulatory vehicle). In conditioned settings solutions are likely safe for a longer period of time. Storage in a refrigerator may preserve MKX for an even longer period. Leftover TKX (Telazol-ketamine-xylazine, typically no diluent) can be frozen and seems to work fine when thawed – haven’t tried it with MKX, but wouldn’t be surprised if it worked fine.

References

Morphine, Ketamine Stun for Colic Pain, Transport Analgesia

Eric J. Abrahamsen, DVM, DACVA

Appropriate analgesic support makes continued treatment humane and improves the general well being of the patient. Heart and respiratory rates decrease while appetite and demeanor improve with effective analgesic therapy. Effective analgesic support also reduces the risk of overly stressing support limbs. The benefits of reducing patient stress on the convalescence process are less defined. Pain restricts use of "damaged" tissues. Overzealous analgesic support can produce deleterious consequences in certain situations.

I use an efficacy-based pain grading system:

Mild pain – adequate relief provided by NSAIDs
Mid-moderate pain – adequate relief provided by opioid agonist-antagonist +/- NSAID
Moderate pain – adequate relief provided by mu opioid +/- NSAID
Severe pain – adequate relief provided by multidrug pain infusion (Pentafusion)
Extreme pain – partial relief provided by multidrug pain infusion (Pentafusion)

MORPHINE

Note: 15 mg/ml multi-dose vials of morphine have been discontinued due to new FDA regulations. See Meet the New Morphine notes for important information regarding the safe use of preservative free morphine.

Morphine, a mu opioid agonist, is a very useful drug for treating moderate levels of pain in the horse and is an important component in the treatment of severe pain. Unlike opioid agonist-antagonist such as butorphanol, morphine does not experience a "ceiling effect", though side effects limit the dosage that can be used clinically for prolonged analgesic support. Morphine is approximately half the cost of butorphanol on a clinical dose basis. Morphine is a controlled substance, but most equine practitioners routinely use controlled substances and are familiar with their associated regulatory requirements.

What about the side effects of morphine?

Morphine administration in horses has been historically associated with two very important side effects; decreases in gastrointestinal (GI) motility and central nervous system (CNS) stimulation. Much of the scientific literature regarding these complications is dated and involves older chemical restraint techniques that combined large doses of morphine (0.34 – 0.67 mg/kg IV) with either acepromazine or xylazine. The contemporary analgesic dose of morphine (0.1 mg/kg IV or IM) is modest by comparison.

GI Motility Morphin decreases GI motility. If the effect is profound and/or persistent enough an impaction can form along with symptoms of colic. Disturbances in GI motility may also lead to the formation of gas pockets and transient symptoms of colic.

A 2006 study using large old-fashioned doses of morphine (0.5 mg/kg IV) reported GI dysfunction lasting up to 6 hours after administration (1). Frequency and weight of bowel movements decreased by 60-70%. Borborygmus score was decreased by 50% along with a mild reduction in fecal moisture content. This dose is 5 times the contemporary analgesic dose used in horses.
The incidence of colic with contemporary doses of morphine is quite low \(2, 3\). The low incidence does not mean negative effects on GI motility have been eliminated, but rather reduced. Given the temperament nature of the equine GI system patients receiving opioids should be monitored frequently for signs of GI disturbance. Pain also reduces GI motility and likely plays a role in the incidence of GI complications attributed to opioid analgesic therapy.

Central and local GI opioid receptors may both contribute, but the GI receptors are likely more important in morphine’s negative effect on GI motility. Co-administration of poorly absorbed oral opioid antagonists or systemic administration of opioid antagonists incapable of crossing the blood brain barrier has reduced opioid-induced constipation without adversely affecting analgesia in people \(4\). The approach may one day prove useful in treating equine patients with decreased GI motility.

**CNS Excitement** Manic behavior, most notably incessant stall walking was common following the older high dose morphine-based chemical restraint techniques. The duration of the large morphine dose was greater than the acepromazine or xylazine used in the techniques. The behavior was easily controlled with the administration of a dose or two of xylazine, or more frequently acepromazine. CNS excitement is rarely observed when contemporary doses of morphine are used for chemical restraint. This is likely due to a shorter duration and the use detomidine for these procedures.

The presence of pain is generally thought to reduce the likelihood of excitatory behavior. Analgesic use of morphine is even less likely to result in CNS excitement than chemical restraint applications. Though risk is low, co-administration of acepromazine or an alpha2-adrenergic agonist can also be used to suppress the CNS excitatory effects of morphine.

**What about the risk of overuse?**

Analgesic management of post-operative fracture cases and laminitis patients is challenging, but crucial to successful outcome. These patients typically require long-term analgesic support, but the risk of overuse is a constant concern. The ability to titrate the level of relief provided is especially important in these patients. The level of analgesia should be should be evaluated at least twice during each dosing interval (more frequently during the early stages of analgesic therapy). The trough level just prior to administering the subsequent dose is used to evaluate patient progress and/or presence of any problems such as cast sores. The peak level at onset of subsequent dose (10-15 minutes IV, 20-25 minutes IM) is used to evaluate efficacy of the analgesia. Both are important to proper analgesic management of fracture and laminitis patients.

**Laminitis** In horses with severe laminitis, we use the amount of time spent standing as a guide in regulating the level of analgesic support. We want the horse to be comfortable getting up for short periods to eat, urinate, defecate or to change positions, but reduce the level of analgesic support if they spend too much time standing. As their feet improve analgesic support is reduced to control the amount of time spent standing. Morphine doses as low as 5 mg/450 kg administered every 4-6 hours have proven to be useful in providing relief in the later stages of convalescence in these patients.

**Post-operative Fractures** In post-operative fracture patients a primary concern is preventing the development of laminitis in the support legs. Analgesic therapy is necessary to encourage use of the repaired limb. Intramuscular (IM) morphine (0.1 mg/kg IM q4h) does not totally
eliminate pain during the early stages of convalescence in patients with more serious long bone fractures and overuse has not been a problem. Morphine dose is reduced as patient becomes more comfortable on the repaired limb to minimize the risk of overuse. Patients with less painful fractures still benefit from analgesic support, but the level must be titrated to the patient’s condition to minimize the risk of overuse. Appropriate analgesic therapy should make the patient more comfortable while leaving enough pain to prevent overuse. The general well being of fracture repair patients as judged by heart rate, respiratory rate, appetite and demeanor improves with the acepromazine-morphine analgesia.

Acepromazine (0.011-0.022 mg/kg IM or 5-10 mg/450 kg, depending on demeanor of patient) is administered every 4 hours along with the morphine. Acepromazine was initially added to prevent the CNS excitement historically associated with morphine use in horses. Smaller contemporary doses of morphine and presence of pain make this less likely, but we find the calming influence of acepromazine reduces unnecessary activity in patients at risk of overuse. Equine surgeons that have used this approach believe it contributes to the successful recuperation of their fracture repair patients. This small dose of acepromazine does not typically produce overt tranquilization in equine patients and is reduced should this occur.

Increasing the dosing interval reduces the mean analgesic effect, but peak effect remains unchanged and the risk of overuse increases. Analgesic support gradually declines over time following bolus administration of morphine and extending the dosing interval drastically reduces the level of analgesia provided in the later stages of the interval.

**Clinical use of morphine in the horse**

I routinely use morphine to help alleviate pain in equine patients. Equine patients expected to experience significant pain upon recovery from anesthesia receive morphine (0.1 mg/kg IM for most cases, 0.1 mg/kg IV when more extreme levels of pain are expected) near the end of the procedure. Onset time is approximately 10 minutes with IV administration and 20 minutes with IM administration. The goal is to have a peak level of morphine analgesia when awareness returns in the recovery stall. Although there are numerous factors involved, the use of morphine seems to improve recovery quality. Morphine is also routinely used to manage pain in hospitalized patients. Morphine (0.1 mg/Kg IM, or 45 mg/450 kg) is administered every 4 hours initially. Administering morphine (0.05 mg/kg IM) every 2 hours would provide a more consistent level of relief, but doubles the number of injections required. Acepromazine (0.011-0.022 mg/kg IM, or 5-10 mg/450 kg, depending on patient’s demeanor) is generally included in the syringe with the morphine injection. Acepromazine was initially added to counter the potential of CNS excitement resulting from the use of morphine. While this level of acepromazine does not typically produce overt tranquilization (and is reduced if this occurs), it’s calming influences seem to limit the level of patient activity, which we find desirable. If greater analgesic support is required, a higher dose of morphine (0.15 mg/kg IM, or 60 mg/450 kg) can be used transiently, but the combination of higher morphine and pain levels increases the risk of GI complications.

Morphine dose is reduced over time as patient comfort dictates. An alternative method of reducing the level of analgesia provided is to increase the dosing interval to 6 hours, which is often more convenient as well. Peak effect remains unchanged when dosing interval is increased, which may increase risk of overuse in some situations. In practices that do not have 24 hour staffing extending dosing intervals during the overnight hours is often used to reduce the impact of providing care to hospitalized patients. Analgesic support gradually declines over time following bolus administration of morphine and extending the dosing interval drastically reduces the level of analgesia provided in the later stages of the interval.
requirements of the patient should determine when extended overnight dosing intervals are appropriate. I have dispensed pre-loaded labeled syringes (1-2 day supply at a time, with directions) while teaching at Ohio State University and working at private equine hospitals to allow responsible owners/farm managers to effectively manage the analgesic support of their laminitic horses when hospitalization was no longer warranted or affordable.

GI dysfunction and potential colic (gas, impaction) should always be a considered when using morphine in equine patients, especially in the early stages of treatment. GI motility (auscultation of all 4 quadrants), and fecal output (volume and moisture level) should be monitored frequently. A modest reduction in fecal output (& moisture) is generally not a concern as long as GI motility remains acceptable, but does warrant closer observation until proven harmless. Water intake should also be monitored closely and methods used to encourage consumption (multiple bucket locations, etc). Patient should be regularly observed for symptoms of colic in the initial stages of morphine analgesic therapy. It is important to remember that two of drugs associated with decreased GI motility (alpha2-adrenergic agonists & opioids) also possess analgesic properties that will mask milder symptoms of colic. Trough level evaluation is important. With prolonged treatment vigilance can be relaxed somewhat once patient has proven tolerant. The incidence of colic with contemporary doses of morphine is quite low. I have maintained horses on morphine support for as long as 3 months without complications. Many of these patients were also receiving phenylbutazone (2 g/450 kg q24h or 1g/450 kg q12h PO).

If GI dysfunction is suspected appropriate medical therapy should be instituted. Morphine (& detomidine) administration is reduced or discontinued, depending on the severity of the GI disturbance. A lidocaine-ketamine-acepromazine infusion can be used to provide analgesic support in patients where morphine must be discontinued.

Prophylactic administration of mineral oil may alleviate some of the anxiety associated with using morphine in equine patients and perhaps improve success should intervention be required.

COLIC PAIN

NMDA antagonists (Ketamine Stun)

Ketamine is a NMDA receptor antagonist. Ketamine possesses amazingly potent analgesic properties at sub-anesthetic doses (5). A small dose of ketamine (0.11-0.22 mg/kg IV, or 50-100 mg/450 kg) layered over a "modest-moderate" level of α2 sedation/analgesia has produced marked short-term reduction or elimination of moderate colic pain and improvement of more severe colic pain (6,7). Adding ketamine allows much smaller doses of xylazine or detomidine to be used, which reduces their adverse effects on cardiovascular function and gastrointestinal motility. The relief provided by the small doses of ketamine can be dramatic and the short duration of effect facilitates frequent reevaluation of the patient's level of comfort. I have successfully managed colic pain for up to two hours prior to surgical intervention using repeated small boluses of ketamine.

The presence of α2 sedation/analgesia seems to improve the relief provided by the ketamine bolus. The balance between the alpha2-adrenergic agonist (α2) and ketamine is the key to successful use of the Ketamine Stun in horses (see Enhanced Equine Chemical Restraint). Larger doses of ketamine combined with higher levels of sedation increase the risk of transient (1-3 minutes) instability. I generally try to maintain a modest level of α2 sedation with periodic doses of xylazine (0.22-0.44 mg/kg IV, or 100-200 mg/450 kg) when possible to provide greater
flexibility in ketamine dosing. I have encountered instability when pushing the limits of the Ketamine Stun technique in chemical restraint applications. I have been careful to avoid mixing high levels of both drugs when using the Ketamine Stun in colic patients and have not had a problem, though a Lexington practitioner told me he had a very painful colic patient that was initially treated with a large dose of detomidine go down after administering the ketamine bolus, though it got right back up (instability, pain, or a little of both?). Intervening early with ketamine (after an initial modest-moderate dose of an $\alpha_2$) rather than chasing the pain with additional $\alpha_2$ is the preferred approach when using the Ketamine Stun technique to manage colic pain. Additional ketamine and $\alpha_2$ is administered based on patient evaluation. The ketamine dose typically lasts 10-20 minutes and the modest-moderate doses of $\alpha_2$ generally last 20-30 minutes. The patient's pain level and the actual doses administered impact effective duration. I find writing down doses and administration times helps me decide which of the two drugs might need refreshing when pain starts to return (which is typically somewhat abrupt). In situations where heavier $\alpha_2$ sedation is present using the low ketamine dose (0.11 mg/kg IV) reduces the risk of instability, though it will also reduce the level and duration of analgesia provided. As the $\alpha_2$ effect decreases over time the size of the ketamine bolus can be increased. Due to continual redistribution the smaller doses of ketamine can generally be administered somewhat closer together to improve the level of analgesia. Experience with the Ketamine Stun technique improves dose selection and timing. Using the lower ketamine dose can provide valuable experience with the technique. Practitioners are encouraged to experiment to the extent they feel comfortable. Feedback is vital to further development of the technique.

If frequent reevaluation of the patient is not required a CRI of ketamine (10 µg/kg/min) and detomidine (2-5 mg/450kg/hr) can provide a fairly stable level of relief while still allowing periodic evaluations (analgesic effect should be substantially gone 20-30 minutes after CRI is discontinued). An analgesic CRI of lidocaine-ketamine-acepromazine can be used to minimize adverse effects on GI motility.

**Transport options**

Providing relief from colic pain during transport of the patient to a referral hospital is a very important consideration. Injury and/or respiratory compromise can occur when horses go down in the trailer. The expected duration of the trip and potential for pain to escalate during transport must be considered in making therapeutic choices. Banamine and/or detomidine can often provide sufficient coverage for patients exhibiting moderate colic pain if transport time is not excessive. If transport time is expected to be long or the patient’s level of pain is likely to worsen, options for providing additional pain relief during transport should be included in the plan. A dose of morphine (0.1-0.2 mg/kg IV or IM can be administered to augment the level of analgesia. With adequate instructions drugs can be dispensed to the owners for use during transport, if needed. Alternatively, you can follow behind the transport rig to provide assistance, if required. For patients experiencing more severe colic pain IV infusion of drug(s) can be used to provide a stable level of analgesic support.

**Colic Pain Analgesic CRI**

Note: Constant Rate Infusion notes provide more detailed information on delivery and control methods.

A “constant” rate infusion (CRI) of analgesic drugs is the best method for managing more severe colic pain during longer trips. When a simple resistive device is used to control IV infusion delivery rate, changes in pressure head (the distance from the fluid bag to the patient’s
catheter) influences the delivery rate. If the patient does go down during transport delivery will increase. If trailer does not allow vehicle passengers to visually monitor patient's position periodic stops for inspection are warranted. Delivery rate must be adjusted if the patient becomes recumbent. If a solution administration set is used to control delivery rate the owner should be provided with target drip rates for the infusion (70 ml/hr = 1.67 ml/min, with a 10 drop solution administration set that is ~12 drops/min, or 1 every 5 seconds, with a 15 drop solution administration set that is ~18 drops/min, or 1 every 3 seconds). An in-line flow control device such as the Dial-A-Flo (LifeShield Regulator IV Extension Set, Dial-A-Flo with Option-Lok, list # 11742-48, Hospira, numerous manufacturers produce similar devices) is less likely to have its setting inadvertently altered by contact with the patient. A Dial-A-Flo is also easier for the owner to adjust should the patient become recumbent during transport, especially if prior testing is performed to determine reductions required for certain alterations in pressure head.

A coiled extension set (Coiled Extension Set, CE8010, International Win, Ltd.) can be used to provide additional free-play. Using a Dial-A-Flo type device to control CRI delivery adds approximately $7 to cost. Dial-A-Flo settings (ml/hr) OFF, 20, 30, 40, 50, 60, 70, 80, 100, 125, 165, 210, 250, and OPEN (but intermediate delivery rates, e.g. not indicated on dial, can be set to accommodate patient size) allow stock 450 kg solutions to be safely delivered to a wide range of patient sizes (Table 1). One disadvantage of the Dial-A-Flo approach is the line can become tangled and occluded if the patient rolls in the trailer. The Pentafusion section of the Hagyard Equine Medical Institute Formulary contains information on creating and delivering analgesic CRI solutions. Detomidine infusion rates used in chemical restraint and colic pain applications are often higher than those used in Pentafusion.

A drip rate monitor makes monitoring and adjusting CRI delivery easy for owners. Drip Assist (Veterinary DripAssist Infusion Rate Monitor, 000A2773, Hallowell EMC, $225) is a simple battery powered (1 AA) device easily placed around the drip chamber of a solution administration set to continuously monitor delivery rate and volume. Display can be set to read drops/min, ml/hr, or total volume delivered (ml). It does not control delivery rate, but makes it easier to set and adjust (display responds to new setting within 3-4 drops). Unit includes an audible alarm function that warns user if delivery deviates +/- 13% of selected rate. If solution concentration allows DripAssist can be used in series with an IV flow control device (e.g. Dial-A-Flo). Adding a simple remote monitoring system (audio or audio/visual) to the DripAssist – Dial-A-Flo combination provides a pretty respectable cost effective CRI delivery setup.

450 kg stock CRI solutions for colic pain control

Delivery rate determines the drug concentrations required in the carrier solution. Drug concentrations are then used to determine the amount of each drug required for a pre-selected volume of carrier solution. First you select a flow setting. I prefer 70 ml/hr as the base rate for my stock 450 kg solution because I use similar flow rates in many other CRI applications and there are several Dial-A-Flo settings near this value to facilitate easy adjustments in delivery, if necessary. The patient’s base delivery rate is simply a ratio of the stock 450 kg base rate (e.g. for a 337 kg patient delivery rate would be 337/450 = 0.75 x 70 ml/hr = "50" ml/hr). Greater latitude in selecting a delivery setting is acceptable when lidocaine is not used as the carrier solution. Next, select a desired duration for the infusion (provide a cushion in case transport takes longer than planned). The combination of flow rate and intended duration determine the amount of carrier solution required (e.g. for a 3-hour duration at 70ml/hr a 210 ml volume of carrier solution is required). Select the drug combination to be delivered (detomidine, detomidine-ketamine, detomidine-ketamine-opioid, lidocaine-ketamine-detomidine-opioid, acepromazine-ketamine-lidocaine, etc.). Empty a bag of electrolyte solution to a final volume
selected (200 ml in the above example, mild rounding is allowed). Add the proper amount of each drug based for the drug combination selected (Table 2). If not recently administered a loading dose of detomidine (0.022-0.044 mg/kg IV), morphine (0.1 mg/kg IV) or butorphanol (0.022 mg/kg IV) quickens the onset of relief.

(Table 1) Patient weight (kg) and corresponding Dial-A-Flo settings for safe delivery of undiluted 2% lidocaine. Table can also be used as a guide in determining the appropriate setting for a patient when using a "stock 70ml/450kg/hr" analgesic mixture

<table>
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<th>30</th>
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</tbody>
</table>

(Table 2) Drug options for 450 kg stock colic analgesic solutions.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Dose</th>
<th>Bottle concentration</th>
<th>Volume per 70 ml (1 hr) of 450 kg stock solution</th>
<th>Volume per 200 ml (3 hr) of 450 kg stock solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Detomidine</td>
<td>0.0044 mg/kg/hr</td>
<td>2 mg/450kg/hr</td>
<td>10 mg/ml</td>
<td>0.2 ml</td>
<td>0.6 ml</td>
</tr>
<tr>
<td>Detomidine</td>
<td>0.011 mg/kg/hr</td>
<td>5 mg/450kg/hr</td>
<td>10 mg/ml</td>
<td>0.5 ml</td>
<td>1.5 ml</td>
</tr>
<tr>
<td>Ketamine</td>
<td>0.6 mg/kg/hr</td>
<td>270 mg/450kg/hr</td>
<td>100 mg/ml</td>
<td>2.7 ml</td>
<td>8.0 ml</td>
</tr>
<tr>
<td>Morphine</td>
<td>0.033 mg/kg/hr</td>
<td>15 mg/450kg/hr</td>
<td>15 mg/ml</td>
<td>1.0 ml</td>
<td>3.0 ml</td>
</tr>
<tr>
<td>Morphine</td>
<td>0.067 mg/kg/hr</td>
<td>30 mg/450kg/hr</td>
<td>15 mg/ml</td>
<td>2.0 ml</td>
<td>6.0 ml</td>
</tr>
<tr>
<td>Butorphanol</td>
<td>0.022 mg/kg/hr</td>
<td>10 mg/450kg/hr</td>
<td>10 mg/ml</td>
<td>1.0 ml</td>
<td>3.0 ml</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>3.0 mg/kg/hr</td>
<td>1,400 mg/450kg/hr</td>
<td>20 mg/ml</td>
<td>68 ml</td>
<td>200 ml</td>
</tr>
<tr>
<td>Acepromazine</td>
<td>0.0022 mg/kg/hr</td>
<td>1 mg/450kg/hr</td>
<td>10 mg/ml</td>
<td>0.1 ml</td>
<td>0.3 ml</td>
</tr>
</tbody>
</table>

* For special circumstances - avoid if anesthesia is contemplated or patient is hypotensive.

Lidocaine (50 mcg/kg/min, or 68 ml/450 kg/hr of 2%) can be safely substituted as the carrier solution in this CRI technique, enhancing the analgesia provided and also adding anti-inflammatory activity that recent studies indicate may be important in minimizing the incidence of post-colic laminitis. When lidocaine is used as the carrier solution its safe delivery rate determines the flow that must be used. I have consulted a number of experts and none have experience with delivery significantly higher in equine patients than the dose provided in these notes. To minimize risk of overdose lidocaine delivery should be "as close as possible" to the calculated dose. Some latitude is allowed, but extremely large or obese patients (greater surface area/body weight divergence), hypoproteinemia, and inaccurate weight estimates can
reduce whatever cushion exists. Examples of safe delivery rates for lidocaine are provided for a number of body weights (Table 1). Intermediate delivery rates, e.g. not indicated on dial, can be set to accommodate patient size. **Delivery rate should be verified initially and periodically evaluated when using a Dial-A-Flo type device (using drip rate and drops/ml conversion factor for the solution administration set).** When lidocaine is used as the base solution for the CRI, a loading bolus of lidocaine (1.3 mg/kg IV, given slowly) should be administered just prior to starting the CRI to counter its rapid clearance rate.

**Sample Protocol using the Dial-A-Flo method** *(450kg patient, 70ml/hr, 3hr duration):*

Attach solution administration set and empty bag of electrolyte solution to a final volume of 200ml. Add 10 mg of detomidine (intermediate dose), 800 mg ketamine (and, optionally, 45 mg of morphine or 30 mg of butorphanol). Attach Dial-A-Flo device and coiled extension set (optional). Fully open the flow adjustment of the solution administration set and the Dial-A-Flo device to fill fluid lines with drug containing solution. Adjust Dial-A-Flo to desired 70ml/hr delivery rate and attach to IV catheter. If not recently administered a loading dose of detomidine (0.022-0.044 mg/kg IV), morphine (0.1 mg/kg IV) or butorphanol (0.022 mg/kg IV) quickens the onset of relief.

**Elastomeric Infusion Pump**

Another option for controlling an analgesic CRI is the use of an elastomeric infusion pump, such as the Homepump Eclipse (Homepump Eclipse E401000, Halyard Health, Inc, numerous manufacturers produce similar devices, a variety of delivery rate / volume combination are available). The elastomeric infusion pump is attached directly to the patient so the risk of delivery interruption should the patient roll is reduced. Elastomeric infusion pumps are more expensive than the Dial-A-Flo and the limited number of fixed delivery rates and volumes require a more customized approach. Medical supply vendors sell elastomeric infusion pumps by the case (24 or 48 units depending on model). Prices seem to vary markedly from vendor to vendor (Homepump Eclipse E401000 per unit pricing ranged from $17.38 to $32.95, January 2016). I have been able to purchase individual units at reasonable prices from local human pharmacies with infusion departments. It may take several phone calls to locate a willing local pharmacy and/or obtain best pricing. The only way to ensure 24/7 availability is to have a unit or two in stock.

Under ideal (specified) conditions delivery accuracy is ± 15%. Understanding how flow is controlled and factors that affect delivery rate is important to the safe and effective use of elastomeric infusion pumps.

Drug compatibility is critically important when using elastomeric infusion pumps. The microscopic bores used to regulate flow are easily plugged with drug precipitate or contaminants. I have tested various combinations of drugs from Table 2 in concentrated and dilute solutions and found no macro or microscopic signs of precipitation.

**Concentrated Solutions (for 2.5 m/hr delivery rate)**
- Butorphanol-acepromazine-saline
- Butorphanol-detomidine-saline
- Butorphanol-acepromazine-ketamine
- Butorphanol-detomidine-ketamine
- Butorphanol-acepromazine-detomidine-ketamine
Morphine-acepromazine-saline
Morphine-detomidine-saline
Morphine-acepromazine-ketamine
Morphine-detomidine-ketamine
Morphine-acepromazine-detomidine-ketamine

Dilute Solutions
Pentafusion (ketamine, morphine, detomidine, & acepromazine in 2% lidocaine)

Creating a mixture to be delivered using an elastomeric infusion pump is actually quite simple. Delivery rate determines the drug concentrations required in the carrier solution. Drug concentrations are used to determine the amount of each drug required for a pre-selected volume of carrier solution. The Homepump Eclipse E401000 delivers 100 ml/hr with a nominal capacity of 400 ml (package insert provides information on minimum and maximum fill volumes, which are 200 ml and 500 ml respectively for this unit, and their impact on delivery rate, which varies slightly). When filled with 400 ml this unit provides a 4-hour duration of delivery. In this example you would add a 4 hr amount of each drug selected (based on the doses provided in 2nd column of Table 2) to the device and then q.s. to 400 ml with carrier solution. Attach reservoir to patient (using braid in mane) and device tubing to extension set of catheter. DO NOT encapsulate items in bandage system that must remain at ambient temperature. The Homepump Eclipse reservoir and distal flow limiter tubing segment are designed to operate at room temperature (68°F). The Homepump C-Series and MILA 7100 reservoirs are designed to operate at room temperature (68°F); their distal flow limiter tubing segments are designed to operate at skin temperature (88°F). When lidocaine is included in the CRI, a loading bolus of lidocaine (1.3 mg/kg IV, given slowly) should be administered just prior to starting the CRI to counter its rapid clearance rate.

Interval between filling and use affects delivery rate. The Homepump C-Series is intended for immediate use (delays greater than 8 hours decrease delivery by 10%). Unfortunately the current delivery rate / volume options are not very useful for equine applications. Using the Homepump Eclipse within the first 4 hours after filling increases delivery. Testing with the 100ml / 100ml/hr model revealed initial flow was + 50%, but the average flow rate for the entire hour infusion period was + 20%, indicating the peak effect is somewhat short-lived. For emergency applications fill the Homepump Eclipse as early as feasible. The risk associated with short-term increases in delivery for many of the drugs listed in Table 2 is minimal (ketamine and lidocaine being the exceptions). Dosages for the others can be reduced by 20% to compensate for the difference in delivery. Using D5W as a carrier solution decreases delivery rate by 10, a simple method of reducing the impact of immediate use. A 50% increase in ketamine or lidocaine delivery might result in unintended recumbency. If I were put in a situation where I thought ketamine and/or lidocaine were required I would try the following adjustments. Reducing ketamine dose by 50% (titrated level) and then by an additional 20% (i.e. 0.6 x .5 = 0.3, 0.3 x 0.8 = 0.24 mg/kg/hr) might allow it to be used safely. Reducing the lidocaine infusion by 20% and the loading bolus by 50% might allow it to be used safely.

Elastomeric infusion pumps with flow-limiters designed to operate at a set ambient temperature are more vulnerable to differences in environmental temperature. The impact of temperature differences (88°F increases delivery rate 28%) will depend on the drugs infused. Dosages for the drugs in Table 2 can be adjusted by an amount equal to the calculated percentage of flow change for a given temperature. Using D5W as the carrier solution decreases delivery rate by 10%, a simple adjustment may be adequate for 7-10 increase in temperature. Using an insulated cover will slow temperature change of reservoir. Temperature correction tables can be
created for common solutions to simplify adjustments. Compensating for very cold temperatures is more challenging. Placing the flow-limiting segment in contact with the skin should theoretically increase flow by 28% (20°F x 1.4%). Adjust solution for the increased flow rate and place the elastomeric reservoir in an insulated cover to slow cooling that may alter its performance.

The MILA 7100 is commonly used in equine hospitals to deliver antibiotic infusion to septic joints and tendon sheath. It possesses a delivery rate / volume combination that accommodates some equine analgesic CRI combinations. The package states it is not intended for IV access, but I have used the MILA 7100 on several occasions when it was the only option available to provide continuous pain relief in equine patients with severe pain. The human version is labeled for IV use according to the president of MILA.

It is more difficult to visually estimate remaining volume when using an elastomeric infusion pump. Placing a stopcock at the junction between the device tubing and catheter extension set facilitates evaluation of delivery status (flow rate, catheter patency) as well as administration of ancillary IV drugs.

**Sample protocol using a Homepump Eclipse E401000 elastomeric infusion pump (450kg patient, 4 hr duration):**

Inject 12 mg of detomidine, 1000 mg of ketamine (and, optionally, 60 mg of morphine or 40 mg of butorphanol) into the device. Fill device to 400 ml with carrier solution (which in this detomidine-ketamine example could be "394" ml of electrolyte solution or 270 ml of lidocaine (3 mg/kg/hr x 450 kg x 4 hr, divided by 20 mg/ml) plus "124" ml of electrolyte solution (minor rounding is allowed). Attach, administer loading doses if appropriate, and initiate infusion.

**Anesthesia to control colic pain**

Violently painful colic patients are especially challenging. They frequently arrive down in the trailer and respond poorly to aggressive analgesic intervention. Anesthesia makes further evaluation safer for both the patient and personnel involved and can also be used to provide humane relief while owners make a decision regarding surgical intervention. Anesthesia can be used to provide the same benefits in the field setting, if necessary. Loading an anesthetized horse into a trailer for transport to a hospital is no easy task, though it can be accomplished.

**References**

Pentafusion: Analgesia for Severe Pain

Eric J. Abrahamsen, DVM, DACVA

Note: 15 mg/ml multi-dose vials of morphine have been discontinued due to new FDA regulations. See Meet the New Morphine notes for important information regarding the safe use of preservative free morphine.

Pentafusion

In patients with severe pain, IM morphine in conjunction with NSAID administration does not provide sufficient relief. Increasing the dose of IM morphine can improve the level of relief provided, but the combination of higher morphine and pain levels increases the risk of GI complications. Using a constant rate infusion (CRI) technique to deliver a steady state of analgesic support provided by a combination of drugs was the next logical step to try in these patients. The drug mixture used in the pain CRI has been modified and refined over time. The five-drug mixture (lidocaine, ketamine, morphine, detomidine, and acepromazine) that resulted from this quest has been used in dozens of horses and proven to be effective in alleviating all but the most extreme pain in these patients (2,3). Due to its five components and CRI delivery it was given the name Pentafusion. Pentafusion recipe is included the Hagyard Equine Medical Institute Formulary.

Food animal clinicians soon requested a similar approach for ruminant patients, but were concerned about the continuous delivery of the alpha2-adrenergic agonist (detomidine) and acepromazine components of Pentafusion. Having no experience with continuous delivery of these drugs in ruminant patients to neutralize their concerns, I modified the technique, creating Trifusion. Trifusion is a mixture of lidocaine, ketamine, and an opioid. I have used both butorphanol and morphine as the opioid component of Trifusion. I have used Trifusion in four adult cattle and two camelids with severe to extreme pain (4,5). The first ruminant patient received a butorphanol-based combination, while the next three received a morphine-based combination. Both of the camelids received butorphanol at the request of the primary clinician. Trifusion provided obvious relief in these patients but did not eliminate the pain. The small number of patients makes comparison of the opioid component difficult at this point. The initial pain level in these patients was quite high. Based on experience in horses, detomidine enhances the analgesic efficacy of the pain CRI and its inclusion may have provided greater analgesic support to these patients. Because detomidine is dosed similarly in cattle and horses, Pentafusion may eventually prove to be useful in ruminant patients as well. I have not used detomidine in a camelid patient to date, but expect an infusion rate could be worked out. Trifusion was administered for several days in each of these patients with no adverse effects noted.

Stock solutions simplify CRI techniques

A CRI technique may seem complicated, but it is much easier than it first appears. Using a stock solution (created for a prototypical patient such as the 450 kg example for equine patients described below) simplifies the technique. Delivery rate is adjusted to the patient's weight using a ratio. If the base infusion rate for a 450 kg patient is 68 ml/hr, the base infusion rate for a 45 kg patient is 6.8 ml/hr (45 kg/450 kg = 0.1, 0.1 x 68 ml/hr = 6.8 ml/hr). Smaller volumes of the
stock 450 kg mixtures can be created to reduce expense and waste when Pentafusion or Trifusion are used in smaller patients. Placing all the drugs to be delivered in one bag (1-bag administration) requires less equipment and disposables, though you lose some of the flexibility provided by delivering drugs via two bags (2-bag administration). I typically use the 1-bag approach to deliver Trifusion and the 2-bag approach to deliver Pentafusion.

**Pentafusion: development and use**

Lidocaine infusions (50 µg/kg/min) used to promote GI motility in postoperative colic patients do not seem to provide much systemic analgesia when used alone. Lidocaine becomes much more important when combined with other analgesic drugs. As an example, we had a horse with clostridial myositis that was extremely painful on presentation. IV and IM doses of detomidine and morphine had not produced much improvement when the primary clinicians on the case asked for a pain consult. I put the horse on a CRI of lidocaine (3 mg/kg/hr) and morphine (0.025 mg/kg/hr) (along with small doses of IM acepromazine). The horse remained uncomfortable, but there was a noticeable improvement with the infusion. Because higher blood levels of detomidine and morphine had not provided the same degree of relief, I surmised that the lidocaine contribution was much greater when it was combined with other analgesic drugs. The lidocaine-morphine CRI did not provide the level of analgesia required to make this extremely painful patient comfortable (it was eventually euthanatized), so I decided to add a small CRI of ketamine (0.6 mg/kg/hr) the next time I used the pain CRI technique. Ketamine is an NMDA receptor antagonist. Ketamine has been shown to possess potent analgesic effects when administered at subanesthetic doses. Ketamine also reduces *mu* opioid tolerance, improving the efficacy of morphine. I was concerned about the potential for adverse behavioral effects (mania) resulting from morphine accumulation or excessive CNS stimulation from ketamine accumulation as infusion duration increased. I added a CRI of detomidine (0.0044 mg/kg/hr) to replace the small boluses of acepromazine I typically administer to equine patients receiving morphine. Detomidine possesses potent analgesic effects, and though the dose used was low, I hoped it would enhance the level of relief provided by the CRI technique while providing protection against drug-induced behavioral changes. This combination proved effective in treating severe laminitis pain (e.g., laterally recumbent with rapid heart and respiratory rates, "groaning" with each exhalation) in several patients.

Attempts to alter the rate of administration of some of the drugs contained in the pain CRI provided some insight as to the relative importance of those components in treating severe pain in the horse. In the next few laminitis patients I tried to substitute acepromazine (0.0022 mg/kg/hr) for the detomidine because it was being administered in IV boluses as part of the routine therapeutic approach in these patients, but I was not as satisfied with the relief provided. Detomidine was returned to the mix, but acepromazine was retained to help counter the vasoconstrictive effects of the detomidine and ketamine. The detomidine and acepromazine components do not produce overt sedation or tranquilization of the patient, though a calming influence may be present. A lower initial infusion rate of ketamine (0.3 mg/kg/hr) was tried in a couple of horses but yielded a less satisfactory level of relief. Patient pain also increased noticeably approximately 30 minutes following reduction or discontinuation of the ketamine component of the CRI, further indicating its importance and the potential for titrating the level of relief provided.

Pentafusion has been used successfully in dozens of horses to provide relief from severe pain. Many of these patients were facing imminent euthanasia when Pentafusion was instituted. The relief provided by Pentafusion allowed owners to be comfortable with continued treatment and several of these patients were eventually discharged from the hospital. I have used Pentafusion
for up to 17 days without complication, though the typical duration is shorter. I have encountered two horses to date with pain levels so severe that Pentafusion was not able to provide adequate relief. Clinical improvements such as return of appetite and the ability to get up for brief periods were evident in both of these patients with the use of Pentafusion, but even when a supersized rate (1.5×) of Pentafusion was used, adequate relief was not obtained. One of these patients experienced GI stasis and bloating during the enhanced rate of administration, though the problem persisted well beyond the discontinuation of the morphine and detomidine components of Pentafusion therapy and may have resulted, at least in part, due to unrelenting pain. Both of these patients were eventually euthanatized. Three horses developed mild impactions that were successfully resolved with the use of sodium sulfate and IV fluid therapy. Pain produces an adverse effect on GI motility and likely contributes to the GI complication rate in patients receiving opioid analgesic support.

GI dysfunction and potential colic (gas, impaction) should always be a considered when using morphine or Pentafusion in equine patients, especially in the early stages of treatment. GI motility (auscultation of all 4 quadrants), and fecal output (volume and moisture level) should be monitored frequently. A modest reduction in fecal output (& moisture) is generally not a concern as long as GI motility remains acceptable, but does warrant closer observation until proven harmless. Water intake should also be monitored closely and methods used to encourage consumption (multiple bucket locations, etc). Patient should be regularly observed for symptoms of colic in the initial stages of morphine analgesic therapy. It is important to remember that two of drugs associated with decreased GI motility (alpha2-adrenergic agonists & opioids) also possess analgesic properties that will mask milder symptoms of colic. CRI delivery does not provide trough levels; reduction or discontinuation of delivery can be used to evaluate patient status and perhaps improve titration of analgesic support if GI motility becomes a concern.

If GI dysfunction is suspected appropriate medical therapy should be instituted. Morphine and detomidine administration is reduced or discontinued, depending on the severity of the GI disturbance. A lidocaine-ketamine-acepromazine infusion can be used to provide analgesic support in patients where morphine must be discontinued.

Prophylactic administration of mineral oil may alleviate some of the anxiety associated with using morphine in equine patients and perhaps improve success should intervention be required.

When pain relief allows, we reduce the acepromazine-morphine-detomidine infusion rate (typically by half initially, and then perhaps by half again) before reducing the lidocaine-ketamine infusion rate. This allows us to alter the degree of analgesic support while at the same time reducing the administration rate drugs with the greatest concerns regarding GI motility. Initial reduction typically occurs within the first 12 hours of Pentafusion delivery. The negative GI effects are constant with CRI delivery so the early reduction may be an important factor in the low incidence of problems.

Pentafusion and Trifusion CRI techniques

The same basic approach is used to mix and deliver both Trifusion and Pentafusion. In ruminant and camelid patients a smaller loading dose of lidocaine (1 mg/kg IV) is used (loading doses of lidocaine should be administered slowly to prevent adverse cardiovascular or CNS effects). Lidocaine, ketamine and opioid infusion rates are the same in both Trifusion and Pentafusion. Butorphanol can be substituted for morphine when using
Trifusion or Pentafusion, though analgesic support may be reduced. The loading dose of butorphanol (0.05-0.1 mg/kg IV or IM in smaller ruminants, 0.02-0.05 mg/kg IV or IM in larger ruminants) and the CRI dose for butorphanol (0.022 mg/kg/hr) are substituted in the Pentafusion protocol described below.

**Pentafusion: equipment required**

I use a 2-bag approach to deliver Pentafusion. The ketamine and lidocaine are combined in one bag and the morphine, detomidine, and acepromazine combined in the second bag. The 2-bag approach allows greater flexibility in altering the levels of the drugs being delivered, which facilitates titration of the relief provided, adjustments to counter potential side effect produced by the drugs used, and eventual weaning when desired. The 2-bag approach requires 2 infusions pumps and a slighter greater amount of disposables (Table 1). A 1-bag approach can be used, if desired, by placing the morphine, ketamine, detomidine, and acepromazine in the bag containing the 2% lidocaine. The 1-bag approach only requires one infusion pump and fewer disposables, though wastage will be higher if solution must be reformulated to alter delivery (Table 2).

**(Table 1) Equipment Required for 2-Bag Administration**

<table>
<thead>
<tr>
<th>Equipment</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 Fluid Pumps</td>
<td>Heska Vet/IV 2.2, Heska Corporation, Loveland, CO (compact rechargeable unit made for the veterinary market; many other models of infusion pumps are available including used human units)</td>
</tr>
<tr>
<td>2 Solution Administration Sets</td>
<td>Baxter Healthcare Corp., 10 drops/ml and approximately 68 inches long</td>
</tr>
<tr>
<td>4 Coiled Extension Sets</td>
<td>CE8010, International Win, Ltd</td>
</tr>
<tr>
<td>1 liter normal saline</td>
<td></td>
</tr>
<tr>
<td>1 Sterile 1 liter bag (a liter bag of fluids can be emptied)</td>
<td>Intra Via Container, Baxter Healthcare</td>
</tr>
<tr>
<td>1 High Flow Double T Extension Set</td>
<td>8575, Mila International, Inc</td>
</tr>
<tr>
<td>1 Needle-lock device (if pain CRI is to be connected to IV fluid line)</td>
<td>2C7833, Baxter Healthcare Corp</td>
</tr>
</tbody>
</table>

**(Table 2) Equipment required for 1-Bag Administration**

<table>
<thead>
<tr>
<th>Equipment</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Fluid Pump</td>
<td>Heska Vet/IV 2.2, Heska Corporation, Loveland, CO (compact rechargeable unit made for the veterinary market; many other models of infusion pumps are available including used human units)</td>
</tr>
<tr>
<td>1 Solution Administration Set</td>
<td>Baxter Healthcare Corp., 10 drops/ml and approximately 68 inches long</td>
</tr>
<tr>
<td>2 Coiled Extension Sets</td>
<td>CE8010, International Win, Ltd</td>
</tr>
<tr>
<td>1 Sterile 1 liter bag (a liter bag of fluids can be emptied)</td>
<td>Intra Via Container, Baxter Healthcare</td>
</tr>
<tr>
<td>1 Needle-lock device (if pain CRI is to be connected to IV fluid line)</td>
<td>2C7833, Baxter Healthcare Corp</td>
</tr>
</tbody>
</table>
The drug volumes added to the bags are small enough to not alter the effective concentrations, and removal of a commensurate volume of carrier solution is not required when using this technique. The infusion mixtures should be created in the bags before attaching and filling the lines. Otherwise, the extensive void volume of the lines will prevent delivery of medication for quite some time, limiting the usefulness of any loading boluses administered and delaying the onset of relief.

**Pentafusion: sample protocol (450-kg patient)**

The 450-kg mixture(s) can be used as a stock solution(s) with adjustments in delivery rate made to accommodate variations in patient size and/or alter the level of analgesic relief provided. The "450-kg base rate(s)" of delivery is adjusted to patient size using ratios (e.g., "patient base rate(s)" for a 337-kg horse is 75% or 0.75 of the "450-kg base rate(s)"). The "patient base rate(s)" determined in this manner can be further adjusted to alter the level of analgesia provided.

**Bag #1**
The empty sterile bag is filled with 1 L of 2% lidocaine. Lidocaine is administered at 50 µg/kg/min. For a 450 kg horse this is 22,500 µg/min, or 22.5 mg/min. Because 2% lidocaine is 20 mg/ml, one needs to administer 1.125 ml/min, or 67.5 ml/hr. This sets the flow rate for bag #1 at 68 ml/hr.

Ketamine is added to bag #1. Ketamine is administered at 10 µg/kg/min. For a 450 kg horse this is 4500 µg/min, or 4.5 mg/min. The amount of ketamine required per hour is 270 mg, which must be contained in the 68 ml of lidocaine delivered per hour. The concentration of ketamine in the bag required to provide this level of administration is 4 mg/ml, so 4 g of ketamine are added to the liter of 2% lidocaine.

Attach solution administration set and two coiled extension sets to bag #1 and fill the line. Set fluid pump for bag #1 at 68 ml/hr.

**Bag #2**
I typically set the infusion rate for bag #2 at the same level as calculated for bag #1. In this example it will be 68 ml/hr. Using the same infusion rate for bag #2 allows the drug amounts calculated for morphine, detomidine, and acepromazine to be added to the lidocaine solution for a 1-bag technique. Morphine, detomidine, and acepromazine are added to a 1-L bag of normal saline in the following manner.

Morphine is dosed at 0.025 mg/kg/hr. For a 450 kg patient this calculates out to 11.25 mg/hr, which must be contained in the 68 ml of saline delivered each hour. Concentration of morphine in the bag required to provide this level of administration is 0.17 mg/ml, so 170 mg of morphine is added to the liter of normal saline.

Detomidine is dosed at 2 mg/450kg/hr, or 2 mg/hr in this example. This amount must be contained in the 68 ml of saline delivered each hour. Concentration of detomidine in the bag required to provide this level of administration is 0.03 mg/ml, so 30 mg of detomidine is added to the liter of normal saline.

Acepromazine is dosed at 1 mg/450kg/hr, or 1 mg/hr. This amount must be contained in the 68 ml of saline delivered each hour. Concentration of acepromazine in the bag to provide this level
of administration is 0.015 mg/ml, so 15 mg of acepromazine is added to the liter of normal saline.

Attach solution administration set and two coiled extension sets to bag #2 and fill the line. Set the fluid pump for bag #2 at 68 ml/hr.

A chart can be created to organize information used to create the infusion mixtures (Table 3 & 4).

**Pentafusion: clinical application**

Create 450 kg stock solutions for bags #1 and #2 (using Tables 3 & 4) with adjustments in delivery rate to fit patient size. Generally creating "12 hour" volumes (e.g. 1000 ml @ 68 mL/hr = 14.7 hr, 100 mL @ 6.8 mL/hr = 14.7 hr) minimizes waste should mixture adjustments be required. The patient’s fluid status should be evaluated to determine if a reduction in maintenance fluids rate may be necessary to accommodate the analgesic volume delivered.

((Table 3) Pentafusion: Recipe for 1 L of 450 kg stock solution. Adjust to patient size using ratio.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Amount Required</th>
<th>Base Delivery Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bag #1</td>
<td></td>
<td></td>
<td>68 ml/450kg/hr</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>3 mg/kg/hr</td>
<td>1 liter</td>
<td></td>
</tr>
<tr>
<td>Ketamine</td>
<td>0.6 mg/kg/hr</td>
<td>4,000 mg</td>
<td></td>
</tr>
<tr>
<td>Bag #2</td>
<td></td>
<td></td>
<td>68 ml/450kg/hr</td>
</tr>
<tr>
<td>NaCl</td>
<td>NA</td>
<td>1 liter</td>
<td></td>
</tr>
<tr>
<td>Morphine</td>
<td>0.025 mg/kg/hr</td>
<td>170 mg</td>
<td></td>
</tr>
<tr>
<td>Detomidine</td>
<td>0.0044 mg/kg/hr</td>
<td>30 mg</td>
<td></td>
</tr>
<tr>
<td>Acepromazine</td>
<td>0.0022 mg/kg/hr</td>
<td>15 mg</td>
<td></td>
</tr>
</tbody>
</table>

((Table 4) Trifusion: Recipe for 1 L of 450 kg stock solution. Adjust to patient size using ratio. Morphine can be placed in Bag #1 for 1-bag administration.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Amount Required</th>
<th>Base Delivery Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bag #1</td>
<td></td>
<td></td>
<td>68 ml/450kg/hr</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>3 mg/kg/hr</td>
<td>1 liter</td>
<td></td>
</tr>
<tr>
<td>Ketamine</td>
<td>0.6 mg/kg/hr</td>
<td>4,000 mg</td>
<td></td>
</tr>
<tr>
<td>Bag #2</td>
<td></td>
<td></td>
<td>68 ml/450kg/hr</td>
</tr>
<tr>
<td>NaCl</td>
<td>NA</td>
<td>1 liter</td>
<td></td>
</tr>
<tr>
<td>Morphine</td>
<td>0.025 mg/kg/hr</td>
<td>170 mg</td>
<td></td>
</tr>
</tbody>
</table>
Fill lines and insert the administration sets in the fluid pumps and set volumes to be delivered. The attached coiled extension sets leading from each of the treatment bags are looped over a swiveling fluid bag hook above the stall and connected to the primary IV fluid line or catheter using the High Flow Double T Extension Set. A needle-lock device is used to secure the High Flow Double T Extension Set to the Y-injection port of the primary fluid line if using that approach. Loading doses of several of the drugs can be given before starting the CRI to speed the onset of relief. I always administer a loading dose of lidocaine (1.3 mg/kg IV equine, 1.0 mg/kg IV for ruminants and camels, administer slowly). If the patient has not recently received morphine as part of its prior therapeutic regimen, I generally give a loading dose of it as well (0.1 mg/kg IV) to speed onset of relief. I generally do not administer a loading dose of ketamine, detomidine, or acepromazine. Time to peak relief is typically several hours, so some patience is required.

When pain relief allows, I reduce the acepromazine-morphine-detomidine infusion rate (typically by half initially, and then perhaps by half again) before reducing the ketamine lidocaine infusion rate. This allows me to alter the degree of analgesic support while at the same time reducing the administration rate drugs with the greatest concerns regarding GI motility. Based on personal experience, the analgesic effects of the ketamine infusion are markedly diminished within 30 minutes of its discontinuation. Initial reduction typically occurs within the first 12 hours of Pentafusion delivery.

**NOTE:** The use of a CRI analgesic technique makes evaluation of pain level more challenging because there are no peaks and valleys in the level of analgesic support. Delivery must be reduced (or less desirably discontinued) to evaluate the patient's comfort level. This is especially crucial in patients with GI pain, where the technique can mask symptoms requiring surgical intervention.

**Controlling Pentafusion Delivery**

**Note:** Constant Rate Infusion notes provide more detailed information on delivery and control methods.

Infusion pump(s) are the preferred method for controlling Pentafusion delivery.

Drip Assist (Veterinary DripAssist Infusion Rate Monitor, 000A2773, Hallowell EMC, $225) is a simple battery powered (1 AA) device easily placed around the drip chamber of a solution administration set to continuously monitor delivery rate and volume. Display can be set to read drops/min, ml/hr, or total volume delivered (ml). It does not control delivery rate, but makes it easier to set and adjust (display responds to new setting within 3-4 drops). Unit includes an audible alarm function that warns user if delivery deviates +/- 13% of selected rate. If solution concentration allows DripAssist can be used in series with an IV flow control device (e.g. Dial-A-Flo). Adding a simple remote monitoring system (audio or audio/visual) to the DripAssist – Dial-A-Flo combination provides a pretty respectable cost effective CRI delivery setup.

Elastomeric infusion pumps such as the Homepump Eclipse or C-Series (Homepump Eclipse E401000, I-Flow Corporation, numerous manufacturers produce similar devices, a variety of pre-set delivery rates and volumes are available) attach to the patient, but their limited volume require more frequent filling. Creating a mixture to be delivered using an elastomeric infusion pump is actually quite simple. Delivery rate determines the drug concentrations required in the carrier solution. Drug concentrations are used to determine the amount of each drug required for a pre-selected volume of carrier solution. The Homepump Eclipse E401000 delivers 100
ml/hr with a nominal capacity of 400 ml (package insert provides information on minimum and maximum fill volumes, which are 200 ml and 500 ml respectively for this unit, and their impact on delivery rate, which varies slightly). When filled with 400 ml this unit provides a 4-hour duration of delivery. In this example you would add a 4 hr amount of each drug (based on the doses provided in 2nd column of Tables 3 & 4) to the device and then fill remaining volume with carrier solution (which again can be a properly adjusted lidocaine solution if desired). Attach to IV catheter.

With close supervision in-line flow control devices such as a Dial-A-Flo (LifeShield Regulator IV Extension Set, Dial-A-Flo with Option-Lok, list # 11742-48, Hospira, numerous manufacturers produce similar devices) can be used. The Dial-A-Flo has "settings" (ml/hr) of OFF, 20, 30, 40, 50, 60, 70, 80, 100, 125, 165, 210, 250, and OPEN, but intermediate delivery rates (e.g. not indicated on dial) can be set to accommodate patient size. With 70 ml/hr representing full delivery for a 450 kg patient, the appropriate weight for other settings is easily determined (e.g. 50 ml/hr ÷ 68 ml/hr = 0.73 x 450 kg = 330 kg) (Table 5). Dial-A-Flo type devices are designed to work within a certain specified height differential (solution container to patient catheter). They seem to provide steady delivery outside of this range, but rate may vary from setting. Delivery rate should be verified initially and periodically evaluated when using a Dial-A-Flo type device (using drip rate and drops/ml conversion factor for the solution administration set). Periodic inspection is also required to detect occlusive problems in delivery system.

(Table 5) Patient weight (kg) and corresponding Dial-A-Flo setting for "maximal" safe delivery when using a "stock 70ml/450kg/hr" analgesic mixture (with or without undiluted lidocaine as a carrier solution).

<table>
<thead>
<tr>
<th>Setting (ml/hr)</th>
<th>20</th>
<th>30</th>
<th>40</th>
<th>50</th>
<th>60</th>
<th>70</th>
<th>80</th>
<th>100</th>
<th>125</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>130</td>
<td>200</td>
<td>265</td>
<td>330</td>
<td>400</td>
<td>450</td>
<td>530</td>
<td>660</td>
<td>830</td>
</tr>
</tbody>
</table>

With extremely close supervision solution administration sets can be used.

References:


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Enhanced Equine Chemical Restraint: Ketamine Stun & Ketamine Stun CRI

Eric J. Abrahamsen, DVM, DACVA

Note: 15 mg/ml multi-dose vials of morphine have been discontinued due to new FDA regulations. See Meet the New Morphine notes for important information regarding the safe use of preservative free morphine.

ENHANCED CONTROL FOR SHORT PROCEDURES

Ketamine Stun

In the early 1990's I began experimenting with small doses of ketamine added to chemical restraint combinations to augment the level of patient cooperation and systemic analgesia in a variety of species. I call this approach the Ketamine Stun because of the stunned appearance it produces in recumbent applications. A fairly intense level of systemic analgesia accompanies this semi-anesthetized state.

Alpha₂-adrenergic agonists possess potent sedative and analgesic effects. Opioids are typically thought of as analgesic drugs, but they possess central nervous system (CNS) effects that when combined with a tranquilizer or sedative produces a greater level of mental depression. Ketamine is a dissociative anesthetic commonly used in veterinary medicine. Ketamine is a N-methyl-D-aspartate (NMDA) receptor antagonist that possesses potent analgesic effects at sub-anesthetic doses. Ketamine was initially included for its analgesic properties, but likely contributes to the mental aspects of the enhanced cooperation exhibited by patients under the influence of the Ketamine Stun technique. Using a combination of drugs to achieve the desired effect allows smaller doses of the individual drugs to be used, which reduces the impact of their adverse side effects.

The Ketamine Stun techniques for camelids and food animal patients are fairly well developed. Moderate chemical restraint of camelids and small ruminants generally results in recumbency. Aggressive dosing of the Ketamine Stun in these patients produces excellent short-term chemical restraint and systemic analgesia. The Ketamine Stun has proven very popular with camelid and food animal practitioners that have tried the technique. In horses and adult cattle the Ketamine Stun must be dosed more conservatively to ensure the patient remains standing, which has limited the impact relative to what is achieved in camelids and small ruminants.

Dramatic improvements in patient cooperation one to two minutes following administration of a small bolus of ketamine in horses that were totally uncooperative under prior detomidine-morphine chemical restraint indicates the potential of the Ketamine Stun technique. Is it foolproof? No, but consistent enough to justify its use when conventional chemical restraint proves inadequate. The Ketamine Stun has facilitated successful completion of numerous standing procedures that would have otherwise required anesthesia. The Ketamine Stun technique has also proven very useful in providing short-term relief of colic pain (see Morphine, Ketamine Stun for Colic Pain, Transport Analgesia).

Proper balance between the alpha₂-adrenergic agonist (α₂) and ketamine is the key to successful use of the Ketamine Stun in standing patients. A high level of α₂ sedation combined
with a high peak effect of ketamine (approximately 60 seconds after IV administration) can result in a transient period of instability. Instability typically resolves in 1-3 minutes as ketamine blood levels are reduced by redistribution, but the systemic analgesia and improvement in patient cooperation produced by the ketamine bolus are also reduced.

Small doses of ketamine added to xylazine-butorphanol chemical restraint did not consistently improve patient cooperation for dental work or provide systemic analgesia against skin pricks in equine patients (4). Patient instability was mentioned as an adverse consequence of the technique. The use of xylazine instead of detomidine likely contributed to the instability observed in this study. The use of xylazine-butorphanol rather than detomidine-morphine may have reduced efficacy. I have not tried the Ketamine Stun for equine dental procedures, though it has proven very effective in camelids.

The Ketamine Stun technique is generally used in situations when more conventional chemical restraint is not effective. Achieving the proper balance between the \( \alpha_2 \) and ketamine components to obtain the desired level of improvement in these patients can be challenging. Experience improves dose selection. The titrated approach can be used to gain experience with this very useful technique.

Several different drugs combinations can be used to produce the Ketamine Stun effect. The following protocols are based on my experience using the Ketamine Stun technique in clinical patients. Practitioners are encouraged to experiment to the extent they feel comfortable. Feedback is vital to further development of the technique (abrahamsen@earthlink.net).

**Detomidine-Morphine-Ketamine**: Patient is initially sedated with detomidine (0.0055-0.011-0.022 mg/kg IV, or 2.5-5-10 mg/450 kg). Lower doses reduce the risk of instability, but larger doses may be used provided adequate time elapses prior to administering the ketamine bolus. A moderate level of sedation seems to work best when using the Ketamine Stun technique in horses. Morphine (0.1-0.15 mg/kg IV, or 45-60 mg/450 kg) can be administered immediately after the detomidine. Onset time for IV morphine is approximately 10 minutes. The ketamine bolus (0.11-0.22 mg/kg IV, or 50-100 mg/450 kg) is administered a minute or two prior to starting the procedure to gain full advantage of its relatively short (10-15 minutes) duration of action. Peak effect and effective duration are typically reduced with the lower ketamine dose. Titrated administration of ketamine (incremental doses of 0.11 mg/kg, or 50 mg/450 kg IV) reduces the risk of instability in patients with more profound sedation. Due to continual redistribution the peak effect produced by two smaller doses administered several minutes apart will not reach the level produced by a single full dose. Systemic analgesia and patient cooperation peak approximately 60 seconds after IV ketamine administration and diminishes over time. The Ketamine Stun CRI is preferred for longer procedures, but I have extended the duration of the Ketamine Stun using incremental boluses of ketamine. Smaller doses administered more frequently provide a more consistent level of patient cooperation and systemic analgesia, but full doses can be used if properly spaced. Small doses of detomidine (0.0022-0.0044 mg/kg IV, or 1-2 mg/450 kg, again smaller more frequent dosing provide more consistent level of effect) may be required when extending duration of the Ketamine Stun.

**Detomidine-Butorphanol-Ketamine**: Butorphanol (0.011-0.016 mg/kg IV, or 5-7 mg/450 kg) is substituted for morphine in the above combination.

**Acepromazine-Morphine-Ketamine**: Substituting acepromazine (0.055-0.088 mg/kg IV, or 25-40 mg/450 kg) for the detomidine produces a steadier patient. My experience with this combination is limited, but it seems to reduce stability problems associated with peak blood levels of
ketamine. Morphine dose (0.1-0.2 mg/kg IV, or 45-90 mg/450 kg) can be increased to replace analgesia provided by detomidine, if needed.

*Acepromazine-Butorphanol-Ketamine: Acepromazine (0.055-0.088 mg/kg IV, or 25-40 mg/450 kg) is substituted for detomidine and butorphanol (0.011-0.016 mg/kg IV, or 5-7 mg/450 kg) is substituted for morphine in the "stun" combination. This combination should provide the same stability benefits as the previous combination.

**ENHANCED CONTROL FOR LONGER PROCEDURES**

**Ketamine Stun CRI**

Note: Constant Rate Infusion notes provide more detailed information on delivery and control methods.

NOTE: The above discussion regarding balance between the alpha_2-adrenergic component and ketamine applies to CRI delivery of the Ketamine Stun.

The level of patient cooperation and analgesia produced by bolus administration of IV drugs decays over time. For longer standing procedures in horses I have been using a "constant" rate infusion (CRI) technique to deliver a steady state of chemical restraint and systemic analgesia. A CRI technique may seem complicated, but is much easier than it first appears and has proven extremely useful. Using a stock solution created for the prototypical 450 kg horse with adjustments in delivery rate to accommodate variations in patient size and/or alter the level of chemical restraint provided simplifies application of this technique.

A solution administration set can be used to control the CRI. Adding a drip rate monitor (DripAssist) makes setting and adjusting delivery rate easier. Drip Assist (Veterinary DripAssist Infusion Rate Monitor, 000A2773, Hallowell EMC, $225) is a simple battery powered (1 AA) device easily placed around the drip chamber of a solution administration set to continuously monitor delivery rate and volume. Display can be set to read drops/min, ml/hr, or total volume delivered (ml). It does not control delivery rate, but makes it easier to set and adjust (display responds to new setting within 3-4 drops). Unit includes an audible alarm function that warns user if delivery deviates +/- 13% of selected rate. If solution concentration allows DripAssist can be used in series with an IV flow control device (e.g. Dial-A-Flo).

An infusion pump (Heska VetIV 2.2, Heska Corp., Loveland, CO, a very compact, rechargeable unit that is very easy to use) or a flow limiting device such as the Dial-A-Flow (LifeShield Regulator IV Extension Set, Dial-A-Flo with Option-Lok, list # 11742-48, Hospira, numerous manufacturers produce similar devices) also make setting and adjusting delivery rate easier. Dial-A-Flo settings (ml/hr) OFF, 20, 30, 40, 50, 60, 70, 80, 100, 125, 165, 210, 250, and OPEN (but intermediate delivery rates, e.g. not indicated on dial, can be set to accommodate patient size) allow stock 450 kg solutions to be safely delivered to a wide range of patient sizes. Clients can be easily guided through delivery adjustments using a DripAssist, infusion pump, or IV flow control device if working without trained assistance, allowing the clinician to remain sterile.

1-bag vs. 2-bag delivery

Combined delivery (1-bag approach) is simpler, but less optimal due to differences in drug clearance. I have used 1-bag delivery extensively; though now favor the 2-bag approach.
Delivering the alpha\textsubscript{2}-adrenergic component separately makes it easier to maintain better balance with ketamine level over time.

**Detomidine CRI options for 2-bag delivery**

**NOTE:** Limiting extraneous "environmental stimulation" reduces the dose of alpha\textsubscript{2}-adrenergic agonist required to achieve a desired level of patient cooperation.

Dr. Abrahamsen’s detomidine CRI technique: detomidine loading dose (0.011-0.022 mg/kg IV, or 5-10 mg/450 kg) and detomidine CRI (0.022 mg/kg/hr, or 10 mg/450kg/hr initially with delivery adjusted to maintain proper level of sedation). Proper sedations is generally the level that "just" keeps head down on padded rest that is high enough to help maintain good front-back balance. The head coming up off the pad in response to "major" stimulation is about right. The head coming off the pad "frequently", especially with little provocation, typically means sedation is inadequate. The head "never" coming off the pad typically means sedation level is excessive.

My detomidine infusion rate was developed through experience using the Ketamine Stun CRI technique in clinical patients. My detomidine loading dose and initial delivery rate is noticeably higher than Dr. Dugdale's and Dr. Wilson's. For comparison, the detomidine delivery rate of Pentafusion is 0.0044 mg/kg/hr, or 2 mg/450kg/hr (along with 0.0022 mg/kg/hr of acepromazine) and produces no overt sedation (5).

Dr. Dugdale’s detomidine CRI technique: detomidine loading dose (0.006 mg/kg IV, or 2.7 mg/450 kg), wait 5 min., then start detomidine CRI (0.006 mg/kg/hr, or 2.7 mg/450kg/hr initially with delivery reduced by 50% when desired level of sedation is reached) (6).

Dr. Wilson’s detomidine CRI technique: detomidine loading dose (0.0075 mg/kg IV, or 3.4 mg/450 kg) and detomidine CRI (0.0006 mg/kg/hr, or 2.7 mg/450kg/hr initially, reduce CRI by 50% every 15 minutes) (7).

**Ketamine Stun CRI combinations and sample stock mixtures for a 450 kg horse**

I have used the Ketamine Stun CRI technique for a variety of standing procedures (laparoscopy, sinoscopy, enucleations, sinus flaps, flank laparotomies, etc.) with good results. The standing chemical restraint CRI I have the most experience with is detomidine-morphine-ketamine. I have tried acepromazine-morphine-ketamine a couple times with good result and detomidine-butorphanol-ketamine on several occasions with good results. I have used the Ketamine CRI technique in freestanding horses, but prefer to have the additional support provided by a set of stocks (patients often lean against the sidebars or gates/rope when more aggressive administration is required). Supplemental padding is often used to improve stability in these patients.

Ketamine possesses potent analgesic effects at sub-anesthetic doses and redistributes rapidly once the infusion is discontinued. The profound level of chemical restraint produced by the CRI resolves fairly quickly once the infusion is discontinued. Though somewhat ataxic, patients are typically walked back to their stall within minutes of completing the procedure. A residual level of sedation remains and gradually diminishes in 1-2 hours. An endless number of concentration and infusion rate combinations are possible. The following protocols are based on my experience using the Ketamine Stun CRI technique in clinical patients. Drug amounts required for one-hour of delivery to a 450 kg patient (450 kg stock solution) are provided for each of the
combinations (Table 1). Altering the volume of carrier solution (NaCl is preferred, but LRS works fine) is used to adjust base 450 kg delivery rate to better fit the control method(s) available. The 1 L (dilute) and 500 ml (middle) versions can be controlled with a solution administration set, though a drip rate monitor (DripAssist) or an infusion pump can be added without changes. The 100 ml (concentrated) version is designed for control with a Dial-A-Flow. Practitioners are encouraged to experiment to the extent they feel comfortable. Feedback is vital to further development of the technique (abrahamsen@earthlink.net).

(Table 1) Drug amounts required for 450 kg stock solutions. When added to 1 L of NaCl the base infusion rate for a 450 kg patient is 1 L/hr (~ 30 drops/10 seconds with a 10 drops/ml drip set, ~ 40 drops/10 seconds with a 15 drops/ml drip set). Reducing the NaCl volume used to 500 ml increases drug concentrations reduces the 450 kg base rate to 500 ml per hour (~ 15 drops/10 seconds with a 10 drops/ml drip set, ~ 20 drops/10 seconds with a 15 drops/ml drip set). Reducing the NaCl volume used to 100 ml further increases drug concentrations and reduces the 450 kg base rate to 100 ml per hour. This allows delivery to be controlled with a Dial-A-Flow. Directions for using the Dial-A-Flow are provided on the package. A ratio is used to adjust delivery for patient size (e.g. patient base rate for a 337 kg horse is 75% or 0.75 of the 450 kg base rate). The patient base rate determined in this manner can be further adjusted to alter the level of chemical restraint provided. For 2-bag delivery place detomidine is a separate bag of equal volume. Adjust delivery using one of the above detomidine infusion protocols.

<table>
<thead>
<tr>
<th>Technique</th>
<th>Detomidine</th>
<th>Morphine</th>
<th>Ketamine</th>
<th>Butorphanol</th>
</tr>
</thead>
<tbody>
<tr>
<td>detomidine-morphine-ketamine</td>
<td>9 mg</td>
<td>10 mg</td>
<td>250 mg</td>
<td>NA</td>
</tr>
<tr>
<td>acepromazine-morphine-ketamine</td>
<td>NA</td>
<td>10 mg</td>
<td>250 mg</td>
<td>NA</td>
</tr>
<tr>
<td>detomidine-butorphanol-ketamine</td>
<td>9 mg</td>
<td>NA</td>
<td>250 mg</td>
<td>10 mg</td>
</tr>
</tbody>
</table>

Technique Specifics

For 2-bag delivery place detomidine is a separate bag of equal volume. Adjust delivery using one of the above detomidine infusion protocols.

Detomidine-Morphine-Ketamine: The patient is sedated with detomidine (0.011-0.022 mg/kg IV, or 5-10 mg/450 kg) depending on its demeanor, with the higher dose generally providing a superior result. A loading dose of morphine (0.1-0.15 mg/kg IV, or 45-60 mg/450 kg) is then administered. Due to the higher loading dose of detomidine I do not generally administer a loading dose of ketamine when using the Ketamine Stun CRI technique. Starting the CRI early in the preparation phase allows adequate time for the ketamine level to gradually build and begin exerting its analgesic and behavioral effects. A CRI of detomidine (0.022 mg/kg/hr), morphine (0.025-0.05 mg/kg/hr), and ketamine (0.6 mg/kg/hr) is started and maintained throughout the procedure. The amount of morphine included in the CRI mixture depends on the level of noxious stimulation anticipated from the procedure. We have found the lower dose of morphine (0.025 mg/kg/hr) infusion is sufficient for many procedures. A supplemental bolus can be administered and/or the bag can be spiked with additional morphine if the level of analgesic support is insufficient.
Adjustments above (up to 1.3x) and below the "patient's base rate" of delivery are used to alter the level of chemical restraint. Prolonged use of infusion rates above the "patient base rate" increase the likelihood of instability. Response to reductions in infusion rate is fairly quick. Response to increases in infusion rate takes longer. A small top-up bolus of detomidine (0.0022-0.0044 mg/kg IV, or 1-2 mg/450 kg) to improve level of sedation and/or morphine (0.0167-0.034 mg/kg IV, or 7.5-15 mg/450 kg) to increase level of systemic analgesia can be used to shorten response time or as an alternative to increasing delivery rate. Infusion should be reduced or temporarily discontinued if weakness or instability occurs. When 2-bag administration is used adjustments are typically limited to the detomidine infusion.

Acepromazine-Morphine-Ketamine: Substituting acepromazine (0.055-0.088 mg/kg IV, or 25-40 mg/450 kg) tranquilization for the detomidine sedation in the above combination can be used in patients where postural stability of the patient is more critical. A somewhat higher loading dose of morphine (0.1-0.2 mg/kg IV, or 45-90 mg/450 kg) can be used to replace the analgesia that would be provided by detomidine. We have found lower doses of morphine (0.1-0.15 mg/kg IV, or 45-60 mg/450 kg) are sufficient for many procedures. Delivery of morphine and ketamine in the CRI mixture remains as above. Acepromazine is not included in the infusion mixture due to its long duration of effect following IV bolus administration. Supplemental acepromazine has not been required in the small number of cases where we have used this combination. If exceptionally long procedures are anticipated, acepromazine (1-2 mg/450kg/hr IV) could be added to the infusion to help stabilize its effect. Infusion should be reduced or temporarily discontinued if weakness or instability occurs.

Detomidine-Butorphanol-Ketamine: Butorphanol (0.011-0.016 mg/kg IV or 5-7 mg/450 kg) can be substituted in the standing chemical restraint CRI in situations when morphine is not available. Butorphanol (0.022 mg/kg/hr) is substituted for the morphine in the above combination. Delivery of detomidine and ketamine in the CRI mixture remains as above. Supplemental dose of butorphanol (0.005-0.007 mg/kg IV, or 2-3 mg/450 kg) is substituted for supplemental dose of morphine in the above combination. Infusion should be reduced or temporarily discontinued if weakness or instability occurs.

References

5. Abrahamsen EJ. Equine pain management with Pentafusion. Hagyard Equine Medical Institute Formulary, or Google Pentafusion, analgesia
Acepromazine, Drug Reversal, Epidural Analgesia, Telazol-based Capture Technique

Eric J. Abrahamsen, DVM, DACVA

ACEPROMAZINE

Chemical restraint is a central element of equine practice. Alpha₂-adrenergic agonists (alpha-2's) with or without an opioid are the predominant choice for producing chemical restraint in equine patients with good reason. There are situations where the alpha₁-adrenergic antagonist effects of acepromazine are particularly useful, either alone or in combination with an alpha-2 and / or opioid. There are several parameters related to chemical restraint (Table 1). The relative importance of each parameter varies with the chemical restraint application.

Table 1: Impact of Alpha-2’s and Acepromazine on Various Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Alpha-2’s</th>
<th>Acepromazine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset (expediency, facilitates titrated approach)</td>
<td>Rapid (2-5 minutes)</td>
<td>Slow (10 minutes “+”)</td>
</tr>
<tr>
<td>Duration (dose dependent)</td>
<td>Short-to-intermediate (romifidine &gt; detomidine &gt; xylazine)</td>
<td>Intermediate-to-long</td>
</tr>
<tr>
<td>Behavior Modification (dose dependent, both improved with addition of an opioid)</td>
<td>Sedation (mild to profound)</td>
<td>Tranquillization (mild to moderate, can be profound with opioid)</td>
</tr>
<tr>
<td>Predictability (consistency of desired response)</td>
<td>Better at high doses?</td>
<td>Better at high doses?</td>
</tr>
<tr>
<td>“Readability” (ability to judge level of effect)</td>
<td>Pretty good</td>
<td>More challenging, especially at low-to-intermediate doses</td>
</tr>
<tr>
<td>Arousal (dose dependent)</td>
<td>More easily aroused by non-painful stimuli at low-to-intermediate doses</td>
<td>Not as easily aroused by non-painful stimuli at low-to-intermediate doses</td>
</tr>
<tr>
<td>Patient Stability</td>
<td>Dose dependent ataxia (less with detomidine &amp; romifidine)</td>
<td>Remarkably stable</td>
</tr>
<tr>
<td>Systemic Analgesia</td>
<td>Dose dependent analgesia</td>
<td>No analgesic effect other than mental indifference</td>
</tr>
<tr>
<td>Reversible</td>
<td>Yes</td>
<td>No*</td>
</tr>
<tr>
<td>Increased urine production</td>
<td>Yes (can be an issue for pelvic scintigraphy)</td>
<td>Modest at most</td>
</tr>
<tr>
<td>Cardiovascular Effects (cardiovascular effects generally well tolerated)</td>
<td>Bradycardia, 2°AV block, hypertension followed by hypotension</td>
<td>Hypotension</td>
</tr>
<tr>
<td>Potential Problems</td>
<td>Bradycardic effects of alpha-2’s exacerbate those produced by hyperkalemia</td>
<td>Paraphimosis, reduced tolerance to significant hemorrhage</td>
</tr>
</tbody>
</table>
Repeated IV Boluses (total dose of 10 mg within a few minutes) of phenylephrine did not produce characteristic transient hypertensive response in anesthetized patients treated with 5 mg of IV acepromazine (unpublished).

Acepromazine can be used alone or combined with an alpha-2. The proportion of acepromazine used (0-100%) depends on the relative importance of the parameters. Acepromazine’s primary virtues are its longer duration (handy for mare-foal separation, shipping, stall confinement, longer procedures, etc.), excellent patient stability (even when combined with an opioid to produce a profound level of chemical restraint), and relaxation of the retractor penile muscle (examination, catheterization, cleaning).

Unlike some of my compatriots, I am not a fan of routinely administering acepromazine prior to anesthesia. Vasodilation produced by the acepromazine can make maintaining arterial pressure more difficult. I use acepromazine (0.011 mg/kg IV, or 5 mg/450kg) to predictably treat tourniquet hypertension. When alpha-2 use is contraindicated I use straight Ket-Val (hyperkalemic foals) or slow guaifenesin infusion (severely compromised colic patients).

**Paraphimosis**

In male horses acepromazine causes dose dependent relaxation of the retractor penis muscle. This effect is useful for examining or cleaning the penis and sheath, urinary catheterization, etc. The level of protrusion (or “let down”) is typically “modest” even with larger doses of acepromazine, but it is noticeably easier to fully coax the penis out of the sheath. The use of acepromazine in male horses has resulted in paraphimosis, or the inability to fully retract the penis into the sheath. Most veterinarians avoid using ace in valuable stallions for this reason. There are veterinarians that will not use acepromazine in any intact male horse, but use it in geldings. Some avoid using acepromazine in all male horses. Surveys have put the incidence at less than 1 in 10,000 (1). Theriogenologists I consulted say that estimate is more appropriate for younger intact males, with the incidence even lower in geldings. They believe risk increases with age in intact males, perhaps due to increased size or fibrosis. Older (4-5+) intact males are more likely to be a breeding stallion, which increases the liability risk.

Risk can be minimized with client and/or staff education. The relaxant effects of acepromazine diminish over time, but may be perceptible as much as four to six hours after administration of large doses. Patients should be observed periodically to make sure they are progressing properly, especially individuals exhibiting an excessive amount of relaxation or at higher risk. Patients should be able to fully retract the penis into the sheath when overt tranquilization is no longer evident. A subtle amount of “dangle” may be present at this point due to residual levels of acepromazine, but the penis should fully retract when stimulated.

Verifying that the penis is or can be fully retracted into the sheath when overt tranquilization is no longer evident is critical to minimizing the risk of paraphimosis. Excessive and/or prolonged “dependency” can lead to vascular engorgement → edema → further impairment of venous and lymphatic drainage → the increased weight produced by this vicious cycle, if not interrupted, produces tension on the Deep Perineal nerve and increases the potential for penile paralysis.

Early recognition and timely intervention (placing a pursestring suture to retain penis within the sheath) stops the progression and can help resolve any edema that has formed while allowing time for the residual effects of acepromazine to dissipate. The farther the cycle is allowed to progress, the more challenging the situation becomes to manage and in some cases has
resulted in penile amputation. Several excellent guides for managing paraphimosis are available (a couple online) (2-4).

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DRUG REVERSAL

An array of receptors classes, or more specifically their ever-growing number of subtypes, regulates most bodily functions. Each receptor class is responsible for regulating a range of activities. The majority of these receptor classes are closely related (perhaps evolving from a “primordial receptor”). Drugs that produce their effect by activating or blocking receptors are generally not the body’s natural ligands so the receptor population impacted can vary even within a drug class (e.g. banamine provides better relief of colic pain than phenylbutazone). Drugs that work by activating or blocking receptors can be very selective, targeting a specific receptor subtype, or fairly non-specific, perhaps targeting not only a range of the desired receptor class, but also a number of other receptor classes (“straying”). Acepromazine interacts with several receptor classes (D₂, α₁, H₁, & mAch). Yohimbine interacts with numerous receptor classes (α₁, α₂, much of the extensive range of 5HT receptor family, D₂, D₃)

The homeostatic processes controlled by receptors typically have a base level activity that can be increased or decreased. Overzealous reversal shifts the equilibrium beyond the normal set point, potentially causing the opposite effects of the drug being reversed (“crossover”).

Administering an overdose (relative or absolute) of reversal drugs can produce serious adverse effects up to and including patient death. A relative overdose occurs when a large dose is administered rapidly IV (or released from a regional perfusion site), reducing the volume of blood the drug is distributed in during the first circulatory pass creating a transient exaggerated effect.

Drug Reversal Principles

Reversal is intended to remove some or all of the residual effects of a previously administered drug. The level of residual effects depends on the dose, route of administration, elapsed time, and the patient’s sensitivity. The relative importance of residual effects can vary. Residual effects may be mediated by different receptor subtypes; it is hypothesized that mu₁ receptor are responsible for analgesic effects and mu₂ responsible for cardiorespiratory effects (bradycardia, hypoventilation) and dependence.

A titrated approach should be used in all non-emergency applications (dangerous animal exception discussed below). The effects of a properly titrated dose should take 3-5 minutes.
(IV) and 7-10 minutes (IM to become evident. The “traditionally recommended” (or “book”) doses are excessive for most applications in my opinion:

- **Yohimbine (0.125 mg/kg IV)**
- **Tolazoline (2 mg/kg IV)**
  - Receptor affinity is much lower
- **Atipamezole (20-60 mcg/kg IV)**
  - Regularly see 50 mcg/kg recommended

I typically administer 50% of these doses IV in emergency situations. For reversal of recumbent chemical restraint (zoo, wildlife, & food animal patients) I use them as my maximal IM dose and reduced to fit the circumstances.

The duration (dose, route of administration) of the reversal agent and drug being reversed must be considered to minimize risk of resedation or renarcotization. If drug being reversed was administered IM, most or all of reversal drug should be administered IM to extend its effects. Splitting the reversal dose (using both intravenous and intramuscular routes) can produce a quicker recovery while minimizing the risk of resedation. Response to the small intravenous component will be faster, so the primary intramuscular component should be administered first.

Allow adequate time for any co-administered drugs (e.g. ketamine, Telazol) to resolve to prevent rough recoveries.

**Alpha-2 Agonist Reversal**

**Yohimbine**
- Not approved for use in horses, but commonly used
- Somewhat selective (alpha-2 > alpha-1)
  - Also effects a myriad of other receptor types
- 2 mg/ml $60 for 20 ml bottle

**Tolazoline**
- Approved for equine use (U.S.?)
- Non-selective (both alpha-1 & alpha-2)
- 100 mg/ml $83 for 100 ml bottle
  - Some evidence to indicate it is more effective than yohimbine in ruminant patients

**Atipamezole**
- Approved for only for IM canine use
- Very selective (alpha-2 >> alpha-1)
  - Much lower “straying” risk, but “crossover” risk remains
- 5 mg/ml $162 for 10 ml bottle

Experience using atipamezole to reverse xylazine and detomidine is still limited. Atipamezole (50 mcg/kg IV) and yohimbine (60 mcg/kg IV) have been recommended for reversal of the sedative and cardiovascular effects of larger doses of detomidine. If this equivalence proves correct, the titrated dose of atipamezole should similar to that of yohimbine, 5-10 mcg/kg. Complete reversal is generally not required in most veterinary applications. Titrated administration can reduce the cost of reversal, perhaps making atipamezole a reasonable choice in the future.
Doxapram
Non-specific stimulation with doxapram has been used to transiently reduce the effects of alpha-2 sedation

Drug reversal is not used frequently in equine practice. Doses must be adjusted to fit circumstances. Experience with a particular drug is valuable. Yohimbine is the alpha-2 antagonist I have the most experience using so is the drug I am most comfortable with and currently prefer to use in equine patients.

**mu Opioid Reversal**

Naloxone (0.4 mg/ml), 10 ml multiuse vial costs $125 (or slightly more than $30/mg)

Naloxone dose (0.002-0.02 mg/kg)

In veterinary medicine the large doses of naloxone are rarely needed. Mini-reversal of opioids is used to improve the level of arousal while maintaining as much analgesia effect as possible in species susceptible to marked opioid-induced CNS depression (small animals, marine mammals, etc). Naloxone (2 mcg/kg IV) is administered every 3-5 minutes until the desired response is obtained (e.g. extubation).

Opioid reversal is rarely used in equine practice. Any adverse GI effects associated with analgesic doses can generally be handled with dose reduction or elimination. The effects of modest chemical restraint doses of morphine (45-90 mg/450kg) rarely cause problems unless the associated sedation is reversed. Large chemical restraint doses of morphine (150-300 mg/450kg) have produced colic symptoms and manic behavior, but they can generally be managed (e.g. banamine, acepromazine) until morphine level resolves. Opioid reversal in equine patients is very expensive. A full dose of naloxone (0.02 mg/kg) for an adult horse requires approximately 4 vials ($300). The mini-reversal dose of naloxone (0.002mg/kg IV) would cost approximately $30.

Co-administration of poorly absorbed oral opioid antagonists or systemic administration of opioid antagonists incapable of crossing the blood brain barrier has reduced opioid-induced constipation without adversely affecting analgesia in people (1). The approach may one day prove useful in treating equine patients with decreased GI motility.

**Opioid Agonist-Antagonist (Butorphanol) Reversal**

Naltrexone (0.05-0.1 mg/kg IM, incremental when safety allows) is often used to reverse butorphanol (as well as mu opioids) in zoo / wildlife recumbent chemical restraint.

**Benzodiazepine Reversal**

Flumazenil (0.1 mg/ml)

The effects of diazepam and midazolam in domestic species do not typically require reversal. Flumazenil (0.005 mg/kg IV) is generally used to reverse the midazolam component of recumbent chemical restraint techniques in zoo or marine park patients.
Reversal to Shorten Injectable Anesthetic Recovery

Arousal occurs abruptly when ketamine (or tiletamine) blood level reaches a critical value due to redistribution. Patients are generally not ready to stand at this point. The residual sedative effects of xylazine delivered in the induction process and any supplemental anesthetic administration are critical in preventing attempts to stand until blood levels of these intravenous anesthetic agents have decreased sufficiently to assure a coordinated effort (braking effect). Equine patients often want to roll up once arousal occurs. Patients ideally remain sternal until sedation has resolved enough so head is consistently up and patient seems mentally engaged. Gentle physical and/or verbal restraint is often effective in slowing these transitions and improving recovery quality.

Reversal prior to the sternal phase of recovery is rarely required. Tiny doses and extreme patience should be used when it is needed. Reversal is typically used when the patient has been sternal for at least 10 minutes (to ensure ketamine or tiletamine has adequately resolved) and significant sedation is still evident (patient’s chin resting on ground or stall floor absent mild stimulation) or progress has “stalled”. Titrated reversal is important to avoid an abrupt transition that may reduce recovery quality. Yohimbine (0.0044 mg/kg IV, or 2 mg/450kg) in administered at 5-minute intervals until head is consistently up and patient seems mentally engaged. Often a dose of two is all that is required to move things along nicely.

Reversal of patients that pose a physical danger to personnel must be handled somewhat differently. Approach will depend on how easily subsequent doses can be administered (hand injection or pole syringe vs. darting) once patient is aroused sufficiently to pose a threat. Initial reversal doses can be more aggressive in short-term recumbent chemical restraint. Initial dose becomes more challenging when interval has been extended with injectable or inhalation anesthesia, especially if subsequent does must be darted. Zoo and wildlife veterinarians tend to dose aggressively when reversing recumbent chemical restraint regardless of the interval (recipe approach).

Reversal of Standing Chemical Restraint

Alpha-2s (with or without an opioid) are commonly used to improve patient cooperation for a wide range of standing procedures. Their effects are generally well tolerated in normal healthy patients and are typically allowed to resolve on their own.

If an opioid is used in conjunction with the alpha-2 and the post-procedure pain not expected to require opioid analgesia some degree of sedation (or tranquilization) may be needed to counter the CNS excitatory effects of the opioid (typically manifested as stall walking) until it resolves. Complete reversal is rarely required. A titrated approach reduces the risk of “crossover” and may reduce cost (a modest reduction is sedation level is often adequate). Initial dose of yohimbine depends on the residual level of sedation evident. A larger initial dose of yohimbine (0.022 mg/kg, or 10 mg/450kg for moderate sedation, 0.044 mg/kg, or 20 mg/450kg for profound sedation) can be used to speed up the process. Subsequent yohimbine doses (0.0044-0.022 mg/kg, or 2-10 mg/450 kg) are based on sedation level 5 minutes after the previous bolus.

Yohimbine (60 mcg/kg, or 27 mg/450kg) costs $40, Yohimbine (0.011 mg/kg, or 4 mg/450kg) increments would only cost $6 each.
Emergency Reversal of Severe Bradyarrhythmias

The combined effects of hyperkalemia (e.g. foal with uroperitoneum) and an alpha-2 can result is severe bradyarrhythmias. The threat posed by the arrhythmia dictates treatment urgency. Life-threatening bradyarrhythmias require immediate and aggressive reversal. Yohimbine (0.044 mg/kg IV, or 2 mg/45kg) plus atropine (0.04 mg/kg IV) successfully converted complete AV block with occasional escape beats to normal sinus rhythm in a patent urachus foal during contrast during a contrast radiography study. Had I the time to calculate, I likely would have used 3 mg, or 0.67 mg/kg, which is slightly more than 50% of the “book” dose. And I wouldn’t argue with someone who used the full 0.125 mg/kg “book dose” of yohimbine in this type of crisis.

Treating Accidental Carotid Administration of an Alpha-2

Accidental carotid administration of an alpha-2 sends an undiluted bolus of drugs directly to the brain. I’ve never witnessed the accidental administration, so can’t describe the immediate patient response, but a couple of minutes later they are typically laterally recumbent with a look I would describe as “unresponsive panic”. Overly aggressive reversal may result in an abrupt, less coordinated attempt to stand and an unsteady patient.

You are treating a massive relative, but typically not an absolute overdose. Titrated reversal should be used, but initial dose is increased relative some of other applications described. Yohimbine (0.022 mg/kg IV, or 10 mg/450kg) is followed by successively smaller doses (e.g. 4mg, 2mg, 2mg…) every 3-5 minutes until the patient is sternal with head up and engaged. Letting patient decide when to stand up minimizes risk of a less coordinated attempt, especially if footing is less than ideal. Patients often roll up sternal after the initial dose (or perhaps two), but still look very sedate.

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EPIDURAL ANALGESIA

Equine epidurals can be divided into two sub-techniques. Caudal epidural anesthesia is a regional anesthetic technique often used to reduce straining during obstetrical manipulations as well as facilitate painful procedures involving the perineal region and pelvic viscera. Caudal epidural analgesia is a regional analgesic technique used to provide pain relief in the above-mentioned areas and with larger volumes the hind limbs. The focus of this talk is caudal epidural analgesia. I’ve included some information of caudal epidural anesthesia for sake of completeness.

Minimizing impact on motor nerve function to the hind limbs is critical to avoid weakness, ataxia, and, in worse case scenarios, unintended recumbency. Na channel inhibition has been demonstrated in several of the drug classes used in caudal epidural anesthesia and analgesia, but of the drugs commonly used in equine patients only local anesthetics, the alpha-2 agonist xylazine, and perhaps very high doses of ketamine are of clinical concern. Local anesthetic agents are often used in caudal epidural anesthesia techniques to improve predictability. Many practitioners now combine xylazine with the local anesthetic to increase effective duration. Total
volume administered must be limited to prevent these drugs (technically a high enough concentration of these drugs) from reaching the motor innervation to the hind limbs.

The first moveable joint caudal to the sacrum, often the first intercoccygeal joint (Co1-Co2) is the most common site for epidural administration in equine patients. The epidural space is a potential space normally maintained under a slight vacuum. Injecting into the epidural space requires barely more than the pressure to overcome resistance in the syringe. Drug solution should be administered slowly – beyond the small amount of space created by the loss of vacuum you are displacing venous blood and perhaps some CSF. Solution expands out from injection point based on a path of least resistance basis – overzealous administration rate can produce unintended cranial spread and excessive pressure on nerve roots. Epidural space is reduced in pregnant and overweight patients.

Drugs administered in the epidural space must diffuse through the meninges to reach target receptors sites in the spinal cord and nerve roots. Lipid solubility is the most important factor in determining how quickly a drug penetrates (the higher the lipid solubility the faster the onset). The epidural space is highly vascular, so drugs with higher lipid solubility are also absorbed systemically faster, reducing the duration of their localized effects and increasing likelihood of producing systemic effects. Uptake by epidural fat initially reduces concentrations in epidural space, but then serves as a modest repository.

Drug concentration also impacts diffusion rate. Dilution (other drugs, saline) increases onset time of a drug. Volume used in equine caudal epidural anesthesia and analgesia techniques is relatively fixed. Increasing dose reduces onset time, increases peak effect, and extends duration, though it also increases the risk of systemic effects for more lipid soluble drugs and risk of hindlimb motor impairment for drugs capable of inhibiting Na channel function.

A combination of drugs acting through different mechanisms should provide a synergistic effect, but even an additive effect allows doses of each component to be reduced, lowering the risk of adverse “side” effects. Combinations can also be used to speed onset, increase impact, and/or extend duration.

Caudal Epidural Anesthesia

Caudal epidural anesthesia is used to produce surgical level analgesia (tail, anus, rectum, perineum, vulva, vagina, urethra, & bladder) and reduce straining (vaginal reflex) during obstetrical manipulations.

Local Anesthetics

Local anesthetics are Na channel blockers capable of inhibiting both sensory and motor nerve transmission. This limits the volume that can be safely administered for solutions containing local anesthetics. Subsequent attempts to locate the epidural space can be more challenging, so a titrated approach is not practical. Dose administered (volume x concentration) determine the success and duration of blockade, so one must be somewhat bold when longer duration is needed using only an intermediate acting local anesthetic (lidocaine, mepivacaine).

There is tremendous variation in onset and duration values provided for the various local anesthetic agents in both the human and veterinary literature. Lidocaine duration ranged from a low of 45 minutes to as much as 2.5 hours. The use of bicarbonate (to speed onset) and/or epinephrine (to prolong duration) complicates comparisons. The level of stimulation affects
efficacy evaluation. Dental studies have demonstrated a difference in perception of onset depending on the level of stimulation. Probing with a sharp instrument overestimated the level of blockade when compared to testing with extreme cold (Endo-Ice). Both were far longer than published values.

Lidocaine and mepivacaine are very similar. Lidocaine onset is perhaps slightly faster while mepivacaine duration is somewhat longer. Mepivacaine is popular in equine practice for diagnostic nerve blocks because it produces less tissue response (swelling) and has excellent spreading properties.

Lidocaine (5-10 minute onset, 45-90 minute duration)
Mepivacaine (10-15 minute onset, 90-180 minute duration)

Bupivacaine and ropivacaine are less soluble, so onset is slower but duration is longer. They are classified as long acting local anesthetics. Bupivacaine and ropivacaine are not commonly used in equine epidurals. The extended duration is rarely, if ever needed for clinical procedures and safer options for long-term epidural analgesia are available that do not include the risk of protracted hindlimb paresis.

Adjuvants can be used to modify the onset and duration of local anesthetics. Buffering (NaHCO₃) can be used to improve lipid solubility and onset time. Vasoconstrictors (epinephrine) can be used to reduce systemic uptake, increasing duration.

Xylazine

Xylazine blocks sensory transmission via alpha-2 receptor activation in the spinal cord. Xylazine is less soluble than detomidine, so onset is somewhat slower (30 minutes) and duration somewhat longer (2-3 hours). Systemic effects (sedation, cardiorespiratory) are negligible at lower doses (0.17 mg/kg) and modest at higher doses (0.22-0.25 mg/kg).

Xylazine (0.17 mg/kg, or 75 mg/450kg) diluted to 10 ml with 0.9% saline provided approximately 2.5 hours of needle probing caudal epidural anesthesia without apparent systemic effects, but somewhat higher doses may be required for actual clinical procedures (1). Higher doses (0.22-0.25 mg/kg, or 100-112.5 mg/450kg) produced tail flaccidity and mild hind limb ataxia. Several members of the alpha-2 agonist family (as well as some opioids) have been shown to possess local anesthetic-like activity. Ataxia is more likely than recumbency and the augmented volume may have contributed to the weakness, which is why xylazine is typically combined with other drugs for caudal epidural anesthesia and is not used in large volume caudal epidural analgesia.

Clonidine increases the duration of co-administered drugs to a degree comparable with epinephrine (which, depending on the source, doubles or even triples local anesthetic duration). Xylazine’s peripheral vasoconstrictive effects likely produce similar beneficial increases in duration.

Caudal Epidural Anesthesia Techniques

I consulted clinicians routinely involved in RV fistula repair for their preferred cocktails. Two basic approaches are being used:
450-500kg patients

8 ml seems to by far the most popular volume for caudal epidural anesthesia in 450-500kg patients

- 8 ml of 2% lidocaine
  - Hindlimb paresis is reportedly fairly rare
  - Onset 5-10 minutes, duration 45-90 minutes

- 3-4 ml 2% mepivacaine + xylazine (0.17 mg/kg, or 75 mg/450kg), QS to 8 ml with saline
  - Using 5+ ml of mepivacaine reportedly increases likelihood of hindlimb weakness and ataxia (perhaps due to its excellent spreading properties?)
  - Onset 30 minutes, duration 2-3 hours

Xylazine doses that are capable of producing ataxia are only 30-50% higher

Caudal Epidural Analgesia

Caudal epidural analgesia can be used to treat perineal region or pelvic viscera pain, but is probably used more frequently to provide relief of hindlimb pain. Regional techniques “target” the affected area, generally reducing 24-hour dosage or allowing it to be more effective.

An epidural catheter assures proper placement of drugs, makes repeated administration easier for clinician and patient, and may facilitate a more cranial spread. Drugs need to reach the level of the 4th lumbar vertebrae to impact the upper hindlimb, which is why volume used is important when using caudal epidural analgesia. Excellent guides are available for placement of caudal epidural catheters in equine patients (all available online) (2-4).

The epidural safety of drugs containing preservatives is a hotly debated topic in human anesthesia. Epidural administration of morphine containing preservatives has been used in large animal patients for decades without problems. Dilution in equine applications (10+ ml of preservative free saline in typically mixture) reduces concentration markedly.

Morphine

Note: 15 mg/ml multi-dose vials of morphine have been discontinued due to new FDA regulations. See Meet the New Morphine notes for important information regarding the safe use of preservative free morphine.

Morphine blocks sensory transmission via $\mu$ opioid receptor activation in the spinal cord. Morphine is the primary analgesic drug used in equine caudal epidural analgesia. Morphine is not very lipid soluble, so onset is slow (visible effects 1-2 hours), but epidural duration is long. Effective duration depends on severity of pain; 6-8 hours with severe pain, 12-18 hours with moderate pain, 24-48 hours with mild pain (mild pain will likely be handled with other approaches in majority of cases). Morphine is relatively cheap (half the cost of butorphanol on a clinical dose basis) and hopefully after listening to my earlier talks regularly stocked in most practices.
**Butorphanol**

Butorphanol has generally not been shown to provide analgesia when administered epidurally, though a couple papers have demonstrated some benefit.

**Detomidine**

Detomidine blocks sensory transmission via alpha-2 receptor activation in the spinal cord. Detomidine is very lipid soluble, providing a quick onset (15-20 minutes). Detomidine is not used alone so duration is unknown, but likely “shorter”. Dose-dependant systemic effects of epidural detomidine (sedation, cardiorespiratory) are similar in intensity and duration to SQ administration.

Detomidine is used to speed onset of relief, but it also increases both the peak level and duration of analgesia. Clonidine increases the duration of co-administered drugs to a degree comparable with epinephrine (which, depending on the source, doubles or even triples local anesthetic duration). Detomidine’s peripheral vasoconstrictive effects likely produce similar beneficial increases in duration.

**Ketamine**

Ketamine is a non-competitive voltage dependent N-methyl-D-aspartate (NMDA) receptor antagonist. NMDA receptors are ion channels activated by glutamate and involved in the transmission of painful stimuli. NMDA receptor activity increases with pain duration (and possibly intensity) producing the phenomenon known as central sensitization or windup. Ketamine is thought to reduce mu opioid tolerance, improving the efficacy of morphine.

Ketamine is very lipid soluble, so onset is quick (15-20 minutes). Ketamine is not generally used alone, so duration information is limited. Ketamine (0.5, 1, & 2 mg/kg) used alone produced 30-75 minutes of caudal epidural anesthesia in horses (5). Analgesic effects extend beyond anesthetic effects. Epidural ketamine (2 mg/kg) produced 2 hours of force plate measured analgesia in dogs with chemically induced synovitis (6). Human post-operative studies indicate epidural ketamine’s analgesic effects are significantly longer (epidural morphine 12.8 +/- 6.2 hr, epidural morphine + 1 mg/kg ketamine 19 +/- 9.8 hr) (7). This seems well beyond the expected duration of ketamine’s analgesia effects – perhaps ketamine’s ability to enhance opioid receptor function requires much lower levels or somehow persists beyond its presence? Epidural ketamine is also generally used in conjunction with detomidine in equine patients, whose vasoconstriction further extends (possibly doubling or tripling) its effective duration. Finally, ketamine is generally used when pain levels are more severe, so epidural dosing intervals are shorter. Even if ketamine does not last the entire interval, the additional analgesia it provides can be very important.

Epidural ketamine produces a brief dose-dependent period of sedation (mild with 0.5 mg/kg) that peaks 15-30 minutes following administration (another reason to keep detomidine does modest).

Ketamine has been shown to inhibit Na channels. Based on results from human and equine studies the concentration seems to be beyond clinically used levels. Magnesium sulfate is also a non-competitive voltage dependent NMDA antagonist. An epidural combination of ketamine (2.5 mg/kg) and magnesium sulfate (2.0-2.5 mg/kg) produced an additive duration of analgesia, but also produced severe ataxia and hindlimb weakness in sheep (8). The Ketamine Stun
technique can produce transient instability when blood levels of ketamine are too high for the level of sedation present (see Enhanced Equine Chemical Restraint). Both epidural ketamine and detomidine produce dose-dependent sedation, so the potential exists if sufficiently large doses of each were administered, though unlikely in the recommended range. Morphine (0.2 mg/kg), ketamine (0.5 mg/kg) and detomidine (0.011 mg/kg) is the most potent equine epidural combination I have used to date and it has proven both safe and effective. I wouldn’t hesitate to increase ketamine and detomidine doses if additional analgesia is needed in the future, but would do so gradually until I had more experience.

Caudal Epidural Analgesia Technique

Drug combinations are generally used to speed onset, increase analgesia effect, and prolong duration. Differing mechanisms of action offer potential of synergistic effect. Epidural analgesia is typically reserved for more significant levels of pain, so dosing intervals are generally BID, TID, or sometimes QID. Milder levels of pain can be managed with SID administration, though generally successfully managed with more conventional analgesic modalities.

The level of analgesia should be evaluated at least twice during each dosing interval (more frequently during early stages of analgesic therapy). The trough level just prior to administering the subsequent dose is used to evaluate patient progress and/or presence of any problems such as cast sores. The peak level at onset of subsequent dose (varies with the drug combination) is used to evaluate efficacy of the analgesia. Both are important to proper analgesic management, especially of fracture and laminitis patients.

Caudal epidural analgesia can provide profound relief of hindlimb pain (4/5 or 5/5 to 1/5) (3). A slow onset and/or long duration make titration more difficult. When managing hindlimb laminitis or fracture repair patients a more conservative initial approach is prudent to minimize the risk of overuse.

Volume

Perineal region and pelvic viscera (0.18-0.022 ml/kg, or 8-10 ml/450kg)
Hind limb analgesia (0.044 ml/kg, or 20 ml/450kg)

Morphine + Detomidine

Morphine (primary analgesic drug)
Dose (0.1-0.2 mg/kg) depends on pain level and duration desired
Larger doses
Faster onset, higher peak analgesia, & longer duration
Balance with other drugs used is important
Systemic effects not common

Onset is slow (visible effects can take 1-2 hours)

Effective duration depends on severity of pain
6-8 hours with severe pain
12-18 hours with moderate pain
24-48 hours with mild pain
Detomidine
Dose (0.011-0.03 mg/kg, or 5-13 mg/450kg) depends on pain level and duration desired
Larger doses
Faster onset, higher peak analgesia, & longer duration
Balance with other drugs used is important
Increased systemic effects (sedation, cardiorespiratory)
Epidural similar to SQ administration

Author’s preferred starting dose 0.011 mg/kg, or 5 mg/450kg
Mild sedation
Ketamine can be added to increase level of analgesia
Separate mechanism adds potential for synergism
Additional detomidine is, at best, additive
Goodrich study dose (0.03 mg/kg, or 13 mg/450kg)
Moderate sedation

Onset is fairly quick (15-20 minutes)
Speeds onset of relief

Vasoconstrictive effect
Prolongs its duration as well as that of co-administered drugs

When dosing interval is properly managed the faster onset provided by detomidine becomes less important, but its contribution to the peak level and duration of analgesia must be considered (and subsequently evaluated) when contemplating its reduction or discontinuation.

Ketamine
Dose (0.5-2.0 mg/kg) depends on pain level and duration desired
Larger doses
Faster onset, higher peak analgesia, & longer duration
Balance with other drugs used is important
Increased systemic effects (sedation, stability with extreme doses)

Onset is fairly quick (15-20 minutes)
Added when additional analgesia is needed
Even more important as pain duration increases
Reduce morphine tolerance

Clinical Approach

Morphine (0.1-0.2 mg/kg depending on pain + detomidine (0.011 mg/kg) is administered. The initial dose of detomidine can be increased to provide more effective early relief in patients with higher levels of pain.

Adjustments in dosing interval and drug combination are based on periodic patient evaluation. Authors preferred progression when additional analgesic support is required (deficit determines how aggressive the adjustment are):
1) Shorten interval (at least temporarily) to speed onset of improved relief
2) Increase morphine dose if using less than 0.2 mg/kg
3) Add ketamine (0.5 mg/kg)
Potential synergism
Improve morphine’s efficacy

4) Increase ketamine and detomidine doses gradually
Increasing ketamine dose intensifies and prolongs its effects. Increasing detomidine intensifies and prolongs its effects, but perhaps more importantly extends ketamine duration. I guess my preference would be to increase ketamine first if analgesic support needs to be improved throughout the interval and increase detomidine first if analgesic support only needs to be improved later in the interval. Increased level of sedation (detomidine & ketamine) can be an issue with shorter dosing intervals.

I have used caudal epidural analgesia primarily in fracture patients. Morphine (0.2 mg/kg), ketamine (0.5 mg/kg) and detomidine (0.011 mg/kg) is the most potent equine epidural combination I have used to date and it has proven both safe and effective.

Drugs doses are reduced (some possibly eliminated) as patient’s condition improves. Increasing the dosing interval reduces the mean analgesic effect, but peak effect remains unchanged and the risk of overuse increases. Analgesic support gradually declines over time following bolus administration and extending the dosing interval drastically reduces the level of analgesia provided in the later stages of the interval. In practices that do not have 24 hour staffing extending dosing intervals during the overnight hours is often used to reduce the impact of providing care to hospitalized patients. The analgesic requirements of the patient should determine when extended overnight dosing intervals are appropriate.

References

6) Hamilton SM. Analgesic effects of epidural ketamine in dogs with a chemically induced synovitis. Master Thesis 2003, Virginia Polytechnic Institute and State University

TELAZOL-BASED CAPTURE TECHNIQUE

Large animal veterinarians are confronted from time to time with situations calling for recumbent chemical restraint. It might be an intractable bull in a setting with limited physical restraint options or perhaps a loose horse. The drug cocktail must be administered by dart, pole syringe,
or in certain situations, rapid hand injection. These administration methods are volume sensitive, requiring a potent drug cocktail.

Reconstituting Telazol (500 mg), a 50:50 combination of tiletamine (250 mg) and zolazepam (250 mg), with ketamine and an alpha2-adrenergic agonist rather than diluent creates a potent cocktail capable of producing recumbency in a 450 kg patient with just 4 ml of volume.

These capture techniques deliver a large dose of alpha2-adrenergic agonist. Dosage may be reduced in patients that may not tolerate the adverse effects of these drugs, though impact may be affected.

RUMINANTS

I have used this technique to knock down smaller beef bulls in stalls, intractable rodeo bull prospects and large exotics in stock trailers (1).

Telazol–Ketamine–Xylazine (TKX-Ru)

Telazol-ketamine-xylazine (TKX-Ru) is a modification of porcine TKX (TKX-P). TKX-Ru can be used to produce recumbent chemical restraint/light anesthesia in ruminants and exotic hoofstock. TKX-Ru is created by reconstituting a 500-mg vial of Telazol with 250 mg of ketamine (2.5 ml) and 100 mg of large animal xylazine (1 ml). Because of the space occupied by the Telazol powder the final volume is 4 mL.

The dosing protocol for TKX-Ru is still evolving. Current recommendations are 1.25 to 1.5 ml/110 to 115 kg for smaller ruminant patients and 1 ml/110 to115 kg for larger ruminant patients. Volume required for large patients is generally deliverable by pole syringe or rapid hand injection. Limitations imposed by dart volume may require a split delivery approach in large patients.

Patient should become recumbent and compliant approximately 5 (ideal) to 10 minutes following intramuscular administration. Onset significantly less than 5 minutes is indicative of an excessive dose or accidental intravenous administration. If onset has not occurred by 20 minutes additional TKX-Ru (one quarter to one half of the original dose, depending on the urgency of the situation and health of the patient) can be administered.

The degree and duration of chemical restraint and analgesia varies markedly from patient to patient. Intravenous administration of Double Drip or Ruminant Triple Drip can be used to enhance or extend the systemic analgesia and patient cooperation produced by TKX-Ru.

Patients are typically awake and sternal by 40 to 60 minutes post–TKX-Ru administration if supplemental anesthetics are not used. Because of the level of residual sedation patients typically remain sternal for an additional 20 to 40 minutes (depending on demeanor and level of environmental stimulation) before attempting to stand. Recovery is generally smooth. Once the patient is awake and sternal, xylazine can be reversed to speed the recovery process, although recovery quality may be reduced somewhat. Letting xylazine resolve on its own generally makes transporting unruly patients smoother.

TKX-Ru is somewhat expensive. Leftover TKX-Ru can be labeled and frozen to preserve its function for up to 6 months.
Reversible Telazol-Ketamine-Detomidine Technique (TZKD)

A recent modification of the TKX-Ru capture technique (TZKD) was evaluated (2).

TZKD is created by reconstituting a 500-mg vial of Telazol with 500 mg of ketamine (5 ml) and 40 mg of detomidine (4 ml). Because of the space occupied by the Telazol powder the final volume is 9.5 ml.

TZKD was dosed at 1 ml/100kg, which resulted in recumbency (50% sternal, 50% lateral) in approximately 6 minutes in 95% of reported subjects* while others required more than one dose.

* Patients that did not become recumbent by 10 minutes post-administration were EXCLUDED FROM RESULTS making efficacy impossible to evaluate!

Local anesthetic was used where appropriate. Additional dose(s) (50% of the initial dose) of TZKD were administered if procedure outlasted anesthesia produced by initial dose. All patients were reversed with atipamezole (0.02-0.06 mg/kg IV) without excitement.

Comparing the component drugs amounts delivered by TZKD and TKX-Ru identifies key differences (Table 1).

**Table 1: TZKD vs. TKX-Ru component delivery comparison.**

<table>
<thead>
<tr>
<th></th>
<th>TZKD @ 1ml/100kg</th>
<th>TKX-Ru @ 1ml/115kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Telazol</td>
<td>0.53 mg/kg IM</td>
<td>1.1 mg/kg IM</td>
</tr>
<tr>
<td>Ketamine</td>
<td>0.53 mg/kg IM</td>
<td>0.54 mg/kg IM</td>
</tr>
<tr>
<td>Detomidine</td>
<td>0.04 mg/kg IM</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(18mg/450kg IM)</td>
<td></td>
</tr>
<tr>
<td>Xylazine</td>
<td>0.22 mg/kg IM</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(100 mg/450kg IM)</td>
<td></td>
</tr>
</tbody>
</table>

TZKD and TKX-Ru seem to produce a similar state of potent chemical restraint (local blockade or supplemental anesthetic required for more painful procedures) and in some cases injectable anesthesia. TZKD delivers half the Telazol dose of TKX-Ru. Both techniques utilize the sedative and systemic analgesic properties of an alpha2-adrenergic agonist, but TZKD depends to a much greater extent on its large dose of detomidine. The reliance on detomidine enhances reversibility, but its adverse cardiorespiratory effects of detomidine must be considered when determining which chemical restraint technique is appropriate for a given situation. Detomidine has been shown to produce greater respiratory depression than xylazine in horses.

Arousal from ketamine or tiletamine occurs when brain drug level reaches a critical value. Arousal is abrupt, but patients are not ready to stand at that point. The residual sedative effects of the co-administered alpha2-adrenergic agonist produce a "braking effect", delaying attempts to stand and improving recovery quality. Premature reversal can reduce recovery quality. Patient’s state of arousal at reversal was not provided.

Intravenous reversal of intramuscularly administered alpha2-adrenergic agonists can result in resedation, rumen atony, and rumen acidosis. Resedation was not evaluated in this study.
EQUIIDS

I have used the equine version to knock down a couple zebras and a Przewalski's horse (3). They were in stock trailers so darting them was fairly easy. The Ohio State University equine field service used this technique to capture a horse that had been running loose in a Columbus suburb for several hours.

Telazol-Ketamine-Detomidine (TKD-Eq)

2-stage technique

Due to their more mercurial nature a 2-stage approach is preferred in equids when feasible. Limitations imposed by dart volume (typically 3-4 ml) may also necessitate a 2-stage approach in large patients.

The patient is sedated with detomidine (0.022-0.044 mg/kg IM depending on initial demeanor and situation, with the upper end of the dose range often providing a better result) prior to administering Telazol (500 mg) that has been reconstituted with 250 mg (2.5 ml) of ketamine. Try to minimize "patient stress" as much as situation allows while waiting for sedation to occur.

The Telazol-ketamine combination is administered at 1 ml/125-150 kg IM. Depending on the dose administered and the degree of prior sedation achieved it generally takes anywhere from 5-15 minutes for recumbency to occur. If recumbency has not occurred by 20 minutes administering an additional partial dose will be required. The level of anesthesia and analgesia produced varies markedly. Patients may tolerate mild to moderately noxious stimulation immediately following knockdown, but will likely require supplemental anesthetic for surgical procedures. Equine Triple Drip can be used to enhance and extend the effects of TKD-Eq. Equine Triple Drip was administered in all three cases I personally managed, so I cannot comment on the quality of recovery from pure TKD-Eq. The field service clinician that used TKX-Eq to capture a loose horse did not report any adverse results.

Single administration technique

In situations requiring a single administration approach dart volume (typically 3-4 ml) may limit the dose of detomidine that can be delivered. Due to the space occupied by the Telazol powder the final volume when reconstituted with 250 mg (2.5 ml) of ketamine is 3 ml. Depending on patient size the volume available for detomidine may be limited to less than an ideal dose.

When a pole syringe or rapid hand injection is used calculate the Telazol-ketamine dose (1 ml/125-150 kg IM) and detomidine dose (0.022-0.044 mg/kg IM depending on initial demeanor and situation, with the upper end of the dose range often providing a better result).

When a dart delivery is used calculate the Telazol-ketamine dose (1 ml/125-150 kg IM) and use remaining volume for detomidine dose (up to 0.044 mg/kg IM). Impact may be reduced when dart volume limits detomidine dose to less than 0.022 mg/kg. A 4 ml dart provides adequate volume for a 450 kg patient.

TKX-Eq is somewhat expensive. Leftover TKX-Eq can be labeled and frozen to preserve its function for up to 6 months.
References

Constant Rate Infusions

Eric J. Abrahamsen, DVM, DACVA

Drug levels and their clinical effects decay over time following bolus administration. The fluctuation between peak and trough levels produced by periodic bolus administration can be advantageous or detrimental depending on the drug and situation. A "constant" rate infusion (CRI) provides a consistent level of drug effect, which can also be advantageous or detrimental depending on the drug and situation.

Intravenous (IV) anesthesia, anesthetic supplements, extended duration chemical restraint, time-dependent antibiotics, and analgesic support for patients with severe pain are examples where the consistent effect produced by CRI is desirable. A CRI can also be used when slow delivery of a drug is important. Rapid administration of bicarbonate can result in acute hyperosmolality, a decrease in ionized calcium, a left shift in the oxygen-hemoglobin dissociation curve, and paradoxical central nervous system acidosis.

CRI techniques are not appropriate for all situations. A patient’s pain level may need to be periodically evaluated to determine if a change in management is required or to help titrate analgesic support to prevent overuse of a recent surgical repair. Concentration-dependent antibiotics rely on peak level achieved rather than sustained presence for their effectiveness. Bolus administration must also be used when trough levels of a drug such as aminoglycoside antibiotics must cycle below a threshold value to reduce the risk of toxic damage to tissues or organs. Clinicians must weight the potential benefits and risks to determine when a CRI technique is appropriate.

Almost all veterinary practices employ CRI techniques of one sort or another. The administration of IV fluids is universal in veterinary practice. Dopamine and dobutamine infusions are commonly used to improve cardiac output and arterial blood pressure during anesthesia. Bicarbonate is frequently administered to combat metabolic acidosis. The precision and attention required of the infusion control methods available varies, as does the level of accuracy required by CRI applications. In normal healthy patients IV fluid delivery during anesthesia is less critical, but cardiac patients often require careful management to minimize the risk of decompensation.

The use and importance of drug infusions in modern veterinary practice is expanding rapidly. Delivery control and solution creation are important fundamental elements common to all CRI techniques.

Methods Used to Control Infusion Delivery Rate

The goal of drug administration is to achieve a desired level of clinical benefit while minimizing or avoiding adverse side effects. For CRI techniques this requires consistent and medically accurate delivery. There is a degree of error introduced in almost every facet of a CRI technique. Subtle variation in the manufacturing process, especially for high volume disposable items introduces a degree of unforeseen error. Rounding during mixture formulation and the challenges of drawing up small drug volumes or gauging drip rates introduce another layer of error. Solution viscosity and changes in patient position also impact delivery rate for some control methods. Using the appropriate delivery control method for the CRI application is crucial to obtaining the desired clinical effect and minimizing risk of adverse effects. Understanding the
advantages and limitations of the various delivery control options available is crucial to reaching this goal.

**Infusion and Syringe Pumps:** Infusion pumps and syringe pumps provide the most precise control of delivery rate. Pumps are the preferred methods for controlling CRI delivery, especially when drugs with potent clinical or adverse effects are administered by IV infusion. Pumps are more expensive than the other control methods discussed, but the long service life of quality unit reduces the prorated cost of ownership.

Infusion and syringe pumps share many common features. Both types are equipped with audible and visual alarms that sound when delivery is impeded, typically due to an occluded line or catheter. Pumps also signal when infusion is finished or battery is getting low. Direct observation is required to detect delivery problems with all of the other methods discussed. Delivery rate is externally controlled (no contact with drug solution). Both types of pumps produce positive pressure in the fluid line so delivery is not altered by normal changes in venous backpressure or patient position. Pumps provide a digital readout of delivery rate, volume infused, and volume to be infused, making adjustments and documentation easier. Most pumps include settings to compensate for more commonly used high viscosity solutions such as total parenteral nutrition (TPN).

Infusion pumps such as the Heska VetIV 2.2 (Heska Corporation, Loveland, CO) regulate flow delivered via a solution administration. Infusion pumps provide accurate delivery (typically +/− 5% of setting) over a wide range of flow rates. In Macro Mode the Heska VetIV 2.2 can deliver 0.1-999 ml/hr (1 ml/hr increments up to 100 ml/hr and then 10 ml/hr increments up to 999 ml/hr). In Micro Mode it can deliver 0.1-99.9 ml/hr (0.1 ml increments up to 10 ml/hr and then 1 ml/hr increments up to 99.9 ml/hr). Infusion pumps are ideally suited for applications requiring accurate delivery of larger volumes. The extensive priming (void) volume of the solution administration set increases wastage in small volume delivery applications. Infusion pumps make tailoring delivery of stock solutions to patient size much easier and more precise. Regulating IV fluid delivery is more precise and requires less personnel attention when an infusion pump is used.

Syringe pumps such as the Medfusion 2010i (refurbished units available from multiple sources) control delivery from a syringe mounted in the device. Syringe pumps accept a variety of syringe sizes and typically offer a wider array of delivery modes, requiring a few more steps in the activation process when compared to an infusion pump such as the Heska VetIV 2.2. Syringe pumps provide accurate delivery (typically +/− 3% of setting) over a wide range of flow rates. Of the 4 delivery modes offered by the Medfusion 2020i, Continuous (ml/min or ml/hr) is the most commonly used in veterinary applications. The Medfusion 2010i can deliver 0.1-378 ml/hr of flow in increments as small as 0.01 ml/hr. It can also be programmed to deliver a bolus amount at the outset of the infusion. Syringe pumps are ideally suited for precise delivery of small volumes. Due to the frequent refilling required, they are less useful for high volume delivery. Syringe pumps are also useful for slow delivery of IV drugs. A microbore extension set (0.2-0.3 ml priming volume, various lengths available) can be used to reduce wastage when using a syringe pump to deliver a small volume of more expensive drugs.

The precision provided by infusion and syringe pumps allows advanced CRI techniques to be used safely to improve patient care and outcome. A midazolam-ketamine-fentanyl infusion can be used to dramatically reduce inhalant level required in compromised patients that might not tolerate the adverse cardiovascular effects of isoflurane or sevoflurane. Titrated infusion of the beta-blocker esmolol or sodium nitroprusside can be used to lower blood pressure to reduce...
bleeding during delicate cardiovascular or neurological surgical procedures. Pentafusion, a 5 drug analgesic infusion, has proven very effective for alleviating severe pain in equine patients.

Infusion and syringe pumps are invaluable tools in modern veterinary practice. It is important to select well-designed and manufactured units from companies with excellent support service. The long service life of quality units reduces the prorated cost of ownership. Units that accept disposables from an array of manufacturers are more desirable.

**IV Flow Control Devices:** In-line IV flow control devices such as the Dial-A-Flo (LifeShield Regulator IV Extension Set, Dial-A-Flo with Option-Lok, list # 11742-48, Hospira, numerous manufacturers produce similar devices) have been used for decades to control delivery rate of infusions. Setting and adjusting infusion rates with an IV flow control device is easier and faster than using the roller or screw clamp of a solution administration set, though studies have demonstrated similar levels of accuracy. Using an IV flow control device adds approximately $7 to cost.

The Dial-A-Flo has "settings" (ml/hr) of OFF, 20, 30, 40, 50, 60, 70, 80, 100, 125, 165, 210, 250, and OPEN, but intermediate delivery rates (e.g. not indicated on dial) can be set. Delivery using an IV flow control device is +/− 10% of setting under ideal circumstance. The distance between the level of fluid in the IV solution container and the patient catheter can be thought of as the pressure head responsible for flow. IV flow control devices are designed to work within a certain specified height differential. IV flow control devices provide steady delivery outside of this range, but rate will vary from setting. Viscosity of the solution and alterations in venous backpressure also impact the accuracy of IV flow control devices. Delivery rate should be verified initially and periodically evaluated when using an IV flow control device. Periodic inspection is also required to detect occlusive problems in delivery system. IV flow control devices have direct contact with the infusion solution. Like the solution administration sets they replace or supplement, they are disposable items intended for short-term use.

IV flow control devices should generally only be used for drug infusions that are closely monitored.

**Solution Administration Sets:** Using the roller or screw clamp of a solution administration set is the most cost effective and readily available method of controlling the delivery rate of an IV infusion. Setting the initial drip rate and any subsequent adjustments generally take more time when compared to pumps and IV flow control devices. Slow drip rates take even more time to set or adjust.

Drip rate (drops/min) is converted to ml/min and ml/hr by applying the conversion factor for the solution administration set used (typically 10, 15, or 60 drops/ml). Counting the number of drops per 10 seconds provides a more accurate assessment of the drip rate. Delivery using a solution administration set is +/− 10% of intended rate under ideal circumstance. Infusion rate set using a solution administration set can change over time, requiring greater vigilance. Changes in pressure head or venous backpressure alter delivery rate. "Cold creep", the outward pressure exerted by the tubing as it resists deformation, can push roller style clamps open slightly increasing flow over time. "Crimping" of tubing under pressure from adjustment clamp does not resolve immediately. As it slowly resolves flow increases beyond the level intended when the clamp was loosened. Delivery rate must be periodically evaluated when using a solution administration set. Periodic inspection is also required to detect occlusive problems in delivery system.
Solution administration sets should only be used to control drug infusions that are closely monitored.

**Drip Rate Monitors:** Optical detection of drip rate and the drop/ml conversion factor for the solution administration are used to determine delivery rate. A drip rate monitor makes monitoring and adjusting CRI delivery easy.

DripAssist (Veterinary DripAssist Infusion Rate Monitor, 000A2773, Hallowell EMC, $225) is a simple battery powered (1 AA) device easily placed around the drip chamber of a solution administration set to continuously monitor delivery rate and volume. Display can be set to read drops/min, ml/hr, or total volume delivered (ml). It does not control delivery rate, but makes it easier to set and adjust (display responds to new setting within 3-4 drops). Unit includes an audible alarm function that warns user if delivery deviates +/- 13% of selected rate. If solution concentration allows DripAssist can be used in series with an IV flow control device (e.g. Dial-A-Flo).

Adding a simple remote monitoring system (audio or audio/visual) to the DripAssist – Dial-A-Flo combination provides a pretty respectable cost effective CRI delivery setup.

**Elastomeric Infusion Systems:** Elastomeric infusion systems are basically a highly engineered elastic bag that when filled properly and used with the associated tubing deliver a “fixed” rate of infusion. They were developed for the human home care market. Elastomeric infusion systems such as the Homepump Eclipse or C-Series (Halyard Health, Inc, Alpharetta, GA) and MILA 7100 (MILA International, Inc, Erlanger, KY) are useful infusion delivery options in certain situations. Elastomeric infusion systems are attached directly to the patient making them ideally suited for applications where the patient is not confined (paddock or pasture settings) or return of the unit could be inconvenient or uncertain (transport analgesia for colic patients sent to a referral hospital). They can also be used to provide targeted delivery of antibiotic to confined spaces such as joints or tendon sheaths. Elastomeric infusion systems are not suitable for small patients due to their weight and bulk.

Elastomeric infusion systems are more expensive than IV flow control devices. The limited number of fixed delivery rates and volumes requires a more customized approach to solution creation, though options are increasing in this rapidly expanding market. Elastomeric infusion systems are sold by the case (24 or 48 units depending on model). Prices have gone up noticeably over the past couple years (Homepump Eclipse E40100 price per unit was $15.50 in 2009, $30 in 2015) and seem to vary markedly by wholesaler. I have been able to purchase individual units at reasonable prices (Homepump Eclipse E40100, $30) from human pharmacies with infusion departments. Best to call around and make arrangements prior to needing the device.

Understanding how flow is controlled and factors that affect delivery rate is important to their safe and effective use. Under ideal (specified) conditions delivery accuracy is ± 15%. Factors affecting delivery rate include: temperature of the flow control segment, temperature of the reservoir, viscosity of the solution, fill volume, pressure head, and duration between filling and use. Delivery is not perfectly linear. Extrapolating from a graph in the Homepump Eclipse Instructions for Use: initial delivery rate is approximately 15% higher than specified, gradually decreasing to specified delivery by 30-35 minutes. A 2.5-hour period of specified delivery follows. At the 3-hour point delivery gradually increases 5% over 20-25 minutes, followed by a steady decline to the 4-hour “endpoint”, at which time delivery is 20% below specified.
It is more difficult to visually estimate remaining volume (delivery progress) when using an elastomeric infusion system. Catheter should be checked periodically to verify patency. A stopcock placed between the tubing of the elastomeric infusion system and an extension set attached to the patient catheter facilitates patency checks and IV access for drug administration. Elastomeric infusion systems cannot be resterlized, but with proper aseptic technique can be refilled a limited number of times to extend usage in a single patient. Pressure head (distance from reservoir to catheter) creates approximately ± 0.5% change per inch depending on which is dependent. Attention to reservoir placement typically minimizes impact. Drug compatibility is critically important when using elastomeric infusion systems. The microscopic bores used to regulate flow are easily plugged with drug precipitate or contaminants.

External pressure on reservoir can increase the delivery rate of elastomeric infusion pumps. Impact depends on the degree and duration, as well as the drug(s) being infused. Reservoir can simply be located to minimize risk of external pressure. If patient is likely to favor one side when laterally recumbent locating the reservoir on the expected non-dependent side of the neck should reduce risk. Reservoir can also be located ventral to neck (making sure to minimize pressure head) if recumbency preference in unknown. A protective sheath fashioned from a partially flattened segment of PVC pipe can be used to shield reservoir from external pressure.

**Homepump Eclipse** The Homepump Eclipse comes in a variety of delivery rate / volume combinations (Table 1). The Homepump Eclipse uses the diameter and length of the attached tubing distal to the filter to control delivery rate. The temperature specified for the reservoir and flow limiting segment is 68°F (20°C). DO NOT place flow-limiting segment next to skin OR under bandage. Isolate reservoir from skin contact if possible. Flow increases 1.4% for every 1°F (0.6°C) rise in temperature (and decreases s 1.4% for every 1°F (0.6°C) drop in temperature).

<table>
<thead>
<tr>
<th>Size (ml)</th>
<th>50</th>
<th>100</th>
<th>100</th>
<th>100</th>
<th>250</th>
<th>250</th>
<th>400</th>
<th>400</th>
<th>500</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flow (ml/hr)</td>
<td>50</td>
<td>50</td>
<td>100</td>
<td>200</td>
<td>175</td>
<td>250</td>
<td>100</td>
<td>200</td>
<td>250</td>
</tr>
<tr>
<td>Duration (hours)</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>0.5</td>
<td>1.43</td>
<td>1</td>
<td>4</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

The Homepump Eclipse is designed for delayed use. Using the Homepump Eclipse within the first 4 hours after filling increases delivery. Testing with the 100ml / 100ml/hr model revealed initial flow rate was + 50%, but the average flow rate for the entire hour infusion period was + 20%, indicating the peak effect is somewhat short-lived. For emergency applications fill the Homepump Eclipse as early as feasible. The impact of delivery increases resulting from immediate use depends on the drugs infused. Saline is the specified carrier solution. Using D5W as a carrier solution decreases delivery rate by 10%, a simple adjustment that may be adequate for drugs with minimal risk. Reducing dosages by 20% is probably adequate for the majority of drugs with limited risk. Elastomeric infusion systems are not suitable choices for infusion of high-risk drugs under ideal circumstances.

Elastomeric infusion systems with flow-limiters designed to operate at a set ambient temperature are more vulnerable to differences in environmental temperature. The impact of temperature differences (88°F increases delivery rate 28%) will depend on the drugs infused. Using D5W as the carrier solution decreases delivery rate by 10%, a simple adjustment may be adequate for drugs with minimal risk in warmer temperatures. Dosages can be adjusted by an amount equal to the calculated percentage of flow change for a given temperature for drugs with
limited risk. Using an insulated cover will slow temperature change of reservoir. Temperature correction tables can be created for common solutions to simplify adjustments. Compensating for very cold temperatures is more challenging. Placing the flow-limiting segment in contact with the skin should theoretically increase flow by 28% (20°F x 1.4%). Adjust solution for the increased flow rate and place the elastomeric reservoir in an insulated cover to slow cooling that may alter its performance.

Homepump Eclipse devices have specified minimum and maximum fill volumes. For the Homepump Eclipse E401000 they are 200 and 500 ml (respectively). Delivery time varies depending on initial fill volume of reservoir (Table 2).

<table>
<thead>
<tr>
<th>Fill Volume (ml)</th>
<th>200</th>
<th>250</th>
<th>300</th>
<th>350</th>
<th>400</th>
<th>450</th>
<th>500</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration (hours)</td>
<td>1.58</td>
<td>2.0</td>
<td>2.58</td>
<td>3.0</td>
<td>4.0</td>
<td>4.75</td>
<td>5.25</td>
</tr>
<tr>
<td>Average Flow (ml/hr)</td>
<td>127</td>
<td>125</td>
<td>116</td>
<td>117</td>
<td>100</td>
<td>95</td>
<td>95</td>
</tr>
</tbody>
</table>

Homepump C-Series Homepump C-Series uses a glass capillary tube at patient end of attached tubing to regulate flow. The flow limiters located in the tubing are temperature sensitive. They are designed to operate at 88°F (31°C), so the distal portion of the tubing containing the flow limiter should be in contact with the skin to ensure accurate delivery. Specified temperature for the reservoir is 68°F (20°C). Isolate reservoir from skin contact if possible. Flow increases 1.4% for every 1°F (0.6°C) rise in temperature (and decreases ≤ 1.4% for every 1°F (0.6°C) drop in temperature). The Homepump C-Series is intended for immediate use. Delays greater than 8 hours decrease delivery by 10%. Saline is the specified carrier solution. Use of D5W decreases flow by 10%. Refer to manufacturers Instructions for Use for minimum / maximum fill volumes and delivery times for various initial volumes.

MILA 7100 The MILA 7100 (100ml volume, $38 in 2015) uses a variety of short, flow-limiting extension sets attached at the distal end of the tubing to control delivery rate. The MILA 7100 comes standard with two delivery rate options (0.8 & 2.5 ml/hr), but other options (5, 20, &40 ml/hr) are available at additional cost ($10–11.55 each). The flow limiters located in the tubing are temperature sensitive. They are designed to operate at skin temperature, so the distal portion of the tubing containing the flow limiter should be in contact with the skin to ensure accurate delivery.

The MILA 7100 is commonly used in equine hospitals to deliver antibiotic infusion to septic joints and tendon sheath. It possesses a delivery rate / volume combination that accommodates some equine analgesic CRI combinations. The package states it is not intended for IV access, but I have used the MILA 7100 on several occasions when it was the only option available to provide continuous pain relief in equine patients with severe pain.

Creating Infusion Mixtures

Step 1 – stock or custom solution: Drug infusions can be either stock or custom solutions. Stock solutions are created for the prototypical patient, with adjustment made in base delivery
rate using ratios to accommodate variations in patient size. Adjustments in delivery can also be made to alter the level of clinical effect. Customs solutions are created for a specific patient, with adjustments in base delivery rate made to alter clinical effect.

**Step 2 – select base infusion rate:** An inverse relationship exists between the infusion rate and drug concentration in the mixture. The lower the base rate, the higher the drug concentration required to maintain desired level of delivery. An endless number of delivery rate and drug concentration combinations are possible. Selecting a base delivery rate makes creating stock or custom infusions fairly straightforward.

The base delivery rate should be modest compared to patients overall IV fluid requirements to provide flexibility. The method used to control infusion delivery impacts the selection of a base rate. Infusion pumps provide accurate delivery over a wide range of flow rates and, therefore, a great deal of flexibility. The Heska VetIV 2.2 can deliver 0.1-999 ml/hr (1 ml/hr increments up to 100 ml/hr and then 10 ml/hr increments up to 999 ml/hr) in Macro mode and 0.1-99.9 ml/hr (0.1 ml increments up to 10 ml/hr and then 1 ml/hr increments up to 99.9 ml/hr) in Micro mode. Syringe pumps provide accurate delivery over a smaller range of flow rates. The range of IV flow control devices are also limited. When possible, selecting a base rate in the finer middle range of "settings" provides greater latitude for adjustments, should they be required. When a solution administration set is used to control infusion delivery, drip rate is the critical factor. If the drip rate is too fast, it is difficult for the user to accurately quantify delivery rate. If the target drip rate is too slow, setting and adjusting delivery is more difficult and time consuming. A drip rate of 1 drop per second is probably the easiest to set quickly using a solution administration set. The ideal target drip rate varies with the likelihood and direction of anticipated changes in delivery rate. A drip rate of 1 drop per second makes increases in rate easier to set. A drip rate of 2-3 drops per second makes decreases in rate easier to set. A pediatric (mini-drip, 60 drops/ml) solution administration set may be required to achieve a proper drip rate when delivery volume must be limited. "Fixed" delivery methods such as elastomeric infusion systems and spring-powered syringe devices severely restrict base delivery rate choices. Delivery rate and volume options are limited, and more importantly, the available combinations of these two parameters must fit the application.

**Step 3 – determine drug concentrations for solution:** Once the base delivery rate is selected, the drug concentration required in the infusion mixture is determined by first solving for patient body weight (µg/kg/min x kg = µg/min). The amount of drug required per unit time must then be converted to match delivery volume per unit time used by the control method selected (typically ml/hr for infusion pumps, syringe pumps, IV flow control devices, and elastomeric infusion systems). If original dose is in µg/kg/min converting to mg at this point makes drawing up drug amounts later easier. The drug amount per unit time and the volume delivered per unit time are used to determine the drug concentration required in the infusion solution (mg/hr x hr/ml = mg/ml). The process is repeated for any additional drugs when a combination is to be delivered.

**Step 4 – select infusion volume or duration:** Once drug concentration has been determined the volume of solution mixed must be selected. Anticipated usage, delivery method limitations, and cost are the primary factors involved in this choice. For analgesic infusions a volume sufficient for approximately 12 hours of delivery is a reasonable compromise between more frequent mixing and wastage should the patients condition require reformulation. For commonly used stock solutions it can simply be the crystalloid bag size appropriate for typical usage.
Drugs can be delivered with or without a carrier solution. NaCl is the crystalloid carrier solution of choice, though many drugs are compatible with lactated ringers solution. 2% lidocaine is used as the carrier solution in some analgesic infusions. When lidocaine is used as a carrier solution its safe and effective dose determines the base rate for the infusion and drug concentration is adjusted accordingly. Lidocaine is typically delivered in these applications at 50 µg/kg/min. (or 3,000 µg/kg/hr = 3 mg/kg/hr = 0.15 ml/kg/hr).

For drugs delivered in a carrier solution select the appropriate final volume for the solution mixture. Determine the amount of drug required to achieve the desired concentration for that volume of solution. Compare the drug volume to the final volume selected. When drug volume is small relative to the final solution volume selected the level of dilution produced by using a carrier solution volume equal to the final solution volume selected will be negligible. The acceptable level of dilution varies with the CRI technique and circumstances. Adjusting carrier solution volume to account for the drug volume to be added eliminates one source of error in delivery.

For drugs delivered without a carrier solution select the appropriate duration of infusion. The amount of drug per unit time and the duration selected are then used to calculate the volume of drug required. Final volume is total drug volume.

**Loading Bolus and Loading Infusion Rates**

Dose rate and clearance rate are the sole determinants of plasma concentration for drugs administered by continuous infusion. It takes approximately 4 elimination half-lives for a drug administered at the maintenance infusion rate to achieve steady state level. An IV bolus (loading dose) or a faster initial rate of infusion (loading infusion) can be used to speed onset when appropriate. When the risk of adverse side effects precludes the use of a loading bolus, a loading infusion can sometimes be used to speed onset. An observable parameter to assess progress is required to safely use a loading infusion.

**Creating a Mixture for an Elastomeric Infusion Systems**

Creating a mixture to be delivered using an "elastomeric infusion system" is actually quite simple. Delivery rate determines the drug concentrations required in the carrier solution. Drug concentrations are used to determine the amount of each drug required for a pre-selected volume of carrier solution. The Homepump Eclipse E401000 delivers 100 ml/hr with a nominal capacity of 400 ml (package insert provides information on minimum and maximum fill volumes, which are 200 ml and 500 ml respectively for this unit, and their impact on delivery rate, which varies slightly). When filled with 400 ml this unit provides a 4-hour duration of delivery. In this example you would add a 4 hr amount of each drug to the device and then q.s. to 400 ml with carrier solution (which again can be a properly adjusted lidocaine solution if desired). Attach to IV catheter.
Meet the New Morphine

Eric J. Abrahamsen, DVM, DACVA

The morphine veterinarians have been using for decades (15mg/ml, 20ml multi-dose vial, Baxter Healthcare / West-Ward Pharmaceuticals, Abbott Labs) has been unavailable due to unspecified “manufacturing delays”. What seemed to be one of an ever-growing number of inconvenient drug shortages is actually more permanent. According to industry pharmacists it was taken off the market because the FDA has recently begun requiring older “grandfathered” drugs to be submitted for approval. Multi-dose vials (containing preservatives) are common in veterinary practice, but not in today’s single use world of human medicine, so would likely require a serious commitment by a veterinary oriented manufacturer to return.

Fortunately, there are several injectable morphine options currently available in the US with a concentration high enough to serve as a reasonable alternative for large animal use:

- Hospira (which is now part of Pfizer): 25 mg/ml, 10 ml single-dose vial
  $7.15 per vial from Henry Schein, which amazingly is a penny per mg cheaper than the discontinued morphine)

- West-Ward Pharmaceuticals: 8, & 10 mg/ml, 1 ml single-dose vials,
  25 vials per box, but you use multiple vials per treatment in larger patents

All are preservative free, which will require some adjustments to ensure safe use.

Labeling

The discontinued morphine was labeled for SQ, IM, or slow IV use. Not for epidural or intrathecal use (thought commonly used in for epidurals in large animal patients without issue).

The morphine from Hospira and West-Ward is labeled for IV use only. Hospira package insert includes suggestions for dilute infusion, though states an IV bolus can be administered for faster onset. According to industry pharmacists the IV only labeling is strictly because that is the predominant route in human medicine so supporting data was more prevalent (easier FDA approval). As before, all veterinary use is off label regardless of administration route.

Preservative Free

The morphine from Hospira and West-Ward is preservative free, so even with sterile technique and proper storage open vials will have a limited safe shelf life. In human hospitals, where preventing nosocomial infections is paramount, single-use / single-dose protocols are strictly defined. US Pharmacopeia (USP) 797 states that single-dose / single-use vials opened in less than ISO Class 5 air quality be used within one hour, with any remaining contents discarded. Single-dose / single-use vials opened in ISO Class 5 air quality can be used up to six hours. This clean room standard is well beyond conditions found in the nicest veterinary hospitals and certainly large animal housing facilities. Microbial growth is exponential; studies indicate microbial count increases dramatically in the 12-24 hour interval after contamination. Hospital pharmacists I consulted suggested 24 hours as a reasonable maximum with sterile technique and proper storage. At a minimum that should include:
1) Select as clean an environment as possible to aspirate drug dose
2) Mask (can be simple dust mask) or at least refrain from talking over top of vial
3) Sterile gloves, or at least freshly washed & disinfected hands (AvaGard, etc)
4) Wipe vial septum “vigorously” (in one direction) with disinfectant agent (70% isopropyl alcohol, etc.) using a low-lint wipe. Allow septum to dry prior to penetrating with needle
5) Nothing should be injected into the vials
6) Label vial with discard date & time
7) Store drugs in a clean dust-free location
8) Periodically culture vial contents prior to disposal to assess compliance

Ongoing treatment of large patients typically consumes a 10 ml vial of 25 mg/ml morphine in less than 24 hours. For single dose applications using multiple 1 ml vials of less concentrated morphine may be more cost effective. No growth was observed when a trial vial was cultured after being open for 5 days (4 separate withdrawals).

**Acidic pH**

Preservative free morphine is acidic (pH of Hospira 25 mg/ml is 3.5, range 2.5-6.5; West-Ward 8 & 10 mg/ml is listed as 2.5-5.5). Perhaps because of the IV only labeling, the 3.5 really caught our attention. The pH range of the discontinued morphine is 2.5-6.5; IM and epidural administration did not cause problems so pH concern may be unwarranted.

A range of syringe dilutions was evaluated using Hospira 25 mg/ml morphine using a digital pH meter:

- 2 ml (50 mg) morphine + 2 ml saline → 4.6
- 2 ml (50 mg) morphine + 4 ml saline → 4.8
- 2 ml (50 mg) morphine + 6 ml saline → 5.0
- 2 ml (50 mg) morphine + 10 ml saline → 5.9
- 4 ml (50 mg) morphine + 20 ml saline → 6.3 (was supposed to be 16 ml saline)

Many practitioners use 2% lidocaine (pH 6.0 without epinephrine) for caudal epidural anesthesia. Saline dilutions (1:4-5) of the Hospira morphine produce a similar pH. Lidocaine with epinephrine is more acidic (pH 4). Modest dilutions (pH 4.6) have been used epidurally in people. Further testing of epidural combinations (ketamine pH is 3.5-5.5) is needed to determine if pH adjustment is warranted.

Volume must be considered when using dilution to improve pH to minimize discomfort and tissue reaction associated with IM administration. For the Hospira morphine the volume required was more than desired, so a syringe mixture of 2 ml (50 mg) morphine and 1 ml 8.4% Na bicarbonate was tested and produced a near physiologic pH of 7.54. Additional testing is needed to determine if a similar ratio is required for larger volumes of the less concentrated morphine solutions. We are currently buffering the morphine for IM administration. Whether that is necessary or not only time will tell.

**Dosing**

Dose remains 0.1-0.2 mg/kg for most applications. Even ml volumes make record keeping easier. In larger patients the rounding required generally isn’t clinically significant. In adult
horses (450kg) even ml aliquots of the 15 mg/ml morphine resulted in doses of 45, 60, 75, & 90 mg). Even ml aliquots of the preservative free morphine variants will produce similar, though in many cases slightly different doses:

Hospira 25 mg/ml

45 mg → 50 mg (11% increase)
60 mg → 75 mg (25% increase)
60 mg → 62.5 mg (4% increase) 2.5 ml
75 mg → 75 mg (0% increase)
90 mg → 100 mg (11% increase)

West-Ward 8 mg/ml

(11% decrease) 40 mg ← 45 mg → 48 mg (7% increase)
(7% decrease) 56 mg ← 60 mg → 64 mg (7% increase)
(4% decrease) 72 mg ← 75 mg → 80 mg (7% increase)
(2% decrease) 88 mg ← 90 mg → 96 mg (8% increase)

West-Ward 10 mg/ml

(11% decrease) 40 mg ← 45 mg → 50 mg (11% increase)
60 mg → 60 mg (0% increase)
(7% decrease) 70 mg ← 75 mg → 80 mg (7% increase)
90 mg → 90 mg (0% increase)

These modest shifts will be clinically insignificant in most applications, especially when weight estimates are involved.
PRE-PURCHASE IMAGING
Myra Barrett DVM, MS, DACVR

Introduction:
Imaging is often a key component of pre-purchase examinations. However, the value of the imaging is closely tied to the rest of the exam. There are four main components of pre-purchase imaging.

1) Which images to acquire and studies to perform
2) Proper image acquisition
3) Interpretation
4) Clinical correlation

Imaging Study Selection:
Which imaging studies to perform depends on multiple factors. This includes the patient’s history and physical exam findings, the intended use of the horse, price and client preferences. Generally, the more expensive the horse, the more imaging that is performed. Radiography is most standardly performed, although ultrasound, MRI and bone scans can all be part of the pre-purchase examination. The primary focus will be on radiography, while briefly touching on other imaging.

Radiographic selection will depend somewhat on the clinical exam and intended use. If there is any area that presents as a possible problem during the physical exam that region should be examined. Areas that have higher likelihood of injury in specific discipline are also included. For example, in many Western performance horses, front feet, tarsi and stifles are regularly included in the study. For race horses, barrel racing horses and eventing horses, the carpi are often included in the study as well. Imaging of the axial skeleton has become more common recently and cervical spine and back radiographs are often included, particularly in dressage and jumping horse disciplines.

Radiographic technique:
In order to obtain the best possible radiographs, the patient should be properly prepared for radiography. This includes brushing the limbs to remove any dirt and debris and cleaning and packing the feet. If possible foot radiographs are best obtained without shoes, although it is rare that shoes can be removed in the pre-purchase examination setting. If shoes are not removed, it is valuable to obtain oblique views of the foot to evaluate the palmar processes as the metal shoe otherwise obscures them. When possible, sedation is recommended to minimize motion artifact and retake examination. It is important that the best possible positioning and technique be utilized to minimize the likelihood of missing an abnormality. Proper labeling is essential and the radiographs should be labeled with the potential buyer’s name as the buyer is the owner of the radiographic images. Laterality markers should be placed on the x-ray plate so that medial and lateral can be identified as well as to ensure that the limb is being imaged correctly.

Radiographic interpretation:
Although the client are often eager for instant feedback, it is easy to overlook an abnormality when in a brightly light area, using a low resolution screen or being rushed in interpretation. Therefore it is best to evaluate the images fully at a later time in the proper environment including a dimly lit room and high quality screen or viewbox. Many clients are open to and willing to pay for radiologist interpretation of prepurchase radiographs. By offering this to your client you provide your self with additional peace of mind and it is a value added service for your client.
Clinical correlation:
The particularly challenging element of screening radiographs is that they are being used for prediction of future problems that may arise from an abnormality. As many radiographic abnormalities can be of variable clinical significance this can be difficult to project. The patient's history, physical exam and future and current level of use are essential components to interpreting these findings. For a patient who is currently working at the same or higher level than will be expected by the prospective buyer and is maintaining this work with no lameness or minimal upkeep, many radiographic lesions may be insignificant, even those that could otherwise be concerning.

On the other hand, for a patient who is not yet in work or will be asked to move to a higher level, the future prediction of the significance of abnormal findings is more challenging. Although there has been research examining the association of screening radiographs in young Thoroughbred race horses and cutting horses, there is minimal data in any discipline for long term association of radiographic abnormalities and clinical outcome.

Other Imaging Modalities for Pre-purchase Studies:
Ultrasound evaluation has increased in prominence in pre-purchase imaging. This is most indicated in cases where there is a palpable thickening or pain on palpation or previous injury. Screening ultrasound in the absence of these findings is performed but is very challenging to interpret as ultrasound abnormalities in the absence of lameness or other physical abnormalities can be incidental.

Bone scans are also sometimes employed for pre-purchase situations. The value of bone scan is for full body screening, particularly for evaluation of the axial skeleton which is otherwise difficult to image. Nuclear scintigraphy is a highly sensitive modality to assess for bone turnover. However, it is not a specific modality and there are many areas of uptake that may be associated with normal adaptive remodeling or incidental variance. Therefore the findings of the bone scan, just as with radiography and ultrasound, must be correlated with the patient's clinical exam.

While less commonly performed, pre-purchase MRI is being done, primarily using the standing magnet systems so as the horses not have to undergo general anesthesia. While these MRIs can be quite valuable for assessing abnormalities in the foot that may go otherwise undiagnosed, it is important that a trained and unbiased observer read them. While there are lesions that may be found in the foot that would likely lead to future risk of injury such as tendon lesions or navicular damage, there often can be incidental findings that may be of minimal clinical significance that should not be over-interpreted. It is important to keep in mind that the low field, standing MRIs offer limited evaluation of articular cartilage and therefore there are other possible abnormalities that may occur within the foot that may not be detected.

In conclusion, it is important to keep pre-purchase imaging as part of the entire picture. Imaging cannot be evaluated in isolation. Obtained the best possible quality studies is essential for the most accurate evaluation. Not all abnormalities are associated with or will lead to lameness and it is possible to have a completely normal imaging study and for the patient to still develop lameness. In the end, the significance of the radiographic or other imaging abnormalities must be taken into account with the patient's physical exam, any history of lameness and the client's tolerance for risk or patient maintenance.
MULTIMODALITY IMAGING OF THE EQUINE DISTAL LIMB
Myra F. Barrett DVM, MS, DACVR

Introduction:
Lameness referable to injury in the distal limb is common and the foot is the most frequently imaged aspect of the equine limb.

Radiography is the typical first line of imaging for the distal limb and provides good bone detail, evaluation of joints and indirect information regarding soft tissue injuries. While ultrasound is typically considered to be most useful for evaluating soft tissue injuries, it is also quite helpful for evaluating surface abnormalities of bone, including peri-articular margins and non-displaced fractures. Advanced imaging allows for evaluation of the limb in multiple imaging planes without superimposition of structures, thereby giving the best overall visualization of the limb. Computed tomography (CT) and magnetic resonance imaging (MRI) are often employed to further evaluate the distal limb when traditional imaging does not provide a complete diagnostic answer. Nuclear scintigraphy also can be useful for lameness localization.

Imaging Site Selection:
Imaging of the distal limb is indicated in horses that have lameness localized to the foot via regional or intra-synovial analgesia or by marked palpable abnormalities. The more specific the diagnostic blocking, the more targeted the imaging can be. This is especially important with MRI as long scan times limit the number of areas that can be evaluated.

Although a palmar digital (PD) nerve block is often considered specific to the foot, abnormalities within the proximal phalanx or even fetlock joint will sometimes resolve or improve with a PD block, in which case the imaging area must be extended. Similarly, while the abaxial nerve block is commonly associated with lameness isolated to the pastern/fetlock region, horses that respond to this block can have lameness originating from the foot to the mid cannon region. The anatomic range of imaging for horses that respond to abaxial nerve block will depend on the physical exam as well as the response to other diagnostic analgesia. For example if a horse blocks out partially to a PD nerve block and the rest of the way to the abaxial nerve block, the exam is more likely to be focused in the foot and pastern region whereas if a horse blocks partially to the abaxial nerve block and the rest of the way to low 4-point, it is sensible to begin the study in the fetlock area.

Palpable abnormalities may also help direct the first area of imaging. For example if there is digital sheath effusion or thickening of the distal interphalangeal joint capsule, the exam starting point may be led by these findings. However, it is important to recognize the range of locations of abnormalities that can be associated with improvement of diagnostic analgesia of this area and not limit the studies to a single region.

Radiography:
For a horse that blocks to a palmar digital nerve block, appropriate imaging would include radiographs of the foot, pastern and, in some cases, fetlock region. Some horses with laminitis, solar pain and collateral ligament injuries of the distal interphalangeal joint are examples of type of cases that may not improve or may only partially improve with the PD nerve block but resolve with an abaxial nerve block, yet the primary abnormality is in the foot. Additionally, some horses with severe navicular disease or deep digital flexor tendon injuries may also not improve significantly with a PD nerve block. Therefore a full radiographic study of the foot should include lateral, DP, dorsal 60° palmar view of the navicular bone and solar margins of the distal phalanx and skyline view of the navicular bone for the foot. Lateral and DP views of the fetlock as well
as oblique radiographs of the fetlock joint are recommended. If the pastern can be adequately
included in the foot or fetlock series this can be assessed in these images. In some cases due
to patient conformation, the pastern should be imaged separately to be tangential to the joint.
Oblique radiographs of the pastern are helpful for identification of subchondral bone defects.
These often reside in the palmar aspect of the proximal interphalangeal joint and are frequently
best assessed on the oblique images.

Commonly diagnosed abnormalities include malalignment of the hoof-pastern axis,
osteoarthritis (OA) of the distal (DIP) and proximal interphalangeal (PIP) joints, bone change of
the distal phalanx such as pedal osteitis and navicular bone changes. OA of the PIP joint is
very common and milder forms may be incidental. When more advanced, subchondral
sclerosis, lysis and joint space narrowing are evident changes that are more likely to be
clinically significant.

OA of the DIP joint is more likely to be clinically significant. Periarticular proliferation due to
osteofy formation and joint capsule enthesopathy are the common hallmarks of DIP OA. It is
important to evaluate the periarticular margins on all views, because in certain cases palmar
periarticular osteofy formation will be more prominent than dorsal remodeling. Joint capsule
enthesopathy is manifested as irregular bone production on the dorsal cortex of the middle
phalanx. It is important to recognize that the DIP joint responds differently to the OA process
than the PIP joint and there can be significant damage to the articular cartilage of the DIP joint
without evidence of subchondral bone sclerosis or lysis.

Changes of the navicular bone can range from equivocal, such as enlarged synovial
invaginations, to highly significant, such as flexor cortical erosive lesions. Changes of the
navicular bone also can indirectly suggest concurrent soft tissue damage to the deep digital
flexor tendon and navicular bursa. Lucencies on the distal phalanx at the insertion sites of the
deep digital flexor tendon and impar ligament are also suggestive of concurrent soft tissue
damage.

**Ultrasonography:**
Indicated ultrasound evaluation for a patient that blocks out to a PD nerve block includes the
pastern, including the palmar foot region between the heel bulb and distal interphalangeal joint.
In some cases, imaging through the frog (transcuneal approach) can be indicated. The
practicability of this is somewhat dependent on the climate; in wet climates this region can be
imaged with only minimal foot preparation whereas dry climates may require prepping and
soaking the foot for up to 48 hours. For ultrasound evaluation of lameness that resolves to an
abaxial nerve block the entire pastern region should be evaluated as well as the soft tissues on
the palmar aspect of the fetlock joint and collateral ligaments of the distal interphalangeal joint. If
the patient is markedly lame or only partially improves of the PD nerve block, evaluation of the
soft tissue structures between the heel bulbs is also recommended because as mentioned
previously in some cases severe deep digital flexor tendon injuries do not improve to a PD
nerve block.

Digital sheath effusion is a common indication for ultrasound of the distal limb. Tenosynovitis
typically occurs due to underlying pathologic processes within the digital sheath, such as a
flexor tendon injury. Tenosynovitis may be purely effusive, or can be accompanied by soft
tissue proliferation, sheath wall thickening and adhesion formation. Proliferative tissue typically
is an indicator of chronicity. Severe synovial proliferation and effusion is generally seen in
cases of septic tenosynovitis.
Tears of the deep digital flexor tendon (DDFT) are common and can occur anywhere but are most common in the pastern and foot. Primary deep flexor tendon tears in the pastern usually affect the dorsal margin and result in asymmetric lobe enlargement. Damage to the DDFT in the pastern region most commonly occurs just distal to the fetlock joint. There is often accompanying digital flexor sheath effusion. DDFT injuries can also be found as core lesions in the pastern region, which in many cases are proximal extensions of further DDFT lesions in the foot. DDFT injuries in the foot are a common cause of lameness and can be manifested as dorsal margin fraying, core lesions and sagittal splits. These changes can be accompanied with navicular bursitis and/or flexor cortical damage of the navicular bone. Ultrasound evaluation of tears that extend to the insertion on the distal phalanx is limited with ultrasound due to the restricted window provided by imaging through the frog and decreased resolution. Ultrasound of the collateral ligaments of the distal interphalangeal joint is limited to the proximal 1/3. As many lesions are found in the mid to distal aspect of the collateral ligaments, a normal ultrasound exam does not exclude injury to these structures.

MRI:
MRI provides excellent soft tissue detail free from superimposition, allows for characterizing bone changes that may not be advanced enough to identify radiographically, and is the best modality for assessing articular cartilage and bone marrow lesions (“bone edema”). The downside of MRI is typically cost and time. In considering types of MRI, there is a tradeoff between low-field standing MRI with decreased resolution versus the improved image quality of high-field MRI, which requires general anesthesia. While there are many benefits to MRI, a particular value of MRI in the foot is the visualization of the soft tissues structures within the hoof capsule, such as the distal DDFT and the distal portion of the collateral ligaments of the DIP joint.

CT:
CT has the advantage of having much faster scan times than MRI, requiring less time under general anesthesia and allowing for a larger scan range. CT images can be reconstructed in any plane and 3-D volume renderings can be constructed. CT has excellent bone detail and is particularly useful for fracture repair assessment and planning. Soft tissue detail is limited with CT and requires regional and/or intra-synovial iodinated contrast material for adequate visualization and assessment of injury.

Putting It All Together:
Each modality has strengths and weaknesses and there is no one “best” modality. Although MRI is typically considered the gold standard, and has the advantage of providing the greatest overall amount of information about bone and soft tissue, it is not the best modality for absolutely everything, nor is it always necessary. In many cases, sufficient diagnosis can be achieved with radiographs and/or ultrasound. Additionally, ultrasound is superior to MRI in detection of small surface bone proliferation and dystrophic mineralization. Depending on the resolution of the MRI and the sequences utilized, radiographs can be more reliable than MRI for detection of periarticular osteophytes and small fragments. However, it is also important to realize the limitations of ultrasound and radiography, particularly in cases where the standard imaging findings do not sufficiently explain the clinical signs or the patient is not responding to empiric treatment. In working through cases, it makes sense to take a stepwise approach, starting with the standard imaging examinations and then deciding if more advanced imaging is warranted. Interpreting all imaging modalities in conjunction will provide the greatest degree of diagnostic information and allow for the most tailored treatment plan.
Figure 1: Radiographs, ultrasound and MRI of the foot. The MRI has been flipped to match the ultrasound image, which is obtained in the transverse plane at the level of the proximal recess of the navicular bursa. Dorsal margin tearing of the deep digital flexor tendon and navicular bursitis is noted on both the ultrasound and MRI (short arrows). There is a severe flexor cortex erosion of the navicular bone, seen on both the skyline radiograph and MRI (dashed arrow). The degree of navicular bone degeneration is better appreciated on the MRI. Additionally, there is core-type tearing of the deep digital flexor tendon that extends to the insertion on the distal phalanx, seen only on the MRI (long arrow).

References:
References available from the author on request.
ULTRASOUND SCANNING TIPS FOR THE HINDLIMB PROXIMAL SUSPENSORY LIGAMENT
Myra Barrett DVM, MS, DACVR

Anatomy:
The hind suspensory ligament primarily originates from the plantar cortex of the third metatarsal bone (MT3) and is nestled between the fourth and second metatarsal bones (MT2 & MT4). A small bundle of fibers originates from the fourth tarsal bone and joins the rest of the fibers originating from MT3. The suspensory ligament contains central portions of fat and muscle fibers. The exact distribution will vary from horse to horse and can even vary slightly between right and left limbs. Sometimes the fat and muscle are confined to the inner and dorsal portion of the ligament and sometimes they extend abaxially or plantarly through the periphery. The suspensory ligament changes shape as it extends distally, starting as a trapezoidal structure to more heart shaped and then ovoid. The lateral aspect of the ligament is normally thicker than the medial. There is fat and connective tissue surrounding the ligament, and distal to the origin there should be space between the suspensory ligament and plantar cortex of MT3.

Ultrasonography:
The complex anatomy and anatomic positioning of the hind SL can create a diagnostic imaging challenge. Ultrasound is the most commonly used tool for evaluating the hind suspensory ligament. Including multiple and non-traditional ultrasound techniques can improve the evaluation of normal tissue types and differentiation of normal variation in tissue from pathologic changes.

Preparation:
Ideally the limb should be clipped and the skin washed. If clipping is not possible, the limb should still be washed and soaked with warm water and gel applied. A linear transducer is used, generally with a frequency of 8-10mHz. A standoff pad is often not necessary, but can be used to help improve probe contact with the limb.

Approaches:
A complete hind suspensory ligament study will include four approaches: 1) standard plantar medial weight-bearing transverse plane 2) standard weight-bearing long axis plane 3) non weight-bearing on and off angle plantar medial transverse plane 4) non weight-bearing on and off angle plantar transverse plane (Figure 1). A long-axis non weight-bearing approach can also be included. While this may seem overly time consuming and extensive, combining these multiple approaches allows for the most complete evaluation of the ligament and in fact makes it easier to define the margins of the ligament, the tissue types and detect pathologic changes. Including off-angle imaging is a key element for visualizing the true margins of the ligament and differentiating fat, muscle and ligamentous fibers within the ligament. When examined from a traditional, on-angle approach, the ligamentous fibers are hyperechoic and there are areas of decreased echogenicity that correspond to fat and muscle. When the transducer is angled slightly so as to be off-angle to the ligament, the ligamentous fibers appear hypoechoic and the areas of fat and muscle are hyperechoic. The off-angle approach also is better for evaluating the size and shape of the ligament. This is because when the ligament appears hypoechoic with off-angle imaging, there is better contrast between the ligament and surrounding connective tissue, allowing for more accurate assessment of the actual borders of ligament. Cross-sectional area (CSA) can be measured and compared to the contralateral limb.

Take Home Points:
• Multiple approaches including non weight-bearing are essential for complete ultrasonographic evaluation of the suspensory ligament
• When examined off angle ligamentous fibers appear hypoechoic and fat and muscle bundles appear hyperechoic

Pathologic Changes:
Pathologic findings can include diffuse enlargement, discrete linear tears, dorsal margin tearing and enlargement, diffuse fiber damage, fibrosis/scarring and osseous changes. If there is an area that remains hypoechoic no matter what the angle of beam incidence, this is generally indicative of areas of fiber damage. However, there is commonly a central area that appears somewhat hypoechoic both on and off angle that occurs approximately 2-4cm distal to the origin of the suspensory ligament. This appearance is more obvious in the plantaromedial approach. Normal variants such as these are important to recognize, as this site is commonly confused for a core lesion.

Areas of increased echogenicity on all approaches indicate fibrosis/scar tissue formation. Dystrophic mineralization can also occur and if subtle can be difficult to distinguish from scarring. Enlargement is common in desmopathy. In addition to measuring the CSA, it is very helpful to compare the size to the opposite limb. Assessing mild enlargement can be difficult, particularly as there is no guarantee that the contralateral limb is normal. It is also important to remember that there is no “one size fits all” on CSA. Normal CSA for a Warmblood is not going to be the same as a reining Quarter Horse, for example. It is helpful to get a sense of normal variations in measurements in non-lame horses that are similar in size, breed and discipline. Additionally, the measurements that have been obtained and reported from the traditional plantar weight-bearing on-angle approach will differ from the CSA on the off-angle non weight-bearing exam and should not be directly compared.

Pathologic changes to the bone can occur in addition to ligamentous pathologic change or can be the primary abnormality. Both long axis and transverse imaging are important for evaluating the plantar cortex of MT3. Abnormalities can include bone proliferation, resorption and avulsion fragmentation. Although in many capacities MRI is very good for assessing changes to bone, ultrasound can actually be superior for identifying small avulsions and enthesopathies because of the improved contrast between ligament and bone with ultrasound when compared to MRI. The axial margins of MT2 and MT4 should also be evaluated for proliferative changes that could impinge on the suspensory ligament.

Take Home Points:
• Diffuse enlargement is one of the most common abnormalities
• Areas of fiber tearing or degeneration appear hypoechoic no matter what the angle of the beam
• Scar tissue and mineralization will appear hyperechoic even when the ligament is examined off-angle
• Comparison studies to the contra-lateral limb are essential
Figure 1: Standard images of the proximal hind suspensory ligament. WB: Weight bearing, NWB: Non weight-bearing, O/A: Off-angle imaging.

References:

RADIOGRAPHY OF THE CERVICAL SPINE
Myra Barrett DVM, MS, DACVR with content contributions from Kurt Selberg, DVM, MS, DACVR

Introduction
The equine cervical spine is receiving increased attention as a source of dysfunction in the equine patient. This includes neurologic abnormalities, pain and stiffness, decreased performance and forelimb lameness. In most practice circumstances, imaging of the equine neck is confined to radiography and ultrasound.

The cervical spine is a complex region of anatomy. Accurate acquisition and interpretation of cervical imaging studies requires understanding the complexity of the anatomy and identifying normal variants and incidental findings.

Technique
A complete study of the cervical spine should include the caudal skull through the first thoracic vertebra. Lateral radiographs are often obtained in the field with a portable x-ray generator and detector. Coordinating both x-ray generator and x-ray detector can be challenging as direct visualization from the x-ray generator is not typically possible. This may be easily overcome by palpating the transverse processes of the cervical vertebral and marking them with tape. Centering the x-ray generator light and x-ray detector at the transverse process/tape will typically allow for adequately positioned radiographs. The addition of lead markers in the jugular furrow with overlapping x-ray images will also allow for easy identification of location on the cervical vertebral column. The resultant images should have the articular processes superimposed centrally. Towards the periphery of the x-ray image the articular facets may be slightly oblique/off set in a craniocaudal fashion due to x-ray beam divergence (figure 1). A common artifact in obtaining cervical vertebral column radiographs is underexposure, which results in a grainy appearance of the digital radiographs. This may be overcome in the field by using a shorter distance to the x-ray plate.

Oblique radiographic projections to better visualize the facet joints individually have been described and can be utilized to more completely assess the pathologic changes within the cervical spine. Oblique radiographs project the dorsolateral and ventrolateral aspects of the cervical vertebra on both the right at left sides. This aids in the separation of symmetrical anatomy and allows for lateralization of bone pathologic change. To accurately evaluate both sides, orthogonal (opposite) oblique images must be obtained. Oblique radiographs are typically obtained from a right/left dorsolateral to left/right ventrolateral fashion at approximately 45 to 55°. These are often more easily obtained while the neck is in a neutral position. The x-ray generator light is approximately 6 to 8 cm dorsal to the tape, while the x-ray detector is placed slightly ventral to the neck to accommodate the angle of the x-ray beam. Obtaining a right dorsal to left ventral oblique will highlight the left articular facets dorsally, and the right transverse process ventrally (figure 2). This allows for more complete evaluation of the pathologic changes and lesion localization.

Common Radiographic Abnormalities
Well-positioned lateral images are imperative in order to accurately assess articular facet changes and look for any evidence of cervical vertebral malformation and stenosis. Small
changes in obliquity can easily create the appearance of facet enlargement and alter the appearance of the cervical canal.

Cervical vertebral malformation (wobblers) is a common differential for horses with a neurologic condition. Radiographs can be helpful for screening, and can often accurately diagnose cervical vertebral malformation but cannot definitively allow for diagnosis of spinal cord compression. Myelography is needed to further evaluate the sites of suspicion for evidence of spinal cord compression, although even it can produce ambiguous results.

Enlargement or malformation of the cervical facet joints can result in multiple clinical signs, including neck pain, poor performance, neurologic deficits and forelimb lameness. Enlargement can occur secondary to multiple pathologic processes, including osteochondrosis (figure 3), osteoarthritis and previous trauma. With facet enlargement, the intervertebral foramen becomes narrower and less distinct; this is a useful radiographic gauge to help assess the severity of the enlargement. However, it is important to recognize that enlargement can also be found as incidental remodeling with no associated clinical signs.

Classic signs of osteoarthritis that are frequently seen in other joints, such as osteophyte formation and subchondral bone sclerosis are less commonly identified radiographically in the cervical facet joints. More common signs of degenerative change include enlargement, periarticular bone production and joint capsule enthesopathy. The radiographic changes must be interpreted in conjunction with clinical signs as there is often poor correlation between radiographic changes and clinical signs in the cervical spine, and radiographic abnormalities may be incidental.
Figure 2. An adequately positioned lateral radiograph of the cervical vertebral column. Note: there is a slight obliquity of the C4 – 5 dorsal articulation due to x-ray beam divergence (arrow). This should not be mistaken for an abnormality.
Figure 4. Right 55° dorsal-left ventral Oblique radiograph of the caudal cervical spine. The left dorsal aspect and periarticular margin of the articular facet is highlighted in this image (arrow). The right articular facet is superimposed over the vertebral body, highlighting the joint space (arrow heads). Note: the cranial aspect of the joint is widened while the neck is in a slightly extended position.

References


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PRACTICE MANAGEMENT PROGRAM
IS YOUR PRACTICE GETTING THE DATA IT NEEDS FOR SUCCESSFUL MANAGEMENT?
Karen E. Felsted, CPA, MS, DVM, CVPM, CVA

Introduction

In the same way that the TPR gives an initial look at a pet’s basic health status, periodic financial and operational reports give the practice owner and manager a look at the practice’s financial health status. Now more than ever, a business must understand and regularly measure the metrics that define financial success. Both internal trending and external comparison to published benchmarks are critical to future growth and prosperity. Several types of metrics should be assessed regularly: revenue, expenses, profits and a broad group of operational benchmarks measuring doctor and staff productivity and client activity. Practices need this data to make decisions about revenue growth, expense management, profitability, marketing program analysis, efficiency, productivity and other issues. One of the oldest clichés is that you can’t manage what you can’t measure. Counting the paperclips on a regular basis would be ridiculous but in this lecture, we will discuss the critical metrics every practice must review each month. Knowing your profit margin isn’t enough, you also must understand what drives the profitability and how to review the success (or not) of programs implemented in the practice.

Starting Out

The first question to be asked before launching into data gathering and analysis is: Why do we think we need data? What are we going to do with it? Practices generally turn to data to either get an overall understanding of how well the practice is doing operationally and financially or to solve a particular problem they perceive in the practice. There are many practice problems for which data can be useful in understanding and solving but some of the most significant ones include:

- Flat or declining revenue
- Poor profitability or cash flow
- Doctor production that is too low
- Staff or other costs that are too high

As noted above, one of the primary reasons the management team of a practice looks at data is to gain an overall understanding (a baseline) of how well the practice is doing operationally and financially. This gives the management team the best chance of selecting the areas where change will have the most impact on growth in revenue or profits. What data should be gathered for this initial review?

Data that will be typically helpful in getting this baseline snapshot of the practice’s financial and operational performance includes:

- Revenue growth/decline (%) in this year/quarter/month compared to the same period in prior years
- Revenue per full-time-equivalent doctor (a measure of overall doctor efficiency)
- Transactions per full-time equivalent doctor (a measure of overall doctor efficiency)
- Medical (doctor) revenue per full-time-equivalent doctor (a measure of individual doctor efficiency)
• Medical transactions (doctor) per full-time equivalent doctor (a measure of individual
doctor efficiency)
• Average transaction charge for the practice as a whole and for medical (doctor)
revenue
• Revenue/transactions/ATC for individual doctors (a measure of individual doctor
performance)
• Revenue by category (dentistry, vaccinations, product sales, etc.)
• New clients
• Active clients
• Lost clients
• A/R aging
• Expenses, particularly drugs and medical supplies, laboratory costs, doctor
compensation, staff compensation, benefits, facility costs
• Staff and doctor hours per transaction
• Revenue and transactions by species
• Operating profit margin

Most of this data will come from the PIMS (Practice Information Management System) used in
the practice. The expense information will come from the profit and loss statement and/or tax
return. Staff and doctor hours worked information will come from the payroll system. The
operating profit margin is calculated using data from the tax return, profit and loss statement,
PIMS and other sources.

Data quality is based on good quality system design and checks and balances that need to be
in place to insure ongoing integrity. Some of the questions that need to be asked include:

• Is all revenue captured in the system?
• How much does the practice lose in missed charges and random discounts?
• Is revenue accurately entered for each doctor?
• Are services included in the correct service category?
• What is the definition of a new client in the practice’s PIMS?
• Are the accounts used to categorize expenses appropriate for a veterinary clinic?
• Are expenses entered accurately into the practice accounting system?
• Are bank accounts reconciled regularly and reconciling items followed up promptly?
• Does the person doing the profit calculation have the appropriate knowledge to do it
correctly?

The most useful baseline analysis includes comparison of the metrics over time in the
practice—this year, last year, two years ago as well as a comparison to other typical practices
based on published studies, such as AAHA’s Financial and Productivity Pulsepoints. This kind
of analysis is excellent for determining where a practice is doing well and what areas need
further investigation or improvement. The practice should first start with the question: Is the
practice truly profitable? And then: If not, why not? The rest of the data can then be used to
determine the answers to this second question. Is it due to high inventory costs? Lack of
productivity by doctors or staff? Poor revenue growth? Declining new clients? The analysis is
also useful to determine where further investigation should be made and to measure progress
as the practice makes changes to improve its operations and financial position.
The Next Step

Once the big picture analysis has been done, the practice should drill down further into areas that may need improvement. For example, let’s say that the practice owner or manager analyzes the revenue of the practice at the doctor level and finds that the average revenue per doctor is lower than that seen in most practices and that there is a great deal of variation in productivity amongst doctors. Improved doctor productivity becomes a goal of the practice. What additional data should be gathered?

• Number of hours worked each week by the doctors—revenue variability may be a function simply of the time spent in the practice
• Number of appointments, surgeries, dentals done by each doctor during this time frame
• Support staff help utilized by each doctor—some doctors may be able to produce more because they have access to and use more support staff
• Number of key procedures (CBCs, chemistry panels, x-rays) performed by each doctor in relation to the number of transactions they generate—revenue may vary because of different approaches to cases which should be more consistent
• Measurement of client compliance with key recommendations by doctors and staff
• Dollar amount of discounts and missed charges per doctor

As the data gets more detailed, a wider variety of sources may be necessary to obtain it. Occasionally practices will have doctors clock in and out the same way non-doctor team members do. If this is true, the practice may have good quality “hours worked” information for doctors although the in-clinic hours may need to be adjusted for any substantial amounts of work done at home (record writing, client callbacks, case research) or for trips back to the clinic outside of normal hours. If the practice doesn’t have this information, it will have be created. Support staff utilization is a more subjective measure that is generally gained by observation. Key procedure information can be obtained from the PIMS. Measure of client compliance with key recommendations isn’t available in all practices but is an important piece of data. When available, it can usually be found via medical record audit or in the PIMS if service codes are used to track when recommendations are made, accepted or declined. Discount and missed charge information comes from a medical record audit.

A number of questions should be asked at this point as well to ascertain data quality:

• Is revenue accurately assigned to the right doctor when charges are entered?
• Is the sample used for the medical record audit representative of the entire year?
• Are the doctor work hour estimates of good quality?
• Is the discount information broken out between approved marketing discounts and random discounts?

The findings from the above analysis will drive what the practice does. For example, after controlling for hours worked, if one doctor is doing fewer dentals than another, it may be because the doctor doesn’t do a good job of discussing dental needs with the client either because they are rushed in the exam room or because their communication skills aren’t as strong as they should be. If it’s the first reason, checklists or a consistent exam room technician may help. If it’s the second reason, communication training is the answer. Doctor productivity may also suffer because the practice simply doesn’t have enough patients coming in the door; in this case, improved marketing may need to be the focus.
General Guidelines For Effective Data Gathering And Use

In order to compare one practice to another, it is necessary to set up the revenue and expense categories in both the practice management and accounting systems in a fashion similar to those commonly used in veterinary medicine and used in the published studies the practice will be compared to. An excellent veterinary chart of accounts is available from AAHA.

Record information in the same categories or perform calculations in the same way each time they are done in order for the numbers to be comparable over time. In order to achieve this comparability, it is important to carefully set up the categories and define the calculations when the accounting system and PIMS are set up. Some aspects of the system will need to be changed periodically; the practice must keep in mind the impact on comparability when it’s time to do this.

Comparison of raw numbers is useful to a point in financial analysis but, in general, it is necessary to do some kind of ratio analysis or use a common basis of comparison to get the best results. The most common ratio used in veterinary financial analysis is that of expressing types of revenue or expenses as a percentage of total gross revenue. This kind of ratio analysis is critical to accurate comparison over time or between practices of different sizes. Another frequently used common basis for comparison is the calculation of doctors on a full-time-equivalent basis. This makes it possible to compare information from one practice to practices that have different numbers of doctors or to compare the figures in your own practice from year to year as you add doctors.

Most practices use a cash basis of accounting for internal purposes. This means that when the bill is paid, the expense gets recorded in the financial statements. Fluctuations in when bills are paid (whether deliberate or accidental) can have a significant impact on analysis. For example, let’s assume a practice normally spends about $30,000/month on drugs & medical supplies expense. When comparing monthly expenditures for the last month, the owner notices:

<table>
<thead>
<tr>
<th>Drugs &amp; medical supplies expense</th>
<th>Apr</th>
<th>May</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$30,000</td>
<td>$25,000</td>
</tr>
</tbody>
</table>

At first, it looks like the practice’s efforts to better control inventory are paying off. However, it turns out that the decrease occurred because the bookkeeper went on vacation during the last week of May and didn’t pay the rest of that month’s bills until June. June’s expense was $35,000. The timing of the bill paying must always be considered in expense analysis. A review of the outstanding accounts payable will help. The practice should consider using some of the accrual accounting features available in most accounting software or doing the five or six journal entries necessary to convert cash to accrual every quarter or every year. It is important that the people doing the bookkeeping and the financial analysis have enough real accounting knowledge to understand the implications of cash vs. accrual accounting.

Key factors to remember when comparing the practice to published data—Is the data meant to represent an average practice or “best practices”? Is the data reliable?

The methodology section of the reports explains how the data was collected, how many practices responded and this helps with understanding how comparative the data is. It is also important to understand how certain calculations were performed in order to know if that metric is comparable to your practice. No study will be perfectly comparable to a particular practice. This doesn’t mean the study is useless. It is still possible to get very valuable information to
help operate the business more effectively. It simply means using these comparisons with a grain of salt and as one tool in running the business, not as the final word about how well the practice is doing. There is more published benchmark information for combined dog/cat practices than there is for feline only practices. The most commonly used are:

- Benchmarks: A Study of Well-Managed Practices (available for purchase from Advanstar)
- Financial & Productivity Pulsepoints and Compensation and Benefits (available for purchase from the American Animal Hospital Association)
- AVMA Business Measures Study (available for purchase from the AVMA)

Finally and most importantly, identifying trends or problem areas is not enough. It is critical that the management team investigate the changes or potential problem areas and determine if action is needed to correct an issue, then implement the changes and track the results.
I CAN’T FIND ALL MY MONEY! CONTROLLING INVENTORY
Karen E. Felsted, CPA, MS, DVM, CVPM, CVA

One of the most significant expense categories in veterinary practices is Cost of Medical Services. This includes the costs of drugs and supplies, laboratory expense, animal disposal, etc. In a small animal practice, the average drugs and medical supplies component is about 15-16% of gross; however, some practices keep this much lower. Effective inventory management is key to keeping these costs under control. Inventory control is sometimes seen as a boring and tedious task, but it can have a huge impact on your profitability and is actually one of the easier things to do well in a practice.

The goals of an effective inventory system include:

• Maintenance of the smallest quantity of drugs and supplies needed by the practice, procured at the lowest possible cost while providing the practice with everything needed to provide the highest quality care and without incurring stock-outs
• Systems and controls are in place to keep theft and other shrinkage to a minimum, to insure accurate records are kept and that drugs and supplies are available when needed
• Accurate records are readily available in order to evaluate the efficiency of the system and to improve upon it
• System is simple for all to use
• Inventory is well organized within the facility, easy to locate and not vulnerable to theft or misplacement
• Vendor numbers are kept to a minimum
• Vendors selected are reputable, interested in your success and the success of our profession and provide products necessary within the practice, as well as good service and fair prices
• All medications and products sold to clients are included on their invoice and appropriately charged for
• Inventory is sold to clients before bill has to be paid (there will be some exceptions to this when good deals present themselves); generally this means inventory needs to turnover once a month
• A reasonable profit is realized on sales

In order to improve your inventory control, you first must get a handle on the current situation by answering the following questions:

• What is the current or most recent dollar value of inventory? As of what date?
• Does this information come from your balance sheet or the practice information management system (PIMS)? Are either or both of these reports accurate?
• What was the total dollar value of inventory purchases for the past fiscal year and as a percent of gross revenue?
  o Drugs and medical supplies
  o Food
• What was the inventory turnover for the past year? This should be calculated for pharmacy items, food and all inventory combined
• Print a list of all inventory items sold during the most recent month and compare this to the items on the shelves. What items are on the shelves that were not sold in the
past month? For items sold during the month, how many units are on the shelves compared to how many were sold?

Now it is necessary to define the critical steps in the process and see if these are being done in your hospital at all times.

- Who is in charge of the inventory system?
- Who normally orders the inventory?
- How does the practice determine what is needed?
- How many distributors does the practice regularly order from?
- What, if any, group purchasing arrangements does the practice participate in?
- How frequently are prices checked amongst vendors? Who does this?
- Are purchase orders used to order inventory? If not, is a written list of each item ordered kept?
- When the order is received, is the list of items ordered compared to the items received?
  - Who does this?
  - Is the order list initialed by the person doing the comparison?
  - Who follows up on discrepancies?
  - Are back ordered items tracked?
  - How?
- When supplies are delivered to the practice, is the packing slip or invoice included in the box checked against the items actually received?
  - Who does this?
  - Who follows up on discrepancies?
- Where is the packing slip or invoice placed after it is checked?
- Are the quantities received and item prices entered into the computer after the order is received?
  - When?
  - By whom?
  - Where is the packing slip or invoice put after computer entry?
- Are the prices charged to clients changed when the clinic cost changes?
- Are items included in group codes or packages checked each time an order of the item is received?
- Who tracks short dated or out of date product?
  - How is this done?
  - Who arranges for the return of product to the vendor?
  - How is this information communicated to the bookkeeper?
- Are packing slips compared to invoices?
  - Who does this?
  - Who follows up on discrepancies?
- Are invoices compared to statements?
  - Who does this?
  - Who follows up on discrepancies?
- Who writes the checks?
- Are the invoices reviewed by the check signer when signing the check?
- Who has the authority to sign checks?
- Is there a limit to the amount?
  - What is it?
One of the most important inventory management procedures is regular counting of the products on the shelves. Most practices do not count their inventory on a regular basis. At best, they do it once a year for tax purposes. The count done for tax purposes is not sufficient to make sure that your inventory system is working effectively. All items need to be counted on a more regular basis.

The items most susceptible to theft are food, heartworm preventative and flea/tick products; these should be counted monthly to make sure they are not being given to clients without being charged for or stolen. In the beginning, you may need to count them more frequently if you are having problems keeping track of your inventory. Make a list of all of these items (list each size individually) and then divide it by four so that each item is counted once a month.

Count the product on hand and immediately check the balance indicated in the computer for this product. It is critical to do these two steps right after each other so that the comparisons are between “apples and apples.” If the product is counted and the computer balance checked later, product could be sold or received and added or deducted from the computer balance which would then not agree with the amount counted.

The counts and computer work should initially be done by a practice owner and should be “visible”; i.e. done during business hours so that the staff is aware that this procedure is taking place. The counts should not be done before or after hours and they should be done when several staff members are around. The counts shouldn’t be treated as an unusual procedure nor should it be suggested that they are being implemented due to the possibility of staff theft, but do let it be known that this is a new procedure that will be done regularly. If asked why the counts are being implemented it should be said that the cost of inventory is one of the biggest expenses in the hospital and with the growth of the practice, the owners want to control this cost a little better by improving the inventory system.

If there are discrepancies in the counts, ask the appropriate questions of the staff people:

- Are there any product purchase invoices that haven’t been entered into the inventory module?
- Was any product used in-house that hasn’t been recorded in the inventory module? (i.e. through a dummy client account?)
- Was any product sent home with either clients or employees that hasn’t yet been recorded on an invoice? This is more often a problem with hospitalized or boarding patients than with out-patients.
- Was any product returned to the manufacturer that hasn’t been deleted from the inventory module?
- Was product used for any other reason and not deleted from the inventory module?
- Is product stored in some other location which may not have been counted?
- Does the staff have any other ideas as to why the discrepancies exist?

Depending on the level of the discrepancies and whether or not reasonable explanations can be found for the discrepancies, it may be necessary to institute more stringent inventory control procedures until the problem can be found.

Once you have this part of the system in place for food, heartworm preventative and flea and tick products, expand your counts to include other products. Unless you are experiencing a problem, the counts on the other products usually do not need to be done as frequently.
Frequency will be determined by the $ value of the item, its likelihood of being stolen or given away and your experience with this product in your clinic. Don’t forget that controlled substances should be counted weekly until you know you do not have discrepancies.

Good physical control of the inventory is important for several reasons:

- Helps insure inventory is properly stored based on its physical requirements; i.e. temperature and light
- Inventory that is well organized and easy to find makes it easier for you to assess how much is on hand, facilitates keeping track of short-dated product and allows for quicker and more accurate physical counts
- Proper organization and storage is a deterrent against theft and makes it easier to keep track of in-house usage
- Sensible organization facilitates good record-keeping

In general, good physical control requires:

- A locked central storage area with limited access—even here only small quantities of product should be kept
- Small quantities of products kept in exam rooms, pharmacy, lab area and other areas easily accessible to employees
- Empty boxes displayed in public areas

Other inventory control issues to be considered include:

- Frequency with which inventory prices are reviewed
- What is the dispensing charge added to each product sale?
- Is there a minimum charge for each product sale?
- What is the markup for food? Flea & tick products? Heartworm preventative? Other prescription drugs?
- If product is purchased at a special price, is this passed through to the client?
- Are inventories adequately insured?
- How frequently are medical record audits done?
- Are inventory costs as a % of gross revenue reviewed from period to period?
- For what kinds of inventory items are more than one similar product carried?
- Are employee accounts reviewed periodically to assess the reasonableness of purchase quantities?
- Do practice owners take inventory items from the practice without paying for them?
- Are the following reports used from the practice’s inventory computer module? Usage, order history, inventory quantities, reorder points
DRIVING PROFITABILITY IN YOUR PRACTICE
Karen E. Felsted, CPA, MS, DVM, CVPM, CVA

Introduction

The gold standard measure of a practice’s financial success is the operating profit margin. Unfortunately, most practices don’t really know what how profitable they are. Since this profitability drives the value of a practice and is essential to making good operating decisions, an analysis of true profitability and what drives it is essential.

Operating Profits

Overview
Understanding the profitability of a practice is one of the most important concepts necessary to manage a veterinary hospital well. The operating profit of a practice is the one single number that tells a practice owner or manager whether or not the practice is really financially successful. Before delving down into where profits come from, it is first necessary to discuss why this concept is so important and how to calculate the overall profit margin of the practice.

In addition to the obvious impact on current cash flow, profitability also is a critical determinant of practice value. Historically, practice owners have assumed (and with good reason) that when they decided to sell their practices there would be buyers ready to purchase them and willing to pay a good price. In other words, they have assumed there was value in these businesses that could be transferred to someone else. Of course, there have always been a few practices for which this assumption didn't hold true. A buyer couldn't be found or what buyers wanted to pay wasn't remotely what the seller thought the practice was worth. Typically these practices have been easy to identify and had several traits in common. They tended to be smaller practices with owners who had not focused much on the business side of things. Often the facility and equipment were old and the doctors hadn’t kept up with the changes in medicine as much as perhaps they should have. These practices had little profit in them and, because the bulk of practice value is determined by profitability, the practices had little value. Fortunately there weren’t too many of these practices.

However, in the last ten years, the number of practices with no or little value has been increasing—to the point where the Veterinary Valuation Council of VetPartners coined the term “No-LoSM SM practice” to describe these practices.

“No-Lo” stands for no or low value practices and is also used to mean no or low profitability. More and more practices, when appraised, did not have the value that would normally have been expected. And, in almost all cases, the owners of these practices were totally unaware of the problem. Some of these practices had traits in common with the practices that have historically had little or no value. They were small practices with a low level of profitability and couldn’t keep up with changing client demands regarding service, quality of medicine, advanced technology and improved facilities. The other practices with no or little value, however, were a surprising group. On the surface, these practices would appear to be doing very well. They are located in very attractive facilities, practice good medicine, have all the latest equipment and a large support staff, offer comparatively high compensation and benefits to their employees and, in the owners’ eyes, cash flow is strong. However, practice value is largely based on profits and the very factors that make these practices look attractive on the surface are those that are reducing profitability.
Calculating the true operating profits of a practice is, however, not a simple task. None of the standard financial or management reports a practice usually gets includes this figure. Neither the taxable income as shown on the tax return nor the net income from the profit and loss statement represents true profitability. This doesn’t mean those reports are improperly prepared; it simply means the reports required by your taxing authority or accounting standards for small businesses weren’t designed to determine profitability. No one report will give a practice all of the financial information it needs to make intelligent operating decisions; unfortunately, the report that seems to be prepared least often is the one that calculates true practice profitability. Because practice owners and managers aren’t used to getting this kind of information, they generally don’t know what the true profitability of their practice is. The first time many owners realize their true profitability is when their appraiser talks to them about it.

The operating profit is the difference between the operating revenues and expenses of a practice. Operating revenue and expenses include only items normally and necessarily seen in the day-to-day operations of the practice such as fees for professional services and drugs and medical supplies expense. These items should be stated at fair market value rates. For ease of comparison with other practices, the profit margin is generally stated as a percentage—this is calculated as practice profits divided by gross revenue. Some of the items that must be calculated differently to determine operating profit versus taxable income or net income include: practice owner payments, facility and equipment rent if these items are owned by the practice owner and leased to the practice, services provided by family members to the practice, depreciation, interest on debt and perks.

How is the operating profit calculated?
The following steps are generally those needed to get to this figure. Taxable income per the tax return is usually the starting point. Various adjustments are made from there; the specific ones applicable to an individual situation depend on the format of the tax return and the individual practice:

- Add back: depreciation, amortization, equipment lease payments treated as an expense in the tax return and interest on debt
- Deduct the estimated average amount spent on equipment per year—purchasing equipment is a true operating expense of the practice but depreciation as determined by tax law is not the best estimate. A reasonable estimate in many practices is 1.5% of gross revenue.
- Determine how much the owner was paid in compensation and rent (if the practice owner owns the practice facility as well) during the year
- Adjust owner compensation to represent a fair compensation for medical/surgical work
- Adjust owner compensation for management work—management expense generally averages 3-5% of gross revenues—if the practice has a practice or office manager, the owner should get less than this amount as management compensation—1.5% is generally used to represent owner management compensation when the practice has a manager.
- Adjust rent expense to fair market value if paid to owner at a rate greater or less than fair market value
- Determine the $ amount of personal perks paid by the practice and remove this expense—perks would be items not necessary to the operation of the practice but paid by the practice generally to gain a tax advantage (examples include excess meals and entertainment, excess auto costs, swimming pool payments, personal
• Deduct the cost associated with free services provided to the practice—family members may provide bookkeeping or other services to the practice at no charge—if the practice had to hire someone to do this work, there would be a cost involved and this should be included as an expense
• Add back any compensation and benefits paid to family members that don’t provide equivalent services to the practice
• Remove any true non-recurring income or expenses such as one-time insurance proceeds or expenses related to a natural disaster
• Subtract interest and dividend income
• Recalculate net income
• Divide the new net income by gross revenue

The resulting percentage is the true operating profit of the practice—how does it compare to other investments you have? And to other practices? 18% or above would be considered superior, 16-18% excellent, 13-15.99% good, 8-12.99% fair and less than 8% poor. The above figures are for companion animal practices. While no specific figures exist for feline only practices, the current thinking is that these figures would still be generally applicable.

**Profit calculations by service or hospital department**

Once the overall profit margin of the practice has been determined, a further analysis can be done to determine which areas of the practice contribute the most to profitability. Both revenue and expenses must be allocated to each department or division of the hospital. Departments can be determined in several different ways; in a general practice they would usually include: outpatient, pharmacy, surgery, dentistry, hospitalization, boarding and grooming. A mixed animal practice might first look at profits by species; for example by equine, dairy cow, companion animal and the other species seen in the practice. It is unlikely there is much difference between the profitability of the services offered to dogs and cats in a typical companion animal practice but there generally IS a significant difference between companion animal and food animal/equine services in a mixed animal practice.

These departments generally have different cost structures and different levels of profitability and unless the departments are reviewed as independent businesses within the larger entity, it will not be possible to manage them most effectively.

It is generally easy to categorize revenue separately for each of these divisions but expense allocation is harder to do and most practices don’t even try. The goal, however, is to create a profit and loss statement for each division. For example, total staff costs may look a little high when they are reviewed in total for the general practice with the large and elaborate boarding component. After costs are allocated between the 3 entities (medical practice, boarding and grooming), however, it may become apparent that staff is used efficiently in the medical practice but costs are very high in the boarding/grooming departments in comparison to the revenue brought in.

Setting up systems to accurately allocate expenses is a little time-consuming in the beginning but will pay big dividends in the end: Ways of allocating some common expenses are shown below:

• Drugs & medical supplies—set up the inventory system to track usage by service; for example, all drugs & supplies may be received into central supply and subsequently issued to each department.
• Outside laboratory test costs should be assigned individually to each department
• Usage of internal laboratory or imaging facilities/equipment—the number of tests used by each department must be captured and total costs for the equipment (including related personnel, supplies and an allocation of the original purchase price) is allocated to each department based on usage
• Compensation—amounts related to doctors or staff assigned solely to one service should be assigned to that department. Costs related to floating or shared staff need to be allocated monthly by usage. For example, receptionists’ compensation may be allocated based on the % of transactions for each department. Benefits may be assigned individually (often done for doctors) or allocated using payroll %s (more commonly done for staff)
• Facility rent—assign costs based on the square footage used by each department. Shared space can be allocated based on the # of transactions per department
• Other facility costs (janitorial, utilities, property taxes, etc.)—allocated based on same %s used to allocate rent
• Marketing—individual marketing programs can be assigned to the department involved; joint marketing can be allocated based on the # of transactions per service
• Credit card fees should be allocated based on % of total or credit card revenue per service

There needs to be a balance between the complexity of the allocation methodology and the usefulness of the information obtained. In general, a reasonably based allocation system (even if not perfect) is better than none.

The same adjustments that were included in calculating the overall profitability of the practice also need to be considered at this level of analysis. For example, if the owner doctors’ salary was adjusted to fair market value in the first calculation, this adjusted number is the one that should be used when allocating his/her salary by department.

Where To Go Next?

If the practice’s profits aren’t at the desired level, what can be done about it? A lack of profitability either comes from revenues that are too low, expenses that are too high, or a combination of the two. Understanding not only the profitability of the practice but the kinds of factors that lead to this state is critical. Until the practice has an idea of the root causes of the problem, it is difficult to determine what the correct solution is.

There are many outstanding management resources available to practitioners from various veterinary and management organizations. Working with a financial advisor or practice consultant may help in not only gaining a greater understanding of the issues impacting profitability but in identifying and implementing solutions. VetPartners (www.vetpartners.org) can help in locating an appropriate individual.
BUYING OR SELLING A VETERINARY PRACTICE PARTS I AND II
Karen E. Felsted, CPA, MS, DVM, CVPM, CVA

Introduction

Buying or selling a veterinary practice can be difficult logistically, financially and emotionally and the hardest part is often just knowing where to start.

Why Are You Doing This?

Both buyers and sellers need to first think through why they want to buy or sell and, more importantly, be sure that this action will accomplish their goals. Common reasons to sell include:

- Ready to fully retire
- Want to decrease workload
- Desire to change careers
- Desire to practice more medicine
- Health issues
- Divorce
- Location/demographics
- Management frustrations
- Financial issues
- Want to bond associate to practice

Common reasons to buy include:

- Increased earnings and equity in the practice
- Increased ability to do things your way
- Ultimately, increased flexibility in schedule
- Personal satisfaction of having created and managed a successful business

Understanding why you want to buy or sell will help you make the best choices.

Types of Practice Purchases/Sales

Outlined below are the most common types of practice purchases and sales and key characteristics of each.

Associate Buy-In (Full or Partial)

- Common transaction type-easiest to complete
- Greatest opportunity to know if buyer/seller are right fit and have right skills
- Good method of seller succession planning
- Some financial investment from the purchaser is good, but seller shouldn’t count on it—associates often have limited financial resources
- Seller must be willing to provide full disclosure during the sales process and allow participation in management and financial decisions afterwards
- Usually lock in timeframe for rest of practice purchase (if applicable) as well as real estate purchase at time of initial transaction
- Usually best option for C corps because is generally a stock sale (assuming only selling part of the practice)
• Seller often carries note on partial sales because doesn’t want to give lender lien on 100% of practice assets
• Need to do all the same documents that would done if seller didn’t know the buyer or wasn’t doing the financing (if owner-financed)—don’t get lax because it’s a friendly deal—these can go sour
• If this is a partial buy-in, both buyer and seller need to be sure they can work together as owners and have similar goals for the future
• Buyer should perform thorough due diligence even though he/she “knows” the practice

Sell/Buy the Entire Practice to/from an Unknown Veterinarian
• Harder for seller to find buyer—may use broker
• Seller should finance the deal only if absolutely must—outside financing is preferable
• Seller has less control over what will happen to practice after the sale
• Some financial investment from the purchaser is good, but seller shouldn’t count on it—associates often have limited financial resources
• Seller must be willing to provide full disclosure during due diligence period and should have reasonable expectations as to sales price, terms, etc.
• Usually lock in timeframe for real estate purchase at time of initial transaction (if applicable)
• Buyer should perform thorough due diligence

Corporate Buyers
• Sellers may have this option if they meet the corporate buyers’ criteria—generally they want profitable practices with minimum of 2-3 doctors and over $1,000,000 in revenues in good locations
• If they want your practice, seller may get a great price and mostly in cash
• Corporate buyers are very sophisticated and knowledgeable; agreements are complex and seller must have a good attorney to help them through the process
• Seller needs to understand the employment provisions—usually can’t leave right away
• Corporate buyers often don’t purchase real estate-lease rates may be low
• Deals can be quick
• Generally asset purchases
• Will have to adapt to corporate expectations—this is not easy for all sellers

Gifting (if Seller Has a DVM Child)
• Uncommon transaction
• Tax issues must be addressed
• Community property issues may make the deal more complex
• A good lawyer and CPA familiar with both federal and state issues is critical in these transactions

Merger
• A great idea if seller/buyer are the right partners
• Can be very slow process but is still the fastest way to grow practice and increase potential associate buyers
• The quantity of practice mergers is increasing and that trend is likely to continue; however, absolute #s of mergers is still small
Finding a Buyer/Seller

If you are a seller, finding a buyer can be difficult and time-consuming. Use as many options as possible when looking for this person. Speak with your own associates, advertise in various publications, contact brokers and use networking wherever possible. You can also consider non-veterinarians if your state allows it. Determine what buyers in your area are looking for and then set about creating a plan for marketing your practice.

Key things buyers are typically looking for include:

- Location
  - Primarily urban and suburban
- Type of practice
  - Primarily SA
- High quality medicine and surgery
- Profitable practice
- Lifestyle--$$ and hours
- Attractive, freestanding facility

If all of these factors don’t apply to the practice being sold, the practice will need to be stronger in the remaining categories and the seller will need to work hard to emphasize the practice’s good points. Fix the problems that are fixable.

Buyers need to recognize that no practice will be perfect and the more flexible they are, the greater likelihood of finding the right practice. Look everywhere—veterinary periodical ads, broker websites, etc. and network like crazy. Make a list of the things that are deal breakers, that are important to you but not a deal breaker or those that are not important. Use this to guide your choices.

Planning is essential for a successful practice sale. Start early--10 years in advance if you need to do an S corp election or need to significantly improve the practice value; 3-5 years in advance for fine-tuning value. A baseline practice appraiser 5-10 years prior to sale can be very useful in planning for the sale.

Advisors

Both sides will need advisors to help with process; listed below are some of the common ones:

- Practice appraisers
- Practice consultant
- Brokers
- Accountant
- Attorney
- At a minimum, each side needs their own accountant/financial advisor and attorney—a good quality appraiser will prepare the valuation on a “non-advocacy” basis

Practice Appraisal and Feasibility Analysis

A valuation of the practice is essential. This is typically done by the seller.
A feasibility analysis, sometimes called an ability-to-pay analysis, looks at the reasonableness of the appraiser’s value determination from the perspective of the buyer. The analysis attempts to answer the question, “Can the buyer successfully afford to purchase the practice at the value determined by the appraiser?” Buyers will typically have a feasibility analysis done to see if the earnings from the practice (salary and profits) will cover:

- Repayment of debt incurred to purchase practice
- Increased income taxes related to practice purchase
- “Decent” living

Determining a FAIR value (versus one that favors either a buyer or seller) is important for several reasons:

- A value that is too high decreases the chances of finding a buyer for the practice and, should it be sold, increases the likelihood the buyer may be unable to meet the debt obligations resulting in business failure and the seller being forced to “take back” what is left of the practice.
- A value that is too low fails to reward the seller for efforts put into building the practice.
- An inaccurate value diminishes the chance of realistic personal or business planning.

Having a practice appraised is one of the single most important tasks a hospital owner will have done throughout his or her practice career, because, for most practitioners, the practice is the most valuable financial asset they own. Valuing a business properly takes skill, experience, knowledge and training. In order to determine a fair value, a competent appraiser is needed—one trained in accepted valuation techniques and experienced in valuing veterinary practices.

Many people currently perform appraisals of veterinary practices including licensed or certified appraisers, accountants, brokers and consultants. Appraisers use varying methodologies to determine practice value. Because of the significant variations seen amongst veterinary practice appraisals, it is critical that the user of an evaluation understand what they are getting. The areas a valuation user should know something about are:

- Method(s) used to value a practice
  - Rules of thumb
  - Income based methods
- Kind and amount of adjustments made to a practice’s financial statements
- Capitalization rate determination
  - Problems with standard capitalization rates
  - Risk factors
- Feasibility analysis
- Using qualified consultants
  - Qualifications of the appraiser

How do you determine the qualifications of an appraiser? Just as in any profession, there are more qualified and less qualified appraisers and it is often difficult for a layperson to determine which is which. The answers to the following questions can help you make an informed choice.

- What course of study or training have you undertaken to qualify you for appraising small businesses in general and veterinary practices in particular?
- What appraisal credentials or certifications have you earned? What organization issued the certification? What were the requirements necessary to qualify for the certification?
- Do you belong to any appraisal professional organizations?
• What experience do you have in performing appraisals for veterinary clinics? How many practices have you valued?
• Can you provide references?
• What methodologies do you use?
• If you rely predominantly on the Excess Earnings method, do you perform an equipment appraisal?
• What kind of information do you require to do the appraisal?
  While an extensive document request list and questionnaire may seem onerous to compile, it is an indication that the appraiser is gathering the large amount of information that is necessary to do a thorough job.
• Do you require an on-site visit?
  Most appraisers do not unless there is something unusual about the practice they are valuing. The appraiser should, however, ask for a video or photographs of the practice.
• Do you follow the guidelines of national appraisal organizations in preparing your appraisal report?
  Almost all professional appraisal organizations endorse the standards put forth by the Uniform Standards of Professional Appraisal Practice (USPAP).
• Will you provide a sample Table of Contents of your reports?
  The table of contents should indicate discussion of the following: the appraisal assignment, the history of the practice, the environmental and demographic environment of the practice, the financial condition of the practice, the appraisal methods used, an explanation of any normalizing adjustments, a discussion of how the capitalization or discount rate was developed, a feasibility analysis from the perspective of the buyer and a reconciliation of the final value.
• Does your appraisal report include a feasibility analysis examining the ability of the buyer to purchase the practice under customary sale terms?
• What do you charge for an appraisal?
  This should be the last question asked. As with anything else, you get what you pay for. A low fee almost certainly indicates the appraiser is not spending the time or gathering the information necessary to do a thorough job, but a high fee doesn’t necessarily mean the work is being done in accordance with current standards.

Purchase/Sale Structure

The seller will also need to determine exactly what will be included in the sale and how will the sale be structured. Is this a stock or an asset sale? Are you also selling the real estate where the clinic is located? In a corporate stock sale, the buyer is buying a % of the business as a whole, not part of the individual assets and liabilities and the buyer becomes responsible for known and unknown past liabilities of organization. In an asset sale, the buyer purchases individual assets—tangible or intangible and DOES NOT purchase liabilities. In general, sellers want to sell stock because of better tax advantages and buyers want to buy assets, both because of better tax advantages and the fact that they don’t inherit the liabilities of the old business. From a practical perspective, however, if the sale is a partial sale to an associate, it will be a stock sale. Almost all sales of 100% of a practice will be asset sales.

Obtaining the real estate associated with the practice is a critical issue for most buyers that must be resolved at the time of purchasing the practice. When and at what price will they get to buy it? If you’re going to lease the real estate for a while, you must deal with these issues:
  • FMV rent
• Term
  o Renewal options
  o 15-20 years
• Who pays for taxes, maintenance, insurance, major repairs

Legal Documents

An attorney can advise both the buyer and seller on the preparation of legal documents, from the pre-purchase agreement through the sales contract. Do not skimp on legal services. Common documents include: pre-purchase confidentiality agreement, letter of intent, letter of offer, memorandum of understanding, the purchase agreement, and a buy-sell agreement.
There is not an owner or manager who can honestly say they have never made a bad hire. People look strong on paper, we like them on the telephone interview, we click with them in person and two weeks after we put them on payroll the Dr. Jekyll become Mr. Hyde. What went wrong?

The veterinary industry has a notoriously high employee turnover rate. According to the AAHA survey from 2008 we have about 44% for lay staff. A study by Harvard University indicates that 80% of employee turnover is due to hiring mistakes. These errors are costly! Entry level employees average costing 1.5 times their annual salary to replace. Those are the calculated hard costs. The uncounted soft costs are unhappy clients, lost opportunities, patients who receive inadequate or incompetent care, damage to the company reputation, low morale and hours of wasted manager time.

The question is how do you avoid these mistakes?

First you must have a HIRING PLAN. Who do you want? By this we mean, what attitude, skills, behaviors, beliefs, values and ambitions do you want to add to your team? Past experiences show these traits are difficult to determine through traditional interviewing methods.

According to the authors of “How to Hire the Right Person for the Right Job Every Time” Lori Davila and Louise Kursmark the top ten hiring mistake are as follows.

1. Not knowing who you are looking for
2. Not preparing for the interview
3. Not asking the right questions
4. Not taking the time to hire right
5. Not being cautious of the “Halo” effect
6. Hiring people like you
7. Raising standards unrealistically
8. Not using multiple interviewers
9. Not having a “subjective” process in place
10. Not checking references and backgrounds

In questioning the attendees of many of my lectures I ask for a show of hands of who has a written job description for their position. On average about 5% – 10% respond affirmatively. So by extrapolation we can determine that 90% of the people working in those veterinary hospitals did not truly know 100% for certain exactly what the manager or owner expected of them.

Our starting point therefore must be the job description. Creating job descriptions seems to be a cause for great distress in practice owners and manager. The common response is always, “I don’t have time”. Good news – you don’t have to have time. The best way to create a good job description is to allow the staff doing the work WELL to create the list of tasks, behaviors and motivations necessary to shine in the position. Be aware that a good job description is more than a list of duties. It is the tool that we begin with to screen applicants for “fit”. You must understand your company’s values, goals, and unique
Veterinary hospitals are very much like people – they all have distinct personalities. The job description is the place where we tell people what we expect. Not just task performance but also behaviors. A mistake often seen in employee manuals is all the “don’t do’s” – don’t park here, don’t wear that, don’t say this. Both employee manuals and job descriptions should say “here is what we expect”. We expect every day you will have a positive attitude, a willingness to learn, you will give great client care, you will exhibit strong team work skills, you will be punctual and respectful of your fellow team members, our clients and our patients. Never assume people know what to do, especially if this is an entry level position or someone’s very first job. When you ask your top performers to help you create job descriptions ask them to look at three things.

1. **The Skills Needed**
2. **The Behaviors needed to be exceptional**
3. **The Motivation needed to WANT to be exceptional**

A good job description lets new hires know what to do from the very first day and makes it simple to eliminate the unqualified applicant. A job description should include:

1. **The Heading** – The Title of the JOB
   i. Reports to who
   ii. Location(s)
   iii. Salary grade
   iv. Exempt or non-exempt status
   v. Travel requirement
   vi. Date
2. **The Purpose** – The Objective – why does this job exist
3. **Duties and Responsibilities** – This is the LIST of skills, tasks, behaviors required to shine (Also Called Core Competencies)
4. **Qualifications** – Certifications, Educations, Licenses, Time on the job
5. **Other** – physical requirements, how success will be measured, the work environment (busy or laid back), team and company VALUES

The Nielson Group offers a good list of Core Competencies as does the Virginia Government and Workforce


**List of Core Competencies**

- Adaptability
- Analysis
- Attention to detail
- Assertiveness
- Collaboration
- Commitment to task
- Conflict Management
- Control
- Creativity
- Crisis management

- Listening
- Logic
- Negotiation
- Oral Communication
- Persistence
- Persuasiveness
- Planning an Organizing
- Presentation
- Priority Setting
- Rapport Building
- Resiliency
- Resourcefulness
- Customer Focus
- Deadline Responsiveness
- Dealing with Change
- Decision Making
- Delegation
- Fact Finding
- Follow up
- Goal Orientation
- Impact
- Independence
- Initiative
- Integrity
- Interpersonal Skills
- Leadership
- Learner Attitude

- Problem Solving
- Risk Taking
- Sensitivity to Others
- Staff Development
- Strategic Planning
- Team Building
- Team Work
- Technical and Professional Knowledge
- Time Management
- Tolerance for Stress
- Versatility
- Work Standards
- Written Communication

### Example of Job Competency Chart

<table>
<thead>
<tr>
<th>SKILLS</th>
<th>REQUIRED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Technical</td>
<td>Computer data entry Knowledge of keyboarding, Microsoft Word, Microsoft Windows, appointment scheduling software</td>
</tr>
<tr>
<td>Telephone</td>
<td>Proficient with multiline phone systems, voice mail and transferring calls</td>
</tr>
<tr>
<td>Customer Service</td>
<td>2 years working in a CS position in a customer facing job such as hospitality or retail</td>
</tr>
<tr>
<td>Behavior</td>
<td>Interpersonal skills Ability to build a positive Experience for Clients by utilizing emotional intelligence to gain trust</td>
</tr>
<tr>
<td>Conflict Management</td>
<td>Ability to solve client problems And retain strong relationships</td>
</tr>
<tr>
<td>Motivation</td>
<td>Integrity Earns and maintains the trust Of both team members and Clients</td>
</tr>
<tr>
<td></td>
<td>Learner attitude Seeks self-improvement Through multiple means in Desire to help the clients and The company</td>
</tr>
</tbody>
</table>
Now that you understand the groundwork behind defining who it is you seek it is time to discuss the process of advertising and interviewing.

**How to Advertise and What to Say.**
Working in a veterinary hospital is difficult, labor intensive, sometimes frustrating and most of the time rewarding work. We are fortunate that so many people love animals and desire to work with them for a living. We are also unfortunate that we sometimes attract the unrealistic, off the beaten path, animal hoarder wackos. The challenge is to advertise in a way that will pre-screen and eliminate many of the undesirables and attract the wonderful.

Online job sites have taken over from the traditional newspaper advertisements and this is fortuitous for hiring managers. We now have a country wide pool of potential hires where before we were limited to our local area. Still advertising locally is a good option for entry level staff such as kennel attendants or part time CSR’s. Most of the time hires for these staff positions are not able to move to take a job. The solution is a locally focused online site.

The ad’s ability to give better job duty details, to post drug and background check policies and to describe company culture all help pre-screen candidates. I encourage all hospitals to become a “Drug Free Workplace” for multiple obvious reasons. One huge plus is you will automatically eliminate anyone who knows they can’t pass the entry screen or subsequent random tests. Posting in your ad that you perform criminal background checks also screens out undesirables.

Ads in many online sites will allow you to also require a pre-application skills test. These tests can be spelling, word definitions, math equations, “making change” in cash situations and even essays on how the applicant cares for their personal pets. The essays show written communication skills. If your candidate can’t write proper sentences, then what will you get in your patient histories?

**Good Job Posting Sites**
Indeed.com
“Cityname”helpwanted.com example (Atlantahelpwanted.com)
WhereTechsConnect.com
Craig’s List
ZipRecruiter.com (posts your ad to multiple job sites for you)
VHMA.org (for hospital managers, CVPMs)

**Recruiting Firms**
[www.myveterinarycareer.com](http://www.myveterinarycareer.com) Powered by AAHA (AAHA accredited hospitals get a discount)
Veterinarians
Many Practice Management consultants offer interviewing services – Debbie Boone 2 Manage Vets

**SOCIAL MEDIA**
Recently recruiting though social media has come to be a new resource for practices. Use caution when hiring clients – especially really good ones. If they don’t work out you lose twice – your hire was a mistake and your client leaves the practice because it is too uncomfortable to return. Still if your team is
happy in their work environment and loves the practice they can be your own private recruitment force. Most technicians know other technicians. Perhaps they were in school with some or worked in another practice with others. They could have met at a continuing education meeting. Your front office team probably knows other CSR’s who are both animal and people lovers. Your veterinary associates keep up with classmates and former colleagues. Ask them to use their contacts to help you find the perfect new hire. Realize that people don’t refer people they think will not do a good job or be a good fit because it reflects poorly on them and their ability to judge people.

It is suggested you create a good ad discussing the job, core competencies, the mission and values held dear by the team and the basis of the schedule such as weekends, holidays, evenings, etc. Also the physical demands of the work, the abilities and skills needed and the ideal behaviors necessary to do the job well should be laid out. Recent studies are showing increased use of social media to generate candidates and that the candidates are more qualified and culturally suited to the position. *(Harvard Business Review)* The results are less turnover and happier new hires and existing team.

**SAMPLE AD: Customer Care Representative**

Full Time Energetic, outgoing Animal Lover needed for a customer care position assisting our wonderful clients and their pets. Hours are Monday – Thursday 1 – 6 pm and every Saturday 9-5. Pay range $13.50 - $15.00 depending on experience level.

Ideal candidate will have outstanding communication and customer service skills, 2 years minimum experience in a customer service field with both telephone and face to face client interactions. Veterinary experience is a positive but not required. Professional appearance and dress along with good manners and strong social skills are important. The desire and proven ability to give over the top service, resolve client conflicts and build relationships with new and existing clients is central to good performance.

Chosen candidates will join a team dedicated to preventative health care, client and patient pampering, state of the art veterinary medicine and supporting each other. Benefits include continuing education, step by step training, room for advancement, 401 K, Health Insurance, PTO and a pleasant work environment. ABC Animal Hospital is a DRUG FREE workplace and utilizes background screening from 3rd party providers.

Having a great team of patient and client champions is our goal. If you would like to be a part of something special, please apply online by filling out the following application _________link______ and taking the short skills evaluation. ABC Animal Hospital is an Equal Opportunity Employer.

**ALWAYS HIRE “UP”**

*“You give me the right people and I don’t care what organization you give me. Good things will happen. Give me the wrong people, and it doesn’t matter what you do with the organization. Bad things will happen.”* – Colin Powell

For links to helpful resources visit  [www.dboone2managevets.com](http://www.dboone2managevets.com) and look at our resources page along with recommended reading lists.

References: “How to Choose the Right Person For the Right Job Everytime” Lori Davila & Louise Kursmark copyright 2005
THE BEST STAFF- Part II- FINDING & INTERVIEWING

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In part 1 of this series the basics of creating a good job description, developing core competencies lists and how and where to place ads was addressed. In this section we will discuss the actual interview process.

There are basically 3 types of interview Questions: Traditional, Situational, Behavior Based. Each type of question has its place in the process but the most effective to delve into the way candidates truly will behave and perform is the behavior based question.

Traditional questions are typically used to open conversation with a candidate. They are useful to relax a nervous interviewee and gain some insight into how the person views themselves and their past performances. Examples:

1. Tell me about yourself
2. What are your greatest strengths and weaknesses?
3. Where do you want to be in 5 years?
4. What have you done to improve your professional skills this year?
5. What are you seeking in compensation?

Situational Questions require the candidate to use their imagination to place themselves in a situation and tell us how they plan to react. Situational questions are helpful to rule out unqualified candidates who would be unsuitable due to their lack of understanding of how the position should work. If we are interviewing a technician and ask about a common medical situation she/he would typically encounter and the candidate has no idea or understanding of the proper response, then we know they probably have overstated their skills. On the other hand, if they are “spot on” we can figure they do know the job. The candidate could also create an answer that is what they imagine you want them to do in this situation. Without further investigation when it is time to actually do the job their performance may be completely different from what they told you in the interview.

Examples of Situational Questions:

1. How would you describe your ideal position?
2. What would you do if a fellow team member was not contributing to the work load?
3. How would you handle an irate client?
4. How do you deal with stress on the job?
5. What would you do if you were asked to do something that was outside your job description?
Behavior based interview questions help us to determine how an employee will behave based on how they behaved in the past. Past behavior is the best indicator of future performance. All behavior based questions ask the candidate to tell us a STORY. With behavior based questions we avoid asking anything that could be answered with a simple “Yes” or “No” much in the same way we “interview” clients about their pet’s condition. If we learn to ask the right questions we can get past the rehearsed responses to potentially “see” how a person will act in the future. All questions should be designed to elicit a “SAR” response. They should tell a STORY resulting in an ACTION creating RESULTS.

As you question the potential hire you are soliciting a DETAILED story. Our work is to uncover how they reacted in situation that will commonly occur during their working day. Perhaps we have determined that a required core competency for an RVT is the ability to persuade clients to accept necessary services for their pet. She must have good communication skills and be able to find the proper descriptors necessary to let the client see value. A good question to determine these competencies would be, “Tell me about a time when you had a patient who needed medical treatment and the client was reluctant or refused to allow you to perform them?” The candidate should be able to relate a story with specifics such as the client and patient name, what services were being discussed and what the client said. As the story unfolds we then question further – “Tell me the outcome” “What process did you use to get her to change her mind?” “What happened to the patient?” “What did the client think after the treatment was over?”

You can see these questions show EXACTLY how this situation was handled in the past and gives strong indication of how the same or similar situation will be handled in the future.

Behavior Based Interviews also show candidate weaknesses. Perhaps this technician has very impressive technical skills. She may even have advanced credentials. But, you discover on interview her people skills are lacking and in your hospital you highly leverage your RVT’s to educate clients and deliver treatment plans. Unless we plan to change our protocols or have her work as a surgical tech she is not a good practice fit.

One of the keys to a successful interviewing process, and hire, is to know when to utilize each type of interview question throughout the process. Our process starts first with the creation of the job description and then job ad, as we discussed in our last session. Not until we have a clear picture of what we need/want, and our expectations, can we hope to find the right candidate. Once the foundation has been laid, we can begin the actual process.

Our first step, upon receiving a candidate’s resume, is to review the resume looking for red flags and formulating our strategy for future interview questions. Red flags that always catch our eye can be:

1. Gaps in employment
2. High turnover
3. Self-centered “objectives” or “profiles”
4. Lack of results-oriented detail
5. Lack of numbers
6. Poor formatting/grammatical errors
Assuming we are interested in a candidate based on her resume, we typically respond asking for a cover letter, if one was not received, as well as sending out a basic written interview form. The goal behind the cover letter is three-fold:

2. We want to see if the candidate is serious enough about the position to take the time to write a cover letter. There is generally not a shortage of candidates looking for work and we want to make sure we hire someone who wants to work for us, not just someone looking for a job
3. Any candidate seeking a job should be looking for ways to add value to the practice. The cover letter gives the candidate the opportunity to research our practice and pitch how their unique skills/interests will benefit the clinic, clientele and staff.
4. A well written cover letter can also serve as a good prompt for the phone interview assuming we decide to move to that step

The “pre-screen” form we send out serves some of the same purposes as the cover letter. We want to see if the candidate is serious enough to answer the three to five questions we pose and we can use those answers as fodder for additional questions. A few examples of questions we use are:

1. Why are you looking to make a career change?
2. What do you most enjoy about veterinary medicine and what aspect(s) do you find the most rewarding? Important?
3. Have you been in a situation when you thought it necessary to convince your employer that a medical standard of care needed to be upgraded or changed? If so, please describe.
4. What are your long-term goals and where do you see yourself in 5 years?
5. To help us better understand your production history and salary requirements, please share what you produced in gross revenue last year (not salary, but gross revenue for the practice):

Requesting the above two documents help us reduce wasted time spent on unqualified or uninterested candidates.

If we like a candidate our next step is a general phone interview. We make our first phone interview fairly general and focus more on the candidate’s experiences, interests, strengths and goals, and less about job specifics.

If the candidates perform well during the first, general phone interview, we move to a second interview, either in-person or via Skype. Nothing beats face-to-face interaction and since it is not always economical to bring a candidate in to the clinic early in the process, a Skype interview can serve as a great conduit for reading body language, watching eye contact, etc. This interview is more practice/position specific and utilizes more behavioral based questions.
Once it is determined that the candidate is a possible fit it is time to proceed into a face to face interview. It is in the face to face interview that our skills in body language reading will help enable us to discover our ideal new staff member.

As stated above behavior based interviews require our candidate to tell us a DETAILED story. Most of the time these stories are true because they are based on memories rather than imagined situations. But, true hucksters understand how to make up believable stories and may even have some prepared as they go into the interview.

The knowledge of non-verbal communication can greatly assist us when faced with a deliberate falsehood or embellishment of the facts. 53% of all job applications have some false information on them (SHRM 2003). You must make a concerted effort to observe body language to succeed in hiring right. It is imperative that you be seated so you can see the interviewee’s entire body. An across the table interview is not as effective as one seated at an angle to the candidate in the open.

Interviews are typically a nervous situation for people but a skilled interviewer can break the ice with traditional questions and relax the candidate. It is important that the interviewer be relaxed because only when we are calm can we be truly observant. When we are upset or nervous our limbic brain takes over and we narrow our field of vision to a protective level of focus. We must be subtle in our surveillance or the reactions won’t be true.

During the interview we should look for a baseline of movement. This could be a mild foot jiggle or a pacifying tie adjustment. But when the foot begins to kick more vigorously and the necktie adjustment becomes a ventilating collar tug we should circle back to the question that elicited this behavior and see if we can discover the trigger. Perhaps you have asked a RVT candidate about her skill placing catheters. Up to this point she has been relaxed and smiling but suddenly her eyes flash closed and her hands rub down her legs as if drying them. These movements require more investigation of her catheter placement skills. The eye “shuttering” is a blocking behavior and the “leg cleanse” is a self-pacification behavior we use to calm ourselves when uncomfortable or distressed. You may find that though she has placed a few catheters that the doctors at her prior employment did most of them and she is out of practice.

In general, when we meet our candidate face to face we want to see a good first impression. They should be standing with good posture; their movements should be purposeful and confident. Their shoulders should be straight but relaxed and they should be balanced equally on both feet. The feet are the most honest part of our body. We are taught to control our features but not our feet. The limbic brain still takes over our tootsies. The candidate should give a genuine smile involving the “crow’s feet” muscles around the eyes. When those muscles are not involved the smile is fake. Since we count on our team to react well in emergencies we want to see “calmness” in gestures - not wild gesticulations. Whether at the front desk, in the exam room with clients or in surgery we want to “make it look easy”. The SWAT teams for the FBI call this “smooth is fast” reactions.

The voice tone is also a non-verbal. Is this person using verbal filler phrases as in, “uh, like, you know” and is the tone pleasant and positive or negative with poor attitude? How are their
manners? Here we are not talking about what fork to use at a dinner party but how do they greet others? Do they have an awareness of how the people around them are acting? Do they offer to shake hands? Do they make good eye contact? Where are their hands? If they are hiding in pockets this is a sign of standoffishness. Giving a tour of the hospital and introducing the interviewee to the team is a good way to judge how they react to others – and how they will react to your clients.

How are they groomed? Many people are under the impression that they don’t have to look good to work in an animal hospital. However, we are professional medical facilities where gaining the trust and respect of our clients is the key to giving our patients great care. Clothing should be clean and pressed; hair should be neatly cut and styled with no unnatural colors or styles that hang in the face. The fingernails and hands should be neat and well-manicured. Excessively long false and natural nails should not be allowed per your dress code. They are inefficient, look as if they would hold germs and do not belong on medical care personnel. People should not be wearing excessive jewelry such as multiple rings on every finger and earrings lined up the ears. I realize this is a common part of our culture but to the consumer it states lack of professionalism. Tattoos are another issue. Many companies require tattoos be covered at all times. Multiple tattoos may be a part of your practice culture and your clientele’s culture, if so take that into consideration, but if it is not then when you hire make sure to observe tattoos and their display – or lack thereof - as part of your dress code. Perfume is another non-verbal flag. Many people are allergic to perfumes and professional candidates will not arrive at your office smelling as if they are heading on a date.

As you question the applicant monitor things like how much they handle their cell phone. Will they be able to work without it constantly in their hand? If so it should not be visible on their interview. Do they use slang, or mumble as they answer? Remember they will be viewed by your clients and must make a good impression.

List of behaviors to watch:

1. Eye Blocks – lower eyelid tension, rapid blinking, or touching the eyes or forehead – sign of discomfort
2. Tucked chin – lack of confidence
3. Lip biting – anxiety
4. Lip compression – the lips disappear - negative emotion like anger
5. Lip licking or tongue movements – self pacifications
6. Blowing out air – releases tension (do this with upset clients and they will tend to mirror your calming breathes)
7. Clothing adjustments – tugging on shirt collars, playing with a watch or pendant on a necklace, rubbing the neck or tie are self- pacifications
8. Pulling a jacket or sweater closed – blocking behavior (when someone hears something they don’t like they use this behavior)
9. Rubbing legs, crossed arms with finger pressure, rubbing palms or interlaced fingers together – self pacifications
10. Hands that disappear or freeze in place. This indicates that limbic “flee, freeze or fight”
response
11. Hands with open palms asking to be trusted or believed
12. Legs crossed away from you to block you out, sudden increase in foot kicking or jiggling
or a dead stop in foot movement (freezing)
13. The voice – hesitant or monotone speech could be rehearsed, clearing of throat or a
quaver in the tone indicates stress

**Emotion always overrides logic.** Having multiple people interview a candidate is a necessity
to avoid what is commonly called the “halo” effect. We interview a candidate, find some
common ground like our college or a breed of dog and then the candidate becomes “like us”.
The problem is they are not – only in where they went to school or their choice of pet -so always
have others interview the prospect. Have a preset list of questions similar to the ones you
asked. You are looking to see if the same answers are given every time.

The best use of non-verbal communication is to gather intelligence that is useful to further
investigate the issue or remark that caused the body to make a change in baseline behavior –
not to discover a lie. The very best FBI profiler was only accurate in discovering deception 60
percent of the time.

Once you have interviewed and are ready to make an offer it is time continue the process with a
background check and a drug test. The background check can be done in house by a simple
Google search and the state criminal records website. The problem is that many state, county
and city databases do not link to federal information. Background screening companies are
able to seek information across all these fields.

You may question the need for background checks with part time or lower paid staff but the
reality is that is who steals the majority of cash and products in the veterinary industry. Also this
will allow you to protect your company from a negligent hiring lawsuit. If you hire someone that
becomes violent and they harm another employee you are responsible. This is not just physical
harm but also verbal damage like bullying. Background checks can be performed by private
investigators, companies specializing in employment screening, online background check
specialist or the employer. As the employer you must determine if you have time to do the work
involved in the screening.

If you chose to hire an outside source look for accreditation through the National Association of
Professional Background Screeners (NAPBS) which means the company has passed an on-
site audit and complies with all areas of background check standards, protection and practices.
Confirm that the company you choose will be able to offer comprehensive coverage –
sometimes in foreign countries. Some screeners are focused on certain industries so those
comfortable with medical and veterinary checks are ideal.

[www.veterinarybackgroundchecks.com](http://www.veterinarybackgroundchecks.com) is an example.
The Fair Credit Reporting Act gives information about what employers can and cannot do when conducting a background check. The link below will give you complete information.

http://www.consumer.ftc.gov/media/video-0026-employee-background-checks

Keep in mind that states vary by what you can seek on a background screening.

You are allowed to check:

- Driving Records
- Bankruptcy
- Military Records
- Drug Testing Records
- Sex offender lists
- Credit reports
- Court Records
- State Licensing Records
- Past Employers
- Criminal Records
- Personal References
- Social Security Number

You CANNOT check – Bankruptcy after 10 years, Records of arrest after 7 years, Accounts placed for collection after 7 years and Facebook or other social networking passwords.

Just because you can check all these things doesn’t mean you should. If your employee will drive a company vehicle you will want a driving record. If you are hiring a customer service person you would check credit reports and criminal history. Fifty five percent of the cash stolen from a hospital is taken by the front office.

According to the 2013 HireRight Small Business Spotlight Report about 95% of small businesses now do some type of background checking for potential hires. This statistic jumped 14% since 2012. The most popular background check is criminal records (90%) followed by identity checks (87%) and employment verifications (55%).

Once your candidate passes the background screening it is time to see what they can do in the hospital. Working interviews are PAID and shadowing interviews are not.

Following the initial face-to-face interview, we bring our remaining top picks in for a shadowing or working interview. There is a difference between the two and it’s important to use the nomenclature correctly. Depending on the type of practice and the role of the candidate (doctor, manager, tech, etc) the layout of the working interview will vary, but regardless of the position, we still look for some common themes:

1. Candidate’s interaction with the staff (not just the owner!)
2. Candidate’s comfort with the clients
3. Candidate’s engagement/willingness to participate
4. Quality of questions
5. Pertinent technical skills
6. Core competencies (as discussed in the previous section)

The ultimate goal of the working interview is to determine not only if the candidate has the appropriate technical competencies, but just as importantly whether or not she will fit within your practice’s culture, medical philosophies and clientele.

We’ve described what probably seems like a fairly in-depth screening process, but putting in the extra time and work on the front end will pay off in spades when compared to the costs, both hard and soft, of replacing a poor hire.

Resources:

“How Every Body Is Saying” by Joe Navarro
“How to Choose the Right Person for the Right Job Every Time” by Lori Davila and Louise Kursmark
“Non-Verbal Communication Science and Applications” – David Matsumoto, Mark Frank, Hyi Sung Hwang
“Louder Than Words” – by Joe Navarro with Toni Sciarra Poynter
THE BEST STAFF PART III: PERFORMANCE REVIEWS
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What is a Performance Review?

“A formal record of a Manager’s Opinion of the Quality of an Employee’s Work”

Dick Grove, “How to Be Good At Performance Appraisals”

Informal – given on a regular basic as work is performed. Can be up to a boss, down to a direct report or cross organizational to a fellow manager. In the moments feedback.

Formal – Annual or Semiannual performance assessments. Designed to summarize all informal evaluations given throughout the time period.

At the beginning of our evaluation cycle we sit down with our worker and discuss goals, expected behaviors, and key job responsibilities.

Goals are best for MEASURED areas such as: What can we do better, what can we do cheaper or what can we do faster.

“Setting specific, difficult goals consistently leads to higher performance
Goals are energizing. High goals generate greater effort that low goals
Tight deadlines lead to a more rapid work pace than long deadlines
Making a public commitment to a goal enhances performance
Whether set mutually or by the manager doesn’t make much difference in achievement”

Professors: Edwin A. Locke & Gary P. Latham

The GOALS GRID:

1) What do we want to achieve – or what do we want that we don’t have
2.) What do we want to preserve? - what do we already have?
3.) What do we want to avoid? – or what don’t you have that you don’t want?
4.) What do we do now that we want to eliminate? – or what do we have that we don’t want?

The best way to discover these KRA’s or Key Results Areas are to ask your team members. Let your techs define their 3 -4 areas, receptionist should do the same.
Humans have a tendency to do what they like and avoid what they don’t. By defining only 3 – 4 areas of responsibility we help our team member focus on what is important in their job. They also know we will be monitoring performance in all those areas so avoiding one of them will reflect on the evaluation.
There are 3 kinds of competencies – CORE, Job – Family and Job – Specific.

Ask yourself:
What would a true role model do?  
What would be an example of skill mastery?  
What would make you say – wow!

10 Core Competencies of High Performers
   Ability to Prioritize
   Works well in teams
   Organizational Awareness
   Effective Problem Solving
   Self-Awareness
   Proactivity
   Ability to Influence
   Effective Decision Making
   Learning Agility
   Technical savvy

Calendar Driven
- Start of Year - either calendar or employee (recommended)  
- 3 months update – 90 day touch back to see if goals are being met
- Midyear – To make adjustments to goals if necessary

Event Driven
- Completion of major project
- having job issues
- expressing frustration
- unusual moods or behavior lasting several days

It is important to Remain Unemotional and Objective
It’s about the JOB – not the person

YOU are the COACH
Give advice for improvement
Provide guidance to develop skills
Give support when needed – don’t hover or take over
Instill confidence so they can manage themselves
Help them gain competence so they can grow and move up

COMMON ASSESSMENTS
Improvement of performance
Attendance
Attitude
Communication skills
Company orientation
Task focus
Stress management
Team work
Integrity
Asks for help
Job knowledge
Productivity
Quality of work
Dependability
Ambition

Be aware of the Fundamental Attribution Error – this simply means as managers we tend to attribute performance problems to **character** rather than **situations** in the workplace.

What should be on the Evaluation Form?

Behavior and Results
Goals
Comments
Rating Scale
Scale definitions
Space for employee comments
Development plan

When Rating the Individual
Comparison with Super Stars
Who ranks with them
Who is in the middle
Who falls behind
HOW FAR BEHIND?

**Rating ERRORS:**
- *Malcolm in the Middle*
- *Angels or Demons*
- *Just like Me*
- *Now or then*
- *Hired or Inherited*
- *Pretty is as pretty does*

- *Dick Grote*
Rating Scale
5 - Excellent or greatly exceeds expectations
4 - Exceeds Expectations/Superior/ Above Standards
3 - Meets expectations/ Fully Successful or Satisfactory
2 - Substandard, Needs Improvement
1 - Unsatisfactory

Meeting with 3 – 5’s
Gather materials
Location
Time
Send copy to employee 2 days before
Allot 45 – 60 minutes
3 things done best – 2 things need improvement

Meeting with 1 - 2’s
Location- your office POWER
No copy before hand
Time – possible day end
No positives
Direct and to the point

If you are judging your receptionist a 2 on customer service, then you should provide two strong examples of this lack of client care. They should be documented as to date and time, perhaps you had some informal discussions along the way that your employee signed off on. Consider it to be like building evidence in a court case – pro or con.

THE GOAL
To obtain the employees understanding – not their agreement

Paul Falcone – VP of HR at Time Warner Cable has a model which states:
“State your precise concern, give specific examples that generated your perception, then close with a request for the employee’s reaction to that perception or with a specific request for change.

Baby Boomers ask for feedback to gain a prize – to win – to be judged on performance – one or two times a year works for them

Generation X – feedback also relates to judgment but the results are not to gain a prize but to gain a longer leash or more freedom to operate in the way they prefer
**Generation Y** – Are constant learners because they live online – and reach out to friends, family and resources for on demand knowledge. They start a task then see a need for more information and seek it out then move on. This may happen multiple times a day. So to Y feedback means I want to learn more. …not be judged or assessed. Y’s want to be taught. They want FEED FORWARD. If they don’t get it they believe you don’t expect much of them.

**Typical $$$**
1- 2 rating  
Raise of 0 – 1.5%
2.5 – 3.8 rating  
Raise of 2 – 3.5%
4 – 5 Rating  
Raise of 4 – 5 %

Money is oddly enough not a big performance motivator. In fact, base pay adjustments only improve performance by 3.8 percent while annual bonuses only effect performance by 2%. Why? The rewards are too far away from the actions. This is why immediate praise for good performance is so important.

The real job of a manager is to be a good steward of the resources of the practice. Often we have the dilemma of the excellent employee who has reached the top pay level for their position. They perform at 3.8 – 4.5 level consistently but the reality is the market determines the pay rate for the top of the scale. Just because a performance may be worth more does not mean the JOB is worth more. This is when we must be creative as managers. Can we offer a better shift? Can we be more lenient on time off allotments? Can we promote in title or responsibility? Or even better what can the employee do to bring more value to the job so he or she can be paid more.

**Resources:**
“How to Be Good at Performance Appraisals” Dick Grote

“f you Have Employees, You Really Need this Book” Jerry Osteryoung, Ph.D. & Timothy J. O’Brian, M.S.
BEST STAFF PART IV: PERSONALITY MATCHING
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Personality profiles are certainly not a new concept. The basic theory behind the four quadrants of DISC Personality Styles actually can be traced all the way back to 444 B.C.

The History of DISC began with the elements of Fire, Earth, Air, and Water. The theory behind these four quadrants of personality style was originally written by Empedocles in 444 B.C. He recognized that people seemed to act in four distinctly different ways but he believed it was external environmental factors that affected us. Astrologers still use these elements today in their predictions. By 400 B.C. these four “earth signs” had shifted to internal factors when Hippocrates defined them as Choleric, Sanguine, Phlegmatic, and Melancholic, and called them the 4 Temperaments. DISC profiling as we know it today has been around since 1928 when William Marston published his book “Emotions of Normal People” (he also created Wonder Women the comic strip character). In 1940, Walter Clark took Marston’s theory and developed the first DISC Personality Profile. Even with its long history many people outside the psychological community are not familiar with this extremely useful tool.

Let’s begin with the basics. First of all Wikipedia defines personality as “the totality of somebody’s attitudes, interests, behavioral patterns, emotional responses, social roles, and other individual traits that endure over long periods of time”. There are four distinct personality types used in DISC profiling:

- **Dominance** – relating to control, power and assertiveness
  (Note: Sometimes the word Drive is used in place of Dominance)
- **Inducement** – relating to social situations and communication
  (Note: Sometimes the word Influence is used in place of Inducement)
- **Submission** – relating to patience, persistence, and thoughtfulness
  (Note: Sometimes the word Steadiness is used in place of Submission)
- **Compliance** – relating to structure and organization
  (Note: Sometimes the words Caution or Conscientiousness are used in place of Compliance)

Details of Personality Styles

**People with the D** style place an emphasis on shaping the environment by overcoming opposition to accomplish results.

A person with a D style is motivated by winning, competition and success. They prioritize accepting challenge, taking action and achieving immediate results. They are described as direct, demanding, forceful, strong willed, driven, and determined, fast-paced, and self-confident. They may be limited by lack of concern for others, impatience and open skepticism.
may fear being seen as vulnerable or being taken advantage of. values competency, action, concrete results, personal freedom, challenges. Hates wasted time and touchy feely behaviors

Goals:
unique accomplishments
new opportunities
control of audience
independence

Needs others who:
weigh pros and cons, calculates risks, use caution, research facts, deliberate before deciding, recognize the needs of others

When communicating with the D style individuals, give them the bottom line, bullet points are well accepted, focus your discussion narrowly, avoid making generalizations, refrain from repeating yourself, and focus on solutions rather than problems.

Team Placement –
D personality dominate people are well placed in positions that need decisiveness – they will do well as surgical techs, emergency triage, managers overseeing lower level managers – for example Hospital Administrators who supervise Office Managers, Head Techs or Kennel Managers who have more soft skills and people orientation. You will often find these people as practice owners because they are brave and willing to take a chance and a challenge to achieve big results. D personalities are good in task oriented positions where speed is more important than attention to detail because their goal is to finish the task and check it off a list and move on. If they had a “tag line” it would be “Get ‘er Done!”

People with the C style place an emphasis on working conscientiously within existing circumstances to ensure quality and accuracy.

A person with a C style
is motivated by opportunities to gain knowledge, showing their expertise, and quality work.
prioritizes ensuring accuracy, maintaining stability, and challenging assumptions.
is described as careful, cautious, systematic, diplomatic, accurate and tactful.
may be limited by being overcritical, overanalyzing and isolating themselves.
may fear criticism and being wrong.
values quality and accuracy

Goals:
unique accomplishments
correctness
stability
predictable accomplishments
personal growth
Needs others who:
Delegate important tasks, make quick decisions, use policies only as guidelines, compromise with the opposition, state unpopular positions, encourage teamwork, initiate and facilitate discussions

When communicating with the C style individual, focus on facts and details; minimize “pep talk” or emotional language; be patient, persistent and diplomatic.

Team Placement –
C personalities are well placed in positions that require attention to detail such as inventory management, Laboratory technician, and bookkeeper. They are data gatherers and do well in nitpicky work. They are great organizers and should be utilized for tasks such as scheduling staff, writing manuals, shopping for equipment and researching data plans. They will not be happy in fast paced areas where they do not have time to ponder decisions. When extreme in their style they may have decision gridlock which can be paralyzing and inefficient. These people hate making a mistake and use gathering information to avoid it.

People with the - I style place an emphasis on shaping the environment by influencing or persuading others.

A person with an I style

is motivated by social recognition, group activities, and relationships
prioritizes taking action, collaboration, and expressing enthusiasm
is described as convincing, magnetic, enthusiastic, warm, trusting and optimistic
may be limited by being impulsive and disorganized and having lack of follow-through.
may fear loss of influence, disapproval and being ignored
values coaching and counseling, freedom of expression and democratic relationships

Goals
victory with flair
friendship and happiness
authority and prestige status symbols
popularity

Needs others who:
concentrate on the task, seek fact, speak directly, develop systematic approaches, prefer to deal with things instead of people, take a logical approach. demonstrate follow-through

When communicating with the I style individual, share your experiences, allow the I style person time to ask questions and talk themselves, focus on the positives, avoid overloading them with details, and don't interrupt them.
Team Placement

– I Personalities are well placed in positions that deal with people. They will do well at the front desk and as a technician or assistant. They can be frustrating as a doctor because they lose focus and writing through records is too tedious and boring but they will be able to influence client to accept services with their persuasiveness. They are creative and do well in marketing areas such as monitoring your team Facebook page, thinking of community service activities, going to events to promote the practice such as visiting schools or groups. They do well at the front desk and in client education. They do lack a strong attention to detail and are bored with tedious tasks. Working alone is misery for I personality. Their natural charisma makes clients drawn to them and builds strong relationships and trust. Place them in areas that move quickly and that have many diverse tasks that keep their attention. When very strong in this type they can be “flaky”.

People with the S style place an emphasis on cooperating with others within existing circumstances to carry out the task.

A person with a S Style

is motivated by cooperation, opportunities to help and sincere appreciation
prioritizes giving support, collaboration and maintaining stability
is described as calm, patient, predictable, deliberate, stable and consistent.
may be limited by being indecisive, overly accommodating and tendency to avoid change
may fear change, loss of stability and offending others.
values loyalty, helping others and security
hates cold and unfeeling behavior

Goals:
personal accomplishments
group acceptance
power through formal roles and positions of authority
maintenance of status quo and controlled environment

Needs others who:
react quickly to unexpected change, become involved in more than one thing, are self-promoting, apply pressure on others, work comfortably in an unpredictable environment, help to prioritize work, are flexible in work procedures

When communicating with the S Style individuals, be personal and amiable, express your interest in them and what you expect from them, take time to provide clarification, be polite, and avoid being confrontational, overly aggressive or rude.

Team placement –
S personality type will do well in people oriented positions like the front desk. They are often the “glue” that binds a team together because of their kindness and loyalty. They are good
client educators because of their natural patience with people. Chances are good they will not be the innovators in your team but can be counted on to follow protocols, protect the hospital and always show up for work. They will do well in kennel care and in lower management positions where steadiness is more important than creativity. As a veterinarian they are often best in charitable hospitals because they tend to want to give services and save the world more than generate income for a business.

Most people have two major personality types although some will be extreme in their dominate type which can eclipse their secondary types. As people gain life experience they tend to learn to temper their natural tendencies in order to accomplish harmony with others. This is called emotional intelligence.

Refer to the core competencies in my proceedings part 1 of “Best Staff” to match personality type with the competencies you determine are necessary for the positions on your team.

If you want to get a free profile just go to the following link for a free report. You may also consider using this tool for potential new hires.

www.discpersonalitytesting.com

Resources:

www.discpersonalitytesting.com

www.peoplekeys.com
COMPLIANCE- HOW TO IMPROVE THIS REVENUE DRIVER

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Compliance. It seems as if we have been struggling with this subject forever – at least for the 27 years I have been working in practices. Why is this so difficult? In the days before computers it was more difficult to collect this data but now the majority of hospitals have a computerized data base. The problem arises when we don’t know how to correctly set up the service and diagnostic codes to allow us to pull this information with ease. There is the additional issue that we simply never look – only assume- we have good compliance. This leads to not only unhealthy and unprotected patients but a practice that is not performing up to potential.

We must start with the definition of compliance. In 2003 AAHA did a landmark study and formed the acronym CRAFT for compliance.

Compliance = Recommendation+Acceptance+Follow Through

The formula for compliance calculation is

\[
\% \text{ compliance} = \frac{\# \text{ of patients receiving a service or procedure}}{\# \text{ of eligible patients needing a service or procedure}}
\]

Measuring the number of dogs eligible for heartworm preventative is relatively easy – we just run a report on all canines in our database over the age of 1 year and 3- 4 months and then compare that to the number heartworm tests we have performed. The problem arises when we want to know how compliant our clients are on dental cleanings when we only have patient history to review. The solution is to set up diagnostic codes with dental grades which will allow us to query all patients with Grade 2, 3, or 4 dental diseases who have not received a dental cleaning in the last – 1 year or 6 months or whatever time frame we choose. When we don’t set up good codes the only way to retrieve this information is manually with a record review. It is possible to set up a patient log for compliance so when records are filed the staff can record the date, patient name, dental grade and whether or not they received dental care. These logs can be tracked and tabulated at the end of each month to show compliance on dental cleanings. No matter how you track compliance you must be consistent and use the same formula each time or your numbers will be skewed.

Occasionally manufacture’s sales reps will perform compliance audits for their products which are helpful but not always accurate. They will look at number of patients eligible and compare it to doses purchased. This purchased amount doesn’t necessarily mean this is the sold amount. You could easily have had stock left from a big buy in the year before or have significant stock still sitting in your pharmacy both of which will distort the compliance numbers. Someone could have stolen product unknown to you and caused you to purchase more. The only accurate number will be eligible patients / PMS generated sold inventory or service quantities.
AAHA is working with software companies to develop a standard diagnostic code just as they have developed a standard chart of accounts for financial reporting. There are companies that will mine this data for you by utilizing your practice service codes and benchmarking your compliance.

Why does this matter? You can’t measure what you don’t know. Measurement and paying attention to these figures is the first step in gaining compliance. To rally your team behind the mission of outstanding compliance goals must be set. Many practices set goals and push for only one area at a time where others work towards multiple goals at the same time. No matter what is decided staff training is the key to attaining success.

Let’s explore why we fail with compliance in order to overcome these issues.

1. **We don’t offer.** Perhaps veterinarians haven’t changed the perspective on the pet’s place in client’s lives and struggle to overcome the “they won’t pay for it” syndrome. In the 2003 AAHA compliance study when owners were questioned 90% wanted to be offered the best option for their pet no matter the cost. Dr. Marty Becker says pets have transitioned from “the barn to the bedroom” and yet many practices fail to realize this change of attitude. We are “furry children” pediatricians and when we realize that we will not fear the “no”. A good way to overcome this fear is to actually track how many “no” verses “yes” responses you receive when you offer best medicine. Human nature tends to remember only the bad and become gun shy of making offerings. You may also see when you track that some of your associates are much better at getting to “yes” than others. Track production for all doctors (whether you pay that way or not) and monitor the amount of medical progress exams, wellness panels, diagnostic panels, doses of preventative products and credits or accounts receivable per provider as indicators of good communication skills and quality medicine. These numbers can vary greatly within a practice with multiple doctors and indicate whether certain associates are following protocols. Monitoring service code numbers is the only way to follow up on what specific doctors are offering without following them into the rooms. When people know you are paying attention they tend to make greater effort to perform.

2. **We don’t monitor frequently.** Running these numbers once a year will not move the needle towards 90% compliance. Monitor monthly. Doctors reports as mentioned in (1.) should be run at the end of every month and given to them to review. Managers or owners should sit down with poor performers and question low numbers. You should also praise those high performers and perhaps have them coach struggling team members. Excitement and confidence in an area of medicine will often show in these measurements. A doctor who is “into” dental care will have a much higher production in dentistry than those who are not as enthusiastic. The same pattern holds for nutrition or geriatric care. This is unfortunate for the patient – and the practice.

3. **We don’t train.** This training pertains to owners, associates and lay staff. Building compliance is like laying a foundation for a fortress. Layer upon layer of offering your best care and constant client education creates a strong underpinning for patient health. But when our team is not a team – they are just workers going in various directions – we can’t accomplish our goals. First the doctors must establish consistent protocols.
more veterinarians working in a hospital the more often we have “opinions” about almost every aspect of medicine coming into play. We certainly don’t expect “cookie cutter” medicine but we must have minimum standards of care in order to develop training for our staff. When there is no reliable answer to the question “what does my pet need” the staff is confused and put in a precarious position. The response to this question should never be, “which doctor do you see” to be able to determine the answer. When these conditions exist the staff will not put themselves out on a limb with the chance of being wrong so they offer nothing. It is imperative that the staff be comfortable and “buys in” to the standards of care.

Once the protocols are determined – and a good base for care are the new AAHA Standards of Care – then staff training on the reasons for the protocols can be implemented. It surprises me how few hospitals really know how to train their team. Having an occasional staff meeting or a lunch and learn is not training. Good solid training is crafted with step by step phase training and then followed by testing. Many hospitals train but very few test. Without the test how will you know what your staff retained? The added value of testing is that it makes the training more important. A well trained team is confident in their knowledge and this is apparent to clients. When great confidence and “true belief” in the value of a product or service is perceived by a client their acceptance of that product or service will increase dramatically. Role play and developing scripts are vital in building smooth delivery of hospital offerings. Although everyone hates doing this there is no better way to learn than to practice. Hesitation to a client means you don’t believe what you are telling me – you only want me to spend more.

4. **We don’t write it down.** So, we all get together and agree on our standards but then we don’t take the time to produce information for our clients that relay this information. Clients retain only 20% of what they hear (10% of that is incorrect), 40% of what they see and 70 -75% of what they see and hear together. This is proof positive that the time spent in creating information for clients is never wasted. Few hospitals have a life plan for wellness for their patients. The Bayer Veterinary Care Usage Study revealed that owners did not think older pets needed as much care as younger pets when we know the opposite is true. Having a plan for care for the entire life of a pet is a key component in client education. Brochures, handouts, website information, You Tube videos, Facebook posts, pet portals utilizing email and text messages all support this written or visual information. Involve as many senses as possible in client education. Verbalizing an exam is a perfect example of increasing client involvement and understanding. When doctors don’t articulate the exam clients come to the front desk objecting to paying for the service. “She just petted my dog! I am not paying for that.” If instead the steps of the exam had been spoken and things like tarter shown, bad breath smelled, crepitus in the knee felt (by the client too), early cataracts observed and shown to the client the acceptance of not only the exam but a dental cleaning and perhaps some medication for the arthritis would occur. The cataracts would boost the acceptance of a senior panel because clients understand that cataracts come with age. Always give a report card. This is tangible proof of the exam and a reference for future care.
We should also write care plans down for the staff. This is part of a written training protocol and if our protocols change we must write the changes down, post them in the hospital, train the team and test them on the new material. Hoping everyone got the” memo” or “heard it through the grapevine” is not the way to improve your compliance.

5. **We don’t market our services enough.** In 2003 the AAHA study found clients don’t mind getting as many as 5 reminders yet most hospitals send 2 and think they are proactive. Send out reminders for core services, for senior panels, for preventative medications, for chronic medication refills, therapeutic diet refills, for NSAID and Phenobarbital panels and anything else you feel is necessary for the good health of your patients. Dr. Robin Downing said in a roundtable discussion on compliance that there are three things that influence the health of a pet. 1. Genetics 2. Environment 3. Nutrition. As medical team members we can have influence on both environment and nutrition and we should be working to gain compliance by motivating clients to listen to our recommendations and accept them. Measure your response to reminders. The addition of one more reminder will often generate as much as an additional 15% response. Utilize technology to reach your clients in the manner they wish to be approached. In a discussion with VetSource, the online pharmacy, the adherence to medications is greatly enhanced when it arrives at client’s home on autoship. This is a great opportunity to help our patients and our business with increased compliance. Mail information to the client about their pets condition – many manufacturers offer digital information that can be emailed to all the members of the household. When all residents of the house are educated we get better compliance. Tell your story in multiple forms such as messages on hold, notations on the bottom of your invoice, posters and bulletin boards in the hospital, the sign out on the street and through email newsletters. Retailers are messaging our clients daily and if we fail to keep in front of them with our message we will see them drift off to other providers.

6. **We don’t capture their return before they leave.** When training front office staff I will often ask, “How many of you make the following visit’s appointment before the client leaves?” It is the rare hand that is raised. Then I ask, “Do you make your dentist appointment, internal medicine appointment, eye exam appointment before you leave?” Almost universally the answer is “Yes.” What is the impediment in a veterinary hospital? Some excuses I hear are:” We don’t know the doctor’s schedule that far out, the client won’t agree, we don’t have time or it never occurred to us to try.” Commitment to return is a huge boost to greater compliance. A goal for appointment scheduling should be 70 – 75% of all eligible calls are converted to an appointment but when we have a client standing in front of us the goal should be 99.9% commit to their next appointment.

7. **We don’t praise good work.** As the pet care expert in our client’s world we fail to realize the importance our positive reinforcement carries. My mantra of managing teams is “what gets praised gets repeated” and it is equally valid for clients. If clients are compliant on certain areas of pet care then we should acknowledge and give them “Atta Girls (or Boys)” for doing the right thing. The utilization of a compliance audit form plays a role here (see handout). The client relations specialist or veterinary nurse will pull the patient record before the visit and review the history for compliant and non-compliant
care. When the client arrives appropriate praise is given for the good compliance and
time is spent in education about the necessity of the non-compliant care. These forms
can be additionally utilized to reward the team for overcoming client objections and
 gaining care in non-compliant areas by simply compiling the information on a simple
spread sheet at a slow time of day. Reinforce your staff’s good work educating and
 gaining client acceptance of our offerings with praise. Set team goals for in room service
acceptance.

8. We don’t follow up with calls. Some veterinarians hide from clients but nothing we do
is more impressive to a client than to have a doctor call to follow up. Obviously this is
not for every service but is certainly appropriate for a severe dental issue. In my work
with dental specialists I find that they always follow up a dental procedure with a
progress appointment about 1 – 2 weeks later. On this follow up visit the client’s
decision to have the work performed is reinforced by asking open ended questions about
the improved breath, eating habits and overall demeanor of the pet. During this visit
owners are instructed on home care with written recommendations for products,
instructions on brushing and dietary recommendations to keep the teeth clean.
Compliance on home care improves significantly. I find doctors trying to discuss home
care on the discharge appointment when the patient has a sore mouth and is not going
to cooperate at home with anything having to do with its teeth. But waiting those 2
weeks allows for healing and then home care will be better accepted. Calls equal “we
care” in the mind of the client. Data codes should be linked to services to generate a list
of follow up calls to be made. Discover for your area what time of day is best to actually
speak to a client on these calls. Leaving a message is appreciated but never generates
a dialog where we can offer help or detect an issue unrecognized by the owner. Only
56% of clients get follow up calls. Work to improve this number. Many hospitals call
every client that visited the hospital for services the evening they leave. This is an
optimal time because this will be when clients have questions or have attempted to give
the first dose of medication unsuccessfully. A knowledgeable ear and some helpful tips
are welcome when clients are unsure.

Getting great client acceptance is something that we can track with data and percentages but
gaining increases has more to do with communication, trust, sincerity and confidence. A client
who has been made to wait an hour past her appointment time is not as accepting as one who
has been given an enjoyable experience leading up to her time in the exam room. A client who
was treated rudely by the front office team will balk when offered expanded care. A client who
perceives the team doesn’t truly believe the service they are offering is in the best interest of the
patient will only think the clinic is gouging them for money. Increased compliance comes from
relationship building with our clients so they trust that we are there for them and their pets and
honestly have the best interest of the patient at heart.

Action Plan:

Standardize Protocols and train staff to them
Create Diagnostic codes and use them to be able to measure compliance
Take communication classes to learn to better offer high quality services with confidence
Add reminders for all possible services –measure response-send as many as 5
Make appointments for follow up visits before the client leaves
Praise clients and staff for good work and utilize a compliance audit sheet for all visits
Create a link to phone call reminders on all appropriate service codes
Measure, Measure, Measure –set goals for your team to improve- reward them when they do

http://www.aahanet.org/PublicDocuments/ComplianceExecutiveSummary0309.pdf
THE ART OF EUTHANASIA
Mary Gardner, DVM

Learning Objectives:

The euthanasia appointment is one of the most emotionally challenging appointments for the entire staff (and owner). This lecture will go over all aspects of the appointment including how to handle the initial phone call, discussing the processing, handling payment, technical aspects of euthanasia and body care.

Being Good at Death:

We are not taught to be good at death. No one taught me how to walk into an exam room for a euthanasia, what to say to a crying teenager, or whether or not to hug the old man that just lost the last piece of his late wife. I received no direct guidance about the proper verbal and non-verbal techniques that make this “most difficult appointment” just a bit easier on everyone, including myself. And from our numerous discussions with new grads, it’s a common theme; about 75% of veterinarians graduate without ever administering the life-ending medication. It’s no wonder why our lectures are packed at conferences and why our hospice practice has more requests for externs than we can handle. We simply weren’t taught the intricacies of death, and as the only medical profession licensed to euthanize, we have an incredible privilege and responsibility to handle this procedure properly.

Euthanasia:

If there is one thing to think about when approaching the euthanasia appointment, it’s “What would I do for my own family’s pet?” This involves not only you, but your immediate non-veterinary family as well. What could you do to help the ones you love through the process? Now make sure that is the minimum standard of service and care you give each of your patients and their caregivers! Here are some tips to put this into practice:

The entire euthanasia process can be broken down into 4 stages:

1) Setting up the Euthanasia Appointment

   a. Be the first to say the “E” word. Clients hate to be the first ones to bring up “euthanasia.” They think you will judge them for not caring about their pet or that you will be mad at them for giving up too early. Be the first to say it. And even if they’re upset at you for the suggestion, at 2:00 am when they’re stressed because their dog is pacing all night or their spouse is yelling at them because their elderly cat has peed outside the litter box for the third time that day, they will know that you gave them permission to think about the next step.

   b. Making the appointment: How your support team handles this initial contact with the client is crucial. It took the owner a lot of nerve and emotion to call; many feel that they are making the appointment to kill their best friend. Guilt, worry, anxiety, sorrow are just a few of the ingredients in their emotional cocktail. The
receptionist should have nothing else on their mind but assisting that client. They should not be put on hold, the receptionist should not be checking out another client at the same time, and if at all possible, background noise should be kept to a minimum. Most importantly, empathy must be conveyed; I'm so sorry you're facing this. Do not be scared to show them some emotion, they want to know that you care.

2) During the Appointment

a. The Arrival: When the time for the appointment comes, everyone in the clinic should be prepared. The paperwork should be ready, dated, and IN the room. The room itself should be set up properly and one person should be prepared to assist the client. Meet the family at their car prepared to help them into the clinic. Even holding the door open while the owner manages the cat carrier is a huge help to the client. And of course, shuttle them to the room immediately. Paperwork is best completed at this time before reality sets in with the family. Again, emotions will only get deeper from here, not lighter!

b. The Space. The room itself is very important. Regardless if it's a separate comfort room or a regular exam room, you must do your best to make it as warm and comfortable as possible (it should not be the 'cold sterile' environment owner's dread).

c. The veterinarian should go into the room and preferably not leave again until the pet has passed unless the owner requests time alone. Go in with sedation and euthanasia already pulled up in syringes in your pocket, or given to your technician. Speak to the client and make a visual assessment of the pet. Do not pass judgement or appear to be uncomfortable with the decision unless you are certain you will not euthanize. Your discomfort will leave a family with guilt for years.

d. When explaining the euthanasia process, it is important to give the owner peace of mind that it is a gentle process. Explain that euthanasia means “good death” and that the medication is an overdose of anesthesia, in which they go to sleep and don’t wake back up

e. Offer them some time alone with their pet. If they want time alone, hand them the 'ringer' portion of a wireless doorbell. Have the 'bell' portion in the treatment room or give it to the technician assigned to the case. That way the owner does not have to leave the pet to find someone when they’re ready. The human animal bond should never be broken. Generally people do request a few minutes alone, but it’s usually a very short amount of time.

f. The Procedure: Intra-muscular or subcutaneous sedation is crucial for the client’s experience and we are always discouraged to learn how many do not sedate pets before euthanasia, or provide only IV sedation (in which their pet rapidly goes from consciousness to unconsciousness, appearing dead). Having 5 minutes for the pet to slowly relax gives the owner time to watch their pet get
comfortable. Many times I hear “I haven’t seen him this calm and relaxed in months!” We call this “secondary sedation of the owner.”

g. When it comes time for the final medication, ask the owner “Max is ready, are you?” Never proceed without them fully knowing what is about to happen. They should also know that their pet will pass in 30-60 seconds. All too often owners do not realize it occurs as fast as it does. Whether you use an indwelling catheter, butterfly catheter, or straight needle, do your best to stay out of the way of the owner. Let them hold their pet and instruct them to “keep talking to her, she can hear you.” Giving them something to do keeps their focus off you and this surreal moment for them.

h. After administration, listen for the heart and remain silent unless the owner speaks. This is an important moment and must be honored.

i. Stay present in the room for a few minutes as you gather the syringe and supplies. Watch for agonal breath(s), twitching, or any other movements, which generally happens within 1-5 minutes post mortem. Since we do not recommend warning about all these side-effects before, this is the time to explain them if/when they occur.

3) Memorial Items

a. The paw print is the most traditional and cherished memorial item, even more than cremains sometimes! Every pet owner should be given one at the time of the appointment and given to the owner to take home that day (at no charge!). With air dry clay like Crayola Model Magic, this is inexpensive and takes very little time. Many clinics make the paw print after the clients leave but you are missing a huge opportunity to make the owners feel a little bit of joy at such a devastating moment.

4) Body Care

a. Never allow the owner to leave their deceased pet alone. If they need time alone after the euthanasia, allow them that time and hand them the wireless doorbell again. This way, a technician can come back into the room as they leave.

b. Know your crematory well. Understand how they do things and be confident they are providing the level of service your client’s deserve.

If there’s one thing we can tell you to improve your end of life care for pets and their families, it’s to provide the best from the get-go. Provide the kind of care that exceeds the expectations of 95% of the population out there. Do not cater to the 5% of people that are irregular.

The euthanasia appointment should not be the end of the client relationship, it should be the beginning of the next relationship you have with them! And remember, if it were your own pet, what would you do?
DON’T UPSELL… UPSERVE
Mary Gardner, DVM

Learning Objective:
This course will provide attendees tools to enhance their customer service skills by looking at some of the best in multiple industries. Leading a conversation, finding out what’s important to the owner and providing outstanding customer service – from all team members will build trust with your clients and therefore encourage them to do what is best for their pet within their means.

Proceedings:

Like it or not, we are all in sales - not in the traditional sense of the word ‘sales’ - but in the broader sense we are all trying to get others to part with resources – these resources are not just monetary; they include resources like time, attention and trust. Leave aside the physical exam, reading bloodwork and radiographs – veterinarians spend a lot of their time in sales. Whether it is to get the owner to clean the ears and apply medications twice a day, the technician to help during surgery when they would rather go to lunch, negotiating with suppliers or gaining trust with clients to allow for diagnostics – veterinarians need to learn the art of sales and how to build trust. And the first step is learning to Up-Serve instead of Up-Selling.

If building relationships is the key to sales success, then trust is the foundation. Most top sales performers will say that the factor that contributes to their success most is building trust with customers. But how do you build trust? Usually, it’s the little things you do over time that make the difference but sometimes, in our industry – we have 5 minutes to build trust. There are 5 secrets that I like to follow to building trust.

1. Truth: We can do many things to lose business: not deliver on time, not communicate effectively, not follow up. But from your customers’ point of view, lying is the number-one way to lose their trust and business forever. Do not lie about their pet’s condition or the treatment options. Period!

2. Reliability: You build trust every time you get back to the client with the information they requested, every time you follow up after the customer receives your service, and every time you respond to a problem immediately and solve it right away. If you forget to call back with bloodwork results or not send the information you promised – you broke that trust.

3. Understanding through uncommon efforts: When you invest the time to understand your client’s needs, concerns, thoughts, what is important to them, etc - you are building the trust by making the effort to see the world through their eyes.

4. Service: There’s no better way to build and maintain your client’s trust than through ongoing personalized service.

Look around your practice and figure out ways to be personalized and earn your client’s trust. And ask yourself the following questions about your clients:

• If one of my clients leaves, do I know why?
• If I don’t know why, do I ask?
• Do I ask every client I have, "Is there anything I'm not doing that I could be doing to serve you better?"
• Do I consider myself a resource for my clients?
• Do I create added value for my clients by going beyond what's expected?
• Do I look for ways to help my customers increase their bottom line?

5. **Take your time**: Building trust does not happen overnight but rather a long relationship of building that trust. The follow-up calls and visits, solved problems, on-time delivery of promises, and caring about them and their pet – honestly – will build trust.

To build trust, your customers need to believe three things about your company:
1. You have their best interests at heart.
2. You are capable of delivering on your promises.
3. You are honest and authentic.

This session will be interactive and go over ways great companies build trust and therefore Upserve vs Upsell!
YOUR PERSONAL CURB APPEAL: BODY LANGUAGE FOR THE EXAM ROOM

Mary Gardner, DVM

A veterinary clinic’s curb appeal does not stop at the clinic door. It extends all the way into exam room and, most importantly, to the entire team! Every person our clients interact with will receive a “snap judgement” from their first impression. How long does this take? For years the general rule has been 7 seconds, but a few years ago a group of psychologists found that it takes about one tenth of a second to form an impression of a stranger, simply from their face (1). They also found that longer exposure to the stranger does not significantly alter the impression, it only boosts confidence in the initial judgment.

What does this mean to a veterinary team? It means that we have a very, very small amount of time to make a positive impression on our clients. This positive impression is not only essential from a business standpoint (you want them to come back!), but also from a medical one. Our clients need to trust us; they need to believe that we care about their pet the same way they do. Without the belief and trust that the client and the doctor have the same desired outcome, trust and rapport will not be established and the client may not accept the treatment plan that the veterinary professional team has offered. Which is, after all, the reason we are in business; to care for, treat, heal, and support animals.

Of course, the importance of body language or non-verbal communication is not a new concept. The “7-38-55 Rule” was first developed in 1971 by UCLA psychology professor Albert Mehrabian (2): 55% of what we convey when we speak comes from our body language, 38% from our tone of voice, and a mere 7% from the words we choose. This study has been widely misinterpreted by stating “97% of what we convey is non-verbal” instead of garnering a greater understanding of vocal (tone, cadence, etc.) and body language cues, which are inappropriately combined to come up with the “97%”.

Mehrabian more clearly states the following on his website:

\[
\text{Total Liking} = 7\% \text{ Verbal Liking} + 38\% \text{ Vocal Liking} + 55\% \text{ Facial Liking}
\]

Please note that this and other equations regarding relative importance of verbal and nonverbal messages were derived from experiments dealing with communications of feelings and attitudes (i.e., like–dislike). Unless a communicator is talking about their feelings or attitudes, these equations are not applicable.

Although this landmark study is riddled with criticism and misinterpretation, it remains an important and highly cited illustration of the value of nonverbal communication. Many other studies have arisen since, each with a new methodology, and with the continued conclusion that non verbal cues are 3 to 4 times more influential than verbals cues.

Before we dive into the real content of this talk, it’s important to understand that reading body language is not the same as mind-reading. This is the difference between “observation” and “evaluation.” Reading someone’s non-verbal cues is about observation; we want to find natural tendencies in someone’s physical behavior (called their “baseline”), then look for deviations from their baseline, and finally ask open ended questions to find the root cause of the change.
For example, you may walk into a room and find two people seated, both have their arms crossed while one has both feet flat on the floor and the other has her legs crossed at the knee. You might assume that the closed off body postures means they are both are upset, and perhaps the female is even more upset because her legs are crossed as well. This may be true, but probably not. Jumping to conclusions so quickly and, for example, immediately putting your guard up or responding with your own closed off body language may start you off on a bad foot (no pun intended) by eliciting defensive behavior from these clients. In this example, crossed arms might be this gentleman’s natural baseline, and the female may simply be cold!

Remember, reading body language is about observing someone’s baseline, finding where there are deviations from that baseline, and using powerful questions to find the underlying cause of the deviation.

**THE BASICS**

The basics of body language are pretty simple. Across species lines, animals (human and non-human), use adaptations to increase or decrease their physical presence. A bear stands on his back legs to appear taller, cobras expand their hood when they are threatened, and the mantis lifts her front limbs while displaying a conspicuous eyespot in order to scare or distract a predator.

Humans present similar non-verbal “tells” by puffing their chest and standing taller when an attractive woman walks by or throwing both hands up in the air after accomplishing a huge milestone (even humans who have been blind since birth exhibit these behaviors).

The opposite is true as well; a dog cowers in the back of a cage or tucks his tail, an embarrassed child covers her face. We tend to minimize our physical presence when we want to disappear!

Each unique area of our body displays our emotions differently. The face is the most important when it comes to first impressions, and the feet most important when you want to know whether a negotiation is being tipped in your favor.

**PERSONAL CURB APPEAL**

When you want to make the most positive impression possible on a client, there are 4 main areas to consider: Initial facial expressions, the introduction to the client, non verbals while speaking, and physical appearance. Each of these areas have been proven to influence the impression someone has on another person.

1. **Facial Expressions**

Judgements based on facial appearance or expression play a very powerful role in how we get treated (2). In fact, in a court of law, it’s been shown that “mature faces” receive harsher judicial outcomes than those with a “baby-faced,” and having an face that is thought to be “competent” (as opposed to trustworthy or likable) may be highly predictive of whether a person gets elected to public office (3). Also, like it or not, attractive people are more favorably viewed in general, leading to overall better outcomes in life in addition to being thought of as more trustworthy (4).

What is a good way to use your facial expressions to improve your curb appeal? Smile. Yes, simply smile. Of course we have all been subjected to the “fake smile” versus “genuine smile”!
This distinction has been researched for quite some time; so much so that a genuine smile is now described with the name “Duchenne smile” after the French physician Guillaume Duchenne, who studied the physiology of facial expressions in the nineteenth century (5).

The *Journal of Personality and Social Psychology* described the difference from the anatomical perspective (5):

A. The Duchenne smile involves both voluntary and involuntary contraction from two muscles: the zygomatic major (raising the corners of the mouth) and the orbicularis oculi (raising the cheeks and producing crow’s feet around the eyes).

B. A fake smile involves the contraction of just the zygomatic major since we cannot voluntarily contract the orbicularis oculi muscle.

Interestingly, the fake smile is controlled by the motor cortex while more complicated emotion-related expressions, like the Duchenne smile, are controlled by the limbic system.

Yes, our clients can tell the difference! A genuine, warm, sincere expression of happiness that conveys a welcoming greeting is related to emotion, while the cheesy grin is simply a forced muscle action. So make sure your greeter (whomever that might be) smiles because they are happy to be there, not because they are forced to!

2. The Non-Verbals of Introduction

Upon being greeted with the warm, genuine smile, the customary introduction ensues. Even if this is a long standing client, there is still a formal greeting ritual we all engage in. The first 7 seconds may be too long for a first impression, but it’s the perfect amount of time for a good introduction.

In our current Western society, the handshake occurs first and, as long as it’s a good one, is the universally accepted sign of professionalism, politeness, and confidence. A good handshake is an art! Whether you’re the veterinarian or the support staff, make sure you initiate the handshake before the client does to show a confident welcome. Remember, they are coming into your “home” (the clinic) and you want them to feel that you genuinely appreciate their presence. Make hand contact palm to palm, web to web (the “web” is the flap of skin between your thumb and pointer finger) while keeping the angle of your hand either perpendicular to the ground, or palm facing slightly up. Palm down in a handshake indicates power. Don’t squeeze too tightly, nor too loosely, and maintain consistent tension as you say your greeting. Also, make sure to shake everyone’s hand in the pet’s family, not just the primary owner, even the children. (What a way to inspire a new generation of veterinarians!)

While shaking the client’s hand, maintain good eye contact and introduce yourself, even if you believe they know your name (but not with close friends of course!). They may have forgotten your name since their last visit, and setting your client up for success by knowing your name helps build their confidence. (More on verbal techniques, including how to say the client’s name, in another lecture.)

Since the introduction is about 7 seconds long, make sure it’s meaningful. Step in front of the receptionist’s desk to shake their hand, use a two-handed handshake (both of your hands around their one hand), lean gently forward to show appreciation for them coming in, and/or bend down to pet their dog (cats may not appreciate this though!).
3. Non Verbals to Gain Rapport

After you’ve made an amazing first impression, followed by a confident introduction, it’s time to complete the circle so that the client builds the trust, rapport, satisfaction, and connection with the entire veterinary team. These skills all enforce the concepts of active listening, engaged interaction, and supporting the client’s concerns.

These concepts are broken into 3 anatomical areas, top, middle, and lower body regions.

A- Body Language in the Top ⅓

Eye contact is incredibly important! But how much is too much? At what point does it start to become creepy? One study in the Royal Society Open Science (6) found that, when asked to stare at a video of an actor staring back at them, participants had a “preferred gaze duration” of 3.3 seconds (give or take 0.7 seconds). They also found that the rate of pupillary dilation (an automatic reflex) was a good indicator of how long they wanted to gaze; the longer their preferred gaze, the faster their pupils expanded. (Don’t get too attached to this difference, however. The change was so subtle that it was only seen with eye tracking software, which would be awkward to follow in real life!)

Make your eye contact consistent by looking only inside the imaginary triangle between the two points about 1 inch above each eye and the tip of the nose; going further down to the mouth or chin is more indicative of a social or amorous relationship.

Aside from the eyes, do not bite, tense, purse, or conceal your lips. Janine Driver, re-known body language expert, says “when we don’t like what we see or hear, our lips disappear.” This is evidenced by turning both lips into our mouth, similar to spreading chapstick once it’s been applied.

When nodding your head, a gentle, 1 second nod implies active listening, whereas faster head nods may tell your listener “hurry up, I don’t have time for this.” Make your nods slow and small with a closed mouth (which indicates you are listening).

Hands and arms are the second component of this category. Many of us will find ourselves wringing our hands or picking at our fingernails at any given moment. This may increase when we are nervous and evolve from a normal, baseline behavior into what is considered “pacifying” behavior. This is a normal reaction to nervousness or discomfort. (Again, we don’t know WHY someone may be nervous or uncomfortable, but we can simply make the observation then follow up with a powerful question.)

CONCLUSION

Curb appeal does not stop at the clinic’s entrance. And fortunately for veterinary professionals, those clinic doors are human sized, not small doggy-doors (until pets earn a monetary income, this will be the case)! We have to interact with, connect with, and ultimately, win the trust of our clients if our professional knowledge is to be put to good use. Without that rapport with our clients, something every person of the veterinary team is responsible for upholding, our treatment plans may not be accepted and/or compliance may not be achieved. Only through immediate, consistent, and appropriate maintenance of this bond will the patients receive the best possible medical care, and our clients happy to see us again!
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THE VERBAL IMPACT – YOUR WORDS SAY MORE THAN YOU THINK

Mary Gardner, DVM

As veterinarians we have two parties to serve in almost all areas of our profession; first, the owner/client, and second, the patient. In human medicine, the client and the patient are generally the same entity. Even in pediatric care, the parent is the guardian of the child, not the owner of that child. The parent generally has the levy to make decisions, but if that decision is not in the best interests of the child (as reasonably determined in a court of law), then the parent will in fact lose the ability to make decisions for that child. In fact, it took a groundbreaking case in 1984 (*In re Guardianship of Barry*, 445 So.2d 365 (Fla. 2d DCA 1984)) to determine that a parent can serve as proxy for their dying infant child’s wishes, allowing the removal of life support in this case.

In veterinary medicine, however, our clients served as proxy for their pet’s wishes in almost every interaction they have with a veterinarian; from the decision to amputate a limb, chose surgical versus medical treatment, and even the choice to removal of “life support” and initiate euthanasia is a common path that the veterinarian must walk the client through on behalf of the pet. Legally, the clients are in fact owners of the patient and our communication and established rapport with that owner is imperative if we are to gain the trust such that our medical knowledge will be put to use for the betterment of the pet and/or the treatment of a disease. Learning how to gain that rapport is where the rubber meets the road!

As we move through this discussion, remember that veterinary medicine these days is more like pediatrics than the “horse mechanics” we were generations ago. Pets are family now. They have moved from the barnyard to inside the home to our bedroom… and even under our sheets! Even the survey conducted by the American Veterinary Medical Association (AVMA) (1) found that of 63.2% considered their pets to be family members. Another 35.8% considered their pets to be pets or companions and only the remaining 1% considered their pets to be property.

In the previous lecture, Body Language - Your Personal Curb Appeal, we discussed non verbal communication and its role in the interaction we have with clients. This next part of the communication picture relates to the words we say, and how we say them.

**The Use of Pronouns**

The words we choose to use when describing pets must be reflective of the importance they hold in the family. Sure, some people may view their pet as “just a dog,” but those people will be only slightly offered by your endearing use of the word “baby” as compared the owner that refers to herself as “Charlie’s mom,” she will be much more offended by the use of the pronoun “it”!

Through many discussions with 1000’s of veterinary professionals, veterinary students, veterinary receptionists, and our highly trained team of care coordinators, I have personally come to the conclusion that more than half of us are willing to say the word “baby” when referring to a client’s pet. Of course, that doesn’t mean we all prefer this term. Personally, I’m not completely comfortable with its use, but I’ve adopted it based on the reaction from pet parents (also a phrase gaining traction). In our Support Center, we use the term “baby” (not fur-
baby, I personally don’t like this one at all), only before we have the name of the pet, which we ask for immediately. Once the pet’s name is known, there is no greater word than the name given to him/her by his owners.

Along these same lines, we have adopted the use of “pet parent” in our practice, but still generally use the word “owner” when referring to the case amongst colleagues. Again, “pet parent” is not my personal first choice, but the upside of conveying we understand the importance of the pet in the family is much more beneficial than risking the downside of appearing “cold” or “rude.” It’s rare that someone is genuinely offended by the use of these overly fluffy words, even if it’s not their first choice either. But with 84% of pet owners referring to themselves as “mom” or “dad,” this doesn’t seem too far off the mark (2).

Tone of Voice

Cats and dogs both use different vocal tones at different times of stress, attraction, play-seeking, or almost any other behavior. Humans also deepen their voice while making their speech sound “more pleasant” when talking to someone they find attractive. A recent study illustrated this point (3):

We examined how individuals may change their voices when speaking to attractive versus unattractive individuals, and if it were possible for others to perceive these vocal changes. In addition, we examined if any concurrent physiological effects occurred when speaking with individuals who varied in physical attractiveness. We found that both sexes used a lower-pitched voice and showed a higher level of physiological arousal when speaking to the more attractive, opposite-sex target. Furthermore, independent raters evaluated the voice samples directed toward the attractive target (versus the unattractive target) as sounding more pleasant when the two voice samples from the same person presented had a reasonably perceptually noticeable difference in pitch.

The idea of using a lower pitched voice to influence others in a multitude of ways has been known for quite some time. Even Margaret Thatcher was known to have too “shrill” of a voice at the beginning of her career; so much so that she was not allowed on party broadcasts. But before her election in 1979, she worked with a speech coach to help lower her pitch and develop her infamously calm, authoritative tone. Her biographer Charles Moore later wrote, “Soon the hectoring tones of the housewife gave way to softer notes and a smoothness that seldom cracked except under extreme provocation on the floor of the House of Commons.” (4) Aside from lowering the vocal tone, a common mistake is the use of “up-speak.” A frequent mistake in women (though men can do this as well!), this offender ends every sentence on a higher note than the rest of their speech. Doing this makes everything that’s said sound like a question and, most importantly, gives up the confidence we wish to convey to our clients. Some professionals feel this kind of tone is very “California-Valley-Girl.” With the perception this speech pattern makes its users appear young, immature, and overall uncertain. Instead of ending a statement on a high note (literally, not figuratively), try ending it on a consistent, or even lower pitch (NOT softer), to convey a strong sense of confidence.

Salutations

We’ve all been there, the typical “hi, how are you” followed by the “great, how are you?” and then, it’s really bad, one more “I’m great, how are you”… and then you’re lost. When responding to the customary “how are you?” find and use (and re-use!) a phrase that you really
love: “loving life and living the dream!” or “this is the best day of my life” or “it couldn’t be better, I get to play with animals all day!” Any of these will leave the client feeling happy (hopefully), and at minimum, spark a curiosity in them that may lead to an interesting conversation.

**Sounding Persuasive**

Though there are 100’s of tips on sounding persuasive, we have chose our top 3 below: Talk moderately fast, use just enough pitch, and use powerful pauses.

1. **Rate of Speech.** Speaking at a regular rate, perhaps even moderately fast, has been shown to be positively correlated with perceived intelligence. “Interviewers who spoke moderately fast, at a rate of about 3.5 words per second, were much more successful at getting people to agree than either interviewers who talked very fast or very slowly,” said Jose Benki, a research investigator at the U-M Institute for Social Research (ISR) (5). Throw in a bit of humor, and you have a recipe for winning someone over!

2. **Pitch Variation.** Some researchers have shown that the more active the pitch and variation, the more energetic and engaging someone may appear. This isn’t always the case, however; “We found only a marginal effect of variation in pitch by interviewers on success rates. It could be that variation in pitch could be helpful for some interviewers but for others, too much pitch variation sounds artificial, like people are trying too hard. So it backfires and puts people off” said Benki (5).

3. **Powerful Pauses.** “When people are speaking, they naturally pause about 4 or 5 times a minute,” according to Benki. “These pauses might be silent, or filled, but that rate seems to sound the most natural in this context. If interviewers made no pauses at all, they had the lowest success rates getting people to agree to do the survey. We think that’s because they sound too scripted. People who pause too much are seen as disfluent. But it was interesting that even the most disfluent interviewers had higher success rates than those who were perfectly fluent (and did not use pauses).”

Particularly in a high paced, knowledge based profession like veterinary medicine, you are best to make your verbal deliveries with minimal variation, focusing instead on tone, include natural... steady... frequent... pauses!

**Sounding Honest**

In Alex Peatland’s book, “Honest Signals: How They Shape Our World,” the authors point out three to keep your eye on (6):

1. Speech mimicry and behavioral mimicry. Are they using the same words you use? Speaking at a similar speed and tone? Are they sitting the way you sit? Is a subtle, unconscious game of follow-the-leader going on? This is a sign the other person feels emotionally in sync with you. It can be faked but that’s rare and difficult to pull off consistently across a conversation.

2. Consistency of emphasis and timing. This is a sign of focus and control. Someone who is less consistent is less sure of themselves and more open to influence.

**Win Them Over Again**
If all else fails, what are 2 things you can do to win someone over? Robert Cialdini, author of the must-read book “Influence,” provides these important tips:

1. Give Honest Compliments. It may not be easy, especially if the person has been distancing themselves from you for a while. But if you’re objective, they probably have some qualities you admire. If you take a positive action and compliment them, it may well break the ice and make them re-evaluate their perceptions of you.

2. Ask for Their Advice. Cialdini notes this strategy – which involves asking for their professional advice, book suggestions, etc. – comes from Founding Father Ben Franklin, a master of politics and relationship building. “Now you’ve engaged the rule of commitment and consistency,” says Cialdini, in which they look at their actions (giving you advice or a book) and draw a conclusion from it (they must actually like you), a surprisingly common phenomenon in psychology. “And suddenly,” says Cialdini, “you have the basis of an interaction, because now when you return it, you can return it with a book you think he or she might like.”

Verbal communication is, indeed, extremely important in the communication we have with clients. The delivery, consistency, and accompanying non-verbal cues give the client the feeling that we are either listening and engaged or detached and uninterested. We have a choice, and with proper education, we can be in a better position to choose the best route for our patient, our client, and our team.

**SOURCES:**

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PLENARY SESSIONS
ZOONOTIC DISEASE WATCH LIST – ONTARIO

Maureen E.C. Anderson DVM, DVSc, PhD, Dip ACVIM

As more and more attention is paid to infectious diseases and “one health” issues, the role of veterinarians as a source of information and expertise in zoonotic diseases for animal owners, the general public and even public health and human health professionals will continue to increase. The following list highlights a few of the emerging, seemingly-emerging and endemic zoonotic diseases in Ontario of which veterinarians need to be aware.

**Echinococcus multilocularis**

Also known as the fox tapeworm, this small nematode parasite produces eggs identical to taenid eggs. While endemic in certain regions of Asia and North America, it was first diagnosed in a dog in Ontario in 2012. Since then, there have been 4 additional cases found in unrelated domestic dogs from several different parts of southern Ontario. All of these cases presented with lesions caused by the intermediate stage of the parasite (alveolar hydatid cysts in the liver or other parts of the body), which is usual because dogs are considered a definitive host, and are therefore considered more likely to develop intestinal infestation with the adult worms, unless they are exposed to massive numbers of parasite eggs. Two cases have also been confirmed in captive lemurs in Niagara, and a chipmunk west of Hamilton. Humans can also develop alveolar cysts following ingestion of eggs from the feces of infected canids. The incubation period in humans can be 5-15 years. Treatment can be difficult depending on the location of the budding cysts (which behave similar to a malignant tumor) and typically involves surgical resection and long-term chemotherapy.

Intestinal infection with adult worms in dogs and other canids is the greatest concern, because it is the eggs passed in the feces of such animals that are a risk to humans and other potential intermediate hosts (typically rodents). Intestinal infection is easily treated with commonly available antiparasitics as for other tapeworms. Eggs are immediately infective when they are passed, and may remain infective in soil for up to a year, therefore the environment can become a significant reservoir. Deterring hunting and scavenging in dogs prevents exposure to the intermediate stage and thus intestinal infection. Cystic masses, particularly in the liver, should be submitted for histology and additional testing if warranted, as dogs with extra-intestinal lesions may also harbour adult worms and thus pose a risk to human health.

It is speculated that the parasite may have been introduced into Ontario via dogs imported from other countries where it is endemic, and it is likely that it has become established in the wildlife population in parts of the province. A prevalence study in wild canids in Ontario is currently being conducted by researchers at the University of Guelph, with support from Bayer and the Ontario Animal Health Network (OAHN).

**Rabies**

Despite dramatically decreased numbers of cases compared to a couple of decades ago, rabies remains alive and well in Ontario. Most recently, a new incursion of raccoon-variant rabies in the greater Hamilton area resulted in over 200 cases in terrestrial animals between December 2015 and October 2016, including raccoons, skunks, one red fox and one stray cat. Bat rabies remains endemic in Ontario as well, with at least 28 rabid bats detected in the same time period from across the province. Rabies is considered one of the deadliest diseases on the planet, and while avoiding contact with the reservoir species that carry the virus (bats, foxes, raccoons and skunks) is the best defense, the next line of defense is ensuring pets are currently
vaccinated against rabies from the age of 3 months. This is required by law in Ontario for all
dogs and cats in 31 of 36 health units under the Health Protection and Promotion Act. Even
indoor cats need to be vaccinated, because unfortunately sometimes cats get out, or bats get in!

Since the transition from the Canadian Food Inspection Agency (CFIA) rabies program in 2014,
private veterinarians are now the front-line for managing potential domestic animal exposures
(human exposures should continue to be referred to the local public health unit, as always). It is
critical for all Ontario veterinarians to familiarize themselves with the components of a rabies
risk assessment, as well as post-exposure management of exposed animals. If there is a
significant risk of exposure, even currently vaccinated animals require a booster vaccination
within 7 days, while unvaccinated or under-vaccinated animals also require a precautionary
confinement period (PCP) for 3-6 months to monitor them for signs of rabies. Remember that a
10-day observation period is only for a pet that has bitten a person – it is NOT sufficient for a pet
that has itself been bitten by a potentially rabid animal.

Veterinary staff at the Ontario Ministry of Agriculture, Food and Rural Affairs (OMAFRA) are
available to assist veterinarians with risk assessments and post-exposure management
recommendations, as well as sample submission in cases where the offending animal is
available for rabies testing. Animal owners should always be advised to contact their
veterinarian first, and veterinarians can contact OMAFRA for assistance through the Agricultural
Information Contact Centre (AICC 877-424-1300). A complete set of resources and information
for veterinarians is also available on the OMAFRA rabies website at

Leishmaniasis

Leishmaniasis is caused by the protozoa Leishmania infantum, and occurs in two forms:
cutaneous and visceral. Clinical signs in dogs may include epistaxis, weight loss, muscle
atrophy, seizures, alopecia, dermal lesions, swollen limbs and joints, and renal failure (leading
to death). Dogs can also be subclinically infected. The parasite is endemic to the
Mediterranean basin, the Middle East, Southern Asia, Iran, Armenia, Afghanistan, Central Asia
and South America, and there is a significant risk of the disease occurring in dogs in Canada
that have been imported from these regions. Leishmania is typically transmitted by
phlebotomine sandflies (Lutzomyia or Phlebotomus spp.), which are not currently thought to be
present in Canada but there is no active monitoring for their presence. In 2000, canine visceral
leishmaniasis infection was implicated in high rates of illness and death among foxhounds in
New York. Surveillance found that Lutzomyia vexator sandflies, not previously detected in New
York, were widespread in the area. Direct transmission between dogs may also occur, e.g.
bites, breeding, needle re-use, and vertical transmission. A 2000-2003 survey of foxhounds,
other breeds of dogs, and wild canids showed that canine visceral leishmaniasis was enzootic in
18 US states and 2 Canadian provinces (ON, NS). Treatment of infected dogs primarily
involves long-term antibiotic therapy but it is rarely curative and relapses are frequent.

In humans, the most common form of leishmaniasis is localized cutaneous leishmaniasis (CL),
which usually appears as one or more painless ulcers. Visceral leishmaniasis (VL) is a febrile
illness which may result in weight loss, enlargement of the spleen and liver and decreased red
blood cell production that can lead to anemia, bleeding and co-infections. Without treatment,
this form of the disease is nearly always fatal. Transmission from cutaneous lesions of dogs to
humans can occur by direct contact. Immunosuppressed individuals and children are at higher
risk of acquiring the infection via direct contact. To date there are no reported human cases in
Canada acquired locally or associated with infected dogs. If the vector range expands due to
climate change, the probability of human and animal exposure will be greatly increased, and it
could become established in the wildlife population as well. At this time, it is primarily important to educate owners and groups that rescue dogs from other countries about preventing carriers of this parasite from entering Canada, and to ensure clinic staff are aware of the zoonotic potential when handling dogs with compatible clinical signs.

**Multidrug-resistant organisms (MDROs)**

Antimicrobial resistance (AMR) is nothing new, but the issue is becoming more and more of a concern as the threat of the "untreatable superbug" looms ever closer. In general, MDROs don't tend to cause any worse disease than their susceptible counterparts, but they can be much harder to treat, leading to prolonged illness with more complications and more expense. Many of these bacteria aren't particular about whether they infect humans or animals. People often share certain components of their microbial flora with their pets, and this can include antibiotic-resistant bacteria. In veterinary clinics, personnel can also become unwitting vectors for these organisms (particularly if basic infection control precautions are not diligently used), resulting in spread from one animal to another, or from a staff member to a patient and back. Infections are more likely to occur in pets with other illness or comorbidities, such as clinic inpatients.

One of the best-recognized MDROs is methicillin-resistant *Staphylococcus aureus* (MRSA), which is primarily an opportunistic pathogen of humans which has spilled over into the domestic animal population (including pets, horses and swine in particular, as well as others). In contrast, MRSA’s cousin, methicillin-resistant *Staphylococcus pseudointermedius* (MRSP), is primarily an opportunist in dogs. It has lower potential for zoonotic spread to people, but spillover infections do occur periodically, and MRSP has an even greater propensity for picking up resistance genes to additional types of antibiotics. There are others to be wary of as well, such as Enterobacteriacea (e.g. *E. coli*, *Klebsiella* spp.) producing extended-spectrum beta lactamases (ESBLs) and carbapenem-resistant Enterobacteriacea (CRE). Enterococci (particularly *E. faecium* and *E. faecalis*) are intrinsically resistant to cephalosporins, clindamycin, trimethoprim-sulfa and aminoglycosides (as monotherapy) and are adept at acquiring additional resistance genes as well.

Here are a few key items to remember with regard to MDROs:

*Prevention is better than cure:* A lot of infections with MDROs are secondary to other problems, or occur in animals with an obvious breach in their normal defensive barriers (e.g. surgical patients, trauma victims). Preventing or controlling underlying problems (e.g. allergic skin disease) and paying close attention to basic infection control measures (especially around high-risk patients) can dramatically reduce the number of infections that occur, as well as the need for additional antibiotic therapy (which can lead to selection of more MDROs).

*Cleanliness is king:* Animals with active infections are likely to shed higher numbers of bacteria, so it’s important to be aware of and address contamination of hands, materials, equipment and surfaces with high-risk substances such as wound discharge, nasal secretions, urine or feces. This applies to both the clinic setting and at home if the animal is treated as an outpatient. Also bear in mind that an animal infected with an MDRO at one site (e.g. a wound) may be colonized with the same bug at another site (e.g. intestinal tract). Pause to clean your paws!

*Culture culture culture:* As AMR becomes more common, and especially in cases of serious infection or those that may require long-term treatment, the importance of performing culture and susceptibility testing cannot be over-emphasized. Collecting samples before treatment is initiated to help maximize yield is critical, even if empirical treatment must be initiated before results are available. Reassessing the animal, response to treatment and the need for ongoing
therapy is also crucial, and narrowing or de-escalating therapy based on laboratory results and clinical response can help spare the “big guns” in the antimicrobial arsenal for when they’re really needed.

Try topical: Relatively superficial skin infections can often be effectively treated with topical biocides such as chlorhexidine-based shampoos, sparing systemic antimicrobials for infections that can’t be treated any other way. Topicals can also be used as adjunct therapy to help decrease the duration for which systemic antimicrobials are required.

Other diseases

There are a number of additional zoonotic diseases in Ontario on the “watch list” that are worth mentioning briefly as well:

Leptospirosis: This bacterial disease is endemic in Ontario and can cause acute renal and hepatic damage in dogs, and can also infect people. Specific infection control precautions are warranted when handling lepto suspects. For a helpful infographic on managing lepto patients in clinic, visit http://oahn.ca/resources/networks/companion-animals/

Lyme disease: Although not directly transmissible between animals and humans, people and pets can share common-source exposure to the causative agent (Borrelia burgdorferi) through specific ticks (Ixodes scapularis). There are now multiple areas in Ontario that are considered higher risk for transmission, but the risk of developing disease depends on multiple factors. An infographic covering this topic can also be found at http://oahn.ca/resources/networks/companion-animals/

Anaplasmosis: This is another tick-borne disease that can affect humans and dogs, as well as ruminants and horses, that is beginning to encroach on Ontario. The organism is species specific (e.g. Anaplasma marginale in cattle versus Anaplasma phagocytophilum in horses and humans), and is transmitted by black-legged ticks, which are already found in certain parts of the province.

Influenza A: Unusual or novel strains of influenza can potentially have a major impact on public health, certain agricultural sectors (e.g. poultry, swine), or both. Influenza also circulates in the equine population, and two canine influenza strains have caused outbreaks in North America, though no cases have been detected in Ontario to date. It is important to provide perspective in all cases, as the level of risk to humans or specific species depends on the strain.

Salmonella Dublin: This particular serotype has been associated with severe infections in both people and cattle (especially veal calves), making it both an animal health and public health/food safety concern. It has been found on a relatively small number of Ontario farms to date, and additional research is being conducted by the Ontario Animal Health Network (OAHN).

Mosquito-borne viral encephalitides: Although case numbers vary from year to year and it is the minority of infected humans and animals that become ill, disease caused by viruses such as West Nile and Eastern Equine Encephalitis can be devastating. Therefore it is important to remain vigilant and take precautions to reduce mosquito exposure and vaccinate where applicable (i.e. horses).

References available from the author on request.
References & Resources

The Ontario Animal Health Network has developed infographics for companion animal practitioners on several of these topics (Lyme disease risk, management of lepto patients, reducing antimicrobial use in pets). They can be found at http://oahn.ca/resources/networks/companion-animals/

Echinococcus:


Rabies:


Leishmaniasis:


MDROs:


BEYOND CONVENTIONAL CHEMOTHERAPY
Sue Ettinger, DVM, DACVIM (Oncology)
Dr Sue Cancer Vet PLLC and Animal Specialty and Emergency Center, Wappinger Falls, NY

Many clients have great interest in dietary supplements for treating pets with cancer. Like drugs, supplements have risks, side effects, and also potential benefits. Many owners start cancer supplement with little input from veterinarians, and it can be challenging to find good information to help our clients make informed decisions. In this talk, I will review some of the supplements that I have incorporated into my cancer patients’ care, and how you can use them in your practice.

WHY INTEGRATIVE MEDICINE?
Integrative medicine services complement mainstream cancer care. They are supportive measures that control symptoms, enhance well-being, and contribute to overall patient care. In contrast, alternative treatments are those used instead of mainstream therapies, but are often called “unproven, expensive and unsafe.” An integrative approach is of interest to many pet owners, and becoming more popular in both human and veterinary oncology. Memorial Sloan-Kettering Cancer Center Integrative Medicine services include touch therapy, mind-body therapy, massage, music, acupuncture, creative therapy, nutrition counseling, exercise programs to improve strength and promote relaxation.

CHEMOPREVENTION
Dietary substances and/or synthetic substances can be used to block, inhibit, reverse, or retard tumorigenesis. These are naturally occurring molecules which support apoptosis when introduced into the body. They support the normal turnover of deranged cells to help build new, healthy cells and affects the death receptor mediated pathway and the mitochondrial mediated pathway.

The effects of dietary components on apoptosis are likely among the central mechanisms underlying associations between cancer and diet consumption. Epidemiologic studies show protective effects of diets rich in fruits and vegetables and the possibility that naturally occurring phytochemicals can exert anti-carcinogenic activity as well. There is evidence accumulating that supports a combination of dietary bioactive agents as more effective. Some examples of dietary bioactive agents are: Epigallocatechin gallate (EGCG) flavonoids which are a major green tea constituent (EGCG in physiologically attainable concentrations causes induction of apoptosis and cell arrest in many cancer cell types without affecting normal cells), Resveratrol from grapes, Polyphenolic compounds, Lycopene from tomatoes, Carotenoids, Genistein from soy products, and other foods and herbs with anti-cancer activity such as: garlic, soybeans, cabbage, ginger, licorice, onions, flax, turmeric, cruciferous veggies, tomatoes, peppers, brown rice, wheat, carrots, celery, parsley. Dietary compounds have been shown to have chemopreventive effects on cancer development. Dietary agents are also known to induce apoptosis by interfering with both pathways (Pan 2008).

APOPTOSIS AND APOPTOGENS
Apoptosis is programmed cell death. It is involved in the regulation of development & maintenance of homeostasis, eliminates potentially deleterious cells and irreversibly damaged cells. It is normal, necessary and critical to physiologic functions, such as cell deletion in embryonic development and normal physiologic cell turnover.

The mechanism of apoptosis is a normal quiet process activated in aged and damaged cells. As apoptosis machinery is activated, there is shrinkage of cell and nuclear volume, membrane...
blebbing, chromatin condensation and nuclear DNA fragmentation, and subsequent phagocytosis. It is a process without inflammation.

There are two pathways of apoptosis. 1) Intrinsic: Mitochondrial-mediated pathway (responses to modulators within the cell) and 2) Extrinsic: Death receptor-mediated pathway (responses to mainly extracellular stimuli).

Why should we care about apoptosis? A universal hallmark in cancer is the lack of apoptosis. Unregulated apoptotic death allows affected cells to continue to proliferate. In degenerative diseases such as myocardial infarction, atherosclerosis, diabetes and reperfusion injury, apoptosis rates are enhanced. In proliferative diseases such as autoimmune disease and cancer, apoptosis is inhibited.

Apocaps®
Apocaps® are a nutraceutical to induce apoptosis with a unique formula of apoptogens. Apocaps® contain luteolin, apigenin, curcumin, beta glucans, silymarin, and gingerols. Apocaps® were developed by Dr. Dressler. Peer-reviewed publications show that there is an increase in anti-cancer effects in vivo. They work on intrinsic and extrinsic pathways to promote apoptosis. Clinically, Apocaps® are highly safe.

The following is a breakdown of each ingredient in Apocaps®:

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Effect</th>
<th>Dietary Sources</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Luteolin</strong></td>
<td>Bioflavonoid - Promotes apoptosis, interferes with glycolysis, blocks angiogenesis, anti-inflammatory</td>
<td>Dietary sources: celery, green pepper, peanut hulls, thyme, dandelion, perilla, chamomile tea, carrots, olive oil, peppermint, rosemary, navel oranges, oregano</td>
</tr>
<tr>
<td><strong>Apigenin</strong></td>
<td>Turns on apoptosis genes in cancer cells, can increase cell arrest in some cancer cells, slows angiogenesis, blocks COX-2, decreasing inflammation</td>
<td>Dietary sources: artichokes, basil, celery</td>
</tr>
<tr>
<td><strong>Curcumin</strong></td>
<td>Increased apoptosis in vitro in cancer cells without cytotoxic effects on healthy cells, slows angiogenesis, downregulates COX-2 LOX, NOS, MMP9 decreasing inflammation, blocks topoisomerase enzymes, critical to DNA replication</td>
<td>Principal curcuminoid of the popular Indian spice turmeric, member of ginger family</td>
</tr>
<tr>
<td><strong>Beta glucans</strong></td>
<td>Mushroom-derived polysaccharides - &quot;biological response modifiers&quot; ability to activate the immune system including NK cells, increase cell arrest and apoptosis, interfere with angiogenesis</td>
<td>Sources: cell wall of baker’s yeast, bran of some grains (oats &amp; barley), some types of seaweed Various species of mushrooms, such as reishi, shiitake, and maitake</td>
</tr>
<tr>
<td><strong>Silymarin</strong></td>
<td>Liver support supplement, role in chemoprevention, may protect liver from Lomustine toxicity and heart from doxorubicin-induced lipid peroxidation</td>
<td>Extract of milk thistle seeds, artichoke, coriander</td>
</tr>
<tr>
<td><strong>Gingerols</strong></td>
<td>Investigated for anti-cancer effect on tumors of bowel, breast, ovaries, pancreas; May reduce nausea</td>
<td>Active constituent of fresh ginger</td>
</tr>
</tbody>
</table>
Apocaps® benefit a canine cancer patient’s quality of life. Using Human pharmaceutical standards, Human-grade materials, following FDA-defined Good Manufacturing Practices, and are Plant FDA inspected and approved for pharmaceuticals. Apocaps are well-tolerated by canine patients but the MTD has yet to be determined.

I started using Apocaps® in my practice in the summer of 2010 in just a few cases. Currently, I use Apocaps® in about 80% of my patients. My patients are prescribed Apocaps® during and after chemotherapy, as well as following radiation therapy. In my experience there has been mild incidence of side effects, and occasionally mild diarrhea. The majority of my clients continue to use Apocaps® long-term after completing their chemotherapy protocols.

When giving Apocaps® to your patients DO: Give on an empty stomach, a minimum of one hour before or one hour after a meal. If you must, a very small amount of food, preferably less than 1-2 tablespoons is ok. If the dog experiences GI upset, give Apocaps® with meals. Apocaps are okay to use with non-NSAID pain medications but decreasing the dose with NSAIDs is advised.

When giving Apocaps® to your patients DON’T: give if the dog is allergic to the ingredients in Apocaps®, the dog has an allergic reaction to Apocaps®, dogs who are vomiting or diarrhea, and dogs less than 5 pounds should not be given Apocaps®. Safety has not been established for use in pregnant, nursing or breeding dogs. Do not use in cats or people, and do not use with steroids or NSAIDs at full dose.

One may order Apocaps® directly from Functional Nutriments (Call 1-888-936-4226), by visiting the Functional Nutriments website (www.functionalnutriments.com/vet) or by purchasing directly from distributors.

POLYSACCHAROPEPTIDE (PSP) MUSHROOMS
PSP mushrooms increase NK cell activity and cytokines, improve immune function, cause cell cycle arrest, reduce cell proliferation, and increase apoptosis. However, there is a lack of evidence of benefit in patients. PSP mushrooms are from Asian mushrooms, mushroom mixtures, and mushroom-derived polysaccharides. The bioactive agent is from mushroom Coriolis versicolor, a proprietary blend.

PSP mushrooms have been shown to be complementary and used as alternative medicine for canines with hemangiosarcoma. (Brown 2012). In a double-bind randomized multi-dose pilot study at UPenn with no placebo group, Zun-Zhu PSP (I’m Yunity), an extract of Coriolis versicolor, given in high doses delayed the progression of metastasis and proved to have the longest survival time for HSA without chemotherapy. Out of the three dose groups, no statistically significant difference was shown in the survival curves, but the two highest dose groups had median survival time (MST) longer than the longest MST reported in the literature to date: Splenectomy: MST 86 days, 50 mg/kg/day: 117 days, 100 mg/kg/day: 199 days.

YUNNAN BAIYAO (OR PAIYAO)
Yunnan Baiyao (or Paiyao) is a Chinese herbal mixture of notoginseng, from pseudoginseng root. Other ingredients include myrrh, ox bile, Chinese yam, sweet geranium, lesser galangal root, and other antiseptics or astringents. Yunnan Baiyao claims to regulate bleeding and has become popular for treating epistaxis and hemangiosarcoma (HSA). It is also known for its hemostatic and thromboembolic properties. Yunnan Baiyo activates platelets, and decreases bleeding and clotting times (Tang, 2009 - human study). I consider it for epistaxis and hemoabdomens secondary to hemangiosarcoma (metastatic).
GET YOUR GROOVE BACK: RECLAIM YOUR VETERINARY MOJO

Mary Gardner, DVM

Being a veterinarian is a dream of hundreds of thousands of people but only a few of us actually fulfill that dream. But what happens when that dream starts to fade and the reality of veterinary medicine starts to creep into our daily lives. So many veterinarians are facing burn out, client compliance frustration, compassion fatigue and even much worse. We must not let our dreams become buried under stress, discouragement, rejection, fatigue and negative voices. A lot of people give up in the face of opposition and let their dream become buried under discouragement, past mistakes, rejection, negative voices and failure.

Instead we need to learn how to find fulfillment, passion and love for veterinary medicine again – regardless of the path you take in this industry. Your dream may be buried, but the good news is that it’s still alive. It’s not too late to get your Veterinary Mojo Back!

This fun presentation will go over some key aspects of how to get your groove back!

Want to live a more passionate life? What does living a more passionate life mean – what is a ‘passionate life’ and what does being passionate about your job mean? Think about and write down - in your words – what is a passionate life and what that looks like for YOU!

My 3 Ingredients for a passionate career are: Fun, Growth and Contribution

Maintain the 4 Budgets in Life:

1. Financial
   a. Building a budget
   b. Plan for the future
   c. Spend some money on having fun NOW!

2. Time
   a. Huge stressor for people is lack of time
   b. Learn when to say ‘No’
   c. Leave time for fun now!

3. Physical
   a. Budget time for exercise
   b. Eat well
   c. Obtain a good quality and quantity of sleep
   d. Plan for fun activities

4. Emotional
   a. Don’t bottle up emotions
   b. Don’t let anyone take your happiness
   c. Watch out for each other

My Top 9 Secrets that ensure my happiness in Veterinary Medicine

1) Be nice and always do your best
2) Take your teammates seriously and respect them
3) Don’t take yourself too seriously
4) Be open minded
5) Declutter and be clean  
6) Learn to forgive yourself now  
7) Shed expectations  
8) Focus  
9) This too shall pass

Watch out for burnout: 10 Signs of Burnout  
1. Exhaustion  
2. Lack of Motivation  
3. Frustration, Cynicism and other Negative Emotions  
4. Cognitive Problems  
5. Slipping Job Performance  
6. Interpersonal Problems at Home and at Work  
7. Not Taking Care of Yourself  
8. Being Pre-OCCupied with Work … When Your NOT at Work  
9. Generally Decreased Satisfaction  
10. Health Problems

Compassion fatigue is real and a problem in our industry. Making sure you maintain a healthy mind, body and soul will help you combat it. See our Self Care Handout for some tips!
Combating Compassion Fatigue in Veterinary Medicine

Veterinary medicine is one of the greatest professions and I’m grateful that this is my career. Sadly, studies have shown that it is also one of the most emotionally draining professions due to various factors. We are automatically at risk for compassion fatigue due to the nature of our jobs (high stress, long hours, long weeks, & most importantly – not enough funding) and our personalities (perfectionists, highly compassionate and high achievers who cringe at failure). Learning to recognize the signs of compassion fatigue is the first step to understanding and combating this silent affliction. The second step is to walk down a path towards wellness.

A PATH TO WELLNESS AND SELF CARE

ref: compassionfatigue.org

• Know that Self Care begins with you.
• Be kind to yourself.
• Enhance your awareness with education.
• Accept where you are on your path at all times.
• Understand that those close to you may not be there when you need them most.
• Exchange information and feelings with people who can validate you.
• Listen to others who are suffering.
• Express your needs verbally.
• Take positive action to change your environment.

Another good exercise for combating fatigue is to recognize your triggers. Should they occur, pause and reflect on why you entered this field and what you love about veterinary medicine. This helps you Get Inspired! Please take a moment to answers the questions below:

GET INSPIRED

1. What is the most rewarding moment that you have experienced at your job?

2. List 5 things you love about your job?

   1. ____________________________
   2. ____________________________
   3. ____________________________
   4. ____________________________
   5. ____________________________

Prepared by Mary Gardner, DVM
Co-Founder, Lap of Love
Combating Compassion Fatigue in Veterinary Medicine

3. List 5 co-workers/clients whose lives you have touched in a positive way
   
   1. __________________________________________
   2. __________________________________________
   3. __________________________________________
   4. __________________________________________
   5. __________________________________________

4. Why did you take your current job?

5. List 3 things you do well in your position
   
   1. __________________________________________
   2. __________________________________________
   3. __________________________________________

6. Write down 3 compliments you have received from co-workers/supervisors or clients
   
   1. __________________________________________
   2. __________________________________________
   3. __________________________________________

Keep this in a place where you can reference it for those “challenging days”


Prepared by Mary Gardner, DVM
Co-Founder, Lap of Love
**SELF CARE CHECKLIST**

Use the scale below to complete the checklist this week. Then complete the checklist every 3 months to see how your self-care program is progressing.

Your completed checklist should indicate attention to your own self-care. The goal is to achieve a higher score in each 3 month period as time goes on. If it does then you are moving toward wellness. Review the chart to see if there are patterns that you need help in and be sure and seek out assistance in those areas. This chart can be a useful aid in avoiding compassion fatigue and moving towards compassion satisfaction.

**How often did you do the following things in the last full week you worked?**

Use the following scale  1 = Rarely  2 = Occasionally  3 = Often

<table>
<thead>
<tr>
<th>SELF CARE ACTIVITY</th>
<th>NOW</th>
<th>3 MONTHS</th>
<th>6 MONTHS</th>
<th>12 MONTHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respected my own dignity &amp; self worth</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Took responsibility for self care</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thought about my right to wellness</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thought about my own physical rest &amp; nourishment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thought about my own emotional rest</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thought about moderating food, drink, cigarettes &amp; other substances for my health</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sought, found &amp; remembered appreciation from supervisors &amp; clients</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Made it known that I wish to be recognized for my work</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Remembered my commitment to my own self-care</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Set or reviewed self-care plans &amp; goals</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thought about strategies for letting go of work</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Generated strategies that work &amp; followed them</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thought about strategies for gaining a sense of achievement.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thought about acquiring adequate rest &amp; relaxation.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practiced effective daily stress reduction methods.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practiced effective physical exercise to improve my health.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Noticed &amp; worked on my body to improve my health.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practiced effective sleep habits &amp; maintenance for better health.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practiced proper nutrition for better health.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Prepared by Mary Gardner, DVM  
Co-Founder, Lap of Love
**Combating Compassion Fatigue in Veterinary Medicine**

Practiced effective behaviors that sustain balance between work & play.

<table>
<thead>
<tr>
<th>SELF CARE ACTIVITY (Cont)</th>
<th>NOW</th>
<th>3 MONTHS</th>
<th>6 MONTHS</th>
<th>12 MONTHS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Practiced effective relaxation time &amp; methods</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Had contact with nature or other calming stimuli.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practiced effective expressions of creative expression.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practiced effective skills for assertiveness.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practiced effective skills for stress reduction.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practiced effective skills for interpersonal communication.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practiced effective skills for time management.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practiced effective skills for changing my thinking when needed.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practiced effective skills in meditation or spiritual practice that is calming to me.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Practiced effective skills for self assessment &amp; self awareness.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Found social Support</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Asker for & got help when needed

Practiced social activism

Balanced work & home

Practiced boundary setting regarding work & personal time.

Effectively addressed the pressures of multiple roles

Practiced realism about how much I can do in one day.

Found support/help at work through peer support.

Found support/help at work through supervision/consultation/therapy

Interacted with role models/mentors

Sought & noted causes of work satisfaction.

Thought about improvements in my self-care plan.

**TOTALS:**

Ref: Charles R. Figley Ph.D. & Robert G. Roop Ph.D. – Compassion Fatigue in the Animal-Care Community 2006
SMALL ANIMAL PROGRAM:

Maureen Anderson, DVM, DVSc, Ph D, ACVIM
Lead Veterinarian, Animal Health & Welfare
Maureen Anderson is a 2003 graduate of the Ontario Veterinary College. After spending some time in mixed animal practice, she returned to OVC to complete a residency and board certification in large animal internal medicine (2008). Her graduate research focused on MRSA in horses and equine personnel. Since then, she has continued to work in the field of veterinary infectious disease control, and completed her PhD in the spring of 2013 at the University of Guelph. Dr. Anderson is currently the Lead Veterinarian – Animal Health and Welfare at the Ontario Ministry of Agriculture, Food and Rural Affairs, where she continues to work in areas bridging animal and public health, including infection control.

Brian Beale, DVM, DACVS
Co-Owner Gulf Coast Veterinary Specialists
Dr. Beale is a board-certified surgeon and co-owner with Gulf Coast Veterinary Specialists in Houston, Texas. He joined them in 1992 after completing his residency and serving on the faculty of the University of Florida’s College of Veterinary Medicine. Dr. Beale has a special interest in arthroscopy, minimally-invasive fracture management, medical management of osteoarthritis, an perioperative pain management. He has authored many book chapters and scientific articles on these topics and is a coauthor of the Small Animal Arthroscopy textbook. Dr. Beale is a frequent invited speaker to local, national and international meetings. He is a past-president of the Gulf Coast Veterinary Education Foundation, and active in the American College of Veterinary Surgeons.

Jennifer Devey, DVM
Veterinarian, Fox Valley Animal Emergency Centre, Saanichton, BC
Jennifer is a graduate of the Ontario Veterinary College, University of Guelph. She completed an internship and an emergency and critical care residency at the Animal Emergency Center in Milwaukee, Wisconsin, and became a Diplomate of the American College of Veterinary Emergency and Critical Care in 1996. She completed a surgical residency in 2004 studying at several private practices in the US. Jennifer has been Director of emergency and intensive care services at a number of large private referral practices in Canada and the United States. She is currently working as an independent consultant and emergency and critical care specialist. She has published over 50 articles and book chapters, is actively involved in research, and is a member of seven professional veterinary organizations around the world. Jennifer enjoys teaching and is actively involved in training residents as well as lecturing and teaching workshops to nurses and doctors.

Sue Ettinger, DVM, DACVIM (Oncology)
Head of Oncology, Animal Specialty & Emergency Center, Hudson Valley, New York
Dr. Sue Ettinger is a practicing veterinary cancer specialist, international speaker, and book author. She is one of approximately 400 board-certified specialists in medical oncology in North America. She attended Cornell University College of Veterinary Medicine. Dr. Ettinger is currently the head of the busy Oncology Department at the Animal Specialty & Emergency Center in the Hudson Valley, New York.

Also known as Dr. Sue Cancer Vet, Dr. Sue is the co-author of second edition of “The Dog Cancer Survival Guide”, which is a best-selling book in small animal health for the last several years, and She also co-hosts the podcast “The Pet Cancer Vet” on radiopetlady.com. Dr. Sue is most passionate about raising cancer awareness, and she has developed “See Something, Do Something, Why Wait? Aspirate®” to promote early cancer detection and diagnosis.
Jennifer Kyes, BSc, DVM, DACVECC

Criticalist, Mississauga-Oakville Veterinary Emergency Hospital

Dr. Jennifer Kyes received her DVM in 2004 from the Ontario Veterinary College. From 2004-2005, she completed her internship in small animal medicine and surgery at the VCA Veterinary Referral & Emergency Center in Norwalk, Connecticut. An interest in emergency medicine and critical care led her to enter a residency program at Ocean State Veterinary specialist in Rhode Island. She completed her residency and became board certified in emergency and Critical Care Medicine in 2009. In 2009, Dr. Kyes joined the Mississauga-Oakville Veterinary Emergency Hospital and Referral Group to lead the critical care department. Dr. Kyes heads up the blood donor program, the small animal internship, the emergency and critical residency and is on the MOVE Pet Foundation Board.

Gary Landsberg, BSc, DVM, DACVB, DECAWBM

North Toronto Veterinary Behaviour Specialty Clinic

Dr. Gary Landsberg received his DVM from the Ontario Veterinary College in 1976 and is a diplomate of both the American college of Veterinary Behaviorists and the European College of Animal Welfare and Behavioural Medicine (Companion Animals). He offers behaviour consultation services at the North Toronto Veterinary Behaviour Specialty Clinic in Thornhill, is a consultant for VIN, and is Vice-President of Veterinary Affairs for CanCog Technologies. Dr. Landsberg has authored and edited over 100 research papers, articles and book chapters, including co-author of Behaviour Problems of the Dog and Cat 3rd edition from Elsevier in 2013. He received the Companion Animal Behavior Award from AAHA in 2000, and the meritorious service award from the Western Veterinary Conference in 2014.

Fernando Mantovani, DVM, DVS, DACVIM Oncology

Veterinary Oncologist, Veterinary Emergency Clinic and Referral Centre

Dr. Fernanda Mantovani is originally from Sao Paulo, Brazil and obtained her veterinary degree from the Sao Paulo State University. After graduating, Dr. Mantovani spent one year taking clinical courses at the University of Wisconsin-Madison, and completed the American Veterinary Medical Association certification program for foreign graduates in 2009. To seek specialization in veterinary oncology, Dr. Mantovani completed a small animal rotating internship at the University of Missouri in 2011, followed by a one year oncology internship and a three year oncology residency/Doctor of Veterinary Science program at the Ontario Veterinary College. Dr. Mantovani joined the team at the VEC in September 2015.

Christopher L. Mariani, DVM, PhD, DACVIM (Neurology)

Associate Professor of Neurology & Neurosurgery, North Carolina State University

Chris Mariani was born near Toronto, Ontario and graduated from the Ontario Veterinary College at the University of Guelph in 1996. He completed a rotating small animal internship at Michigan Veterinary Specialists, followed by one year as an associate veterinarian in Beverly Hill, Michigan. After this he began a residency in neurology and neurosurgery at the University of Florida, College of Veterinary Medicine. This was followed by graduate work in brain tumor immunotherapy, leading to a PhD in Neuroscience from the College of Medicine, also at the University of Florida. Dr. Mariani is a diplomate of the American College of Veterinary Internal Medicine (Neurology subspecialty) and is currently an Associate Professor of Neurology & Neurosurgery at North Carolina State University. His professional interests include epilepsy, inflammatory brain disease and brain tumor therapy. He currently directs the Comparative Neuroimmunology and Neurooncology Laboratory at NC State.

Conny Mosley, DMV, DACVAA, CVA

Staff Anesthesiologist and Integrative Pain Management, 404 Veterinary and Referral Hospital

Dr. Conny Mosley graduated in 1997 from the University of Leipzig, Germany. She completed her thesis in Munich and an anesthesia internship in Glasgow, Scotland. Her passion for anesthesia and analgesia in non-domestic species resulted from her residency at the University of Washington in Seattle. Dr. Mosley grew and learned from her time as a faculty at North Carolina State University, University of Oregon and most recently at OVC in Guelph (although she probably has learned most during her maternity leave in British Columbia). Dr. Conny Mosley has a broad interest in anesthesia and analgesia in nondomestic species ranging from large felids to invertebrates and has written various articles and book chapters on these topics. Her interest in domestic species are “integrative pain management” (perioperative and postoperative as well as chronic pain treatment options). Dr. Mosley is certified in acupuncture from the Chi institute in Florida, which teaches a traditional Chinese approach to veterinary medicine. Dr. Mosley is currently working as a staff Anesthesiologist at the 404 Emergency and Referral Hospital, where she also provides an Integrative Pain Management Service to help patients with chronic pain to improve their quality of life.
Craig Mosley, DVM, MSc
Staff Anesthesiologist, 404 Veterinary and Referral Hospital

Dr. Craig Mosley graduated from the Ontario Veterinary College at the University of Guelph where he also completed a residency and Masters of Science program in veterinary anesthesia. He has been actively involved in many facets of veterinary medicine since graduation including; mixed animal practice, critical care medicine, teaching, management and of course, anesthesia in both private and academic practices throughout North America. Dr. Mosley’s varied experiences have provided him with the foundation for his practical and “real-world” approach to anesthesia and pain management.

Dr. Mosley is currently working as a Staff Anesthesiologist at the 404 Veterinary Emergency and Referral Hospital in Newmarket, Ontario. He is also an independent consultant, Mosley Veterinary Anesthesia Services, providing direct clinical support, continuing education and consultations to both private and academic institutions.

Dr. Mosley has written several book chapters, peer reviewed articles and has lectured extensively at local, national and international conferences on a variety of topics and issues related to veterinary anesthesia. His interests in veterinary anesthesia are diverse, encompassing everything from wildlife and zoo animal anesthesia through to the equipment used to maintain and monitor anesthesia; and from the management of perioperative pain to the issues surrounding chronic pain and palliative care. His wider interests in veterinary medicine include innovations in veterinary education, the science of clinical decision-making and medical errors.

In his spare time, Dr. Mosley keeps bees, dreams of sailing the world and spends time re-exploring his home province with his veterinary anesthesiologist wife and two young daughters.

Robert John Munger, DVM, Diplomate, American College of Veterinary Ophthalmologist
Owner and President, Animal Ophthalmology Clinic, Ltd

Dr. Robert Munger received his DVM degree in 1973 from Texas A&M University. After graduation, he completed an internship in Large Animal Medicine and his residency in Veterinary Ophthalmology at the Veterinary Teaching Hospital, U.C. Davis. He became a Diplomate of the American College of Veterinary Ophthalmologists (ACVO) in 1979. Dr. Munger was an assistant professor in veterinary ophthalmology at the University of Tennessee School of Veterinary Medicine from 1979 to 1983. He has twice served as President of the ACVO Board of Regents and served on numerous committees. Currently he serves on the ABVO Examination Committee through October 2016.

Dr. Munger founded the Animal Ophthalmology Clinic in Dallas, Texas in 1978, and continues his clinical practice there.

O’Brien, Elizabeth, DVM, DABVP (Feline)
Feline Specialist, Visionary for Cat Healthy and Owner of The Cat Clinic and Village Cat Clinic

Dr. Elizabeth O’Brien graduated from OVC in 1985 and became a Diplomate, American Board of Veterinary Practitioners in feline practice in 1999. She is the owner of two feline only practices in Hamilton, Ontario where she continues to be in the trenches as a practitioner and a passionate advocate for the welfare of cats. “Dr. Liz” is the visionary for Cat Healthy, whose mission is to increase the value of owned, homeless and feral cats in communities across Canada. Through her leadership, Dr. Liz brought together the six board certified feline specialists in Canada and industry partners to create the Cat Healthy Initiative. The cathealthy.ca website promotes regular preventive healthcare in cats and includes resources for veterinary teams, shelters and cat owners. Dr. O’Brien was recipient of the CVMA Small Animal Practitioner of the Year Award for 2014.

Anthony Pease, DVM, MS, DACVR
Section Chief, Diagnostic Imaging, Michigan State University

Dr. Anthony Pease graduated from the Virginia-Maryland Regional College of Veterinary Medicine in 1999. He then participated in a large animal surgical and medicine internship at Marion DuPont Scott Equine Medical Center in 2000 as well as obtaining his Masters Degree in Veterinary Medicine. In 2001, Dr. Pease completed a small animal rotating internship at the Affiliated Veterinary Specialists in Maitland, Florida. He then completed a 3-year residency program in diagnostic imaging at Cornell University and became board certified by the American College of Veterinary Radiology in 2005. Dr. Pease has been lecturing extensively on various aspects of diagnostic imaging with special interest in ultrasound for the past 10 years. He is currently the head of Veterinary Diagnostic Imaging at Michigan State University and the 2014 President of the American College of Veterinary Radiology.

Howard B. Seim III, DVM, DACVS
Professor of Small Animal Surgery, Colorado State University

Dr. Seim graduated from Washington State University, completed an internship in Saskatoon, Saskatchewan Canada, and a surgical residency at the Animal Medical Center in New York City. He obtained Diplomate status in the American College of Veterinary Surgeons in 1983, and is currently on the surgical staff at Colorado State University. Dr. Seim was a recipient of the Merck AGVET Award for Creative Teaching, the CSU Award for Instructional Innovation, and was selected as the North American Veterinary Conference’s Small Animal Speaker of the Year in 2009. Dr. Seim is the founder of VideoVet, a Veterinary Surgery Continuing Education video series. (www.videovet.org)
Kelly A. St. Denis, MSc, DVM, DABVP (Feline)

Owner/Veterinarian, Charing Cross Cat Clinic

Dr. Kelly St. Denis earned a Bachelor of Science Degree in Molecular Biology and Genetics from the University of Guelph in 1992, and a Master of Science Degree in Immunology from the University of Toronto in 1994. After working as a laboratory research technician in cancer research for 2 years, Dr. St. Denis returned to university to study veterinary medicine. She completed her Doctor of Veterinary Medicine Degree at the Ontario Veterinary College in 1999. Dr. St. Denis founded the Charing Cross Cat Clinic in Brantford, Ontario in August of 2007. Dr. St. Denis was awarded certification with the American Board of Veterinary Practitioners (ABVP) in the specialty of feline practice in 2013. There are currently only 6 Canadian veterinarians certified in feline practice with the ABVP.

In addition to continuing her full-time clinical work at her practice in Brantford, Dr. St. Denis works as a Consultant in the Feline Internal Medicine folder on the Veterinary Information Network. She is an active participant in the ongoing development of the Canadian Cat Healthy program (http://cathealthy.ca). She sits on the advisory board for the AAFP Feline Friendly Practice program and she acts as a reviewer for the Journal of Feline Medicine and Surgery. Dr. St. Denis continues to enjoy her growing lecture schedule in cities across Canada and within the United States.

At home, Dr. St. Denis enjoys spending time with her two children. She lives with a Labrador-mix rescue, Noelle and 2 domestic longhair cats, Mamasita and Nala. Her clinic is home to 3 additional rescue cats, Mary, Marty and Fuchsia.

Anne Sylvestre, BSc, DVM, DVSc, CCRP, Diplomate ACVS/ECVS

Surgeon, Sylvestre Mobile Surgical Services

Dr. Sylvestre is board certified with the ACVS and ECVS for over 20 years. She was on faculty at the University of Pennsylvania and then the University of Guelph for several years before leaving academics for private practice. She spent seven years in a busy mobile surgical practice in Southwestern Ontario. In 2004, she joined others to open a full service, 24/7 referral hospital in Oakville, allowing her to open a rehabilitation centre and service a far greater number of animals. Dr. Sylvestre has also spent a lot of time at the Animal Hospital of Cambridge, helping out with the client-owned and rescue animals. In 2002, she formed a continuing education business with the focus being on practicality and hands on learning for veterinarians. Dr. Sylvestre has since given well over 60 workshops through Focus and Flourish. She has been invited to speak to many international and regional meetings. She has published research and clinical papers, book chapters and, several electronic books on veterinary surgery and rehabilitation.

Jinelle A. Webb, DVM, MSc, DVSc, DACVIM (Small Animal Internal Medicine), Adjunct Professor OVC

Co-owner, Small Animal Internal Medicine Department, Mississauga-Oakville Veterinary Emergency Hospital

Dr. Jinelle Webb received her DVM in 2001 from the Ontario Veterinary College. An interest in small animal internal medicine led to a residency and board certification at the OVC, which she completed along with a DVSc in 2005. In 2006, Dr. Webb started the internal medicine and oncology service at the Mississauga-Oakville Veterinary Emergency Hospital, where she remains today seeing clinical cases and performing small research projects. She is an Adjunct Professor at the OVC. Dr. Webb is a published author and speaker.

Shannon Westgarth, DVM, DACVIM, DVSc

Internal Medicine Specialist

Dr. Shannon Westgarth attended Queen’s University to obtain her BSc in life sciences. She then went on to the Ontario Veterinary College and graduated with her DVM in 2011. She did a small animal rotating internship at the OVC and then stayed on for an internal medicine residency. Dr. Westgarth both completed her thesis to achieve her doctor of veterinary science and became a diplomate of the American College of Veterinary Internal Medicine in 2015.

FOCUS ON INFECTION:

Michelle Evasion, DVM, DACVIM

Internist, Michelle Evasion Veterinary Internal Medicine & Nutrition Consulting

Michelle Evasion is a small animal internist. She has previously worked in specialty clinical practice at large private referral centres, in academia as clinical faculty at Tufts University, and in the pet food industry (Royal Canin Canada, and currently Rayne Clinical Nutrition Canada). Her interests encompass ‘just about anything and everything’ that might improve patient care and aid pet owner compliance.
Jason Ward Stull, VMD, MPVM, PhD, DACVPM

Assistant Professor, The Ohio State University, College of Veterinary Medicine

Dr. Jason Stull is a veterinary epidemiologist, holding a VMD from the University of Pennsylvania, Masters in Preventive Veterinary Medicine from the University of California at Davis, and PhD in veterinary infectious disease from the University of Guelph. He is a Diplomate of the American College of Veterinary Preventive Medicine. He is currently an Assistant Professor in the Department of Preventive Veterinary Medicine at The Ohio State University, where his research focuses on infectious disease in companion animals, the role of animals in the transmission of zoonotic pathogens and infection control in the veterinary clinic and non-clinic environments.

Scott Weese, DVM, DVSc, DipACVIM

Professor, University of Guelph

Dr. Scott Weese is a veterinary internist and microbiologist, and a Diplomate of the American College of Veterinary Internal Medicine. He is a Professor at the Ontario Veterinary College, University of Guelph. He is also Chief of Infection Control at the Ontario Veterinary College Teaching Hospital and holds a Canada Research Chair in zoonotic diseases. His clinical and research interests focus on infectious diseases, including antimicrobial resistance, urinary tract disease, emerging diseases and zoonotic diseases.

EQUINE:

Eric J. Abrahamsen, DVM, Diplomate American College of Veterinary Anesthesiologists

Owner, Equine Anesthesia Services

Dr. Eric Abrahamsen graduated from Michigan State University College of Veterinary Medicine in 1978. As a live-in student intern, he spent 15 months assisting with colic anesthesia, which eventually led to daytime assignments anesthetizing research horses. Dr. Abrahamsen began his professional career working on the thoroughbred racetracks of New Jersey. To broaden his experience, he spent a year in a busy Minneapolis area equine practice before returning to the racetracks of New Jersey and Miami. His interest in exercise physiology eventually led him to Washington State University as a rotating equine resident and graduate student. While at WSU he filled in when anesthesia staff was not available for colic surgeries. Finally yielding to his strong interest in equine anesthesia, Dr. Abrahamsen received specialty training at the Ohio State University and the University of Pennsylvania’s New Bolton Center. He is a Diplomate of the American College of Veterinary Anesthesiologists. Dr. Abrahamsen taught large animal anesthesia at the University of Pennsylvania, University of Florida, and the Ohio State University. Since “retiring” he has focused on improving anesthesia care of practices through training and continuing education.

Myra Barrett, DVM, MS, DACVR

Large Animal Diagnostic Imaging Director, Colorado State University

Dr. Myra Barrett earned her DVM from Colorado State University. After graduating, she completed a year long internship at Oakridge Equine Hospital, in Edmond, Oklahoma. Dr. Barrett underwent a non-conforming radiology residency in order to particularly focus on equine diagnostic imaging. The residency was based at Colorado State but included training with multiple equine imaging experts in the US and internationally. At the same time, Dr. Barrett obtained a masters degree through the Orthopaedic Research Center at CSU. After achieving diplomat status in the American College of Veterinary Radiology, she stayed on at CSU as a special appointment faculty and later accepted a position as a tenure-track assistant professor of radiology. Dr. Barrett is the head of the equine diagnostic imaging service at CSU, which includes all modalities of clinical diagnostic imaging of horses, as well as training of diagnostic imaging residents, equine diagnostic imaging interns and fellows and equine sports medicine residents. She is also involved with canine sports medicine service to offer musculoskeletal ultrasound examinations of canine sports medicine patients. Dr. Barrett’s primary research and clinical interests are equine musculoskeletal imaging and comparative imaging, and she works closely with the equine surgery and sports medicine services. She has spoken at multiple large national meetings and is regularly involved in continuing education courses, including courses that draw an international audience. Dr. Barrett is dedicated to the advancement of the specialty of equine diagnostic imaging and is currently the President of the Large Animal Diagnostic Imaging Society, a subgroup of the American College of Veterinary Radiology.
PRACTICE MANAGEMENT:

Glenn Armstrong, DVM, MBA
Managing Partner, Veterinarian, Coventry Animal Hospital
After graduating from OVC in 1992, Dr. Glenn Armstrong practiced as a mixed animal veterinarian for 5 years with the Mitchell Veterinary Services Group. In 1997, he joined the partnership and put his interest in business to work, involving purchasing, personnel management and growth promotion. In 2013 Dr. Armstrong entered the MBA program at Ivey Business School, University of Western Ontario, where he focused on the applications of the curriculum as it applies to current veterinary industry conditions and how business strategy could be simplified to apply in daily decisions. After graduation in 2015, he went back to practice to test and improve these concepts in a clinical setting. Dr. Armstrong’s current passion lies in helping veterinarians achieve their goals through the application of personalized business models with a focus on creating and communicating value.

Debbie Boone, BS, CCS, CVPM
2Manage Vets
Debbie Boone managed AAHA accredited hospitals for 23 years. Her rather unique skill set includes experience with small animal, mixed animal, specialty, emergency and even shelter management. Acknowledged as a leader in change implementation and employee engagement, her skills as a trainer & speaker have been utilized by most major animal health manufacturers to bring value added services to their customers. Debbie’s style as a presenter is to bring real life stories to her students to educate and entertain leading to glowing reviews and a message that “sticks”. She has been a speaker for the WVC, AAHA, Midwest and Atlantic Coast Veterinary Conferences and always fills the room. She is the instructor for Patterson Veterinary Supply’s Communication and Customer Service class, and consults with 2 ManageVets Consulting. Debbie has also co-authored “The Veterinarian’s Guide to Healthy Pet Plans – How to Design and Implement Successful Preventive Care Plans” with Dr. Wendy Hauser.

Chris Doherty, DVM
Economic Analyst, Ontario Veterinary Medical Association
Dr. Chris Doherty completed his BSc at the University of Guelph and his DVM at the Ontario Veterinary College. After a few years in companion animal practice, he began working as an economic analyst for the Ontario Veterinary Medical Association, involved in the research and analysis of veterinary economics, practice management and business development. While his primary role is the business of veterinary medicine, he continues the practice of veterinary medicine, primarily through voluntary veterinary work, as well as locum veterinary work.

Karen Felsted, CPA, MS, DVM, CVPM, CVA
President, Panthera Veterinary Management Consulting
Dr. Felsted is a CPA as well as a veterinarian and has spent the last 15 years working as a financial and operational consultant to veterinary practices and the animal health industry. She also spent three years with the National Commission on Veterinary Economic Issues as CEO. She has written an extensive number of articles for a wide range of veterinary publications and speaks regularly at national and international veterinary meetings. She is the past treasurer of VetPartners, a member of the Veterinary Economics’ Editorial Advisory Board, a past member of the CVPM board of directors and the past treasurer of the CATalyst Council. In 2011, Dr. Felsted was awarded the Western Veterinary Conference Practice Management Continuing Educator of the Year and in 2014, the VetPartners Distinguished Life Member Award.

Mary Gardner, DVM
Lap of Love
Dr. Gardner loves a grey muzzle! Her professional goal is to increase awareness and medical care for the geriatric veterinary patient and to help make the final life stage to be as peaceful as possible, surrounded with dignity and support for all involved. A University of Florida graduate, she found her niche by cofounding Lap of Love which is now the nation’s largest organization of veterinarians (85) dedicated to end of life care in the home. Dr. Gardner not only is a practicing veterinarian, but she is also the company’s Chief Technology Officer and is responsible for the company’s proprietary software, web presence and all internet marketing. She speaks internationally at conferences and veterinary schools on geriatric medicine, marketing, leadership, body language and finding your passion.

When not working, she enjoys playing golf and a rousing game of monopoly!
Darren Osborne, MA Economics

Director of Economic Research, Ontario Veterinary Medical Association

Darren Osborne is the Director of Economic Research for the Ontario Veterinary Medical Association (OVMA) and Economic Consultant for the Canadian Veterinary Medical Association and several State Veterinary Medical Associations. Darren conducts economic research and analyses data in order to provide thousands of veterinarians with Fee Guides, Economic Reports, Personal Benchmark Reports, Reports on Compensation and Benefits for Associate Veterinarians and Non-DVM Wage Reports. More recently, Darren has partnered with Idexx to create a Practice Dashboard Report that automatically extracts financial information from veterinary practice management software to provide monthly metrics that track performance and compare to the average veterinary hospital in the region. Darren attended York University and completed his Master’s Degree in Economics in 1992.

When he is not crunching numbers, you can find Darren playing a guitar, running, swimming or cycling.

Terra Shastri, B. Comn, Dip. B.A.

Manager of Business Development, Ontario Veterinary Medical Association

Teaching and building a client-centric culture is Terra’s passion. With over 25 years experience inspiring multi-disciplinary teams to deliver an amazing client experience, Terra knows how to energize staff and get them motivated to provide “WOW” experiences. She holds a degree in Communication Studies and a diploma in Business Administration from Wilfrid Laurier University, and is a graduate of the Walt Disney Institute (School of Leadership). Before joining the Ontario Veterinary Medical Association in 2008, Terra spent 20 years helping small businesses better market their services, improve client service, increase revenues, improve staff retention and manage change. She also managed 17 teams and was responsible for staff training & development while meeting aggressive business objectives. Currently, Terra holds the position as Ontario Veterinary Medical Association’s Manager of Business Development assisting OVMA members with the business side of their practice and teaching the popular JumpStart! Boot Camp workshop she created.