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## Megan Brashear, BS, CVT, VTS (ECC)

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# Understanding Fluid Therapy

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Megan Brashear, BS, CVT, VTS (ECC)

Fluid therapy is a potentially life-saving treatment performed in veterinary hospitals multiple times a day. Patients are supported during surgical procedures, supported during minor illness, and resuscitated from major fluid losses. A healthy animal is approximately 60% water, and that water is distributed through different compartments throughout the body:

- Intracellular fluid makes up the largest percentage of total water in the body. This is fluid inside the cells and is controlled by the flow of water, electrolytes, and other solutes across cell membranes.
- Extracellular fluid is all of the fluid not inside cells, and where water is lost first during sickness or disease states. Extracellular fluid is further divided into:
  - Interstitial fluid which is fluid surrounding cells in muscles and other tissue
  - Intravascular fluid which is the water portion of plasma in circulating blood
  - Transcellular fluid which is found in dense connective tissue and bone. This makes up a small portion of the body's total water

When a patient becomes compromised due to fluid loss, replacement and support is administered in the form of crystalloid or colloid fluids. Crystalloid fluids are water and electrolytes and are used to replace volume. Crystalloids can be administered in large volumes over short periods of time and can be administered over days at a time. Different types of crystalloid fluids may be chosen to treat various electrolyte abnormalities which may occur depending on electrolyte needs. Crystalloid fluids will shift from the intravascular space into the interstitial and intracellular space; this is beneficial to treat a variety of causes for fluid loss and is important to remember when monitoring patients on IV fluid therapy.

Colloids are fluids comprised of large molecules which will remain in the intravascular space longer than crystalloid fluids. Colloids can also help “pull” fluids into the intravascular space to increase circulating volume and give blood pressure support. Blood products such as packed red blood cells and frozen plasma are natural colloids. Vetstarch™ is a synthetic colloid that can achieve similar results. There is current controversy in both human and veterinary medicine regarding the use of synthetic colloids; there is concern for acute kidney injury in critically ill human patients. At this time there is no evidence that veterinary patients are under the same risks but caution should be exercised when administering synthetic colloids.

When determining fluid therapy needs for a patient, a physical exam with a patient history must be taken so that important questions can be answered. The veterinarian and nursing team must be able to answer the following questions:

### **Is the patient in hypovolemic shock that requires immediate attention?**

Understand the physical signs of hypovolemic shock. Tachycardia, weak pulses, pale mucous membranes, prolonged capillary refill time, cool extremities, and decreased mentation can signal a critical fluid deficit that must be addressed immediately. Hypovolemic shock is treated with crystalloid fluid replacement; large volumes are bolused until the patient begins to respond with a decreasing heart rate and increased blood pressure. This initial rapid administration of fluids is referred to as the resuscitation phase of fluid therapy and is completed quickly before rehydration occurs.

### **Is the patient dehydrated?**

Not every patient that needs fluid therapy is dehydrated but the majority of them are experiencing dehydration. Evidence of dehydration can be determined with both laboratory (elevated PCV and TS) and physical exam results. Dehydrated patients will have a loss of elasticity in their skin, will have sticky mucous membranes, tachycardia, and sunken eyeballs. Dehydration is replaced with fluids after the resuscitation period and it can take 24-36 hours to fully hydrate a patient.

### **Can the patient consume enough water to remain hydrated?**

The answer to this question can help determine if the patient must be treated in the hospital or perhaps as an outpatient. As hospitalized patients begin to recover, water should be offered in the hospital and fluid requirements adjusted as they begin to drink water.

### **What type of fluids should be given?**

This question is dependent on many factors and includes administering crystalloid fluids, colloids, or both. It also concerns the type of crystalloid fluids based on the patient's electrolyte status and make-up of different fluids. Electrolyte additives can be introduced to the fluids to tailor them to the patient needs.

Within the water in the body is electrolytes. Any discussion of fluid therapy cannot be complete without an understanding of electrolytes and the role they play in fluid shifts within the body. Different cell membranes are made with different permeability to allow for these shifts. The following vocabulary is necessary to understand when learning about fluid therapy:

- Osmosis: the diffusion of fluid across a membrane from an area of low solute concentration to an area of high solute concentration in order to make both sides equal.
- Hypotonic: referring to a solution with a solute concentration less than plasma, causing fluid to shift out of the intravascular space.
- Isotonic: referring to a solution with a solute concentration equal to plasma.
- Hypertonic: referring to a solution with a solute concentration greater than plasma, causing fluid to shift into the intravascular space.

The kidneys are responsible for the sodium balance within the body. Through the renin-angiotensin-aldosterone feedback system, the kidneys recognize when perfusion is decreased and release renin. Renin signals the liver to release angiotensin I. In the lungs, angiotensin converting enzyme (ACE) converts angiotensin I into angiotensin II. Angiotensin II promotes sodium uptake by the kidneys and will cause vasoconstriction to increase circulating volume and increase blood pressure thereby improving perfusion to the kidneys. Angiotensin II signals the adrenals to release aldosterone which will further encourage sodium uptake and potassium excretion. In normal body states the kidney will excrete the amount of sodium ingested each day to maintain a healthy balance within the body.

Due to the principle of osmosis, fluid follows sodium. An increase in circulating plasma sodium means the concentration of sodium is higher outside of the cells. Fluid follows this gradient and will increase intravascular volume. When circulating sodium levels are decreased, this means the sodium content in the intracellular space is greater than the circulating plasma, and fluid will flow to the intracellular space. In patients with sodium disorders, it becomes important to monitor levels and anticipate changes in fluid shifts and monitor for complications.

Clinical signs of hypernatremia are often noticed when sodium serum levels reach greater than 170mEq/L. Hypernatremia can be caused by lowered water intake by the animal, fluid losses, or a dramatic intake of sodium (salt toxicity). Increased sodium in the circulating plasma will cause fluid shifts to the intravascular space in an attempt to dilute the plasma sodium levels. As this happens within the brain, vessels can rupture and cause cerebral hemorrhage.

The clinical signs seen with hypernatremia will vary depending on the speed that animal experienced the change. As a general rule, acute changes will cause acute signs, with more gradual changes in sodium the body has time to adjust and the animal may appear normal. At any rate, the clinical signs of hypernatremia are neurologic. Ataxia, depression, seizures, coma, and death can all be seen with an elevation in sodium levels.

When treating patients with hypernatremia it is important to treat any shock and hypovolemia first, as these conditions can lead to death, the focus on addressing any underlying disease. In chronic disease states, the body produces idiogenic osmoles which act as sodium molecules in the brain to prevent large fluid shifts from the intracellular compartment. If correction to normal sodium levels occurs too quickly, the body will respond as if in acute hyponatremia and this is not desired. Correction of chronic sodium abnormalities should happen slowly; the goal is to decrease sodium levels 0.5 (for acute) – 1 (for chronic) mEq/hr. To begin treatment, choose a fluid with a sodium content closest to the patient's sodium levels. In many cases this is 0.9%NaCl. Once dehydration is corrected the patient may need fluid changes to balanced electrolytes replacement or maintenance fluids or a combination of different types of crystalloid fluids to strike the appropriate balance, Blood work should be checked often to make sure levels are not falling too quickly, and the patient monitored closely for any neurologic changes.

Clinical signs of hyponatremia are often evident at serum levels less than 140mEq/L. Hyponatremia results from volume loss (from vomiting or diarrhea), administration of diuretics, hypoadrenocorticism, kidney failure, or increased water intake (water toxicity). Decreased levels of sodium in the circulating plasma will cause a fluid shift from the extracellular space to the intracellular space and can cause cell rupture and cerebral edema.

The clinical signs seen with hyponatremia will vary depending on the speed that the animal experienced the change. As a general rule, acute changes will cause acute signs, with more gradual changes the body has time to adjust and the animal may appear normal. The clinical signs of hyponatremia are weakness, vomiting, ataxia, and seizures.

When treating patients with hyponatremia, it is important to assess their volume status first. Hyponatremia can exist with dehydration, normal hydration, or overhydration and fluid therapy will need to support that; assessing for dehydration must occur throughout the patient's hospital stay. Sodium supplementation will need to be provided to these patients using crystalloid fluids and care must be taken not to raise their sodium level too quickly. The goal is to raise serum sodium levels but 0.5 (for acute) to 1 (for chronic) mEq/hr. Fluid choices can range from hypertonic saline (7% NaCl) to 0.9%NaCl, to a balanced crystalloid solution (NormosolR, LRS) to raise sodium levels to normal. Blood values should be checked often to ensure that levels are not rising too quickly and the patient monitored closely for any neurologic changes.

Chloride will follow sodium and will rarely become hypo or hyper on its own. By treating sodium disorders, chloride levels will become normal as well. Chronic vomiting and loss of body acid can lead to chloride alterations. Severe changes in chloride can lead to ECG changes; these patients must be monitored closely as fluids are administered.

Potassium levels are closely monitored and replaced or diluted with fluid therapy. Potassium is the main intracellular electrolyte; normal circulating plasma levels of potassium are much lower than normal sodium or chloride levels. Clinical signs of hyperkalemia are often noticed at levels higher than 7.5mEq/L. Excess potassium is excreted from the body in the urine, so the most common cause of hyperkalemia is urinary obstruction followed by acute or chronic kidney failure. Patients experiencing a hypoadrenocorticism crisis can also suffer from hyperkalemia. Increased circulating potassium can cause muscle weakness, cardiac arrhythmias and death.

The clinical signs of hyperkalemia are often seen in connection with the heart. Bradycardia can occur, and on ECG you may see tall, tented T waves. As potassium levels continue to climb, the P waves disappear, and the T waves continue to widen until the patient suffers cardiac arrest. Hyperkalemia is a true emergency that requires a quick correction of levels and correction of the underlying disease.

Treatment for hyperkalemia is aimed at diluting circulating levels potassium with intravenous fluids and promoting cell uptake of potassium. In cases of urinary obstruction, balanced crystalloid fluids, while containing some potassium, is safe to use in these patients prior to relieving the obstruction. An IV bolus of dextrose will cause cells take in the excess glucose. As this happens the cells will also take in potassium. In severe cases of hyperkalemia IV insulin may be administered to achieve the same effect.

Clinical signs of hypokalemia are often noticed at levels lower than 3.0mEq/L. Hypokalemia most commonly in chronic kidney failure patients, as they are excreting too much in their urine. The other common cause of hypokalemia is GI loss from vomiting and diarrhea. Muscle weakness (including the heart muscle) is the biggest concern with hypokalemia, and low potassium can decrease kidney perfusion making kidney disease worse.

Patients suffering from hypokalemia will express ventroflexion and profound weakness. Respiratory status must be monitored as some of these patients are too weak to appropriately ventilate and may need support during treatment. It is important to monitor heart rate and blood pressure on these patients as their levels are corrected.

Treatment for hypokalemia is aimed at replacing potassium levels, most commonly this is done through IV crystalloids. Potassium chloride is added to fluids to increase the amount given but should not exceed a rate of 0.5mEq/kg/hr. Technicians and nurses should be comfortable calculating the rate of potassium as fluid rates change. Fluids containing potassium should never be bolused.

Colloid use is tailored to patient need and hospital supply. Natural colloids such as packed red blood cells can be administered to those patients suffering the clinical signs of anemia. Signs such as tachycardia, tachypnea, hypotension, pulse quality, and weakness will all direct choices on blood transfusions. Synthetic colloids may be used to support blood pressure in cases of large volume loss, anaphylaxis, or protein losing disease. Colloids carry the risk of patient immune mediated reaction (in the case of natural colloids) and the emerging knowledge of potential kidney damage with synthetic colloids. This knowledge should lead to judicious use of colloids and when other avenues of therapy have been exhausted.

### **By what route should fluids be given?**

Many patients needing fluids need them to quickly replace losses (such as those in hypovolemic shock) or because the animal cannot take in fluids due to illness or obstruction. These patients will obviously require intravenous administration of fluids. However, a population of patients with minor disease or those that need daily support for chronic disease can receive subcutaneous fluids to maintain hydration. Any patient that suffers from vasoconstriction due to poor perfusion, hypothermia, or shock will be less successful with the subcutaneous route of administration.

## How much fluid should be given?

This question should be asked and answered multiple times during a patient's stay in the hospital. The rate of fluids should relate to the patient's physical exam, hydration status and nutritional status. In states of hypovolemic shock, large volumes of crystalloid fluids are administered intravenously until an improvement in tachycardia, blood pressure, pulse quality, and mucous membrane color is noted. This resuscitation phase of fluid therapy may see patients receiving 20, 50, even 90ml/kg of fluids. Isotonic crystalloids are quickly administered and the patient monitored often. Once physical exam parameters are normal and remain normal on lower rates of fluid can the fluid plan change.

Once the resuscitation is complete, the patient then has their hydration deficit replaced. This volume is calculated by determining their dehydration percentage and multiplying that by their body weight. This volume (in liters) is then divided into 24-36 hours and replaced over that time frame. Added to this is the maintenance fluid requirement, often calculated as 40-60ml/kg/day. For small dogs and cats and very large dogs, the quick calculation may underserve them, and more accurate calculations based on basal energy requirements may be performed.

Lastly, losses must be calculated into a fluid therapy plan. Patients with large volumes of vomit, diarrhea, or urine output need those losses corrected. Urinary catheters can help to quantify urine output, and nasogastric tubes can help to quantify gastric contents. Bedding can be weighed before and again after soiling to determine approximate losses. The nursing team can also guess at fluid volumes of vomit and diarrhea.

All patients on fluid therapy must be monitored closely during their hospital stay. Nurses must be confident in assessing hydration, monitoring for clinical signs of shock, and in looking for signs of fluid overload in patients. Thoracic auscultation, pulse quality assessment, mucous membrane color and nature, blood pressure, and skin turgor should be tested multiple times throughout the shift and when any problems are noted. Urine output and urine specific gravity can be monitored as a way to gauge success of fluid therapy as well as PCV/TS and electrolyte levels.

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# Understanding Acid/Base and Blood Gas Measurements

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Megan Brashear, BS, CVT, VTS (ECC)

A patient's acid/base status is measured to analyze the hydrogen content of blood. Hydrogen is produced by the normal metabolism of protein, and is necessary for enzyme function and cell structure. The body needs to maintain a narrow range of pH in order to function correctly. When we measure pH, we are measuring  $H^+$ . Hydrogen is an acid and is inversely related to the pH. A low pH indicates an acidic environment, and an increased level of  $H^+$ . A high pH indicates a basic, or alkalotic environment, and a decreased level of  $H^+$ .

In order to maintain a normal pH, the body utilizes buffers. A buffer can either absorb or donate  $H^+$  in attempt to keep levels constant and minimize pH changes. Bicarbonate ( $HCO_3$ ) is the main extracellular buffer. It is the main metabolic indicator of acid/base status. Bone is also a source of calcium carbonate and calcium phosphate and can be utilized to buffer blood as needed.

Carbon dioxide ( $CO_2$ ) is the last component of evaluating the acid/base status of a patient. It is the main respiratory indicator of the acid/base status.  $CO_2$  when combined with water in the presence of carbonic anhydrase will create an acid, so  $CO_2$  has the same relationship with pH as hydrogen does. We normally think of  $CO_2$  as an element of respiratory function, but there is a metabolic balance to  $CO_2$  as well, as cell metabolism is necessary for normal  $CO_2$  concentrations. Elevated or decreased  $CO_2$  levels will affect the pH.

The body needs the pH maintained in a narrow normal range and will begin to compensate for even minor changes in pH. When looking for the primary acid/base problem in a patient, you can use the blood gas to determine which is the primary disorder, and which is the compensatory mechanism employed to balance it out. Once you know what the problem is, you can then begin to anticipate the patient's response, anticipate the problems that may arise from that patient, and begin the steps to correct the underlying problem.

The two body systems involved with acid/base regulation and compensation are the renal system and the respiratory system. The kidneys regulate the excretion of  $H^+$  and the uptake of  $HCO_3$ . Once a change in pH is noted, the kidneys go to work either increasing or decreasing these levels, and this work can take hours or days to create a noticeable change. The respiratory system can enact change almost immediately. Remember that  $CO_2$  can combine with water and create acid, so by changing the respiratory rate to either increase or decrease  $CO_2$  levels, the pH can be changed.

Measuring pH is important in certain populations of patients, as minor changes can cause major problems. Patients suffering from acidosis can have decreased cardiac output, decreased cardiac contractility, a decreased response to catecholamines (often leading to hypotension), antagonism to insulin, and hyperkalemia (potassium will shift out of the cells in exchange for H<sup>+</sup>). Patients suffering from alkalosis can experience muscle spasms, stuporous mentation, hypocalcemia and hypokalemia (potassium shifts into the cells in exchange for H<sup>+</sup>).

Metabolic acidosis is the most common disorder seen in patients. We can see in patients with lactic acidosis (a result of shock and poor perfusion), renal failure (as the kidneys are a major player in choosing to retain or excrete HCO<sub>3</sub>), DKA (ketone production contributes to acidosis), and GI losses (vomiting and diarrhea result in a loss of HCO<sub>3</sub> through the GI tract). Remember in acidosis that potassium shifts out of cells in exchange for H<sup>+</sup> and hyperkalemia may result.

For severe metabolic acidosis, treatment with sodium bicarbonate may be ordered. Sodium bicarbonate, when administered, will buffer the acid, but is broken down into water and CO<sub>2</sub> which then the lungs must be able to exhale. Patients with metabolic acidosis and any form of respiratory distress should have an arterial blood gas evaluated before administering bicarbonate. This drug can cause dramatic changes in pH as well as electrolyte abnormalities and must be used with caution. Any administration IV should be slow; many treatment regimens call for a CRI administered over 4-6 hours.

Metabolic alkalosis is seen most commonly with GI outflow obstructions, but can also occur when the patient's chloride levels drop accompanied by poor perfusion. In this situation the patient is attempting to reabsorb water and sodium to increase vascular volume with results in the reabsorption of bicarbonate even in the face of alkalosis. These patients often respond favorably to crystalloid fluids which contain chloride. Remember in alkalosis that potassium shifts in to cells in exchange for H<sup>+</sup> and hypokalemia may result. Patients suffering from refeeding syndrome may suffer from metabolic alkalosis, as well as those with severe potassium deficiency, renal insufficiency, or those on diuretic therapy.

Respiratory acidosis occurs anytime the patient is hypoventilating and not eliminating CO<sub>2</sub>. CO<sub>2</sub> is an acid and will cause a drop in pH. Patients suffering from respiratory acidosis are often critical, as this disturbance is almost always associated with hypoxia. This can be caused by medications which will relax thoracic muscles and depress the respiratory center, neuromuscular disease, upper airway obstruction, pleural space disease (pneumothorax, effusion, diaphragmatic hernia), and gas exchange disorders such as PTE, pneumonia, and pulmonary edema.

Respiratory alkalosis can occur when a patient is hyperventilating and breathing off too much CO<sub>2</sub> causing the pH to rise. There are minimal health effects of this and this primary disturbance is rare to see clinically.

Compensation:

	pH	CO2	HC03	Kidneys	Lungs
Metabolic Acidosis	↓	↑	↓		Increase respiratory rate
Metabolic Alkalosis	↑	↓	↑		Decrease respiratory rate
Respiratory Acidosis	↓	↑	↓	Hang on to HC03, excrete H+	
Respiratory Alkalosis	↑	↓	↑	Decrease rate of H+ excretion	

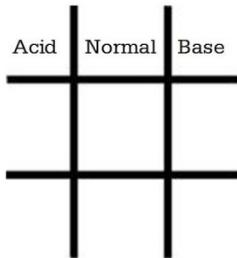
When you are ready to interpret blood gas results, you first want to determine the primary disorder, decide if the body is compensating, and then look at the oxygen values and determine the level of hypoxia (if any). There's no getting around memorizing normal values. To determine the primary disorder and compensation, you'll look at pH, CO2, and HC03.

Base Excess is a number that may be reported on blood gas analysis, and is the amount of strong acid or base needed to convert 1L of blood to a normal pH given a normal body temperature and normal PCO2. If the BE is negative, indicates metabolic acidosis, a positive BE points to metabolic alkalosis. Normal is -4 to +4.

There are a number of relatively complicated ways to determine acid base disorders and many of them involve math and formulas and determining level of compensation. Survivenursing.com has given us a better way. The following is a quick and easy method which makes determining the primary disorder as simple as Tic Tac Toe. To start, know your normal values and what they mean. Write out this chart and keep it close to your blood gas analyzer, in your pocket, or in your brain.

	ACIDIC	→	BASIC
pH	7.35		7.45
CO2	45		35 respiratory component
HC03	18		24 metabolic component

Remember that the higher the CO<sub>2</sub> the more acidic the blood, so keep that high CO<sub>2</sub> on the acid side. Also know that the CO<sub>2</sub> is your respiratory value, and HCO<sub>3</sub> is your metabolic value. Next, draw a tic tac toe grid and from left to right in the top three boxes label Acid, Normal, and Base.



Look at the pH value and place it in the appropriate column. Then do the same for CO<sub>2</sub> and HCO<sub>3</sub>, placing them in the appropriate columns. Wherever there are three values in a row (including “Acid” “Normal” and “Base”), that is the patient’s primary disorder. If the CO<sub>2</sub> is in that three in a row, the disorder is respiratory. If the HCO<sub>3</sub> is in the three in a row the disorder is metabolic. Then look to see where the ‘stand alone’ value is. If it is in the opposite column then the patient is partially compensating for the disorder. For example, a patient has the following values on an arterial blood gas:

- pH 7.288
- pCO<sub>2</sub>: 56.9
- HCO<sub>3</sub>: 31.7

Place the pH in the Acid column. Place pCO<sub>2</sub> in the Acid column. Place the HCO<sub>3</sub> in the Base column. There are three in a row in the acid column, so the primary disorder is an acidosis (which is hopefully recognized with the low pH). Because the CO<sub>2</sub> is also in that acid column, the primary disorder is a respiratory acidosis. The HCO<sub>3</sub> is abnormal in the opposite (Base) column so the patient is partially compensating. It is a partial compensation because the pH is still abnormal.

There are times when the values will make a horizontal tic tac toe, with the pH in the normal category. This is a patient that is fully compensating but still has an underlying disorder that needs correcting. In these cases, look at the pH and determine which side of normal it sits. If the pH is greater than 7.4, the disorder is alkalotic, look in the base column and whichever value is there will be the disorder (if CO<sub>2</sub> is in the base column, then the patient has a fully compensated respiratory alkalosis). If the pH is less than 7.4, the disorder is acidosis and whichever value is in the acid column will be the disorder (if HCO<sub>3</sub> is in the acid column, then the patient has a fully compensated metabolic acidosis).

In those patients with a respiratory issue, after looking at the acid/base and determining the primary problem, it is on to looking at the respiratory component of the blood gas. This will involve examining the PaO<sub>2</sub>, FiO<sub>2</sub>, and performing some math to determine the origin of the hypoxia. It is important to remember the difference between oxygenation and ventilation, and which values are measuring which.

Oxygenation, measured by PaO<sub>2</sub> (the partial pressure of oxygen in arterial blood) is measuring how well blood is flowing through the lungs and picking up oxygen which is then delivered to the tissues. Ventilation (V) and perfusion (Q) must happen in the alveoli in order for proper oxygenation to occur. When blood travels from the left atrium, through the lungs and to the left ventricle without becoming properly oxygenated it is called venous admixture, and is due to a V/Q mismatch. Low V/Q is from disease such as pneumonia, asthma, PTE, inflammation, pulmonary edema, and neoplastic changes in the lungs. No V/Q means no blood is getting to areas of the lungs and is caused by atelectasis and severe pleural effusion leading to alveolar collapse. Diffusion impairment caused by pulmonary fibrosis or COPD is rare in small animals but can cause V/Q mismatch and decreased PaO<sub>2</sub>.

PaCO<sub>2</sub> is the value that represents ventilation. Ventilation is the mechanics of breathing; the body's ability to take in oxygen, perform gas exchange, and exhale CO<sub>2</sub>. PaCO<sub>2</sub> is not only a measurement of ventilation but also perfusion and cellular metabolism. Low PaCO<sub>2</sub> is rarely clinically significant and can be caused by fear and stress, pain, or attempted compensation of metabolic acidosis. High PaCO<sub>2</sub> is caused by the lungs inability to move air. Neurologic disease, spinal cord injury, upper airway disease, trauma involving the thoracic wall or muscles, and drugs used in anesthesia can all cause hypoventilation and elevated PaCO<sub>2</sub> levels.

Always look at the PaO<sub>2</sub> and PaCO<sub>2</sub> numbers in relation to each other. Is the animal breathing faster (lower CO<sub>2</sub>) to compensate for low PaO<sub>2</sub>? A quick method to determining lung function is to use the "120 Rule". To do this, simply add the PaO<sub>2</sub> and PaCO<sub>2</sub> results from the arterial blood gas. If the two added together are  $\geq 120$ , the patient has adequate lung function. If the two added together are  $\leq 120$ , the patient has abnormal lung function. The 120 rule only applies to patients breathing room air at sea level.

Patients that have incredible respiratory effort but normal blood gas values must be watched closely, as they can suffer from respiratory exhaustion and need mechanical ventilation.

The FiO<sub>2</sub> is the fraction of inspired oxygen, and represents the percentage of oxygen the patient is breathing. Room air is 21% oxygen, so the FiO<sub>2</sub> of room air is 0.21. A patient with bilateral nasal oxygen cannulas is breathing 40% oxygen and has a FiO<sub>2</sub> of 0.4.

After evaluating the PaO<sub>2</sub> and PaCO<sub>2</sub> values, the next step is to calculate the A-a gradient. By calculating the A-a gradient we can look deeper into the cause of the hypoxia and differentiate between hypoventilation and lung disease. The capital "A" is the measurement of oxygen present in the alveoli, and the lowercase "a" is the arterial concentration of oxygen. The PaO<sub>2</sub> is the arterial measurement, so we need to calculate the alveolar (A) concentration. This formula is calculated assuming the patient has a FiO<sub>2</sub> is 0.21 (room air). Many blood gas analyzers will calculate the A for us, but memorize the formula so that you can calculate it yourself:

$$A=[FiO_2 \times (P_b - P_{H_2O})] - (PaCO_2 / 0.8)$$

$P_b$  is barometric pressure which will be a constant at sea level (760).  $P_{H_2O}$  is saturated water vapor pressure which is 50 (sources vary on the exact number). The 0.8 is the respiratory quotient and is a fixed number. At sea level and breathing room air we can simplify the equation to:

$$A= 150 - (PaCO_2 / 0.8)$$

If the patient is not at sea level, or you are asked to calculate the A-a gradient for a patient breathing supplemental oxygen, you can do that with the original formula. Once you have calculated A, subtract the  $PaO_2$  value (a) from the alveolar value (A) and you have the patient's A-a gradient.

Normal A-a gradient should be less than 15. With abnormal values we can assume that the patient's hypoxia is due to significant pulmonary parenchymal disease or heart disease. Patients suffering from hypoxia can have abnormal blood gas values but a normal A-a gradient; we can then assume their reasons for hypoxia are not related to lung disease.

Lastly, when the animal is receiving supplemental oxygen (and the arterial sample was drawn with the patient on oxygen) you need to calculate the  $PaO_2:FiO_2$  ratio. This will tell you if the patient is responding appropriately to the amount of oxygen it is receiving. The  $PaO_2$  should be 5 times the  $FiO_2$  (so with a  $FiO_2$  of 21% or 0.21, the  $PaO_2$  should be 100 or close to it). A normal  $PaO_2:FiO_2$  ratio is 300-500. If that number is less than 200 then the patient has significant pulmonary disease and possible ARDS. This is an important number to calculate, as you may have a patient with a  $PaO_2$  of 100 which looks good, until you notice that the  $FiO_2$  is 80%.  $100/.8 = 125$  which is a terrible result! This value is used with mechanical ventilation; if you begin to wean the patient from oxygen and decrease their  $FiO_2$  it is important to monitor their progress. A change in  $PaO_2$  may not seem noteworthy, but in the face of a changing  $FiO_2$  the ratio may change significantly meaning the patient is not handling the decrease in oxygen supplementation.

Venous blood gas values can still be used for important information on patients but the respiratory component is most accurately measured with an arterial sample. There is a 5-10mmHg difference between venous  $CO_2$  and arterial, with the venous sample being higher (as blood is carrying the  $CO_2$  back to the lungs to be exchanged for oxygen and exhaled).

Take the analysis step by step and think through each value and how they relate to each other.

**NORMAL VALUES:**

pH	7.35-7.45
PaCO <sub>2</sub>	35-45
HCO <sub>3</sub>	18-24
PaO <sub>2</sub>	80-100
A-a Gradient	<15
PaO <sub>2</sub> :FiO <sub>2</sub>	PaO <sub>2</sub> ≈ 5 × FiO <sub>2</sub> ; or at least 300

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# Attitude is Everything

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For those who work full time, we find ourselves spending more quality time with our coworkers than those people who we chose to marry or share our homes with. 40 hours a week in a small treatment area with people we are forced to interact with is not always a pleasant experience. Add the stress of a hospital, money, clients, time constraints, and a barking dog or two and we can quickly see how communication breakdowns happens within veterinary teams. If we are lucky we have the support of our team and we work through problems together. Unfortunately, competition and lack of communication skills lead to a breakdown within veterinary teams. By learning good communication skills, we can elevate not only our teamwork with each other, but we can see our clients as team members as well.

The first step to dealing with issues among team members is to actually deal with the problem. Issues that start small can blow up into big problems if they are left to fester. Managers and leaders need to encourage employees to manage small problems amongst themselves, and employees need to feel empowered to generate difficult conversations in order to move past disagreements. Have these conversations outside of work if at all possible – at least begin them on a break away from the front desk. Admit some fault to the misunderstanding and ask what can be done to move past the issue. By confronting problems early in the process we can hopefully avoid a large-scale issue months down the road.

The first step to developing good communication skills is to make sure that we understand ourselves. When we know how we communicate, how we prefer to be communicated with, and know what our stressors are, we are more aware of ourselves and how we react to others. One such method for understanding our communication style is the DISC assessment. Knowing yourself, and your teammates, helps to identify potential communication walls and teach team members how to adapt. People do not fall into one category as personalities are more complex than one field, but they will have dominant traits. The categories are as follows:

Dominant – one who is independent, communicates directly, task-oriented and needs to see results from their work. They are motivated by directness (how they want to be communicated with) and are afraid of being taken advantage of. Their weak areas are being aware of other’s feelings and they can be argumentative. These people are able to create new ideas and will challenge the idea of “this is how we’ve always done things”.

Influence – one who is an optimist, enthusiastic about work, social, persuasive, and trusting of others. They are motivated by public recognition and fear rejection or not being liked by their peers. They can be disorganized and may be easily distracted but they are great motivators and encourage people. They also tend to be more creative when approaching problems.

Steady – one who is a team player, a good listener, and practical in their approach. They are motivated by safety and feeling secure in their position, and their fear is losing that stability. The weakness of this team is that they are resistant to change and may be possessive of their position and duties, but they are reliable, loyal, and dependable employees.

Conscientiousness – one who is precise and accurate in their work and hold themselves to a high standard. They are motivated by logic, organization, and performing detailed task work. They fear criticism and may take it harder than anticipated. They tend to be demanding of themselves and their coworkers but avoid conflict. Their attention to detail and accuracy make them valuable to the team.

The purpose of understanding people and their personality characteristics is not to label and discredit people, but to see strengths and play to those strengths. A person who leans to steady may be one who is not great at training a new employee, but they would be great to participate in creating a list of skills necessary to learn. One who has a majority of influence is great to pair with new hires as they enjoy the social interaction and will be friendly. Taking the DISC assessment as a team and then discussing strengths and weaknesses together can help to build the team and encourage future adult conversations when trouble arises.

Gossip is a problem we encounter from a very young age and unfortunately, seems to increase in frequency and malignancy as we get older and join the workforce. The simplest definition of gossip is talking about something neither person has the ability to change or impact. When we complain about a coworker's tardiness to another coworker, when we discuss the argument we overheard between the practice owner and the new associate veterinarian, or when we can't wait to share embarrassing stories about the new crazy client we are gossiping. Some of the time gossip is harmless chatter, but it can quickly escalate into a larger culture problem and cause rifts between coworkers and teams.

It is unrealistic to think that any hospital will be completely free of gossip, but it is important to pinpoint problems before they escalate and invade a hospital. Leadership must be aware of their employees and how they are feeling. This happens when employees feel empowered to discuss issues and feel they are heard by their manager. If employees feel ignored or feel that nothing ever changes no matter what they say, then they turn to their coworkers to vent frustrations instead of having constructive conversations. Challenge yourself and your coworkers to have constructive conversations as opposed to destructive gossip sessions. Gossip has nowhere to go if people simply do not participate. There may be one or two employees who love to get everyone riled up; but these employees have nowhere to go if no one chooses to listen. If you can, walk away. If you cannot leave the area, change the subject and don't let gossip poison you against your coworkers or your clients.

Bullying is another issue seen in many veterinary hospitals that can fracture teams and make for a miserable work environment. Bullying can happen not only between members of the same team, but also between technicians and doctors, medical and non-medical employees, and managers and employees.

There are many theories behind why people bully in the workplace – but a major contributor is employees who feel powerless and not respected at work. Medicine has a natural hierarchy and as you travel down the chain, respect is truly lost or perceived to be lost and employees lash out at each other to compensate. This often manifests as hoarding information, refusing to train or answer questions, or more obvious outbursts of aggression and gossip. This behavior makes the aggressor feel like they now have control over their situation.

Hospital management has a responsibility to admit to any bullying that may be happening, but employees must also take responsibility for their work culture and move beyond the bullies. Create training opportunities for staff members to teach each other, reward progress, provide skills checklists and ensure that new employees are given learning opportunities. Pair up new employees with someone proven to provide support. Reward empathy and team building. If you are working with a bully – talk to your supervisor and provide specifics. Many times, a bully is otherwise a great employee; often good with patient care or client care and therefore still seen as an asset to the hospital. If you feel that a hospital tolerates a bully and you are not making headway in a professional relationship with that bully, it may be time to look for work at a different hospital. Compassion fatigue is not just limited to the medical staff, and working with a bully will contribute to compassion fatigue and burn-out.

While we have discussed some of the negative cultures than can grow in veterinary hospitals, there is hope! One of the most important steps in creating a positive practice is to pay attention to the new hire process. Are you interviewing for personality, skills, or both? Do you know enough about your work culture that you know how to find the right fit? If your hospital employs a large group of passive aggressive indirect communicators, hiring a brash, up-front, direct communicator can spell disaster. Know your team's strengths and weaknesses and work to your strengths. There is nothing wrong with wanting to become more direct, but warn the team and the new person about potential pitfalls.

It is important to create structure for new hires and set an expectation for their progress. This not only helps new employees see where they are going, it also improves trust between coworkers. When the entire team participates in creating a new hire checklist or skills assessment, they are all ensuring that employees are learning the same skills and will learn to perform them the same way. This can cut down on information withholding and new hires feeling left out of the process. Ensure that new hires are given the opportunity to train and learn; do not throw them to the wolves on their first day. Success comes with confidence and it is everyone's role to make sure we feel confident in our roles.

One of the first steps to creating a positive culture within the practice is to find mutual ground. Service, whether to clients, patients, or each other, should be a common theme. Bring all employees together for a discussion on service. Begin by defining service. Solicit stories and feedback from the entire team regarding the basics of good service. Define service on the phone, in person, and decide the basics that every client will receive while in the clinic. Through this exercise, create a list of responsibilities for each

team and each team member. By including everyone in the process, the teams see that client service is not the responsibility of only one team, but a key requirement of every employee.

Another helpful activity for the hospital is to ask each employee their reason for working in that practice. What are their core values? When the team members have discussed their personal values, discuss the core values of the practice. Allow everyone the opportunity to see the value of service and communication and to understand their role in carrying out the values of the practice. Team members should take the opportunity to see how their reasons for working mesh with the values of the practice. These values should be discussed with each new hire and reiterated at yearly reviews or if any service issues arise at any time.

Practice members need to place a premium on helping each other. While it is beneficial to foster comradery among small groups working together, do not let it turn into hostility for other teams. This can happen between shifts as well as between varying roles. Challenge employees to see each other as internal customers. Treat coworkers with as much respect as a client deserves. Encourage employees to participate in the training process. If a technician is having problems with a process that involves the front desk, a customer service representative can step in and help with the training process rather than waiting for the technician manager to respond. The technician team, rather than complain that the check in process is taking too long, can offer to help by taking phone calls or entering information into the computer.

Set goals for the team and reward progress. With each successful new hire making it to a positive 90-day review, call out those who were instrumental in that employee's success. Reward positive reviews from clients and specifically name the team members who helped that client. Set the expectation that while the entire hospital does not need to be close friends outside of work, everyone will treat each other with respect. Learn about yourself and your coworkers and how you will all best communicate, play to each other's strengths, and be the grown-up in the situation and your hospital is well on its way to a positive and supportive work environment. This will show in your teamwork and will end up providing better service to your clients.

Compassion fatigue affects those of us in helping professions and includes a depletion of the emotional ability to care. Apathy, anger, and depression can follow further distressing the person who entered the veterinary field because of their ability to care and empathize with both animals and people. This fatigue can quickly lead to career burnout and sees many veterinary professionals leaving the field. For many, it is not a matter of if we will experience this feeling of burning out, but when.

Euthanasia can be a major trigger to feeling fed up and burned out. Depending on the patient population, some veterinary professionals are involved with euthanasia (many times multiple) every single shift. While euthanasia can be a great gift for suffering animals, the grieving client also requires compassion.

Sometimes an animal is euthanized for a correctable medical problem but the client lacks the funds. Other times, euthanasia is the appropriate choice but the client cannot bring themselves to authorize it. Animals are removed from the hospital to “die in peace” at home, leaving the staff in frustration. Even if a visit does not end in euthanasia some animals return home with minimal treatment due to the financial position of owners. In these types of situations, the veterinary team must train themselves to look for the good in the client and resist the urge to judge. By giving in and judging that client, we take on that responsibility for the patient and the end result can never be positive. Our role is not to judge the decisions made by others. Our role as veterinary professionals is to provide the best possible care for the pet and to educate the owner with empathy and decency. When we are able to look for and focus on the client experience, we are better able to understand their decisions and focus more on making the pet’s life the best we can with what we are given.

Many veterinary hospitals operate in a guilt culture, where staff members work long hours and extra shifts out of guilt to their coworkers and other patients. Lunch and breaks are not taken because there is always more work to do and more patients to care for. Staff members work through colds and fevers because staying home sick means your team has to work twice as hard. It is up to all of us to take responsibility for ourselves and do what is best by us to keep our longevity in the profession. Make a meal break a priority and communicate with your team when you plan to take a break. Put pressure on teammates to take a minute to eat. Discuss the possibility of a hospital budget for healthy snacks close to the treatment area. While easy to place blame when triage lists are high and staffing is low, it is up to us to empower ourselves and each other to take care of ourselves. While impossible to banish guilt completely, work on becoming self-aware and fight the urge to give in every time.

The tools to combat compassion fatigue and burnout are within us. First is to recognize changes in our attitude and outlook. Quick to anger, apathy, sadness, trouble sleeping, forced isolation, crying at work (or at the thought of work), and lack of hobbies outside of work are all danger signs of burnout. Are you simply going through the motions at work? Do you find yourself treating patients without caring who they are or why they are in your hospital? Do you lack empathy for your coworkers? Recognizing negative trends in your own behavior and thoughts can mean that you are suffering.

Recovering from that “crispy” burned out feeling is possible but it requires self-care and self-inventory. Taking time away from work, whether it be a few days at home watching movies, taking appropriate breaks, or leaving town for a few days can help to reset. Discover hobbies and have something to look forward to outside of work. Eating better is necessary, and means that you have to take breaks at work to eat home cooked food. Exercise, as simple as walking your dog every night after work, can help ease anxiety and bad feelings. Many veterinary practices, invested in the long term health of their employees, are implementing self-care ideas in the practice. Working to change the guilt culture takes work and a progressive manager, but should be a goal in every hospital.

Even if you do not have symptoms of compassion fatigue, look out for problems in your coworkers. Look out for each other by ensuring everyone has appropriate breaks. Make it okay for everyone to discuss their feelings after a tough euthanasia. Plan activities outside of work for everyone to bond together in a non-stressful situation. Discuss the possibility of a wellness program at work. Reward healthy behavior, or have a central location for listing positive changes and encourage discussion. Create a small budget for buying healthy snacks for the team. Even small changes from leadership can show to the staff that mental and physical health is important.

It is also important to resist the urge to complain about clients in front of coworkers. Clients can be a major source of stress due to decisions they make that are outside of our control. It is easy to place our own knowledge and desires onto clients and then experience crushing defeat when decisions are made outside of what we expect. It is easy to slip into anger when we think animals have waited too long for care, or when they are kept alive when we perceive suffering. It is easy to complain when a client over the phone asks “dumb” questions. Take a step back and assume the best from each interaction with a client. They want the best for their pet, and they are doing the best that they can do. Complaining, making fun of client questions, and placing blame will only serve to bring you down and by verbalizing your complaints you will also bring down your team.

Feelings of burnout do not have to be a permanent situation. With recognition, self-care, and time it is possible to return to loving your job, your patients, and your clients. Evaluate yourself every few months and be honest with where you are in your career. What can you do to remain happy and ensure mental health in the future? Rely on your coworkers for support, and be a support to them. Take time to take care of yourself so that you can take care of others.





## The Top 5 Emergencies and What to Do Parts 1 and 2

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Emergencies can arrive at any moment, no matter what type of hospital or clinic. Some emergencies can develop in patients who were stable when they were checked in. Emergencies require quick recognition, and timely treatment can prevent a big disaster down the road. Technicians who are critically thinking and are aware of what to watch for are vital to patient comfort and survival. Be ready to receive the most common emergencies in your practice and you can be the one who everyone looks to in an emergency.

### **Gastrointestinal Emergencies**

Gastrointestinal emergencies are the most common reason for veterinary visits outside of preventive care. Dogs and cats are sneaky at eating anything and everything they can get their mouths around. As humans, we often want to love our pets through food. While minor vomiting and/or diarrhea are usually not a major deal, some of our GI distress patients can arrive already in trouble, or quickly get in trouble if they are not assessed and treated quickly.

The cause of shock in small animal patients is the result of a deficit of oxygen supply and oxygen demand. When a lack of oxygen delivery from poor perfusion occurs, cells cannot produce necessary energy. Every cell in the body requires ATP to protect cell wall integrity and perform cellular functions. ATP has a life span of 3 seconds and the body needs oxygen in order to create ATP and keep up with need. In a shock situation, because the body is no longer able to provide normal blood flow to tissues oxygen distribution is interrupted, ATP production decreases, and cells begin to die. Patients experiencing trauma can be in shock from lung injury (not performing gas exchange well enough to provide oxygen to cells), crushing injury (blood vessels are damaged and cannot carry oxygen to tissues), from obstruction (blood cannot flow to cells due to a physical obstruction), or from blood/fluid loss (not enough volume to carry oxygen to tissues). If left untreated, patients in shock can die.

The body has baroreceptors to warn the body of decreased blood flow in an attempt to avoid this cell death. These receptors are located in the aorta and kidneys and in times of hypovolemia (for example) sense when blood flow is low and signal back to the body that blood flow is lacking. The sympathetic nervous system reacts with vasoconstriction, increased cardiac contractility, and tachycardia. These adjustments will increase cardiac output in an attempt to maintain blood flow and oxygen flow as normal. The kidneys, via the renin-angiotensin-aldosterone system will begin to retain sodium and water in order to increase intravascular volume. These compensatory efforts will display as subtle clinical signs in early shock and are often missed by pet owners.

As shock progresses without any intervention, these compensatory mechanisms are soon overwhelmed and can no longer keep up the “business as usual” blood flow. Vasoconstriction begins to preferentially decrease blood flow to major organ systems (beginning with the periphery, then the gastrointestinal tract, progressing to the liver, then kidneys, and eventually brain) in order to maintain as much perfusion as possible to the heart, lungs, and brain. This whole body decompensation exhibits in the clinical signs of shock: pale mucous membranes, poor pulse quality, decreased blood pressure, and depressed mentation. If these signs are ignored and treatment not administered, organ systems will fail due to lack of oxygen and the animal will die.

Animals can be in shock from either a lack of fluid (dehydration) or a lack of perfusion (hypoperfusion) and in some cases, both. Perfusion and hydration is not the same thing, and we are able to measure these in a patient by using different modalities. Perfusion is monitored by measuring mucous membrane color, capillary refill time, pulse quality, and heart rate. Hydration is monitored by measuring skin turgor, the moisture of mucous membranes, and assessing whether the patient’s eyeballs are sunken.

As patients present to the hospital with shock, or as those in the hospital are monitored closely, the technician team must be aware of and look for the clinical signs of shock. As discussed, the body begins with subtle signs of compensation that may go unnoticed like light pink mucous membranes, mild mental depression, tachycardia, and mildly prolonged capillary refill time. In compensatory shock the blood pressure and pulse quality remain normal.

Early decompensated shock sees the patient still trying to keep up with decreased perfusion. This stage mimics the pain response in animals, so pain management is important to not only keep the patient out of pain, but to rule out this stage of shock. Dogs suffering from early decompensated shock will exhibit pale mucous membranes, tachycardia, dull mentation, hypotension, poor pulse quality, and increased capillary refill time. If patients in early decompensated shock are not assessed and treated, they will continue to worsen. In late decompensated shock the body begins to shut down and can no longer keep up with oxygen needs. The heart rate may be decreased, mm color is white or gray, and pulses may be absent. These patients are minimally responsive or obtunded.

Patients that present with these physiologic signs of shock need immediate treatment. Unless the patient has cardiac disease, the mainstay of shock treatment is intravenous fluid therapy. A large bore short length IV catheter will deliver a large amount of fluids quickly, and should be placed in a cephalic or jugular vein. Crystalloid fluids are electrolyte and water replacement and are administered quickly in an attempt to restore circulating volume and therefore oxygen delivery to cells. The “shock dose” of crystalloid fluids is 90ml/kg in a dog, 45 to 60ml/kg in a cat. This large dose is often divided into smaller amounts (20ml/kg for example) and administered as a quick bolus. Once the bolus is complete, the vital signs are once again assessed. The bolus amount is repeated until vital signs begin to improve.

Colloids are fluids comprised of large molecules that will remain in the intravascular space; they may also help to draw fluid into the intravascular space. In many trauma cases colloids (like hetastarch and VetStarch™) can be used in conjunction with crystalloid fluids to improve patient blood pressure and perfusion parameters. Colloids have a dose limit of 20ml/kg/day but small amounts can be given as a bolus during the resuscitation period. Blood products are natural colloids and can be used for volume expansion and to boost oxygen carrying capacity in animals bleeding from trauma.

Hypertonic saline may be administered to facilitate a quick draw of fluids from the extravascular to the intravascular space. Hypertonic saline has a much higher sodium concentration (usually 7% NaCl) as compared to physiologic saline (0.9% NaCl) which draws fluid into the intravascular space in an attempt to equalize the sodium content. Relatively small volumes are needed (5ml/kg) administered over 5-10 minutes. The intravascular volume expanding effects of hypertonic saline last about 30 minutes, but their administration may keep the patient alive long enough to allow a large bolus of crystalloid fluids or other therapies time to work. The fluid shift that occurs with the use of hypertonic saline can help to reduce intracranial pressure in head trauma patients, as well as modulate inflammation. Hypertonic saline should not be used as the sole fluid used for resuscitation as it can cause dramatic changes in the patient's serum sodium levels.

It is important to note that cats do not respond to shock the same way dogs do. Cats in shock will often vasodilate (not vasoconstrict like dogs), lose body heat, and often experience extreme hypotension. This hypotension is not necessarily from low circulating volume. As cats warm back to a normal temperature their vessels will constrict back to normal size. If large amount of fluids are administered to a severely hypothermic cat the blood pressure may read normal, but as the cat warms vasoconstriction occurs. As their temperature returns to normal the excess fluid from their vascular space can become pulmonary edema. It is important to be judicious with fluid administration on hypothermic cats and focus on warming them as much as providing fluid therapy. While the cat is warming, small (20ml) boluses of crystalloids can be given as they are closely monitored. When their body temperature approaches normal and they are still hypotensive more aggressive IV fluid therapy can begin. Cats can be bradycardic or tachycardic with shock, goals from treatment need to be vital signs approaching normal.

## **Respiratory Emergencies**

Receiving respiratory emergencies takes quick action. These patients need immediate oxygen support in the form of an oxygen cage, flow by, nasal prongs, or an oxygen hood. These patients are fragile and must be handled with care; an important goal during treatment is to minimize stress. Observe the breathing pattern of the animal when they first arrive. Watch their posture and note if they are positioned with elbows abducted and/or their neck stretched out. Watch their thorax and abdomen as they breathe; are the chest and abdominal muscles moving together (outward movement during inhale, inward movement during exhale) or opposite each other? Patients exhibiting paradoxical breathing are at risk for respiratory arrest and should be closely monitored.

Ascultation of the chest is necessary and can assist in localizing the underlying problem causing the distress. Listen for upper airway sounds, absence of lung sounds, harsh lung sounds, crackles, and for cardiac abnormalities like a murmur or arrhythmia. Ascultation can help guide treatment and lead to interventions such as thoracocentesis which can stabilize the patient. Radiographs are helpful in diagnosing respiratory problems but are stressful and can be detrimental to the distressed patient; effort must be made to attempt to stabilize the patient as much as possible prior to radiographs. Ultrasound can also be utilized to identify pericardial effusion, pleural effusion, and pneumothorax and can be performed with the patient in sternal recumbency with minimal stress.

Mild sedation can be administered early in the treatment plan for these patients; sedation can help to facilitate procedures and diagnostics and provide mild relaxation while these animals rest in oxygen. For non-traumatic respiratory distress, butorphanol (0.2mg/kg IV or IM) can help with relaxation. Traumatic injuries need pure mu opioid pain management. In patients that are not responding to therapy, those too fractious to allow handling, or those with severe respiratory exhaustion, the best treatment plan may be general anesthesia and intubation. Sedation with an opioid and benzodiazepine may be sufficient but a slow titration of propofol or alfaxalone can also be utilized for induction and intubation. Although anesthesia in a critical respiratory distress patient can be a frightening thought, intubation provides a secure and open airway and allows full control (if necessary) of respiratory rate.

Take diagnostics slowly with these patients. They are easily stressed and small things (like a temperature or blood pressure) can increase their distress. Prioritize treatments, discuss IM vs IV injections of medications, and allow long rest periods between interventions. When restraining for IV catheter placement, blood draws, or injections, allow the patient to remain sternal if possible and be cognizant of their head position. Do not put any unnecessary pressure on their neck or chest during restraint. If the patient allows, continue to provide at least flow by oxygen during treatments.

Many respiratory patients will eventually need radiographs; minimize time and stress by ensuring good diagnostic images. Be sure your positioning is good; watch the rotation on both lateral and VD views. Ensure your cranial landmark is the thoracic inlet and include all of the lung fields. If at all possible take the radiograph on inspiration. The ventral-dorsal view is the most stressful for the patient; rather than wrestle, discuss taking a dorsal-ventral view if possible.

Once a treatment plan is created for any patient needing oxygen support the next step is monitoring. Along with routinely checking all of the animal's vital signs, their oxygen needs must be assessed regularly. A SpO<sub>2</sub> reading is certainly part of that monitoring but it shouldn't be the only parameter used.

SpO<sub>2</sub> – results for a patient on supplemental oxygen support should be monitored and improve throughout hospitalization. As the patient improves they should be challenged off of oxygen support for a time and a SpO<sub>2</sub> reading taken off oxygen to help wean down to room air. Remember the limitations to SpO<sub>2</sub> (ambient light, patient compliance, mucus membrane color)

Respiratory rate and effort – frequent (every 2 hours) monitoring of respiratory rate should be performed. It is important to try and observe the animals in secret as values may change in the presence of a technician. Effort should be noted as well.

Mucous membrane color should be closely monitored; gray or muddy mucous membrane color can mean the patient's respiratory status is worsening.

Heart rate should be closely monitored. Hypoxia will result in tachycardia; evaluate the patient if sudden tachycardia occurs or if the heart rate increases between treatments.

Arterial blood gas –if your hospital has the ability, an arterial blood can be utilized to accurately record respiratory disease progress or decline.

A patient in respiratory distress may spend only hours in your hospital or may spend days in the ICU with mechanical ventilation. The technician role is to quickly recognize respiratory distress, provide oxygen support, and closely monitor patients for changing needs.

## **Trauma Emergencies**

When a patient presents to the hospital with a trauma emergency a technician should perform a triage in the lobby to determine next steps. In many cases of trauma, the patient should be brought to the treatment area for a full assessment and immediate treatment. Begin with the ABCs:

**AIRWAY** – ensure the patient has a patent airway and is moving air. If not, intubation and ventilation is the first step to take.

**BREATHING** - many trauma patients can present to the hospital with some degree of respiratory distress. Provide at least flow-by oxygen to these patients and observe their breathing pattern to help determine the cause of their distress. Trauma, bleeding, and pain can all cause changes in respiratory rate and effort. Listen to sounds the patient makes while breathing; are the sounds on inhalation or exhalation? Listen for lung sounds; are they increased or decreased? Watch the patient breathing; are they using their abdominal muscles? Is the effort increased on inhalation or exhalation? Watch for flail chest and observe for any open wounds which may penetrate the thoracic cavity; if present these must be covered and bandaged as soon as possible. Have the supplies necessary to perform thoracocentesis as needed.

**CIRCULATION:** Trauma patients are often suffering from hypovolemic shock due to hemorrhage. Team members must be comfortable recognizing the signs of shock and familiar with the appropriate treatment and monitoring of these patients. To assess circulation, evaluate femoral pulses, heart rate, mucous membrane color, capillary refill time, and blood pressure.

Once the ABCs of the patient is evaluated, it is important to record all vital signs. It may be tempting to skip over this step as the patient may require a multitude of treatments and care, but unless the patient needs immediate CPR, admitting vitals are important to the team's ability to monitor and track progress. Move

systematically through the patient while performing a brief exam, and include the blood pressure in the admitting vitals if at all possible.

When treating a trauma patient you must prioritize their conditions and treat first what will kill them first. While a giant wound may look impressive, dealing with respiratory distress or decompensated shock must take priority. Patients experiencing trauma can be in shock from lung injury (not performing gas exchange well enough to provide oxygen to cells), crushing injury (blood vessels are damaged and cannot carry oxygen to tissues) or from blood/fluid loss (not enough volume to carry oxygen to tissues). If left untreated, patients in shock can quickly die.

When resuscitating a trauma patient, goals are set for the patient's vital signs and treatment administered until those goals are met. Some patients may be resuscitated until their heart rate and blood pressure are normal or approaching normal. The role of the technician is to closely monitor these patients and recheck all vital signs after each bolus of fluids. This information is relayed to the veterinarian and the next steps ordered. In some patients, particularly those with abdominal bleeding or thoracic trauma, more conservative goals may be set so as not to cause further bleeding and contribute to respiratory distress.

Pain management is an important part of any trauma treatment protocol. The clinical signs of shock can be confused for the clinical signs of pain, and trauma patients are often experiencing both shock and pain. Pain needs to be alleviated to allow the patient to begin healing. Pain will increase the body's need for oxygen and increase metabolic needs. Pain can also lead to vasoconstriction which can restrict oxygen flow to injured areas. If possible, choose an appropriate full mu opioid to treat traumatic pain and continually assess levels of pain throughout the patient's stay. If your hospital has the ability, a CRI of morphine, lidocaine, ketamine, fentanyl, or a combination of these drugs can be beneficial for these cases as they recover. Technicians can monitor for small changes in patients and advocate for appropriate pain control during the hospital stay.

Performing even minimal blood work is important in the stabilizing process of trauma cases. It is beneficial to draw blood as the IV catheter is placed to save time and patient stress; even using what blood is in the catheter stylet is enough to get started. Every trauma patient should have at least a PCV/TS, blood glucose, and BUN performed at the start of treatment. As the patient stabilizes with fluid therapy and pain management, more detailed blood work can be drawn and performed.

Wounds are common in trauma cases but are rarely the most important treatment. Wounds should be addressed quickly, but only to assess their nature and what it may take to repair them. Any penetrating thoracic or abdominal wounds will require attention early in the stabilizing process to clean and cover. Once the patient has received appropriate pain management, clipping and cursory cleaning can occur

with the focus on making the patient a good anesthetic candidate for definitive repair later in their hospital stay. Bandages can be applied with a plan to reassess as the patient status allows.

Once a trauma patient has stabilized it is necessary to continue monitoring these patients closely. Serial monitoring of heart rate, pulse quality, MM color, CRT, respiratory rate and effort, body temperature and mentation should be performed at least hourly until further directed. Monitoring for pain and bleeding should also be a part of the treatment plan.

### **Urinary Obstruction in Cats**

Urethral obstruction, occurring in more male than female cats, can occur from urine sediment or mineral stones causing mechanical obstruction of the urethra, from inflammation of the urethra, or from urethra spasm. This is a life threatening medical emergency and requires immediate recognition and treatment. The nursing team is instrumental in recognition, preparation for safe anesthesia, and monitoring the hospitalized cat.

If you palpate a turgid bladder (not all cats with a urinary obstruction will present with a large bladder, do not let a small but hard bladder fool you) this patient needs to move to the top of the list of tasks for your day. Thinking ahead to the procedures necessary for a patient suffering from urinary obstruction (anesthesia, possibly major surgery) a complete physical exam with vitals always needs to be performed and abnormalities addressed.

Pain management is extremely important in these cats upon presentation to the hospital. Not only is a urinary obstruction known to be a painful condition, but patient compliance is much improved with the addition of opioids. If possible, treatment prior to de-obstruction should include a pure mu-opioid such as oxymorphone or hydromorphone but buprenorphine is acceptable as well. Pain management is important throughout the hospital stay for these cats and into their transition back home.

IV fluids are also necessary in blocked cats, especially as they prepare for anesthesia to pass a urinary catheter. While not appropriate to leave a blocked cat on hours of IV fluids, starting the diuresis immediately prior to anesthesia will help to improve or maintain blood pressure, dilute out azotemia and hyperkalemia, and improve perfusion.

A minimum amount of blood work should be performed before anesthetizing any patient for relief of obstruction. Even in seemingly healthy patients a PCV/TS, electrolytes, and renal parameters should be evaluated. Urinary obstructions present for significant periods of time (>24 hours) can cause electrolyte abnormalities and elevation in BUN and serum creatinine. The most immediate danger in 'sick' blocked

cats comes from dangerous elevations in potassium. Most of the body's potassium resides inside the cell, and circulating plasma potassium levels are low compared to serum sodium and chloride. When potassium cannot be excreted out of the body by the failing kidneys into urine, serum levels start to rise. Potassium is necessary for nerve impulses and cardiac and muscle function. Extremely elevated levels will change the resting membrane potential of cells resulting in conduction abnormalities in the heart. If it is not corrected, death from cardiac failure can occur. Hyperkalemia must be addressed before de-obstruction occurs. Even without blood results, physical exam findings and looking at an ECG tracing can alert you hyperkalemia. In many hyperkalemic patients you can see tall, wide T-waves on the ECG, a classic sign of hyperkalemia. Many of these cats are “sicker” on presentation, and may suffer from bradycardia, hypothermia, and hypotension.

If there are cardiac arrhythmias evident, hyperkalemia can first be treated with calcium gluconate (10% calcium gluconate at 50-100mg/kg slowly IV). Calcium gluconate will not directly affect the levels of potassium in the blood but it will decrease the amount of damage to cardiac muscle from the potassium. Immediate improvement in ECG tracings can be seen just after treatment with calcium gluconate. To treat the high potassium levels, injections of dextrose or dextrose and regular insulin can be used. Insulin acts as the key that opens up cells to take in glucose. When glucose enters the cell potassium follows closely behind. So by giving regular insulin (0.25-0.5 unit/kg IV) to a severely hyperkalemic patient you can begin to lower the level of circulating potassium. For moderate hyperkalemia, an injection of 50% dextrose can work the same way. Any patient that receives IV insulin needs dextrose added to their IV fluids and frequent blood glucose checks.

When treating a hyperkalemic or azotemic blocked cat, a cystocentesis can be performed to relieve the pressure in their bladder and help to stabilize the cat prior to anesthesia and unblocking. While possibly considered a risky procedure, cystocentesis performed in cats suffering from urethral obstruction can halt the progression of renal damage and bring immediate relief to the cat. While risk exists for tearing the potentially friable bladder wall during the procedure, the benefit to the patient outweighs the risk. Appropriate pain management and patient positioning can reduce the risk of sudden movement and bladder wall laceration.

The procedure of relieving obstructing almost always requires general anesthesia. Alternatively, a coccygeal block can be performed which can greatly reduce the amount of anesthesia needed. A coccygeal block is a simple local block to perform that will block the perianal region and is perfect for relieving urethral spasms, passing a urinary catheter, and even used for tail amputations. 2% lidocaine (0.1-0.2ml/kg) will provide pain relief 5 minutes after injection and last about one hour. This allows for smooth anesthesia recovery. In the sickest patients, an injection of an opioid paired with a coccygeal block can allow unblocking to be performed without general anesthesia.

Remember that these patients may be suffering from some degree of acute kidney injury and need close, careful anesthesia monitoring. Their blood pressure should not be allowed to drop below 80 systolic/60

MAP in order to keep the kidneys perfused. The procedure should be performed in as sterile a manner as possible. An assistant will often be required to pulse sterile saline through the urinary catheter to force the obstruction back into the bladder or force the urinary catheter past the obstruction. This process may take many different kinds of urinary catheters, so be prepared with a selection. The final indwelling urinary catheter should be made of a gentle material (silicone is most ideal) for long-term comfort. A post-procedure radiograph should be performed to confirm proper placement within the bladder.

While hospitalized, any patient with an indwelling urinary catheter should have fluid ins and outs monitored. Every four hours the urine bag is emptied, urine quantified, and fluid rates adjusted as needed. Cats can experience post-obstruction diuresis where they produce urine at such a high rate they can actually become dehydrated while on IV fluids. This occurs because the kidneys temporarily lose their ability to reabsorb salt and water resulting in high urine output. Post-obstructive diuresis cannot be prevented, only managed until the kidneys return to normal function. The nursing team should be aware of normal urine output, and how to calculate urine ml/kg/hr output. Compare the fluid going in to the fluid coming out.

(amount of urine (ml) / hours since last measured) / wt (kg)

This formula will give you ml/kg/hr of urine produced. In previously obstructed animal urine output should be at least 2ml/kg/hr. If the urine output is higher than the fluids going in, an adjustment should be made until urine output slows.

## **Toxin Emergencies**

Dogs and cats are into everything, and this appetite for life often lands them in the hospital after ingesting a toxin. If the toxin ingested is known (medication, cleaning product, rodenticide, etc.) there are veterinary toxicologists at the ready to help with treatment. Pet owners can also call these veterinary specific poison centers for advice and treatment guidelines. Communication with owners can be tricky, especially if illicit substances are involved, but it is important to always ask and make sure the owner knows you only need to know for the health of the pet. Gaining client's trust will only help to serve the pet.

Many times the specific toxin is unknown when an animal presents to the hospital. Sometimes the owners cannot recall or even know anything toxic was ingested. Unfortunately the symptoms of many poisons are non-specific, but these symptoms, regardless of cause, should be quickly evaluated and addressed. Vomiting and diarrhea are common signs of many disease processes as well as a toxic overdose. Neurologic changes, especially ataxia, are warning signs of toxicity; in some cases the animal has generalized muscle tremors that can progress to grand mal seizures. Many toxins increase heart rate, others will cause bradycardia. Toxic plants often cause hypersalivation and GI distress. Because of their job in filtering, the renal and hepatic systems are often affected by toxins. When faced with a patient showing any of these signs without a known cause, toxin ingestion should be considered. It is important

to methodically question owners as to the possibility of drugs, cleaners, foods, plants, and poisons so that treatment can be focused and appropriate whenever possible. Regardless of cause, emergency treatment for seizures, dehydration, and shock should be administered immediately.

Decontamination is an important step when dealing with toxins. If ingestion occurs within a couple hours of presentation, induction of vomiting is the first step in decontamination. While there are many methods, apomorphine (0.03-0.04mg/kg IV) is the most reliable drug to give to induce vomiting in dogs. Cats can receive xylazine (0.44-1.1mg/kg IM or SQ) to induce vomiting. As long as the toxin is not caustic, vomiting in a veterinary hospital is a safe method of decontamination. Vomit induction will not remove all traces of the toxin, as animals will vomit only 40-70% of their stomach contents. Further decontamination methods should be followed even if the patient vomits. Patients suffering from altered mentation or an inability to swallow should not be encouraged to vomit and alternative method for toxin removal explored.

If the animal does not vomit with the methods used or is incapacitated and it is not medically safe to induce vomiting, then gastric lavage can be performed. The animal must be under general anesthesia so the procedure is not without risk. Once anesthetized, ensure the endotracheal tube cuff is properly inflated with the animal in lateral recumbency. Measure the orogastric tube to the last rib and mark that length with tape. By applying gentle pressure, feed the orogastric tube down the esophagus until you reach the pre-measured limit. You may need to make small adjustments until stomach contents are seen within the tube. Flush water in through the tube and allow it to clear until there are no more stomach contents to remove. Be sure to clean the oropharynx before the animal recovers from anesthesia.

Performing a cleansing enema on a patient is also an effective way to clear toxins, especially once the stomach contents have traveled into the small intestine. For patients that present to the hospital hours after ingesting a substance, or if they only vomit a portion of what they ingested, it is possible to encourage elimination of the toxin by performing multiple cleansing enemas. Water is infused into the colon until it comes back out clear. Cleansing enemas can be performed multiple times during hospitalization and can significantly decrease the patient's clinical signs, especially with toxins such as metaldehyde and THC.

Usually the last step in a decontamination protocol is giving the patient activated charcoal. Activated charcoal is comprised of many tiny porous particles that absorb the toxin to prevent absorption by the body. Sorbitol is used as an activated charcoal additive and is a cathartic to encourage faster passage of the toxin through the body. Activated charcoal is most effective the sooner it is administered post ingestion, but for toxins that undergo enterohepatic recirculation it is administered every 4-8 hours for up to 3 days. Not every toxin will bind to charcoal, those with -ol (xylitol, alcohol for example) do not bind, therefore administration is not helpful.

The use of intravenous lipid emulsion is an emerging treatment for certain toxicities. Local anesthetics, ivermectin, pyrethrins, metaldehyde, marijuana, and fat soluble medication toxicities can be treated with intravenous lipids. Most IV lipids are formulated so they can be administered through a peripheral line requiring some extra attention by the nursing staff. IV lipids can be used in conjunction with other treatments (vomit induction, charcoal, etc) and reported side effects are uncommon. Lipids can be difficult to obtain as they come on and off backorder so many advocate saving them for the most critical of patients. General protocols state that with a 20% lipid emulsion, give 1.5ml/kg over 1-15 minutes then continue as a CRI at 0.25ml/kg for 30min-2 hours.

Preparation and training are the keys to handling any emergency well. Use your critical thinking skills and plan two steps ahead; if you know what is coming, you can prepare and be ready.

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# Acute Kidney Failure: Nursing Them Through

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The kidneys are vital to life in our patients – they maintain and manage the composition of fluid in the body by regulating water and electrolytes, they remove metabolic waste from the blood, remove chemicals from the blood, absorb solutes, maintain arterial blood pressure, regulate red blood cell production, maintain metabolic synthesis of calcitriol and glucose, and make urine to remove waste. The kidneys receive 20-25% of cardiac output and are therefore readily exposed to toxins and are sensitive to changes in blood pressure and circulating volume. The renal system is absolutely dependent on kidney function and blood supply to perform.

Because of kidney dependence on adequate blood flow, the renin-angiotensin-aldosterone system is utilized as a back-up to maintain appropriate circulating volume and to ensure the kidneys receive adequate blood flow.

renal blood flow is reduced – detected through baroreceptors

↓

kidney converts prorenin into renin, secretes renin into circulation

↓

renin converts angiotensin (released by liver) into angiotensin I

↓

angiotensin I converted to angiotensin II by ACE (angiotensin converting enzyme) in the lungs

↓

angiotensin II causes vasoconstriction which increases blood pressure

↓

angiotensin II stimulates secretion of aldosterone which signals the kidneys to reabsorb Na<sup>+</sup> and H<sub>2</sub>O which will increase circulating volume and maintain a stable blood pressure and kidney perfusion

If damage occurs to the kidneys and they are no longer able to adequately filter blood, an increase in creatinine and BUN are noted. These are waste products of normal muscle and body function and are filtered out of the blood via the kidneys. This azotemia is categorized into pre-renal, intrinsic, or post-renal. In pre-renal damage, there is a failure of blood reaching the kidneys. This is due to hypovolemic shock, hypotension due to anaphylaxis or anesthesia, or acute blood loss from trauma. The cause of pre-renal damage can often be quickly corrected and if so, elevated creatinine and BUN can return to normal.

However, if the cause of hypovolemia or hypotension is not quickly addressed, permanent kidney damage and eventual failure can occur. These patients require crystalloid fluid administration in large enough doses to replace losses and return circulating volume and therefore kidney perfusion to normal levels. Technicians must closely monitor patient response to fluids via heart rate, mucous membrane color, and blood pressure.

Intrinsic kidney damage occurs with direct damage to the kidney due to ischemia, exposure to toxins, or infectious insult. Common toxins associated with intrinsic kidney damage are:

Lily exposure in cats – renal tubules are sensitive to and damaged by all parts of the lily resulting in acute kidney injury

NSAID overdose – leads to renal vasoconstriction and acute tubular necrosis

Ethylene glycol – causes metabolic acidosis, metabolism of the toxin causes calcium oxalate crystals to be deposited within the kidneys and then failure

Medications such as amikacin, gentamicin, NSAID use in cats, amphotericin B, cyclophosphamide, cyclosporine, and radiographic contrast agents used in MRI and CT studies.

Post-kidney azotemia is caused by an obstruction of fluid after it reaches the kidneys – most commonly seen is urethral obstruction in cats, but trauma leading to bladder rupture, prostatic disease, cancer, and uroliths can all lead to post-renal azotemia. Uroabdomen can be present in these patients either as the result of trauma (like a mauling from a larger animal or vehicular trauma) or iatrogenic from attempting to place a urinary catheter or as the result of cystocentesis. Urine is reabsorbed from the peritoneal cavity, and as urine contains levels of potassium higher than circulating plasma, hyperkalemia is a common effect of uroabdomen. Abdominal fluid in patients suspected to be suffering from uroabdomen should be analyzed for potassium and creatinine. If both levels are higher (creatinine ratio of 2:1 abdominal: peripheral blood) in the abdominal fluid than the circulating plasma, a uroabdomen can be assumed. Pain must be managed in these patients as fluid therapy is started and treatment for hyperkalemia.

Patients that present to the hospital with azotemia require immediate attention. Any underlying toxin or disease process will need to be diagnosed and addressed, but each of these patients require the following minimum diagnostics:

PCV/TS – gaining a baseline is important to determine dehydration and to look for anemia and the presence of protein loss.

Electrolytes – the kidneys are instrumental in the body's electrolyte balance. Major sodium changes can lead to mentation changes; potassium elevation can lead to cardiac arrhythmias and death; potassium decrease can lead to weakness and respiratory difficulty; elevated calcium levels can lead to cardiac arrhythmias. Be prepared with emergency treatment as needed.

Blood gas – in acute kidney injury and failure, the kidneys cannot excrete H<sup>+</sup> and reabsorb bicarbonate leading to metabolic acidosis. Elevated lactate levels from hypoperfusion can also contribute to acidosis and should be monitored.

Chemistry panel – performing a blood chemistry panel gives baseline values of not only creatinine and blood urea nitrogen, but also phosphorus which can be abnormal due to excretion and dietary changes associated with kidney injury. Anticipate monitoring these values at least daily.

CBC – infection can play a role in kidney injury and these patients must be evaluated not only for white blood cell counts and differential but also red blood cell count and morphology.

Blood pressure – prolonged hypotension leads to ischemic injury to the kidneys and must be addressed immediately. Hypertension can signify kidney damage – decreased blood flow (from damage) can trigger the renin-angiotensin-aldosterone system increasing the systemic blood pressure.

Body weight – these patients need a baseline body weight and then comparative weights frequently during their stay. Weight gain or loss can signify an inability of the kidneys to process fluid appropriately and technicians must be looking for all signs.

Urinalysis – results can give information as to where the kidneys are damaged. Look for excessive protein, blood, crystals, casts, and special attention to the specific gravity.

### **Acute Kidney Injury**

Kidney failure is further divided into acute kidney injury and chronic kidney injury. Patients suffering from acute insult will often have enlarged kidneys on radiograph or ultrasound and are often painful upon kidney palpation. Acute injury is often the result of toxin exposure, trauma, or obstruction, but can also be the result of bacterial infection or infectious disease. Depending on the geographical area, consider testing these patients for Leptospirosis, or tick borne diseases such as: Rocky Mountain Spotted Fever, Ehrlichia, Lyme, Babesia, or Leishmania. Regardless of the inciting cause, acute kidney injury patients can present with life threatening electrolyte abnormalities and require close monitoring. Disease processes such as sepsis and SIRS can also lead to multi-organ dysfunction syndrome (MODS) leading to acute kidney injury in patients. Even young patients that present to the hospital without any clinical signs of kidney damage can suffer from acute azotemia. Technicians must be aware of this and monitor this class of patients closely for changes in fluid output, electrolytes, and mentation.

Sodium abnormalities, especially those that develop acutely, can cause dangerous fluid shifts leading to cellular damage and the potential for cerebral edema. IV fluid administration type will depend on electrolyte abnormalities, remembering that rapid changes in sodium are detrimental to patients. Hypernatremia can lead to a free water deficit, meaning the intracellular space is depleted of fluid. Free water is exactly that – water without electrolytes. Replacing free water may require administering D5W, as the metabolism of the dextrose will result in an equal quantity of free water. Calculating free water deficit should be done once the patient has had the extracellular fluid appropriately replaced; once the patient is no longer showing clinical signs of hypovolemic shock. If, after hypovolemia has been corrected and the patient still shows marked hypernatremia, free water replacement may need to occur. Fluid type

should most closely mirror the patient's serum sodium content and may need to be adjusted or changed as the patient reaches sodium levels closer to normal. Technicians are responsible for closely monitoring patient mentation and fluid rates to ensure patient safety.

Potassium derangement is common in acute kidney injury and failure and must be corrected quickly. Elevated potassium prevents muscle cell depolarization leading to deadly cardiac arrhythmias. In cases of cardiac dysfunction, 10% calcium gluconate (0.5-1.5ml/kg IV) can be administered to antagonize the hyperkalemic effects on the heart. Calcium gluconate must be administered slowly – the entire dose over 10-15 minutes – while the ECG is monitored for bradycardia. Its effects will last 30-60 minutes to allow for other methods to lower the serum potassium. In critically ill patients, insulin administration is utilized to drive potassium into the intracellular space. 0.5U/kg of Regular insulin administered IV takes about 30 minutes to accomplish this but will lower levels. Dextrose administered at 0.5g/kg should follow insulin administration to ensure the patient does not suffer from hypoglycemia. This may need to be repeated depending on patient response. Albuterol and terbutaline can also be administered to drive potassium to the intracellular space. The response from the patient may be inconsistent with this method, but it can be used as an adjunct treatment to insulin and dextrose. Terbutaline is administered at 0.01mg/kg IV slowly, or 2-3 puffs of albuterol via inhaler. Monitor these patients for tachycardia. Finally, sodium bicarbonate can be administered to correct potassium levels, as metabolic acidosis (common in kidney injury) will cause an extracellular shift of potassium. 1-2mEq/kg can be given slowly over 10-15 minutes. In cases of urinary obstruction causing hyperkalemia, relieving the obstruction will allow potassium levels to return to normal. In acute kidney injury not caused by obstruction, potassium disorders may linger and must be closely monitored.

Acute kidney injury can be a challenge to treat, as the kidneys cannot process fluids the patient can easily become fluid overloaded complicating treatment. Dialysis or renal replacement therapy is the gold standard of care for many of these patients (depending on cause) but is not always financially available or geographically available. Peritoneal dialysis is an option for these patients and can be performed in any critical care facility. It can be a burden on the staff as it requires a technician to be dedicated full time to the dialysis patient. Peritoneal dialysis works because solutes such as Na<sup>+</sup>, K<sup>+</sup>, urea, and creatinine will permeate across the peritoneal membrane. Dextrose added to fluids and infused into the peritoneal space will draw these solutes into the abdomen where they can then be drained from the body. Peritoneal dialysis catheters are ideally placed surgically where a partial omentectomy can be performed at the same time, but many patients requiring this treatment are critical and may not be ideal general anesthesia candidates. Short term catheters can be placed under sedation, but these will clog with omentum over time and may not be useful after only a few hours.

Furosemide is sometimes utilized in acute kidney injury in an attempt to remove excess fluid from circulation. While diuretics have no proven benefit in these patients, measuring their response to the drug may aid in the prognosis of the patient. Those with no increase in urine output in the face of furosemide carry a worse prognosis. The use of dopamine and fenoldopam because of their effect on

vasodilation and increased kidney perfusion were once thought to be beneficial in patients with acute kidney injury but studies have yet to prove this effect.

### **Nursing Care and Monitoring**

Kidney disease patients require close monitoring by the nursing team. Nursing care must be tailored to the patient, as each patient will have varying severity of disease and response to treatment. Urine output is one of the top monitoring needs and can be evaluated in different ways. Normal urine output is 1-2ml/kg/hr. Once a patient is placed on IV fluids, urine output should approximate fluid input, and 1-2ml/kg/hr while on IV fluids is considered oliguria. Measurement can be obtained by catching urine on walks for dogs, by utilizing plastic cat litter beads and measuring from the litterbox in cats, by weighing bedding prior to placing it in the kennel and again after the patient has urinated on it (1gram of weight = 1ml of urine), or by placing a urinary catheter. Urinary catheters are often a source of urinary tract infections, so it is important to not only place the catheter using appropriate aseptic technique, but to also care of the catheter throughout the hospital stay. Keep the catheter clean of feces and dirt, change the patient bedding frequently, and every 8 hours wipe the entire catheter and collection set with 0.05% chlorhexidine solution. If the patient allows, flushing the vaginal vault or prepuce with the dilute chlorhexidine should be performed.

Fluid therapy must be closely monitored. Those patients with electrolyte abnormalities may have frequent changes of fluid types and may have multiple fluids with frequently changing rates. The nursing team must ensure good communication and check these fluids often to make sure the plan is adhered to. Technicians must understand the clinical signs of rapidly changing sodium levels, and be aware of ECG changes associated with potassium derangement. Fluid additives such as potassium chloride and potassium phosphate may be ordered and will require math skills to ensure the patient receives the appropriate dose.

Body weight must be measured multiple times per day, at minimum the patient should be weighed twice daily. Weight gain can signify the body's inability to process fluids and may be the first clue that necessitates fluid rate changes. The same scale should be utilized each weight session to ensure comparable results.

Patients must be monitored closely for signs of edema. Edema below IV catheter tape is common, worse cases may see pitting edema as excess fluid leaks from capillaries. These patients should be monitored for chemosis as well which can signify possible fluid overload. Clear nasal discharge may appear and should be noted. Along those lines, patient should be auscultated multiple times per day listening for crackles and wheezes signifying pulmonary edema and an inability to handle the IV fluids. Occult cardiac disease in cats can make itself known during treatment for kidney disease and will complicate treatment plans.

Blood work is monitored frequently in kidney disease patients, not only looking for resolution of azotemia, but also closely monitoring acid/base status, electrolytes, and phosphorus. Sampling catheters

can be placed allowing for frequent blood sampling without causing more pain and stress to the patient. These lines require advanced skill to place and require more maintenance and care than peripheral catheters.

Urine specific gravity must be measured when the patient presents to the hospital and can be utilized to guide fluid therapy throughout the hospital stay. Urine specific gravity measures the density of urine – not the absolute solutes in the urine – so the results depends not only on the number of solutes present but also their weight. The specific gravity is a measurement of the tubules ability to resorb solutes and concentrate the urine. Isosthenuria (SpGr of 1.008-1.012) means that the urine osmolality is the same as the body plasma. Measuring the urine specific gravity on undiagnosed patients can assist in determining if the azotemia is due to pre-renal or intrinsic renal damage. In a dehydrated patient with azotemia, a urine specific gravity of >1.030 in a dog or >1.035 in a cat means the kidney function is still good, and the azotemia is most likely due to poor perfusion.

Uremia in these patients causes an increase in gastric acid production which leads to vomiting. These patients require GI protectants to prevent stomach ulceration. Sucralfate and omeprazole are commonly used. Famotidine, if used for these patients, should be used at lower doses as the drug is cleared through the kidneys. Uremic toxins also work on the chemoreceptor trigger zone, so central acting anti-emetics should be chosen to control vomiting. Severe uremia can induce thrombocytopenia; nursing care should always involve monitoring for signs of bleeding. When the medical team is able to overcome the patient's vomiting, nutrition is an important treatment for these patients. As the kidneys are unable to clear phosphorus, phosphorus binding medications such as aluminum hydroxide must be administered while the animal is eating. Nasogastric/nasoesophageal or esophageal feeding tubes should be utilized to provide nutrition in the earliest stages of treatment.

The technician team is vital to the triage, diagnosis, and treatment stages of kidney disease. Patients in kidney failure require close monitoring and communication between veterinarian, owner, and nursing team. Client education on kidney toxins and home care requiring medications and fluid administration is required. With team effort and top notch nursing care these cases can prove rewarding with ample opportunity to learn.

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## Nicole Hoffman CVT Neurology/Neurosurgery

Nicole has had an interest in animal's life long and decided to pursue their well being at a young age. She obtained her degree in Veterinary Technology from Madison Area Technical College in 2005. Working her 1st three years in general practice. She decided that she would like to persue a specialty and started working for the University of Madison Veterinary Teaching Hospital in May of 2008 as a flex tech working in a variety of departments ultimately falling in love with neurology. She hopes to obtain her veterinary specialist certificate in 2020. When away from the hospital she enjoys spending time with her husband and their 2 children Brennan (11yrs), Cassandra (5yrs) and her 4-legged kids, Sid (cat), Hondo (dog) Foxy(horse) and Woody (horse).





# The BAER Necessities

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Nicole Hoffman CVT Neurology/Neurosurgery

## What is BAER?

BAER stands for **B**rainstem **A**uditory **E**voked **R**esponse. What this means is we are recording the neural activity created in cranial nerve VIII, also known as the vestibulocochlear nerve. The sensory organ for hearing is located in the middle ear. The nerve arises from the sensory portion and terminates in the brainstem portion called the medulla. From these brainstem nuclei, information is distributed to the forebrain for conscious perception of hearing along a predictable path through the brainstem as well as to other brainstem locations for auditory reflexes. BAER testing is useful due to it being a minimally invasive test that can be performed quickly and can help assess the brainstem function. It also can help differentiate between conductive hearing loss and sensorneural hearing loss. Conductive hearing loss occurs secondarily to a dysfunction in the outer or middle ear (ex: PSOM or otitis). Conductive hearing loss occurs when dysfunction in the normal movement of sound to the cochlear. Sensorneural hearing loss can occur from an abnormal neural function (cochlear receptors, nerve or brainstem origin).

## How the test is performed

When performing a BAER test, we are using auditory stimuli typically in click form. Each click is a 0.1ms electrical pulse projected through an ear phone placed in the ear canal. By doing this we are testing the function and integrity of the outer, middle, and inner ear along with the auditory portion of the peripheral and central nervous system.

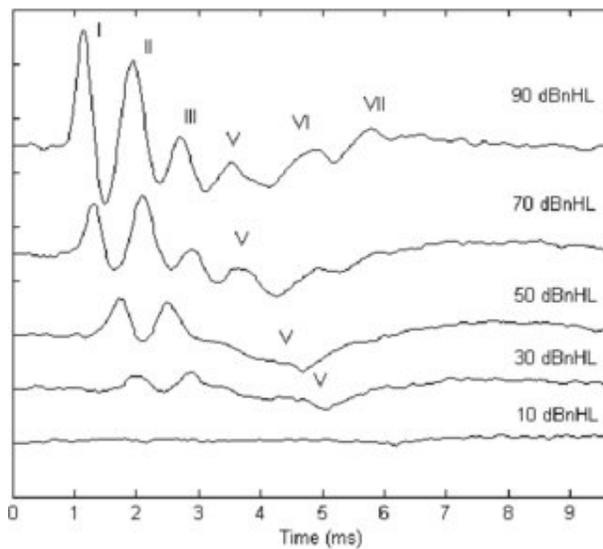
Each ear is tested independently from each other while the untested ear is masked with a lower intensity noise to reduce unwanted stimuli in the untested ear. In each test, we stimulate the ear with 200-1000 clicks. The results are averaged to create a tracing.

We place electrodes in the subcutaneous layer of the skin to record the electrical impulses as they travel from cranial nerve VIII through the brainstem.

BAER tests can be performed on animals that are awake, sedated or fully anesthetized with few changes in the results and minor changes to the size of the waveform.

Each tracing is broken down into seven waves numbered in Roman numerals I-VII. Many times, only six waves or peaks are identifiable and only the first five are clinically relevant.

Wave form	Generator
Wave I	CNVIII
Wave II	Nucleus (medulla)
Wave III	Trapezoid body
Wave V	Caudal Colliculus
Waves IV, VI, VII	Not clearly defined



### What we can test for:

The BAER can test for hearing loss due to several reasons such as: Cochlear agenesis and degeneration, presbycusis (age related), ear pathologies including primary secretory otitis media (PSOM) in Cavalier King Charles Spaniels, chronic otitis, acquired, or degenerative hearing loss.

### Who should we test:

Animals at risk for congenital deafness that are slated for breeding, those suspected to have congenital or acquired deafness or animals with an auricular injury may benefit from a BAER. It's been reported that over 100 canine breeds can be affected by congenital deafness. Congenital deafness can be hereditary or acquired before or shortly after birth. Inherent deafness is most commonly caused by a defective gene that can be a dominant, recessive or sex linked.

Congenital or inherited deafness is commonly recognized in canines with certain pigmentations of their hair coats, such as mono-pigmentation of white hair coats or bi-pigmentation of merle coloring. Although not all breeds with the merle coloring are affected, some of the common affected breeds are the Collie,

dappled Dachshund, and the Harlequin Great Dane. Boxers, Samoyed, Greyhound, Bulldogs, and Dalmatians are a few breeds that are affected by mono-pigmented coloring, also known as piebald gene. Dalmatians are particularly affected with 8% of the US population bilaterally deaf and 22% unilaterally deaf.

Other causes for deafness unrelated to inheritance may include: ototoxic drugs (gentamicin being the most common one), liver disease, intrauterine infections, other toxic exposures and aging. We test aging dogs when owners have expressed that their pet is unable to hear. Another population that is routinely tested are those canines that are in dangerous jobs such as the military and police force. These dogs are exposed to repeated loud noises, such as explosions, that can cause permanent damage. If these dogs lose their hearing, they are unable to keep themselves or the people they are protecting and serving safe. Long term exposure to kennel noise can also cause damage to hearing.

#### **When can we test:**

Puppies have four of the measured waves/peaks readily readable at 12-13 days old, when the ear canal is open. Recommendations are to test between 6-7 weeks old and to re-test at 16 weeks of age if any abnormalities are detected at the first test due to cochlear degeneration.

#### **Clinical usefulness:**

Given the minimally invasive nature of this test, routine use for hearing assessment is its most common utility. Not only is BAER testing useful for detection of deafness in dogs and cats, it may be used to identify brainstem malfunction unrelated to hearing. This is especially true when considering brainstem death.

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## Kristen Cooley, CVT, VTS (Anesthesia/Analgesia)

Kristen Cooley is a Veterinary Technician Specialist in anesthesia and analgesia. She is an Instructional Specialist at the University of Wisconsin, School of Veterinary Medicine where she has spent over decade studying anesthesia and pain management under some of the most brilliant minds. Kristen is the immediate Past President of the Academy of Veterinary Technicians in Anesthesia and Analgesia, she is a board member of the International Academy of Pain Management (IVAPM) and a part of the steering committee for the newly organized North American Veterinary Anesthesia Society (NAVAS). Kristen is a published editor, author and illustrator of anesthesia related text books as well as an internationally recognized and frequently sought after speaker and award winning instructor. She lives in Madison, Wisconsin with her 6 year old twins, a handful of dogs, cats, rodents and chickens and a 29 year old box turtle.





# Thinking Outside of the Box

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Kristen Cooley BA, CVT, VTS (Anesthesia/Analgesia)

Anesthesia carries with it a certain degree of risk and there is no single best way to anesthetize animals. Appropriate drug selection is important and largely dependent on the patient's physical status, temperament, type of procedure, present and anticipated pain, familiarity of drugs available and cost. It is essential that every animal undergoing anesthesia has a thorough and timely physical exam performed by a veterinarian. Essential information for anesthesia includes current and accurate body weight, body condition score for precise drug dosing, resting temperature, pulse and respiration, heart and lung auscultation and pulse synchronicity for an accurate baseline for trend monitoring. The animal's temperament and activity level are also important to consider when choosing an anesthetic drug protocol- anxious, aggressive or excitable patients will require different drugs compared to geriatric, calm or ill patients.

## One size does not fit all

It is generally not recommended to use pre-mixed drug cocktails like BAG or kitty magic. These cocktails do not allow for tailoring of the protocol to the specific temperament of the patient and often result in inappropriate dosing. There is also room for human error in mixing up large volumes of drugs, recording controlled substances and breaks in aseptic technique when pulling from a multi-dose vial. Best practice includes accurately drawing up each drug individually and then mixing them together for delivery. Designing an anesthetic protocol is easy if you remember PIMP, preanesthetics, induction, maintenance and pain management.

## Pre-anesthetics and balanced anesthesia

**Pre-anesthetics** are an essential component to successful anesthesia. Pre-anesthetics help to calm and sedate anxious, frightened or fractious patients. It streamlines restraint and decreases patient and staff stress. Pre-anesthetics reduce the necessary dose of induction drug and help to decrease the amount of inhalant needed for maintenance of anesthesia. Premedicated animals frequently have smoother inductions and calmer recoveries. Their anesthetic plane is often more even and easier to manage. Pre-emptive pain management, or giving an analgesic in anticipation of pain, should be part of the preanesthetic period. Pre-emptive analgesia can make post-operative pain easier to manage. **Balanced anesthesia** is the concurrent administration of smaller quantities of multiple drugs. The drugs work synergistically- combined drug effectiveness is greater than the sum of each part- which allows for the use of less of each drug minimizing side effects and maximizing therapeutic benefit.

## Route of administration

Pre-anesthetics are usually given either intramuscularly (IM), subcutaneously (SQ) or intravenously (IV). The onset and duration of action vary with each route as does the dose- in some instances a lower dose

given via a slower route may not ever reach therapeutic levels. The SQ route has the slowest onset and longest duration of action. This is often the easiest and least invasive route of administration however some medications are not well absorbed via this route and results tend to vary. Intramuscular route of administration has a faster onset time compared to SQ and a slightly shorter duration of action. Any large muscle group can be used for IM injection however the biceps and quadriceps offer the best and most consistent absorption. Patients should be allowed to sedate in a quiet area for 15-30 minutes when premedicated IM or SQ. Intravenous administration of drugs offers a faster onset and shorter duration of action and is often reserved for calm, aged or debilitated animals. An IV catheter should be placed when drugs are given via this route to avoid perivascular administration and patients should be allowed a few minutes to sedate before induction drugs are given. All IV medications should be given slowly and with caution and when applicable, doses should be adjusted.

### **Pharmacokinetics and pharmacodynamics**

Understanding anesthetic drugs requires an appreciation of each drug's pharmacokinetics and pharmacodynamics. **Pharmacokinetics** refers to the effect the body has on the drug and **pharmacodynamics** refers to the effect the drug has on the body. Drugs enter the body via the bloodstream and bind to specific receptors located in target organs and tissues and exerts specific effects- for anesthetics these effects are usually central nervous system depression or stimulation. **Agonists** are drugs that bind to and stimulate a specific receptor found in target tissues. **Antagonists** bind to but do not stimulate receptors- most antagonists competitively bind to receptors and displace agonists effectively 'reversing' the action of the initial drug. A **partial agonist** is a drug that only partially stimulates a receptor and an **agonist-antagonist** binds to more than one receptor, agonizing one and antagonizing another.

### **Anticholinergics**

Sometimes pre-anesthetics contain an anticholinergic to prevent bradycardia and decrease salivary secretions. These drugs competitively block the binding of the neurotransmitter acetylcholine blocking parasympathetic effects (bradycardia, bronchoconstriction, excessive tear and saliva production, respiratory secretions, GI motility and miosis).

**Atropine** is derived from the deadly nightshade plant and has a rapid onset of about 1 minute when given IV with a peak effect occurring in about 3-4 minutes making it ideal for emergency use.

**Glycopyrrolate** is a synthetic quaternary ammonium compound that has a slightly longer onset time and longer duration of action. Glycopyrrolate does not cross the blood-brain barrier and is better suited for routine use.

Drug class effects include secretion reduction, bronchodilation, arrhythmias (glyco is less arrhythmogenic), thickening of respiratory secretions, and inhibition of intestinal peristalsis. Conservative use of anticholinergics is recommended and their use should never be a substitute for diligent monitoring.

### **Neuroleptanalgesia**

Pre-anesthetics can consist of an opioid combined with a sedative or tranquilizer- this is referred to as **neuroleptanalgesia**. These drug classes work together **synergistically** providing better sedation and pain control at lower doses compared to either drug alone. A **sedative** is an agent that causes drowsiness by

depressing the central nervous system (CNS) where as a **tranquilizer** is an agent that decreases anxiety. Most of the sedative and tranquilizers that we commonly use have overlapping effects- they produce both drowsiness and anxiolysis.

### **Benzodiazepines**

Benzodiazepines like diazepam and midazolam are sedatives that work by increasing the activity of an inhibitory neurotransmitter- gamm-aminobutyric acid or GABA. This class of drugs is used for anti-anxiety and calming in ill or geriatric animals, skeletal muscle relaxation, appetite stimulation and as an anticonvulsant. Benzodiazepines do not provide any analgesia and cause very minimal (if any) cardiovascular and respiratory depression. They have the tendency to decrease inhibition in young, healthy animals causing paradoxical excitement especially when given alone. Benzodiazepines are reversible with the drug flumazenil. Onset time is rapid and duration of action is 1-3 hours

**Diazepam:** propylene glycol based, for IV use only

**Midazolam:** water based, can be given IM, SQ or IV

### **Phenothiazines**

The phenothiazine, acepromazine or 'ace', is a tranquilizer often used as a preanesthetic in cats, dogs and horses. Acepromazine is an alpha-1 adrenergic and dopaminergic receptor antagonist that causes dose dependent sedation and a generalized disinterest in surroundings. It has no analgesic properties and it can mask the signs of pain without alleviating them. Ace can cause peripheral vasodilation that can negatively affect blood pressure and body temperature. It also has antiemetic and antihistamine effects. Onset time is around 15 minutes with peak effect at 30 minutes. Duration of action is variable and dose-dependent and can last 4-8 hours in small animals.

### **Alpha-2 adrenergic agonists**

Alpha-2 adrenergic agonists provide sedation, muscle relaxation and analgesia. This class includes the drugs detomidine, dexmedetomidine and xylazine which are commonly used in both large and small animals. Dexmedetomidine is more specific for the alpha-2 receptor making the side effects associated with this drug class less prominent compared to xylazine and therefore safer for our small animal patients. Sedation and analgesia is dose dependent as is the duration of action. Peripheral vasoconstriction leads to pale mucous membranes and a reflex bradycardia. Vomiting and increase urine production are not uncommon. Some animals may be refractory to the drug. Co-administer with an opioid and allow patients to sedate in a quiet place to decrease stimulation. Alpha-2 agonists are fully reversible with atipamezole (dexmedetomidine) or yohimbine (xylazine). The volume of reversal given should be the same as the volume of drug given previously.

**Dexmedetomidine:** onset time is 5-15 minutes IV, 15-30 minutes IM. Duration of action is around 90 minutes.

### **Opioids**

Opioids provide analgesia by binding to specific receptors located in the brainstem and spinal cord. Some opioid receptors can also be found in peripheral tissues. Opioids are great analgesics and they work at various locations along the pain pathway. Specifically they block transduction that occurs peripherally and

they decrease modulation in the spinal cord and dampen perception in the brain. Opioids don't remove pain; they raise the threshold making pain more tolerable. There are two main types of opioid receptors, **kappa** and **mu**. Pain is mediated by mu receptors in mammals and kappa in birds. There is some evidence to suggest that kappa receptors exist in higher numbers in the gut compared to the peripheral tissues in mammals allowing kappa agonists to provide good visceral analgesia.

Pure mu agonist opioids include morphine, hydromorphone, oxymorphone, fentanyl and methadone. This group of drugs fully bind to the mu receptor and are effective at providing analgesia. They can also cause sedation, especially in geriatric, pediatric or debilitated animals. Pure mu agonist opioids cause minimal cardiovascular depression and dose dependent respiratory depression by increasing the resting partial pressure of carbon dioxide or PaCO<sub>2</sub>. Bradycardia from vagal stimulation, vomiting from direct stimulation of the chemoreceptor trigger zone (CTZ), urine retention from decrease sensation and urge, antitussive properties and resetting of thermoregulation are not uncommon side effects of this drug class.

**Morphine:** histamine release when given IV, poor oral bioavailability, vomiting is common, high lipid solubility make it a great for epidural use (preservative free formulation is recommended). Duration of action is 4-6 hours.

**Hydromorphone/oxymorphone:** less vomiting compared to morphine, no histamine release IV, hydromorphone may cause hyperthermia in cats. Duration of action is 2-3 hours.

**IV fentanyl:** very potent synthetic opioid, 3-8 minute onset time IV with a short duration of 20-30 minutes. Given as a constant rate infusion (CRI).

**Fentanyl patch:** variable absorption that may not provide adequate analgesia if used as a sole means of analgesia. Takes 12-18 hours to reach therapeutic plasma levels and can potentially last for 3 days. Dose accordingly!

**Methadone:** pure mu agonist and NMDA antagonist (helps prevent wind-up pain by blocking n-methyl d-aspartate). Good analgesia and less sedation compared to morphine. Duration of action is 3-4 hours.

Agonist/antagonist drugs stimulate one receptor while blocking another. Drugs like butorphanol and nalbuphine stimulate the kappa receptor while blocking the mu receptor making them only effective at treating mild pain in mammals. Because they block the mu receptor they can be used to reverse any unwanted effects of a pure mu agonist opioid while maintaining some analgesia.

**Butorphanol:** mild analgesic with a ceiling effect (more drug does not equal more analgesia), short duration of action lasting 45 minutes- sedation may last longer, controlled as schedule IV

**Nalbuphine:** mild analgesic, short duration of action lasting only 30 minutes, not a controlled substance

A partial agonist opioid is a drug that does not fully bind to the mu receptor making it less effective than a pure mu agonist like morphine.

Partial agonists are drugs that only partially bind to the mu receptor exerting an effect that is not as great

**Buprenorphine:** very high affinity for the mu receptor making the use of subsequent opioids ineffective until buprenorphine has worn off. Slow onset of action taking it nearly 45 minutes to provide pain control

but long duration of action 6-8 hours, great transmucosal absorption in the cat but only about 30% bioavailable transmucosally in the dog, class III controlled substance.

**Simbadol:** High potency buprenorphine. Extended duration of action based on dosage.

**Sustained release buprenorphine (SR)** is not an FDA approved product but is available through a compounding pharmacy. It is given SQ and has been shown to provide up to 72 hours of analgesia. Reversal is difficult and may require hospitalization.

### Opioid antagonists

Antagonists completely reverse the effects (analgesia, sedation and cardiopulmonary depression) of all circulating opioids including endogenous ones. These drugs should only be used in the face of an absolute opioid overdose because their use removes ALL analgesia along with any other effects. An acute awareness of pain can lead to catecholamine release from sympathetic stimulation which may result in cardiac arrhythmias, hypertension and possibly death. Redosing may be necessary due to the short duration of action of naloxone compared to most opioids.

**Naloxone:** onset 1-2 minutes IV, 5 minutes IM, duration of action 30-60 minutes

**Nalmefene:** onset 1-2 minutes IV, 5 minutes IM, duration of action 1-2 hours

### Induction Drugs

Induction drugs consist of injectable agents that allow the anesthetist to induce anesthesia quickly and to secure an airway. Injectable agents provide a safer, less stressful alternative to 'gassing' or 'boxing' down a patient. It is also less expensive, faster, with less waste/pollution and more control. All injectable anesthetics depress some vital organ function making the use of preanesthetics and subsequent lower doses essential.

### Dissociative anesthetics

Also referred to as cyclohexamines- these drugs produce a dissociated state along with sympathetic stimulation. Increases in cerebral blood flow and intracranial and intra-ocular pressure, CNS stimulation, primary cardiovascular depression with indirect sympathetic stimulation leading to increased heart rate, blood pressure and cardiac output. Sub-anesthetic doses of ketamine (and possibly Telazol) act as NMDA antagonists blocking **central sensitization** (wind-up pain) in the dorsal horn of the spinal cord. They are not analgesics in and of themselves and should not be used as a sole means of pain control however; ketamine is a great adjunctive medication contributing to a balanced approach to analgesia.

**Ketamine:** causes muscle rigidity when used alone, commonly coupled with a benzodiazepine for induction. Not recommended for use with an anticholinergic as significant tachycardias can ensue, hepatic metabolism in the dog, renal excretion in the cat.

Ketamine and Telazol are contraindicated in patients that are sympathetically spent- severe trauma, stress, shock etc. This is due to the fact that only the direct cardiovascular effects will be appreciated. Not recommended for use in patients with cardiovascular disease, head trauma, intracranial masses, glaucoma, corneal ulcers, pheochromocytoma, hyperthyroidism.

**Propofol and Propofol-28:** Propofol is an ultra-short acting non-barbiturate induction drug with rapid metabolic clearance. Ninety seconds to peak effect and 5 minutes to redistribute, non-cumulative, administer slowly and steady to avoid over-dosing. Propofol has the potential to cause profound cardiovascular and respiratory depression and should be used cautiously. IV injection can be painful and muscle twitching and seizure-like activity has been reported. Repeated dosing is not recommended in cats as oxidative injury to red blood cells may result. Drug can be re-dosed intraoperatively at 1-2 mg/kg if patient becomes light. Pre-oxygenation is also recommended. Propofol-28 contains benzyl alcohol as a preservative giving it a 28-day shelf life. Regular propofol should be discarded 6-8 hours after opening.

**Etomidate:** Etomidate has little to no negative effect on the cardiovascular system- ideal induction drug for hemodynamically unstable patients. It may have brain protective properties after global ischemia, and inhibits steroid production by the adrenal glands for 3-6 hours after administration- not recommended in patients with hypoadrenocortism. Vomiting and retching is common with underdosing and etomidate can be painful IV because it is based in propylene glycol. This drug is also expensive.

**Alfaxalone:** Alfaxalone is a neuroactive steroid substance that is used extensively in the UK, Australia, Europe and Canada. It is currently awaiting DEA scheduling for use in the US. It produces hypnosis with reasonable muscle relaxation and a decrease in cerebral blood flow and cerebral oxygen demand. Dose dependent hypotension may be seen initially due to myocardial depression and peripheral vasodilation but the effects are often offset by the reflex tachycardia. Respiratory depression associated with the use of alfaxalone is dose dependent; the drug is non-cumulative and approved for IV use only. IM chemical restraint is off-label and works better for cats vs. dogs (very short duration of action). Alfaxalone is not an analgesic, it does not contain preservatives and should be discarded 6 hours after opening.

## Maintenance

Maintenance of anesthesia is often achieved through the use of inhalant anesthetics. Inhalants are potent vasodilators and cause dose-dependent hypotension and respiratory depression. The two most common inhalants used in veterinary medicine are isoflurane and sevoflurane. The minimum alveolar concentration or MAC is the potency of an inhalant. MAC-50 is the amount of inhalant needed to keep 50% of patients non-responsive to surgical stimulation. MAC-95 or surgical MAC is the amount of inhalant needed to keep 95% of patients non-responsive to surgical stimulation and is calculated by multiplying MAC-50 for the species by 1.5. It is important to note that respiratory arrest can occur at 2 x MAC so proper dosing is essential to safety. MAC studies are done on patients who have not been given pre-anesthetics. The use of pre-anesthetics decreases the amount of inhalant necessary thereby reducing MAC.

**Isoflurane:** MAC is 1.3% in dogs and 1.6% in cats, low blood-gas solubility (rapid induction and recovery), 0.2% metabolized in the body

**Sevoflurane:** MAC is 2.3% in dogs and 2.6% in cats (sevo is less potent and requires higher vaporizer settings to maintain anesthesia), lower blood-gas solubility, 3% metabolized in the body.

## Pain management

Pain management is an important part of the anesthetic protocol and balanced techniques should be employed whenever possible. In addition to opioids and alpha-2 agonists, the flowing drugs should augment the pain management protocol.

**Non-steroidal anti-inflammatory drugs (NSAIDS):** NSAIDS (Non-steroidal anti-inflammatory drugs) block the production of specific prostaglandins by binding and inhibiting the cyclooxygenase (COX) enzyme. COX enzymes (COX-1 and COX-2) also have important homeostatic functions. COX-1 mediates prostaglandins responsible for renal and GI blood flow and platelet integrity and COX-2 mediates prostaglandins responsible for inflammation, pain, edema, fever as well as other homeostatic functions. COX-2 selective NSAIDS (carprofen, meloxicam, robenacoxib) are preferred because by sparing the COX-1 enzyme GI side effects are less likely. NSAIDS should be avoided in animals with renal or hepatic dysfunction, coagulopathies, GI disorders, shock, hypotension/hypovolemia and they are not recommended for use in combination with corticosteroids.

**Lidocaine:** Lidocaine is a local-anesthetic and anti-arrhythmic agent with a rapid onset and short duration of action. Lidocaine is a prokinetic that enhances gut motility and help prevent ileus. It has MAC sparing effects when given as a constant rate infusion, a loading dose must be given prior to starting a CRI to achieve blood levels. Cats are sensitive to lidocaine and care should be taken when this drug is used.

**MLK (Morphine, Lidocaine, Ketamine):** Drugs are combined into a bag of IV fluids and delivered at a surgical rate. Loading dose of each drug is required to achieve therapeutic levels.

**Tramadol** is an oral medication that has weak mu receptor effects along with norepinephrine and serotonin reuptake inhibition. Tramadol works well with NSAIDS as post-operative pain management.

**Gabapentin** is effective at reducing hyperalgesia and allodynia associated with neuropathic pain and central sensitization as well as chronic and malignant pain. Gabapentin is not an analgesic but an adjunctive medication that allows true analgesics to work better by calming down the nervous system- gabapentin should be used in conjunction with NSAIDS and/or tramadol for best results.

**Amantadine** is an NMDA antagonist and analgesic adjunctive medication. It is good at reducing central sensitization. Amantadine is excreted almost unchanged in the urine- reduce doses in the renal patient.

**Ketamine** CRI is an excellent and inexpensive adjunctive medication to use on surgical patients to decrease MAC.

**Cannabis** should be considered as a potential drug to help treat both acute and chronic pain. More information to come on this topic.

There will always be risks associated with anesthesia but those risks can be minimized when patients are appropriately evaluated and drug protocols individualized. Anesthetists well-versed in pharmacology are better suited to anticipate potential drug-related complications and side effects. Tailored protocols that include a plan for preanesthetics, induction agents, maintenance drugs and pain management will ensure that a balanced approach is used.



## Thomas Masterson

Mr. Thomas Masterson serves as President of MediVet Biologics a Lexington, KY based animal health company. Mr. Masterson is a RACE certified speaker on the topic of regenerative medicine & immunotherapy. He is an author of numerous articles and publications in animal health. Mr. Masterson is a graduate of the University of Kentucky.





# Translational Biologics in Veterinary Medicine: Regenerative Medicine & Immunotherapy

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Thomas J. Masterson

Our bodies are designed to provide restoration of injured or diseased tissues and to eliminate infectious microorganisms. In some instances, such as advanced age, chronic disease processes may overwhelm the body's capacity to provide these important functions and to maintain normal physiologic functions, in which case therapeutic intervention is needed.

Scientific innovations, such as cell-based therapies, have been translated into new medical treatment plans in human health.

Regenerative Medicine such as Stem Cell therapies are becoming more common for a variety of medical issues. Similarly, Immunotherapy is now seen as the fourth modality in cancer treatment.

These cell-based options for pets are translational approaches which can improve quality of life and strengthen the human and animal bond, leading to synergistic improvements in health.

## Stem Cell Therapy

Stem Cells are found in extremely small numbers throughout the body. They act as sentinels for tissue repair and maintenance of normal tissue function when damage occurs in those tissues. These repair cells are mobilized by injury and disease to carry out their restorative functions.

Stem cells can be isolated from normal tissues and used therapeutically to promote tissue repair. Use of Adult Stem Cells is an alternative to using Embryonic Stem Cells. Adult tissue sources such as adipose (fat) tissue and bone marrow contain resident Mesenchymal Stem cells (MSCs), a type of adult stem cell. Mesenchymal Stem Cells can engraft into host tissues, differentiate or transform into a number of normal tissue cell types and can provide immunomodulatory effects. Additionally, these MSCs exert potent anti-inflammatory and pro-angiogenic properties.

Autologous (from the patient) Adult Stem Cells (ASCs) can be safely harvested from the patient, concentrated, and then re-administered on the same day. Because adipose (fat) tissue contains greater than 100-fold more Mesenchymal Stem Cells than bone marrow, it provides an important source of ASCs for therapeutic use.

Degenerative Diseases such as osteoarthritis are a common problem in pets as they age. Adult Stem Cells from fat can provide a safe, simple and affordable solution to pet owners seeking alternatives to medication or surgery. In addition, stem cell treatments hold promises for many other inflammatory based diseases.

## **Immunotherapy**

Cancer cells can be recognized as foreign by the immune system if those cancer cells are presented to the immune system in a manner that overcomes immune tolerance and immunosuppression, features which are commonly associated with cancer. Decades of research in humans and animal models have shown that tumor cells are very much like normal cells in that they express self-antigens that the body is taught to ignore early in development and throughout life.

An immunosuppressive environment produced by growing tumors promotes an immune response toward the tumor that is ineffective, allowing the tumor to grow unchecked.

An appropriately designed immunotherapy, can break this cycle of tumor immunosuppression, generating tumor-specific immune cells that have effective anti-tumor reactivity, improving survival and quality of life in veterinary patients.

50% of dogs over the age of ten will develop some type of cancer. There is a need for an affordable, broadly applicable and transportable solution to canine cancer. Autologous tumor derived vaccines can fulfill these criteria and provide complimentary treatment to conventional cancer therapies.

*References are available upon request*

## Shawn Hook, DVM

Dr. Shawn Hook graduated from the University of Wisconsin - Madison - School of Veterinary Medicine in May of 1999. Since that time he has been practicing veterinary medicine in the Madison area. Dr. Shawn Hook has served as Vice President and President of the DCVMA (Dane County Veterinary Medical Association). Doctor Shawn Hook is a member of the American Veterinary Medical Association (AVMA), the Wisconsin Veterinary Medical Association (WVMA), The Dane County Veterinary Medical Association (DCVMA), the American Heartworm Society (AHS), American Animal Hospitals of America (AAHA), the Association of Exotic Mammal Veterinarians (AEMV), the Association of Avian Veterinarians (AAV), American Ferret Association (AFA), and the Association of Reptilian and Amphibian Veterinarians (ARAV). Dr Shawn Hook taught Exotic Animal Medicine at Globe University in their Technician Program. His special interests are in exotic animal medicine and surgery, ultrasonography and soft tissue surgery. Dr. Hook took over ownership of the Arbor Ridge Pet Clinic in March of 2007. You may email him with questions at [shawnhookdvm@yahoo.com](mailto:shawnhookdvm@yahoo.com).





## Improving Quality of Life for Exotics Pets – Cage Activities, Foraging, Enrichment

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Why is this important

- Remember that the opposite of stress is boredom. We want to avoid too much stress or bad stress but a little stress is good as it alleviates boredom.
- There are different types of stress – some we want to avoid like a dog that is constantly barking and snapping at a rabbit cage is a bad type of stress that long term will cause physiological and psychological trauma.
- However, people taking a roller coaster is a stress, but it causes excitement and joy by doing it (for some people). Just like a monkey finding a box with a tasty banana hidden inside
- Our goal in the veterinary world is not only to provide medical care, but to improve quality and quantity of our patients lives
- Stress can lead to a whole cascade of events that can shorten a patient’s life.

What are things that we can recommend to improve quality of life?

- Attention – handling of patients – one on one time
- Grooming and bathing
- Auditory Stimulus
- Ocular Stimulus
- Cage Positioning Reducing bad “Stress”
- Thermoregulation
- Keeping environment clean
- Access to clean water and food
- Toys and Puzzles
- Training and games
- Exercise
- Foraging

## Attention – One on One time

- This may seem redundant, but attention for some of our exotic pets is very important. It is important to have a conversation early with our owners the FIRST time that they present the pet for an examination about attention and time.
- The owner should try to be consistent with time spent with the pet. Changing this frequency can lead to destructive behaviors in the cage and sometimes self mutilation.
- Our avian patients in particular are very sensitive to this and it is an important facet to maintaining a healthy living environment for them.
- It can be as simple as 15 minutes every night with the owner when they are home from work. But if done constantly can help prevent stress and bad behaviors tremendously.
- Some patients it is very obvious need attention from the owner like birds, ferrets, guinea pigs, rabbits, but many reptiles, amphibians and even certain fish like attention as well.
- Birds and many mammals like to be petted but animals like reptiles may like just being looked at or talked to. Some fish like to chase or interact with fingers touching the glass of their tank and moved along.
- The important thing to emphasize is for the owner to be consistent with the attention that they give every day as the pet will come to expect this interaction each day.
- This should be one of the easiest things for the owner to provide as that is the reason that they bought the pet in the first place to give it attention

## Grooming and bathing

- Grooming is important for all pets, some like grooming from their owners, some groom themselves
- This can be another form of attention or interaction for pets and their owners that the love.
- Using appropriate brushes and shampoos this can be done safely at home

## Grooming/Bathing - Small mammals

- Making sure to use appropriate shampoos, but oatmeal and aloe Dog/Cat Shampoo diluted with water can be used safely in these pets.
- Rabbits and Chinchillas have very fine fur and easily torn skin so using gently flowing water from a dish side sprayer is a good way to rinse them.
- Using your hand or rubber gloved brushes are a gentle way to groom their coats as slicker or wire brushes are too traumatic to their skin
- With Hedgehogs using a medium or soft bristle toothbrush is a great way to brush their quills safely.

- Some mammals, especially rabbits like being brushed or groomed weekly with rubber or rubber brush tipped gloves.

#### Grooming/bathing - birds

- Some birds love showers or baths. This is best done with warm water and no detergents or shampoos
- If they do not like being bathed they can be slowly introduced to it or have the animal perched in the bathroom with the doors shut and fans off to provide a humid environment for their coat
- Having the owners gently pet the birds feathers and gently breaking down the shedding cuticles when they are molting is a great interaction that the owner can do with the bird. These molting feathers are often itchy and it is a great way to help the bird molt and shed the dry cuticle from the newly grown feather.

#### Grooming/Bathing Reptiles

- Some reptile like showers or baths. This is best done with warm water and no detergents or shampoos
- In reptiles this can help remove retained shed, especially on the toes of small lizards like geckos who are prone to losing toes from retained shed.
- We do not recommend bathing amphibians as they can absorb toxins on your fingers through their skin easily. (always important to clean and rinse your hands well prior to handling – esp. if you smoke).

#### Auditory Stimulus

- Many pets like having the sound of people around them while you are gone.
- This can be achieved by having talk radio at a normal (not loud) level or recordings or your voice or podcasts on while away
- Care needs to be taken to avoid music or sound that the pet does not like or at levels that will stress the patient.

#### Ocular Stimulus

- Some patients like birds like ocular stimulus such as a television.
- Just as the radio care needs to be taken with the volume level and programming played as this could cause stress to the bird depending on the material.
- In addition with birds that speak care should be taken with the language that is used in the programming material that you expose them to as they could learn vocabulary that you do not want them to repeat.

### Cage position and bad stress

- The position of the cage is very important to quality of life for exotic pets.
- Having the cage in a position that is away from stressors like a loud or active children or an excited or barking dog or frequent company or new people.
- Animals need regular sleep intervals. Having the cage in a room where people are up and active late at night and then another person is up and active early in the morning in that room can disrupt the pet's normal sleep cycle.
- Having the pet in another less active room or a sleep cage in a quiet room is a good idea.

### Thermoregulation

- Proper temperature in the cage is important for health of all exotic pets.
- An environment too cold can be a risk for birds or our small pocket pets
- However reptiles, amphibians and fish are probably the most reliant on proper cage temperature.
- Reptiles and Amphibians thermoregulate by going to their POTZ
- POTZ – Preferred Optimal Temperature Zone.
- It is important to provide varying temperature zones in their cage so that throughout the day the animal can go to their preferred temperature zone.
- Digestion and immune system work best at certain temperatures which is why it is important to provide this to animals that can not thermoregulate on their own.

### Clean environment and water

- Having clean food, water bowls and environment is a very important factor for a healthy pet as well.
- Discussing and developing a frequent and consistent cleaning routine with the owner on the first visit is important

### Toys, Puzzles and games

- Giving animals like birds and pocket pets puzzles and games to play can help alleviate boredom
- Safe toys and puzzles can be used if made with plastics or paints that are safe, lead free (recommend not from China).
- Other things can be used as toys and puzzles – like putting knots in rope, shoe strings or strips of leather can be given to your bird to untie.

- As with anything offered new, the pet may avoid the item for a while until they are comfortable with the item

### Exercise

- Exercise can be an important thing to do with your exotic pets, not only to help maintain a healthy weight, but also help alleviate boredom.
- Wheels can be used for rats, gerbils, mice, hamsters and degus.
- It is important to use solid wheels to help prevent the animal from catching its toes and nails to help prevent breakage
- Hamster and guinea pig balls can be used to allow for exercise around the house for pocket pets as well.

### Foraging

- This is a very important one.
- Giving the pet something to do while the owner is away is a good way to avoid boredom and help prevent destructive behavior or self mutilation.
- Foraging consists of using toys, puzzles, cardboard boxes and paper to seal in treats or food stuffs.
- The treat is used a reward for opening or working on the puzzle.
- It causes the pet to be engaged or stimulated cognitively and rewards them with a treat that reinforces the behavior.



# Exotic Nutrition

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## **Exotic Nutrition**

Nutrition is a leading cause of many of the medical problems that our exotic pets

Proper diet and nutrition can lead to not only a longer life, but a healthier one as well.

Some Exotic Pets have commercially prepared diets that are a complete nutrition

Many of our exotic pet's diets are not completely understood and we have to form the recommended diets from what we currently understand about their physiology.

## **The Goal When You Leave Here**

To be able to provide advice for proper nutrition in the exotic pets that you see in your practice

Be able to educate owners on proper diets and the risks and pitfalls of feeding the wrong diet

## **Water is Very Important**

Clean water should be available to the pet at all times

Clean water dishes weekly to try to keep bacteria counts low

If using a sipper bottle, make sure to check to make sure water is coming out as they can sometimes develop "vapor lock" and not administer the water to your pet properly

Most animals get used to "one" was of getting water so it is important to provide this to the pet. It is especially important when introducing a new pet to a cage to ensure that they can drink from the provided bowl or bottle.

Some animals, like Chameleons will only drink water from leaves or tree branches so drippers or misters are needed in the cage at all times.

## **Rats, Gerbils, and Hamsters**

These rodents are omnivores and so need a variety in their diet to provide adequate nutrition

They are prone to obesity, this is especially true when fed high fat diets

They are hoarders, especially rats, so giving them large food articles makes it harder to hoard.

As they need food available all the time it is difficult to “diet” these animals

Providing exercise, foraging and feeding lower fat foods is a good way to prevent obesity.

The majority of the diet should be commercially prepared food.

A good source of pellets is Oxbow Pellets, however Mazuri makes comparable pellets for these pets.

Avoid pellets that are mixed with seeds as these are too high in fat and put the animals at risk for obesity and liver disease

Lab Block Pellets can be used and help with rats to reduce hoarding.

Small amounts of leafy greens and fruits and veggies may be offered each day in small amounts. (Avoid onion, avocado, garlic, and tomatoes)

### **Degus, Chinchillas, Guinea Pigs, and Rabbits**

These mammals are hind gut fermenters.

This means that the majority of their diet should be roughage or non-digestible fiber in the form of Grass Hay (Timothy, Orchard Grass Etc. – Not Alfalfa which is a legume and high in sugars)

75% of their diets should be Grass Hay

25-30% of their diet should be a salad each day made up of leafy greens (collard greens, radicchio, endives, romaine lettuce) and small amounts of non-leafy green veggies and fruits (peppers, raspberries, blackberries, strawberries, kiwi, star fruit, mango, citrus – orange, tangerine, etc).

Fruits should be avoided in Degus as they are VERY prone to Diabetes.

Diets too high in calcium like spinach, alfalfa, sprouts, broccoli can make these hind gut fermenters prone to stones or uroliths in their bladders.

These are normally Calcium Carbonate Stones

Increase water intake and decrease foods high in Calcium to help prevent reoccurrence, in addition can discontinue pellets.

Pellet amounts – for chinchillas, Degus and Guinea Pigs recommend a Tablespoon per day. For rabbits recommend 1/8 cup for a small rabbit and ¼ cup for a large rabbit per day.

### **Guinea Pigs**

Don't forget with Guinea Pigs that they cannot make Vitamin C on their own

They need daily supplementation 80-100 mg

This can be with treats, chewable vitamins or food (Orange, tangerine, Kiwi, peppers, etc)

DO NOT recommend water based Vitamin C as the bacteria in the water breaks it down quickly and it can cause bacterial overgrowth in the water.

## **Ferrets**

We are fortunate that we have many diets available to clients now.

When we used to have only Marshall Farms Ferret Food available an alternative was a kitten food or diet. Unless there are medical reasons to I would not recommend cat food.

Can be prone to obesity so may have to limit the amount fed, however having multiple ferrets can make this more difficult

Brands of Ferret food Marshall Farms, Totally Ferret, Mazuri, ZuPreem, Kaytee (pelleted only).

## **Hedgehogs**

Hedgehogs are insectivores. There are many good pelleted foods available to hedgehogs that are specific to hedgehogs

It is not recommended to feed cat food as this tends to lead to obesity which is a problem with hedgehogs but also it makes the prone to bladder stones. Bladder stones have only been noted when hedgehogs are fed a cat food and not when they are on a hedgehog or insectivore diet. If cat food has to be fed, it is recommended to use a light or low calorie cat food

Insects can be offered like crickets or mealworms though they should only be offered occasionally for enrichment over a normal diet. Ideally the insects should be gut loaded first.

Veggies like leafy greens, fruits and other can be offered in moderation if the hedgehog wants it

## **Sugar Glider**

These guys are nocturnal insectivores.

They do produce a pelleted food.

Mazuri makes a good one that I recommend.

Gut loaded insects like crickets can be given but more for enrichment and occasional treats and not as the main diet.

They DO NOT need nectar the those available are really sugar water and NOT recommended.

I do recommend Calcium supplement three times a week as they are prone to hypocalcemia, especially when younger.

## **Birds**

We will discuss diets for these birds by breaking them into groups to make it easier.

### **Chickens**

Fowl and Anseriformes (ducks, geese, swans)

Psittacines (parrots) and passerines and Mynahs

Most of the chickens that you see in your practice will likely be backyard flocks. Usually 2-5 birds and all laying hens as roosters are usually illegal in town.

If they are young birds it is recommended to feed them a growing or grower diet or starter diet. Fed 1-20 weeks of age.

As they reach adulthood it is recommended to put them on and maintain them on a laying diet or Layer Diet. Fed >20 weeks of age.

Offering mealworms, other insects or parasite free worms can be offered but are not necessary and more for enrichment.

Leafy greens can be offered daily sparingly but are not required.

### **Fowl and Anseriform Diet**

These diets like chicken feed are often acquired through feed shops or stores like Farm and Fleet or Fleet/Farm

They make grower diets like chickens as well as maintenance or layer diets for adults.

Parasite free insects or worms can be offered but are not necessary.

### **Psittacines, Passerines, Tucans and Mynahs**

These birds have the most commercially available diets in the pet stores.

Unfortunately there are a lot of bad foods out there

Many breeders wean birds on to a seed diet.

Birds don't like change and bird seed is high in fat and fat tastes good so it is harder to switch them off seeds to pellets.

It is recommended to feed 75% of the diet as a quality pelleted food.

We do recommend to feed and offer leafy greens on cage clips or in dishes chopped up with fruits and veggies

A small square of non-white bread or toast should be available as a grain source as well.

## **Reptiles**

To make this easier we will break them down into different categories:

Snakes

Aquatic Turtles

Box Turtles

Tortoises

Squamids – lizards, geckos etc.

Herbivorous

Omnivorous

Carnivores

### **Snakes**

All snakes are carnivores.

Except for young snakes where they may only be able to eat insects like small crickets, snakes should be fed frozen thawed food. Make sure to gut load the insects first.

I recommend having a temp gun to test the temperature of the food before it is fed.

With mice and rats when taken out of the freezer place them in a clear plastic bag and soak in hot water for 15-20 minutes, then test the temperature (ideally 90-95 Deg F)

Gut Loading

Gut loading means that you feed the insects for at least 5 days prior to feeding the insects to the reptile.

This can be achieved with Flukers Gut Load diet cubes (recommended) or feeding crickets veggies, fruits, grains or dog/cat food.

Water for insects I recommend sponges soaked in water daily. Crickets will drown themselves easily so giving them a bowl of water will result in many dead crickets.

### **Aquatic Turtles**

Aquatic turtles are carnivorous as well.

A good pelleted food is a good base for aquatic turtle.

Minnnows, feeder goldfish can be offered occasionally but should not make up the bulk of the diet

Leafy greens soaked and floated in the water can be offered and fed if eaten.

A UV-B light should be provided and on for 10-12 hours a day to aid in Vitamin D3 Production needed for their diet.

### **Box Turtle**

Box turtles are omnivores, though as adults the majority of their diet should be vegetation with minimal proteins fed.

Protein can be achieved with Tofu, Soy or beans and not animal protein.

65% of diet should be leafy green vegetables

The rest of the diet should be non-leafy greens and fruits that are safe.

After rinsing the vegetables placing Calcium or Vitamin powder supplements. We recommend applying Calcium and Vitamins three times a week each.

We recommend RepCal for Vitamin and Calcium supplementation

### **Tortoises**

Tortoises are vegetarians.

They should have grass hay (timothy, orchard grass etc) available at all times

75% of their diet should be leafy green vegetables

25% can be non-leafy greens and fruits.

Some flowers or flower parts can be used as well

They tend to be attracted to certain colors like reds, oranges, and yellows

75% of the diet should be leafy green vegetables. Acceptable vegetables include romaine lettuce, dandelion greens (not flowers or flower stems), kale, collard greens, mixed greens, parsley, mustard greens, turnip greens, cactus, and broccoli and spinach (in small amounts). Avoid iceberg or head lettuce and celery as they are fiber rich, but of NO nutritional value.

25% of the diet should be fruits and non-green vegetables. Red, yellow, and orange vegetables should be included since reptiles seem to be attracted to these colors. Food such as tomatoes, peppers, squash, sweet potatoes, bok choy, okra, corn, beans, pea pods, green peas are all acceptable food sources. Fruit can include apples, pears, bananas, mangos, peaches, kiwi, melons, figs, papaya, raspberries, strawberries, blackberries, blackcaps, starfruit, and pomegranate.

### **Nutrition – safe foods**

A few safe foods:

Alfalfa

Apples

Bananas

Beans

Berries

Broccoli

Carrots

Clover

Corn

Dandelion greens

Grass

Guava

Kale

Kiwi

Mango

Mustard greens

Parsley

Peas

Potatoes

Pumpkin

Soybeans

Spinach

Squash

Star Fruit

Timothy Hay

Tomatoes

Turnips

Zucchini

Squamids or Lizards

Herbivorous lizards – follow the same guide as the Tortoises

Omnivorous lizards – follow the same guide as the Box Turtle

**Carnivorous Lizards – follow the nutrition guide for the Snake**

## **Amphibians**

Most Amphibians kept as pets are insectivores, some like the Pac Man frog can become big enough to eat pinkies or fuzzies.

Insects can be crickets, mealworms, hornworms, tomatoworms and even non-insects like earthworms. It is important to try to get insects from a parasite free breeding facility.

I recommend Timberline Fisheries ( [timberlinefisheries.com](http://timberlinefisheries.com) or [Timberlinefresh.com](http://Timberlinefresh.com) ).

Remember it is important to gut load your insects for 5 days before feeding out

It is important to apply Calcium and Vitamin powder 3 times a week.

## **Frank the Tank**

### **Tarantulas and Scorpions**

Just like Amphibians they are insectivores.

It is important to gut load your insects for 5 days before feeding out to your pet





# Dermatology in Exotic Pets

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## **Dermatology and tools needed**

Skin problems can occur in exotic pet the same as in dogs and cats.

We use many of the same tools that are at your disposal for cats and dogs in exotic dermatology

Tools Recommended in your practice for dermatology:

10 blades

Slides

Microscope

DTM auger

Culturettes

In house or out of house lab services

## **Skin Scrape**

Skin scrapes can be done in the same manner as dogs and cats

With mammals scrape until the skin blanches or becomes red or irritated for a deep scrape (careful with rabbits as their skin is thin and can tear easily).

Birds care must be taken as their skin can tear easily.

Gentle scrapes should also be used for amphibians and fish

Reptiles can be more difficult and you will not get as much blanching on the scales as you would a mammal

Use the blunt end of a sterile #10 scalpel blade

Once collected apply to clean slide

Saline and cover slip can be used to look for external parasites and a slide can be stained with diff quick to evaluate for infection or other pathology

## **DTM – Ringworm test**

Samples can be collected with clean forceps and taking some hair or using a clean toothbrush and brushing the coat and inoculating the auger.

If the sample grows fungus and the DTM is pink it is a good idea to take clean tape and place a drop of new methylene blue on a slip and identify the fungus to ensure it is ringworm.

### **Animals we will cover – Common problems seen in Practice**

Ferrets

Small rodents

Rabbits

Hedgehogs

Birds

Reptiles

Amphibians

Fish

### **Ferrets – Adrenal**

Adrenal disease in Ferrets is caused by hyperestrogenism and does not tend to be caused by irregularities in cortisol levels as happens in dogs.

It can cause thinning and hair loss on the caudal aspect of the ferret usually starting with the tail and working its way forward. If left untreated long enough it can work its way up to the front limbs as well.

In males it can cause swelling of the prostate and even urine blockage if severe enough.

Females can show vulvar swelling / edema

It can cause anemia but what is often seen first is the hair loss.

Diagnosis can be made via ultrasound imaging or extended adrenal panel to measure sex hormone levels.

### **Ferret Adrenal Treatment**

Treatment options

Surgical removal of affected gland

Many times both glands are involved or one will later become involved so surgery can sometimes be difficult to completely treat or can lead to post operative complications

Surulan injection

These implants last for a year and are relatively easy to administer.

It is a pellet about the size of a microchip and is placed deep SQ between the shoulders

Melatonin oral or implants

This has questionable results but the oral is cheap and may an option if someone is limited in funds

### **Ferrets Fleas**

The cat flea *Ctenocephalides felis*, can infest ferrets and can carry tapeworms

They can cause intense pruritis and erythema on the skin, especially around the tail head and perianal area

The can get F.A.D. or Flea Allergy Dermatitis

Treatment is a flea prevention for three months to break the life cycle

Advantage Multi for cats

Revolution puppy-kitten

Frontline Spray

### **Ferrets – ear mites**

Ear mites are common in ferrets (*Otodectes cynotis*).

Clean ears and treat concurrent infections if present.

Diagnoses on cytology by visualizing the mites or eggs

Treatment – Revolution puppy-kitten for 2 months or Ivermectin

### **Small Rodents - Fleas**

The cat flea *Ctenocephalides felis*, can infest ferrets and can carry tapeworms

They can cause intense pruritis and erythema on the skin, especially around the tail head and perianal area

Treatment is based off the species – Ivermectin, Selamectin or Permethrin based shampoos can be used. Remember to treat all mammals in the home.

### **Fleas on a Guinea Pig**

### **Sarcoptic Mange Mite**

*Sarcoptic Scabiei* is the mite responsible for sarcoptic mange in humans, dogs, horses, cattle (reportable) and others

This mite can cause severe pruritus (itching), alopecia, erythema, and papules

Diagnosed via deep skin scrape or skin biopsy

### **Guinea Pig Dermatopathy**

External parasites

Ringworm

Pyoderma *Strep zooepidemicus*

### **Small rodents Ringworm**

Ringworm can be seen in rodents but is most commonly seen in Guinea Pigs in practice.

They will present with Scaled skin, pruritis sometimes so intense that they will go into a seizure and hair loss.

The species is generally *Trichophyton species* so does not glow under black light.

Cytology via skin scrape and DTM culture is important to help diagnose this as the presenting signs are common amongst most of the external infections or parasites.

Treatment is topical antifungals for small lesions and can be combined with oral ketoconazole that is compounded as well.

### **Ringworm in Guinea Pigs**

### **Chirodiscooides**

*Chirodiscooides caviae* – hair clasping guinea pig mite

Not as common as the *Trixacarus* mite

It causes intense itching and alopecia

Usually diagnosed on cytology or Clear tape Cytology

### **Trixacarus**

*Trixacarus caviae*

This is a parasite of the guinea pig

This causes alopecia, skin crusting and pruritus so intense that it can cause fits or seizures

Diagnosed via Deep Skin scrape, rarely skin biopsy

### ***Gliricola porcelli***

*Gliricola porcelli* is a sucking louse of the guinea pig

This is a common external parasite of guinea pigs

They can cause intense pruritus, hair loss, and if bad enough seizures (more common in mite infestations)

They can take a year or two to get bad enough for guinea pig to show clinical signs

Clean cage well and treat all g. pigs in household

### **Pyoderma in Guinea Pigs**

Pyoderma can be seen in guinea pigs although usually it is secondary to other external infections or parasites or allergies

It is usually diagnosed via impression smear or light skin scrape

Most common etiologic agent is *Streptococcus zooepidemicus*

### **Dermatopathies in Mice**

External parasites

Ringworm

*Strep zooepidemicus* pyodermas

### ***Myobia musculi***

*Myobia musculi* clasp hairs and infest mice

Causes intense pruritus, alopecia and a secondary pyoderma

First toes have hooks

### **Myocoptes**

*Myocoptes musculinus*

Rodent hair clasping mite

Seen in mice frequently

### **Dermatopathies in Rats**

External parasites

Ringworm

Strep zooepidemicus pyodermas

### ***Radfordia ensifera***

*Radfordia ensifera* infests rats and causes hair loss and intense pruritus

This is in the same family as the myobia and so has claws on its first feet

Diagnosed with Tape Cytology or impression smears

Treated with an Avermectin for best results.

### ***Polyplax serrata***

*Polyplax serrata* chewing lice in rats

If severe enough can cause itching and if very severe anemia and death

Usually diagnosed with clear tape cytology

### **Dermatopathies in Rabbits**

External parasites – Ticks can be seen in rabbits, especially ones kept outdoors (ears are a common spot).

Ringworm

Bacterial dermatopathies

Atopy – not frequent

### ***Otobius megnini***

The Spinose ear tick is more commonly seen in warmer

Larvae and nymphs invade the ears of its hosts

Host are numerous including horses, cattle, rabbits and occasionally man

Individual ticks may remain in the ears of the host for up to one year

### **Notoedres**

*Notoedres cati*

This mite can infest cats, rats, rabbits and humans

These mites cause usually starts at the pinna and face, but can extend to the hindquarters

Usually diagnosed with deep skin scrap or skin biopsy

### **Psoroptes**

*Psoroptes cuniculi*

This mite infests rabbits ears and occasionally other areas of the body

Some rabbits are asymptomatic

Under stress this infestation will usually explode and cause cankers and bacterial otitis

Usually diagnosed with deep skin scrape or skin biopsy

### ***Cuterebra jellisoni***

*Cuterebra jellisoni* or more commonly known as botfly

The maggot or larval stage is the parasite here

The adults have vestigial mouthparts and they do not eat, just mate, lay their eggs and die

These botfly larvae can migrate to the brain in the dog, cat and rabbit and cause acute death

The botflies can be surgically removed, if they are, care must be taken to try not to break the maggot as this will cause severe tissue reaction in the subcutaneous tissue and skin

### **Pasteurella in rabbits**

*Pasteurella sp.* Is normal flora in rabbits but can cause a slew of problems and symptoms in rabbits.

It can cause dermatopathies usually on the paws and face usually secondary to pawing or overgrooming the face

Usually there are respiratory signs along with the dermatological changes

## **Francisella tularensis**

Tularemia in rabbits is a zoonotic disease that you need to be aware of as it is zoonotic and humans tend to have skin symptoms among other symptoms

It is a wild rabbit problem and can be an issue for hunters or people who take in or care for wild rabbits.

CDC site for more information <https://www.cdc.gov/tularemia/>

## **Rabbit Syphilis**

Rabbit Syphilis (*Treponema paraluis-cuniculi*)

Causes skin usually crusted skin on the nares and urogenital areas, but also result in lesions periocular or other areas on the body.

The lesions usually cause alopecia, severely thickened skin or hyperkeratosis and dry/cracked skin

Treatment is INJECTIBLE Pen G, given IM weekly.

## **Dermatopathy in Hedgehogs**

Can get ringworm – usually *Trichophyton species*

Fleas – usually the cat flea is seen in our practices

Ear mites *Otodectes species*

Other mites can cause dermatopathies in Hedgehogs

Skin cancer

## **Mites in Hedgehogs**

Hedgehogs can get many types of mites including: *Otodectes sp*, *Sarcoptes sp*, *Caparina sp*, *Notoedres sp*, *Demodex sp*.

*Otodectes sp* and *Caparina tripilis* are the most commonly seen species

*Otodectes sp* can affect just the pinnas causing crusting and hair loss on the ears only or some species can cause alopecia, crusting through out the body

Diagnosis is usually made by deep skin scrap or less likely skin biopsy

Treatment is usually ivermectin.

### **Hedgehogs skin cancer**

Skin cancer or Squamous cell carcinoma is a common killer in hedgehogs.

It frequently starts in the mouth, however it can appear anywhere on the body. Other common spots are the extremities

It often appears as a pink, pedunculated, cauliflower like mass on the skin or mouth

Diagnosis is made with biopsy

Surgical excision is warranted however prognosis is poor as it does tend to spread

### **Dermatopathy in Birds**

External Parasite – these are more common in your backyard chicken and turkey pets than Psittacines and Passerines kept indoors, with a few exceptions

Bacterial infections are possible but usually secondary to something that decreases immune response and allows for bacteria to break the skin barrier

Neoplasia is possible, especially in older pets - SCC and feather cysts are the most common

### ***Argus Persicus***

The Fowl Tick

The nymphal and adult stages are active at night while hiding in trees, chicken coupes, under houses, etc. during the day

Ticks may travel long distances to find their hosts. Large numbers of ticks may parasitize fowl at night, removing large amounts of blood

During the day, birds will show little sign of parasitism but a striking anemia

This ticks can be involved in the transmission of avian borreliosis and paralysis

Owners will sometimes report that the birds at night are being loud and screaming or crying out

### **Ornithonyssus**

*Ornithonyssus sylviarum* or Northern Fowl Mite

This species of mites infests the feathers and nests of birds

If the infestation is bad enough it can cause anemia and even death

## **Sternostoma**

*Sternostoma tracheacolum*

This blood sucking parasite is found in the trachea and air sacs of canaries and finches

## **Syringophilus**

*Syringophilus bipectinatus* – fowl feather quill mite

*Syringophilus columbae* – pigeon feather quill mite

These mites live their whole lives on the bird, usually on tail or flight feathers

## **House Fly**

In small animals the most common problem occurs with the larval stage

Maggot infestation is a problem where feces is allowed to cake onto the hair or skin

This is most commonly seen in animals that are limited in movement or outdoor animals

This is a common problem in back yard chickens that have been injured or are ill and feces cakes to their cloaca.

Treatment involves manual warm water rinse and debridement and usually an avermectin and addressing the underlying problem

Care must be taken with food animals and medications administered

## **Knemidokoptes**

*Knemidokoptes mutans* – chicken scaly leg

*Knemidokoptes pilae* –scaly leg in parakeets

*Knemidokoptes jamaicensis* – scaly leg in canaries

These mites cause scaling, hyperkeratosis, erythema and pruritus

The mange can affect the legs, beak, vent and backs of the affected birds

Diagnosed with deep skin scrape or chopping up skin chunks and finely ground with scalpel and then placed on slide and coverslip with mineral oil or saline

Treatment is Ivermectin

## **Dermatopathy in Reptiles**

Can get bacterial infections

External parasites

Skin cancers – usually older patients or amelanotic specimens

## **Bacterial infections**

Usually gram negative bacteria.

It is usually a result of poor husbandry – either poor nutrition, lighting, cage temperature or not cleaning the cage appropriately or frequently enough or poor substrate

Their purulent material is very caseous so antibiotics can have a hard time penetrating abscesses

Ideally a cytology and Culture and Sensitivity is performed to ensure the right antibiotics are used.

If an abscess is present, sedation, lancing-flush-cleaning and topical gels or beads are used to help treat the infection.

## **The Snake Mite**

*Ophionyssus Natricis*

These parasites are bloodsuckers

They are commonly seen between scales or in the gular fold on the ventral jaw

If the reptile is small enough and infested with enough mites it can become anemic

## **Ticks**

Many ticks can infest Reptiles.

Care should be taken when examining any reptiles recently brought into this country as they can harbor exotics ticks and exotic infections that can affect people as well.

## **Dermatopathy in Amphibians**

External parasites possible – more common on amphibians that spend more time out of water which can be infested with ticks and mites

Bacterial skin infections are more common with amphibians kept as pets due to poor husbandry – poor temperatures, improper cleaning or frequency and poor water and light conditions.

Gram's stain negative rods--usually *Aeromonas sp*, *Chlamydophila sp.*, as well as *Mycobacterium spp.* identified in amphibians include: *M. marinum*, *M. xenopi*, and *M. ranae*

Viral infections – Herpes virus and Ranavirus (common in frogs) can cause skin lesions

Fungal infections – there are many an up and coming one affecting our environment is the Chytrid fungi

Skin cancer can occur – especially in older patients

Sometimes skin conditions can occur due to an over abundance of internal parasites as well.

### **Dermatopathy in Fish**

External parasites - *Ichthyophthirius multifiliis* (Ich ) most common, larger external parasites occasionally seen like in the Beta (Siamese Fighting Fish)

Bacterial infection – *Mycobacterium* species. (zoonotic), *Staph aureus*,

Fungal infections

Neoplasia

### **Ichthyophthirius multifiliis**

*Ichthyophthirius multifiliis* or more commonly known as Ich or Fresh Water White Spot Disease is probably the most common external parasite seen in clinic

Common symptoms include:

Small white spots on skin

In advanced stages fish can become lethargic and anorexic

Redness or bloody streaks on the fins can be seen in advanced stages – bacterial sepsis can also do this

Fish can be itchy and scratch against rocks and gravel causing secondary bacterial infections if they damage their mucous layer on their skin

Treatment can be difficult and frustrating

Frequent water changes

Salt added to the water with 25% water changes

Malachite green, methylene blue, quinine hydrochloride and mepracrine hydrochloride – care must be taken dosing and treating the tank as some fish and especially amphibians can be very sensitive to this.

**Thank you**

There are many more diseases that could not be covered in this lecture if you have questions please email or use resources like [vin.com](http://vin.com) for more information.

Thank you for your attendance.



# External and internal parasites common to general practice

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## Parasites in exotic pets

Parasites can be common in veterinary practice and the same is true when you see exotic animals in your practice.

With external parasites cytology via skin scrapes, clear tape impressions are great to look for external parasites

With internal parasites it is recommended to look at fresh stool samples, ideally less than twelve hours old.

In our practice we use two fecal floats – zinc sulfate and centrifuged sugar solution

We also do direct fecals – saline, lugol's iodine, and new methylene blue – these are particularly good at finding protozoa.

## External Parasites

External parasites are important pests to our pets for many reasons

They can cause skin irritation and allergic reactions

They can carry many bacterial infections that can cause severe harm to our pets

They can carry intestinal parasites that can infest our pets

External parasite control is a multi-billion dollar a year business, most of which is sold over the counter with dangerous or inferior products, it is up to us to educate our clients

These ticks infest people as well and in many cases these tick borne diseases are zoonotic as well

## External Parasites

These can be broken down into two groups

The Arachnids and the Insects

## The Arachnids

We will start with the Arachnids

These invertebrates (animals without an internal skeleton) have 8 legs in their adult phase

These can be further broken down to two major categories

The Ticks

The Mites

### **Ticks**

Ticks carry many bacterial (Rickettsial diseases)

They are arachnids, in the same class as spiders

Larvae have 6 legs, nymphs have 8 legs, and adults have 8 legs

They are difficult to kill because of their size, making disease difficult to prevent

### **Anatomy and Parasitism of Ticks**

Ticks in Wisconsin

We will break these ticks into two categories

Soft Ticks and Hard ticks

### **Soft Ticks**

Soft ticks are named such because they lack a hard dorsal shield

There are two that are seen in small animal and exotic practice

*Otobius megnini* - the Spinose ear tick and *Argus persicus* – the fowl tick

### ***Otobius megnini***

The Spinose ear tick is more commonly seen in warmer climates

Larvae and nymphs invade the ears of its hosts

Host are numerous including horses, cattle, rabbits and occasionally man

Individual ticks may remain in the ears of the host for up to one year

### ***Argus persicus***

The Fowl Tick

The nymphal and adult stages are active at night while hiding in trees, chicken coupes, under houses, etc. during the day

Ticks may travel long distances to find their hosts. Large numbers of ticks may parasitize fowl at night, removing large amounts of blood

During the day, birds will show little sign of parasitism but a striking anemia

This ticks can be involved in the transmission of avian borreliosis and paralysis

## **Hard Ticks**

These are by far the most commonly seen ticks on ourselves and our pets

They have a hard dorsal shield, where males the shield covers the whole dorsal abdomen, this is what differentiates males from females

There are four main species seen in our pets in Wisconsin

The Deer Tick

The Wood Tick or American Dog Tick

The Brown Dog Tick

The Lone Star Tick

## **The Deer Tick**

*Ixodes scapularis*

This tick is the most common vector for Lyme disease (*Borellia burgdorferi*) in North America

The adults are cool weather ticks, appearing in spring and fall but usually remaining on the host throughout the winter

Larvae and nymphs are abundant in the summer and feed on small mammals and lizards

The life cycle is typically 2 years

In addition to Lyme disease, this tick is responsible for transmitting *Anaplasma sp.* (formerly *Erhlichia Equi*), tularemia, human and animal babesiosis, and tick paralysis

Adults can be identified by the presence of large mouthparts (longer than the basis capituli) and an anal groove that forms an arch anterior to the anus on the ventral side.

## **The American Dog Tick**

The Wood Tick or *Dermacentor variabilis*

This is the most common adult tick seen in Wisconsin mid-April and peaks by June, declining until September

The most common hosts are white tailed deer and wild turkey

It is a common vector for Rocky Mountain Spotted Fever, anaplasmosis, feline cytauxzoonosis and tick paralysis

### **Brown Dog Tick**

*Rhipicephalus sanguineus*

Although common on dogs, it can be found feeding on other mammals but rarely humans

Adult activity is from spring until autumn

When off the host, the adult ticks have a tendency to crawl upward and are often found in roofs and cracks of dwellings. It is one of the most common ticks involved in infestations of houses

This tick is involved in the transmission of canine babesiosis, haemobartonellosis, canine ehrlichiosis and tick paralysis

### **The Lone Star Tick**

*Amblyomma americanum*

It is primarily a problem as a pest but it is involved in the transmission of canine ehrlichiosis, tularemia, Q fever and cervid (deer) theileriosis

Adult females are easily recognized by having long mouth parts and a white spot on the dorsal scutum

### **Mites**

Most mites are microscopic or need magnification to be seen

These parasites are not usually species specific

They are also related to spiders and ticks

They have an egg, larvae, nymph and adult phase just like the tick

### ***Cheyletiella sp.***

*Cheyletiella sp.* also referred to as the “walking fur mite” or “walking dandruff”

This mite is zoonotic, it also can infest guinea pigs, rabbits, cats, dogs and ferrets

## **Chiggers**

“Chiggers” or Trombiculid mites or Harvest Mites

The adult mite actually lives in the dirt and feeds on insect eggs

Larva (which are 6 legged) feed on liquified skin and sebaceous glands

The larvae feed for 4 days then drop off the host, usually at this time the erythema and pustules are noted

The normal host is the vole

## **Chirodiscoides**

*Chirodiscoides caviae* – hair clasping guinea pig mite

Not as common as the Trixacarus mite

It causes intense itching and alopecia

## **Knemidokoptes**

*Knemidokoptes mutans* – chicken scaly leg

*Knemidokoptes pilae* –scaly leg in parakeets

*Knemidokoptes jamaicensis* – scaly leg in canaries

These mites cause scaling, hyperkeratosis, erythema and pruritus

The mange can affect the legs, beak, vent and backs of the affected birds

A good method to find them is to take a piece of thickened skin freshly removed from the bird and chopped finely with a clean scalpel blade, then placed on a slide with a coverslip and either saline or mineral oil

## ***Myobia musculi***

*Myobia musculi* clasp hairs and infest mice

Causes intense pruritus, alopecia and a secondary pyoderma

First toes have hooks

## **Myocoptes**

*Myocoptes musculinus*

Rodent hair clasping mite

### **Notoedres**

*Notoedres cati*

This mite can infest cats, rats, rabbits and humans

These mites cause usually starts at the pinna and face, but can extend to the hindquarters

### **Ornithonyssus**

*Ornithonyssus sylviarum* or Northern Fowl Mite

This species of mites infests the feathers and nests of birds

If the infestation is bad enough it can cause anemia and even death

### **Otodectes**

*Otodectes cynotis* – the Ear Mite

Causes intense irritation of the external ear canal and adjacent tissue of the dog, cat, fox, ferrets and hedgehogs

They cause a large amount of cerumen in the ear

### **Psoroptes**

*Psoroptes cuniculi*

This mite infests rabbits ears and occasionally other areas of the body

Some rabbits are asymptomatic

Under stress this infestation will usually explode and cause cankers and bacterial otitis

### **Radfordia ensifera**

*Radfordia ensifera* infests rats and causes hair loss and intense pruritus

This is in the same family as the myobia and so has claws on its first feet

### **Sarcoptic Mange Mite**

*Sarcoptic Scabiei* is the mite responsible for sarcoptic mange in humans, dogs, horses, cattle (reportable) and others

This mite can cause severe pruritus (itching), alopecia, erythema, and papules

### **The Snake Mite**

*Ophionyssus Natricis*

These parasites are bloodsuckers

They are commonly seen between scales or in the gular fold on the ventral jaw

If the reptile is small enough and infested with enough mites it can become anemic

### **Sternostoma**

*Sternostoma tracheacolum*

This blood sucking parasite is found in the trachea and air sacs of canaries and finches

### **Syringophilus**

*Syringophilus bipectinatus* – fowl feather quill mite

*Syringophilus columbae* – pigeon feather quill mite

These mites live their whole lives on the bird, usually on tail or flight feathers

### **Trixacarus**

*Trixacarus caviae*

This is a parasite of the guinea pig

This causes alopecia, skin crusting and pruritus so intense that it can cause fits or seizures

### **Insects**

The adult form of this arthropod has 6 legs instead of eight

Some of these are parasites only in certain stages of their life or certain sexes

Many insects not covered here are not direct parasites, but harbor intestinal parasites

### ***Cuterebra jellisoni***

*Cuterebra jellisoni* or more commonly known as botfly

The maggot or larval stage is the parasite here

The adults have vestigial mouthparts and they do not eat, just mate, lay their eggs and die

These botfly larvae can migrate to the brain in the dog, cat and rabbit and cause acute death

The botflies can be surgically removed, if they are, care must be taken to try not to break the maggot as this will cause severe tissue reaction in the subcutaneous tissue and skin

### **Deerfly**

A tabanid type fly

These common pests are blood suckers

They cause a painful bite

They can spread or carry tularemia

### **Horsefly**

Another tabanid fly

These flies are blood suckers, they have a huge knife like mouthpart to cut into the hosts skin

They have been implicated in *Anaplasma* sp transmission

### **House Fly**

In small animals the most common problem occurs with the larval stage

Maggot infestation is a problem where feces is allowed to cake onto the hair or skin

This is most commonly seen in animals that are limited in movement or outdoor animals

### **Mosquito**

The mosquito is a common pest in Wisconsin

The adult female is the one that needs the blood meal to grow her eggs

They are responsible for what major internal parasite?

Heartworms

They also can carry west Nile virus and dengay fever

## **Lice**

Lice are broken down into two categories

Chewing or Sucking lice

Their eggs are called knits and are glued to the hair cuticle

They are relatively susceptible to parasitocals

### ***Polyplax serrata***

*Polyplax serrata* chewing lice in rats

If severe enough can cause itching and if very severe anemia and death

### ***Gliricola porcelli***

*Gliricola porcelli* is a sucking louse of the guinea pig

This is a common external parasite of guinea pigs

They can cause intense pruritus, hair loss, and if bad enough seizures (more common in mite infestations)

They can take a year or two to get bad enough for guinea pig to show clinical signs

Clean cage well and treat all g. pigs in household

## **Fleas**

There are many species of fleas

Today we will talk about a few here

The cat flea is probably the most important here in Wisconsin

### ***Ctenocephalides sp.***

*Ctenocephalides felis* the cat flea is by far the most common flea in this state which affect cats, dogs, humans, rabbits and rodents

*Ctenocephalides canis* the dog flea is far less common

These fleas can transmit tapeworms, specifically dipylidium caninum

### ***Xenopsylla***

*Xenopsylla* the rat flea

It is an important vector for *Yersinia pestis* (the plague or black death)

This can be distinguished from the human flea with the vertical rod on the mesothorax

### ***Cediopsylla***

*Cediopsylla* the rabbit flea

It is an uncommon flea in this country

The genal teeth lie at a parallel angle to the pronotal comb

## **Internal Parasites in Exotic pets**

Internal Parasites for Small  
Animal and Exotic Pets

There are two large categories: Protozoans and Helminths

### **Protozoans**

These are single celled organisms

They are in the Kingdom – Protista

Many are free living

Some are symbiotes like the protozoa in the rumen of ruminants and the cecum of hind  
gut fermentors

Then there are the parasitic ones

### **Coccidians**

These parasites develop in the epithelial cells of the alimentary tract, this causes a form of enteritis called  
coccidiosis

They transmit via fecal-oral transmission

They transmit in feces via oocysts pronounced “oh’ oh-sists”

They include *Eimeria*, *Cystoisospora* (*Isospora*), *Hammondia*, *Sarcocystis*, *Neospora*, and *Toxoplasma*

The oocysts do have an operculum (a “cap”) which can sometimes be visualized

### ***Eimeria sp.***

These coccidians mainly infest birds and herbivores

They may be identified by containing 4 sporocytes, which each contain 2 sporozoites

Unsporulated oocysts of *Eimeria* species will look just like *Isospora* species

### ***Cystoisospora sp.***

*Cystoisospora sp.* or originally called *Isospora* are a group of coccidians that infest carnivores and some omnivores

The new name comes from a reclassification of some of the bird *Isospora*’s that were found to be more closely related to the *Eimeria* species coccidians

These can be identified with sporulated oocysts via 2 sporocysts and 4 sporozoites

### ***Cryptosporidium species***

*Cryptosporidium* species are coccidians that cause diarrhea and infest the small intestines

These coccidians are zoonotic unlike the *Eimeria* and *Cystoisospora* species and cause very severe G.I. symptoms and even death in humans, especially elderly, immune compromised and children

The most common species is *Cryptosporidium parvum*, however *Cryptosporidium* have other more rarely seen species like *hominis* (human), *canis* (dog), *felis* (cat), *muris* (rat and mouse), and *suis* (pig)

### **End of coccidians**

### ***Entamoeba sp.***

*Entamoeba sp.* are usually a parasite of the large bowel

In humans they cause amoebic dysentery

Dogs and cats they are not a problem

They are a problem in primates and in our pet reptiles

*Entamoeba invadens* causes severe disease in reptiles infesting the duodenum and the liver and can cause anorexia, diarrhea, listlessness and acute death

### ***Haemoproteus* species**

*Haemoproteus* species are parasites of birds, turtles and lizards

They appear in the erythrocytes (RBCs) of said animals

Use Wright's Giemsa stain to visualize best

These are mainly transmitted by mosquitoes

Look for these on every CBC differential that you perform

### ***Giardia* sp.**

There are many species of *Giardia* species

Some species affect multiple species, some affect one

*Giardia* causes the disease known as Giardiasis

Symptoms can vary from vomiting to bloody diarrhea and sometimes both

It can often be difficult to clear an animal of giardia, sometimes they can not be

*Giardia* sp. infest the small intestines

They replicate by budding – dividing in half

When in the environment and in a dry environment they form cysts which can be difficult to kill

It is usually more concentrated in stagnant, low flow water

### ***Leucocytozoon* species**

Leucocytozoan species are parasites of domestic and wild birds

They can cause acute and sometimes fatal disease

They first invade hepatocytes (liver cells) and vascular endothelial cells (cells that line the blood vessels), these produce merozoites that in turn invade erythrocytes and blasts, lymphocytes, monocytes and then develop into gametes

They tend to distort the nucleus on the cell that they invade

### ***Plasmodium* species**

*Plasmodium* species are the causative agent of malarias in humans, nonhuman primates, rodents, birds and reptiles

Mosquitoes transmit these diseases, however different groups of mosquitoes pass malarial parasites to humans than in birds

Reptile transmission is largely unknown

### ***Trichomonas sp.***

There are multiple other *Trichomonas sp.* that infest birds, reptiles and humans

Treatment of these species are generally with metronidazole and or a benzimidazole drug

### ***Tritrichomonas foetus***

*Tritrichomonas foetus* is a venereal disease that causes infertility in cows and heifers, abortion, pyometra and fetal mummification

In cats it causes diarrhea usually in kittens under a year of age

Species of *Tritrichomonas* can be seen in reptiles and amphibians

Diagnosis is by fecal or gold standard by culture media

Treatment is difficult and long, often > 1 month

## **Helminths**

This phylum of animals is more commonly called “worms”

This phylum includes the following phyla

Platyhelminthes – “flat worms” these include the flukes and tapeworms

Nematoda – “roundworms”

### **Platyhelminthes**

These “flat worms” called so because they are flat

They are broken down into three classes

Turbellaria – “tube worms” which are not usually parasitic

Trematodes – “flukes”

Cestodes – “tapeworms”

## **Trematodes**

The flukes, there are many flukes, many of which infest ruminants, there are many that can infest carnivores as well.

We will discuss the most common ones

Most parasitic “flukes” have intermediate hosts – many of which are our exotics like frogs/amphibians, crustaceans and fish

### ***Heterobilharzia americana***

This trematode is in the family of the Schistosomatidae

The intermediate host is the snail, when it leaves the snail it penetrates the skin of a raccoon, rabbit, or a dog

These parasites migrate through the lungs and end up in the liver, then go to the mesenteric vein mate and the eggs migrate through the intestines and pass into the feces

### ***Nanophyetus salmincola***

This “fluke” is responsible for canine salmon poisoning

Infection acquired by infested freshwater snail eaten by a salmonid fish which is then eaten by a dog or cat

The PPP (Pre Patent Period) is 5-8 days

The “fluke” usually is asymptomatic but carries a bacteria *Neorickettsia helminthoeca*, which causes a severe febrile illness and even death if not treated aggressively

The fluke can be treated with Praziquantel and Drontal Plus for Dogs

## **Class Cestodes**

Cestodes or more commonly known as “Tapeworms”

These worms in their adult phase are made of a scolex or “head”, that attaches and feeds in the intestinal tract, the rest of the body is made up of many segments

Each segment is capable of involuntary movement

Each segment contains many thousands of eggs

These eggs hatch and infest intermediate hosts, encysting in these hosts until they are ingested by the definitive host where the adult develops

Both the adult worm and the proglottid cysts can cause severe disease and even death in their host

Zoonosis is a big factor with these parasites as humans can be infested with the adults of some species, but also hydatid cysts in other species that we act as the intermediate host for

Thus controlling these parasites are very important, not just to our patients, but also their owners

### ***Diphyllobothrium latum***

This cestode uses copepods and fish as an intermediate host

The adults infest the small intestine of dogs

The eggs are passed in the feces

PPP = 5-6 weeks

### ***Dipylidium caninum***

This tapeworm infests dogs, cats and even humans when bitten by or ingests *Ctenocephalides felis* or *Trichodectes canis*

PPP = 3 Weeks

The eggs are passed in segments that look like cucumber seeds, the segments contain egg packets that can contain 3 to 30 eggs

Flea and lice control is the key to preventing this parasite in our pets and thus us

### ***Spirometra mansonioides***

These tapeworms develop in copepods, which are then eaten by amphibians, reptiles, birds, or mammals

They infest the small intestines of dogs and cats

They can cause emaciation, diarrhea, and or vomiting

PPP = 10 days

### ***Taenia species***

There are many species in this group

The dog and cat are almost always the definitive hosts

The intermediate hosts are usually rabbits, rodents, reptiles, amphibians and humans

Reptiles and humans can be the definitive host depending on the species

The PPP = 1-2 months

This is a very important group of parasites, which affect all vertebrates

## **Nematoda**

The Nematodes or “roundworms”

These are further broken down to

Strongyloides

Oxyurida

Ascaridida

Spirurida

Physalopteroidea

Filariordea

Enoplida

### **Strongyloides**

A common parasite in small animals and exotics

Ancylostomatidea – the hookworms

Other parasites in strongyles are

*Syngamus* species

*Aelurostrongylus abstrusus*

*Filaroides* species

### **Ancylostomatidae**

The Hookworms

These strongyles can cause significant blood loss and lead to anemia and even death in puppies, kittens and immune compromised patients

*Ancylostoma* and *Uncinaria* species are the most common species that infest our dogs and cats

These infections can occur through the skin and is infective as soon as the egg hits the ground

Infection can occur transplacentally as well (though not in the cat)

It causes cutaneous larval migrans in people

### ***Ancylostoma* species**

*A. caninum*, *A. braziliense*, *A. tubaeforme* are the most common species seen in our pets

Adults have very sharp teeth

PPP = 2 weeks

Adults can consume 0.1ml of blood, when considering a large worm burden it is no wonder that these species can cause severe anemia in our pets

### ***Uncinaria* species**

This species have cutting plates for mouth parts

PPP = 2 weeks

This species is less common than *Ancylostoma* species

### ***Syngamus* Species**

*Syngamus* species affect our domestic birds

They are also known as the gapeworm

This parasite infests the trachea

It can cause severe respiratory distress, sudden death, coughing, bloody sputum

The intermediate host is the earthworm and snails

### **Order Oxyurida**

Oxyurida or "Pinworms", is not an order that affects dogs and cats

However they do affect our exotic pets like rabbits, rodents, reptiles and humans

These worms are species specific

They can be asymptomatic or can cause weight loss, diarrhea, anorexia, and anal irritation or itch

Treatment to remove infestation can be difficult due to re-infestation from environment

### **Order Ascaridida**

The ascarids are some of the largest and most familiar roundworms

Aquatic species that infest aquatic reptiles will have aquatic intermediate hosts, however terrestrial ascarids have adapted to become directly infective or transmammary or transplacentally

### ***Baylisascaris procyonis***

*Baylisascaris procyonis* is a common roundworm in the racoon

The visceral larval migrans in people which invade the brain and cause acute death in people

It looks like *Toxocara* larvae

This is a very dangerous parasite especially for its zoonosis potential

### ***Dracunculus* species**

*Dracunculus medinensis* parasitizes people

*Dracunculus insignis* parasitizes racoons as well as dogs and cats in North America

There are reports of species that infest reptiles as well (snakes and snapping turtles)

### **Physalopteroidea**

Physaloptera species are parasites of the stomach in carnivores – dogs and cats

The intermediate host is beetles, crickets, and amphibians and reptiles

### **Filarioidea**

The most important member of the species is *Dirofilaria immitis*

They tend to be long thin worms where the male has a spiral flexure on the end of the tail

*Microfilaria* – larval stage 1, circulate in the blood stream

Insects pick them up from the blood stream, develop, and are then passed on to a definitive host

### ***Dirofilaria immitis***

*Dirofilaria immitis* these are the most important worm in dogs, cats and ferrets

They parasitize the pulmonary vessels as well as the right chamber of the heart

Since there are other microfilaria that can be present in the blood stream, microfilaria alone can not be relied on as the sole diagnosis method

In addition not all infested dogs, cats and ferrets have microfilaria present in the blood stream

In dogs a heartworm antigen test is the best method to diagnose the disease

However since the PPP is about 5-6 months, a false negative on the heartworm antigen test can occur

In addition single sex infestations (like no females present) can lead to false negatives on the heartworm antigen test

In cats heartworm diagnosis can be difficult as single worm infestation can occur

Less larvae survive to adulthood in the cat

The larval migration through the lungs can be devastating to the cat, causing asthma like symptoms called Heartworm Associated Respiratory Disease or H.A.R.D.

Diagnosis using a heartworm antigen test and an antibody test is best to look for disease in cats

Ferrets testing using an antibody test is likely the best way to look for disease

Prevention using a larvacidal preventative is the most important method of preventing the disease

### ***Dioctophyme renale***

This is the giant kidney worm

This worm's definitive host is the mink, however other carnivorans – (ferrets), swine, and sometimes humans

The eggs pass in the urine and are picked up on urinalysis

The eggs are large 68x48 mcm

Males are smaller than the female

The worm will slowly devour the kidney, leaving only the renal capsule, infestation usually involves the right kidney or the worm is found in the renal pelvis

If both kidneys become infested it results in death of the infested animal

### ***Capillaria hepaticum***

This species lives in the liver of rats and other rodents

It can also infest humans

### ***Capillaria putorii***

This is an intestinal threadworm of hedgehogs

It can cause diarrhea, weight loss, or be asymptomatic

### **Phylum Annelida**

This phylum of worms includes the earthworm, and contains few parasites, however includes the class Hirudinea – the leeches

Leeches have terminal suckers for locomotion and attachment

The salivary organ hirudin produces an anticoagulant to ensure a good blood flow

If this parasite is ingested it will attach to the oropharynx or laryngeal mucosa and cause severe bouts of coughing, choking, bloody sputum and if severe death

Treatment is by manual removal

# Sedation and anesthesia for the Exotic pet

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Sedation and anesthesia is a necessary tool in veterinary practice, especially for some exotic species

Sedation can be used to make exams and minor procedures less stressful

Anesthesia is used for procedures just like in the dog and cat such as OHE, neuters, dentals, exploratory surgery, lump removals, etc.

Avians and Rabbits tend to be a lot more sensitive to anesthesia and are thus more risk.

High stress animals like birds and rabbits can benefit from sedation even for extensive exams to help lower the risk for respiratory or cardiac arrest

## **Disclaimer about Doses**

Doses for exotic animals are extra-label use. The FDA does allow us to use these drugs extra-label as established standard doses do not exist.

Doses that are provided in this lecture are doses that I have used and gotten from research done by others, however a veterinarian that you work for should always check and calculate doses based on their research and what they are comfortable. Doses used by you, your veterinarian and your clinic are your responsibility and Dr. Shawn Hook does not take responsibility for drugs and doses used in your clinic on any of your patients.

## **Sedation or premeds**

A good combination for sedatives in small mammals and birds is butorphanol and midazolam

These can be administered intramuscular, however to reduce stress it can be administered intra-nasal in birds and intra-oral in small mammals

The midazolam can be reversed with flumazenil

If needed in bigger procedures or very stressed animals dexmedetomidine and ketamine can be used with butorphanol and midazolam

## **Sedation or premeds - Butorphanol**

Butorphanol is great for sedation and minor procedure restraint

It is a  $\kappa$ -opioid receptor agonist and  $\mu$ -opioid receptor antagonist

It is not adequate pain control for major surgeries in our patients except birds as they do not have the same receptors for opiates as mammals do

Butorphanol is a mixed antagonist and agonist agent in mammals

In birds since they lack  $\mu$ -opioid receptors, most opiates like morphine and hydromorphone don't work very well. Buprenorphine has questionable effects in birds since it is a partial  $\mu$ -opioid agonist. Leaving Butorphanol as our best opiate in birds

Intranasal administration in birds take about 3-5 minutes to take effect

It is a relatively safe analgesic

It does not tend to do much for reptiles

## **Butorphanol**

### **Sedation or Premed -Morphine**

Morphine is a pure  $\mu$ -opioid agonist

Care must be taken with opiates with hind gut fermenters as it can cause serious GI stasis.

Birds as they have different pain receptors and pure  $\mu$ -opioid agonists like morphine do not work

In reptiles Morphine is one of the few opiates that have been shown to cause some pain relief

Generally 1mg/kg IM or IV

SQ or Intracoelomically 1.5-6.5 mg/kg and higher

## **Midazolam**

Midazolam is a Benzodiazepine , a sedative drug that is in aqueous form.

Because of its aqueous form it can be delivered in more ways than Diazepam which is an oil based solution and thus does not absorb well when used intra-oral or intra-nasal.

It can be used relatively safely in our exotic pets and can be reversed with flumazenil (0.01–0.1 mg/kg).

Midazolam in small mammals can be used Intra-muscular or orally, dose typically used 0.25-1 mg/kg (Midazolam 5mg/ml).

In birds the dose range is 0.5-3mg/kg. However I tend to use what the UW of Madison VMTH - Christoph Mans, Dr. med. vet., DACZM, uses as a dose range which is 2mg/kg and smaller birds like finches and budgerigars 4mg/kg intranasally for quick sedation procedures. I usually use with Butorphanol.

In reptiles typically it is used with an opiate or other sedation agent at 0.1–2 mg/kg SC, IM, or IV

## **Other sedation meds**

**Ketamine** – Dissociative general anesthetic drug can be used in smaller doses to aid butorphanol and midazolam to help achieve more sedation

Dose ranges from 1-10 mg/kg when used with other sedative. I tend to use 2.5-10mg/kg on many species.

Dexmedetomidine – An Alpha-2 agonist which can also be used in small doses to help achieve better sedation with butorphanol and midazolam and even Ketamine.

Dose range typically 0.005–0.01

Can be reverse with atipamezole at equal volumes

## **Induction Medications**

Induction medications are drugs used to achieve or maintain a full plane of anesthesia for more invasive procedures

The goal for the drugs that we use is to provide a safe and controlled level anesthesia so that are patients are unconscious for the procedure but will wake up in a safe and as short amount of time possible

There are two main categories

Injectable

inhalants

### **Induction medications - Propofol**

Propofol - is a common induction agent. It is a short acting hypnotic agent that is broken down by the liver and subcutaneous tissue. It can cause upon induction temporary apnea so aided respirations are usually warranted.

The drug is eliminated fairly quickly in most animals except reptiles which can last up to 24 hrs on some patients.

Dose is typically 2-10mg/kg depending on the species. It can be repeated as needed to maintain anesthesia. Reptiles typically 0.5-1mg/kg to effect

### **Induction medications - Injectable - alfaxalone**

Alfaxalone is an induction and maintenance drug that can be bolused or administered CRI

Rapid IV administration will produce a short period of apnea.

The manufacturer does not recommend combining Alfaxan with other drugs into the same syringe.

It is labeled for IV use only but it is noted by the manufacturer that if it goes out of the vein it does not irritate surrounding tissue.

Many times it is used IM in exotics

Can be used with Midazolam and an opiate

Dosing varies, small doses can be used for sedation and larger doses for induction of anesthesia

Reptiles take much higher doses for anesthesia induction but if ideal body temperature is maintained, it does leave the system relatively quickly.

### **Induction medications – Injectable - Ketamine**

Ketamine can be used as an induction drug though it is frequently mixed with Midazolam and an opiate

It is metabolized by the liver, some animals can be sensitive to it and should not be used in animals sensitive to it.

Can be administered IV, IM and intra-oral

Recommend not using with animals with CNS, renal or cardiac disease.

For sedation combined with other drugs 2-5mg/kg is typically used in small exotic mammals and 5-10mg/kg for induction combined with Midazolam and an opiate (I tend to start lower at 2.5-5 mg/kg).

### **Induction medications - inhalants**

Two major inhalants are currently Isoflurane and Sevoflurane. Both are halogenated ether inhalational agents.

Both are relatively safe anesthetics

Sevoflurane is more potent so our patients tend to become induced and recover from anesthesia faster with this inhalant.

Since reptiles can hold their breath for a long time, it is not recommended to use inhalants for induction.

Many exotics are masked down especially for minor and short procedures

Hedgehog restraint

Bird restraint for x-rays and bloodwork

Teeth trims on rodents and rabbits

### **Induction medications – inhalants - Isoflurane**

Isoflurane is a readily available and cheap inhalant induction/anesthetic agent.

Smell can be noxious so animals can sometimes fight mask induction and thus add more stress.

Induction is normally 3-5% for most exotics

Not recommended for Reptiles

Maintenance

Mice, rats, gerbils, hamsters, guinea pigs, chinchillas, degus – 2-3%

Reptiles 3-5%

Avians 1.5-2%

### **Inhalants - Sevoflurane**

Tends to be quicker induction and recover

Less noxious smell so if masked may object or fight less and thus the animal is less stressed

98% exhaled 2% metabolized by the liver

A Very potent inhalant always be looking to see if you can turn them down.

Adjustments should be made in quarter increments instead on full turns like in Isoflurane

Sevoflurane typical dosing for patients

Induction in healthy birds at 8% and 2 L/min O<sub>2</sub> flow. Compromised birds induction at 4-5%.

Maintenance usually around 4% Sevoflurane.

Small mammals usually induce at 8% and maintain 3.5-4.5%

Reptiles I do not recommend induction with Sevoflurane, but maintenance is usually 4-5%

Remember that doses will be less if in certain patients, in patients receiving CRI

### **Local Pain Meds**

Local pain meds can be used to numb or deaden pain of a site.

This helps not only control pain if used prior to stimulating the nerves, but also allows you to use less maintenance inhaled or injectable medications.

Care must be taken depending on the patient on what local medications are used.

Lidocaine and Bupivacaine are typically used at 1mg/kg each.

With small animals these may need to be diluted to be able to administer safely.

Other locals that can be used. These are being researched but using meds like buprenorphine and dexmedetomidine with Lidocaine and Bupivacaine has been used by the speaker.

### **CRI – Constant Rate Infusion**

CRI or Constant Rate Infusion can be used by many medications, especially opiates to help control pain better while under anesthesia and lessen the amount of inhaled anesthesia.

Fentanyl can be used depending on the patients 4-20 mcg/kg/hour

Lower doses preferred for hind gut fermenters like Rabbits and Guinea Pigs

It can slow GI transit time, however it is metabolized so quickly that the effects are usually very transient and with the other anesthetics used, you may not notice more GI effects

Other medications used CRI – Propofol, Alfaxalone and Ketamine can be used.

### **Catheters and sites**

Catheters should always be attempted in our exotics patients when performing general anesthesia.

There are multiple sites depending on the patient's species

Many times unless you IV infusion pump can go low enough many times a syringe pump is needed to deliver IV fluids in our really small patients.

Depending on patient using 26g, 24g, 22g IV catheters or 22 g Spinal needle or regular needle (for IO or intra-osseous catheterization).

### **Catheters – Small mammals**

Rats – IV catheters can be used in the cephalic veins using 24 or 26g catheters

Guinea pigs using cephalic or rarely lateral saphenous veins using 26g and 24 g catheters

Rabbits the cephalic, lateral saphenous veins can be used using 24 or 22g needles depending on the size of the rabbit. Aural veins can be used as well using 26 or 24g catheters.

Ferrets the cephalic is typically used using a 24g catheter

### **Intra-venous Catheters - Avian**

Larger birds you can use an intravenous catheter. The veins are very weak, easy to collapse and easy to have the vein blow.

A right jugular catheter and a medial saphenous vein can be used.

In smaller birds or for long term fluids an intra-osseous catheter may be more ideal.

### **Intra-osseous catheter - avian**

Intra-osseous catheters can be placed in the ulna and tibia.

Remember that birds have pneumatic bones and so the femur and humerus can not be used.

These catheters are painful and the animal must be kept on pain killers while the catheter is in place

Osteoarthritis can be caused, especially while using the tibia, care must be taken to not damage the cartilage in the joint while placing.

A post-placement radiograph is a good idea to ensure that the catheter is in place.

### **Intra-osseous Catheter - Reptiles**

Intravenous catheters can be attempted however cut-downs must be performed as you can not see the veins through the scaled skin.

Intra-osseous is more frequently used. The proximal tibia is the typical place of insertion.

These catheters are painful – so pain medications must be administered while the catheters are in place

Post-placement radiographs should be considered to ensure proper placement

A 22g Spinal needle or regular 22 to 20 g needles can be used depending on size of reptile.

### **Conclusion**

In conclusion sedation medications can and should be used to reduce stress in our exotics patients

They can be used for minor procedures, radiographs and venipuncture

Many can be reversed like Midazolam

Pain therapy is very important in our exotic patients – just as in our dogs and cats

Pain therapy can be delivered injectably, but also intra-orally/nasally, via local blocks and constant rate infusions

Anesthesia can be achieved with both injectable and inhalant medications

It is important to remember that anesthesia and sedation many times used more than one drug which allows for lower doses for each drug which allows for safer and less stressful anesthesia or sedation.



# Radiographic and Ultrasound Positioning and techniques

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## **Positioning and techniques**

Radiographs and ultrasonography can be utilized in exotic practice just as you do dogs and cats

Positioning is very important to get the best results

Different techniques are used as exotics such as avians have pneumatic bones and so less exposure is needed.

Digital radiology is the most common standard in most hospitals and will yield the best results.

That said, if digital radiology is not available in your practice then I recommend high detail, rare earth cassettes with single emulsion film.

Mammography film will produce even better detail, however it does require a higher KVP and MA to achieve.

I would recommend making a technique chart or having someone familiar with them help you make one for taking exotics radiographs. Digital companies should be able to give you techniques to take exotic radiographs

## **Small mammals - radiography**

Small rodents like mice you can use dental radiography to get really detailed images

Dental film can be used or if you have direct X-ray plate this works better and easier

## **Small mammals restraint**

When not using sedation if the animal wants to curl up or not sit still plastic tubes, tennis ball tubes and plastic containers can be used to help restrain the animal for x-rays.

Injectable or gas anesthetics can be used for restraint for radiographs or ultrasound

Typically Sevoflurane or Isoflurane can be used relatively safely for quick procedures like radiographs and ultrasound

Make sure someone is monitoring the patient carefully while under sedation

Injectables like Propofol, ketamine, midazolam, telazol can be used but often take longer to induce and longer to recover than inhalants.

Remember that all sedation medications will affect GI transit time so using them for a barium study is not warranted

### **Small mammals - Radiographic positioning**

With hind gut fermenters like guinea pigs and rabbits it can be uncomfortable to have them on their backs to taking DV (dorso-ventral) views may be easier than VD (ventro-dorsal) view.

If you have CR digital radiography and a handheld dental x-ray gun in your practice positioning can be a lot easier as you can have the animal sitting on the table and position the cassette below or behind them

Ideally a DV or VD view and two lateral view right and left for thorax and abdomen

For skull views ideally a VD or DV and left and right lateral obliques

### **Small mammal lateral x-ray position**

### **Small mammal – VD radiographs**

### **Small mammals – Ultrasound positioning**

Rats and ferrets many times will lay on their back or with gas or injectable sedation will easily lie on their back for an ultrasound exam much like a dog or a cat

Rabbits and other hind gut fermenters (guinea pigs, degus, chinchillas) have a large amount of gas in their GI tract so ultrasound will not be as rewarding and different positioning may be warranted.

### **Ultrasound – guinea pigs**

When imaging for cystic ovaries, having the guinea pig stand on a table top and positioning the probe on the back pointed ventral over the L2-L4 epaxial position is the best place to look for cystic ovaries.

### **Radiographs in Avians**

Ideally best done under sedation.

With CR cassettes and dental x-ray gun you can take vd and lateral while the bird is perched however on the lateral view the legs do cover part of the coelomic cavity.

This should be reserved to do if medically warranted or if the owner declines sedation.

## **Radiographs Avian - Sedation**

Intranasal sedation can be used effectively for minor procedures

Midazolam which can be reversed with flumazenil

Butorphanol - best opiate as many birds do not have the mu receptors for pain like mammals do and thus opiates like morphine and hydromorphone have questionable pain relief in birds

Inhalants like Sevoflurane and isoflurane are good options for quick and minor procedures.

Bird tape is a great tool to help hold extremities in position for radiographs.

I recommend 3M's Transpore Tape

## **Avian radiograph positioning**

Lateral view – it is recommended to tape the wings up and out of the way and tape the legs caudally out of the way of the coelomic cavity

## **Avian VD Radiograph Positioning**

With VD position tape the neck, wings and legs out of the way of the coelomic cavity (thorax and abdomen)

Bird perched for radiographs

This really can only be used with a moveable x-ray tube

Position the bird then shoot perpendicular to the bird with the cassette behind the bird

## **Avian Ultrasound - positioning**

Ultrasound

can be difficult due to the abundance of air sacs in the coelomic cavity.

I tend to use the vd positioning for radiographs for ultrasound of the coelomic cavity.

## **Radiographs in Reptiles**

For squamids (lizards) VD/dv and lateral views like you would in small rodents

For snakes usually if you lay down a snake in a box a DV view can be achieved fairly easily if the snake is not super mobile. For lateral views, putting a snake in a tube can help get them into a nice lateral plane, though if you have CR cassettes and a dental x-ray gun you can position the cassette however you need

Terrapins (turtles, tortoises, and box turtles) – a DV, lateral and cranial-caudal view is preferred

## **Lizards dv view**

### **Lizard lateral view – with moveable x-ray tube**

### **Lizard lateral view – without moveable x-ray tube**

This can be very difficult. The best way to achieve this is to tape or vetwrap the animal to a flat surface turn perpendicular to the x-ray beam. Make sure that the animal is secure and not going to be injured. Sedation may be required.

## **Snake restraint for x-rays**

### **Snake DV View**

### **Snake lateral view**

With non-moveable x-ray tube place snake in tube and rotate to side to take picture

With a moveable x-ray tube stretch snake out straight and shoot horizontally

## **Turtle or tortoise restraint for radiographs**

### **Turtle or tortoise DV view**

### **Turtle or tortoise lateral view – with moveable x-ray tube**

### **Turtle or Tortoise lateral view – without moveable x-ray tube**

### **Turtle or tortoise cranial-caudal view – with moveable x-ray tube**

### **Turtle or tortoise cranial-caudal view – with non moveable x-ray tube**

## **Ultrasonography in Reptiles**

Just as in avians this can be difficult to get a full picture as they have air sacs and with terrapins they have shells that block being able to get to the majority of the internal parts

With snakes and lizards restraining in lateral or dorsal recumbency is best. They can also be held with the tail towards the floor and the head towards the ceiling.

## **Amphibian radiology**

It is important to wear gloves when handling amphibians as they can absorb things more easily through their skin – so things such as nicotine and other toxins can negatively affect these animals

Typically using a moistened dish or container is used to place them in and then the container is placed on the cassette or table.

A DV view is relatively easy for any type of tube.

A lateral view is more difficult if you do not have a moveable x-ray tube as turning and restraining these animals safely in a lateral position. A container like a petri dish with lid can be used as long as they fit relatively comfortably and they are taken quickly

For moveable x-ray tubes, placing them in a container and placing the cassette and x-ray tube perpendicular to the animal works best.

## **Radiology DV view for Amphibians**

## **Radiology DV view for Amphibians**

## **Radiology lateral view for amphibian**

## **Radiology in Fish**

For larger specimens usually sedation is required.

Plastic containers or plastic bags can be used as well

The fish will always try to be right side up so once again without a moveable x-ray tube a lateral view can be difficult as the fish even in a bag will not want to be on its side and will fight it.

Radiography lateral view fish

## **Ultrasonography in Fish**

Ultrasonography in fish needs to be done generally under anesthesia and no alcohol or harsh chemicals are used on the probe or fish – generally just saline

Ultrasonography can be attempted with a fish in a Ziploc bag and gel and the transducer is placed on the bag. This method works although restraining the fish with this method can be a bit difficult.

### **Radiology in invertebrates**

The same process is used as amphibian radiology, gloves should be worn and protective plastic or moistened paper towel used so that the animal does not contact harsh chemicals.

### **Ultrasonography in invertebrates**

Invertebrates with an exoskeleton can generally not be ultrasounded very well. Worms and snails can be ultrasounded but as in amphibians and fish care must be taken with handling and materials used with the probe

<https://youtu.be/OW0XuHc5mgo>

Friday  
October 12,  
2018



## Megan Brashear, BS, CVT, VTS (ECC)

Megan is the Technician Trainer at VCA Northwest Veterinary Specialists in Clackamas, Oregon where she enjoys her time on the clinical floor working with technicians and medical staff providing in-the-moment training and teaching the whys of ECC nursing.





## The ABCs Of CRIs

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Megan Brashear, BS, CVT, VTS (ECC)

Constant Rate Infusions (CRI) are commonly used in veterinary medicine for a variety of patient needs. Any drug that is beneficial administered as a continuous infusion as opposed to bolus dosing can be calculated to be administered concurrently with IV fluids or as its own infusion. Drugs with short half-lives must be administered continuously in this manner. Common CRIs include pain medication, anesthesia, electrolytes, blood pressure management, and insulin. Veterinary nurses are responsible for calculating these constant rate infusions, understanding the effects of the drugs given, and managing patients that may be on a variety of medications at once.

Management of CRIs starts with knowledge of the drug being delivered and any special needs of that drug. Some drugs are light sensitive, others may bind to plastic, others may not mix well with concurrent fluids or medications and require a dedicated IV catheter. Veterinary nurses are responsible for a thorough understanding of these drugs and their special considerations and also need to understand the desired effect of administered drugs and monitor patients for those effects. They also need to anticipate any adverse effects and how to manage those. Finally, nurses must have a knowledge of drug dosages for CRIs so that if fluid rates are changed, drug dosages are still administered appropriately. This requires a good understanding of calculations and quick thinking.

Proper labeling of CRIs is vital for communication and accuracy. Each infusion must be labeled with the drug name, the drug dose per milliliter, the time and date the CRI was mixed, an expiration date (if necessary), and the initials of the person who created the CRI. Double checking calculations and having a coworker recheck your calculations is helpful as improper concentrations can be detrimental when administered over time.

Metoclopramide is a drug commonly added to already running IV fluids to enhance gastrointestinal motility. For example, a 23kg dog has fluids running at 110ml/hr. The veterinarian orders a metoclopramide (5mg/ml) CRI at a dose of 2mg/kg/day. How many milliliters of metoclopramide do you add to a liter of fluids that will run at 110ml/hr?

$$2\text{mg/kg/day} = 46\text{mg/day (24 hours) of metoclopramide (23kg} \times 2\text{mg/kg/day)}$$

↓

$$46\text{mg}/24\text{hours} = 1.91\text{mg metoclopramide per hour (46mg per day / 24 hours in a day)}$$

↓

$$1000\text{ml}/110\text{ml per hour} = 9.1 \text{ hours (1 liter of fluids will last this dog 9.1 hours)}$$

↓

$$9.1 \text{ hours} \times 1.91 \text{ mg per hour} = 17.4\text{mg metoclopramide needed per 1 liter of fluids}$$

↓

$$17.4\text{mg}/5\text{mg} = 3.5\text{ml of metoclopramide needed (drug dose/drug concentration)}$$

If you add 3.5ml (17.4mg) of metoclopramide to 1 liter of fluids running at 110ml/hr, a 23kg dog will receive 2mg/kg/day of metoclopramide

As the nurse monitoring this patient, the calculation should be taken one more step to determine how many milligrams of metoclopramide are in each milliliter of fluid so that if the fluid rate is changed, the dose of drug is known. If this patient's fluid rate is increased to 140ml/hr, how much metoclopramide is the dog receiving?

$$17.4\text{mg} / 1000\text{ml} = 0.0174\text{mg/ml (number of milligrams per liter divided by 1000ml)}$$

↓

$$0.0174\text{mg} \times 140\text{ml/hr} = 2.43\text{mg/hr (milligrams per milliliter multiplied by fluid rate per hour to get mg/hr delivered to the dog)}$$

↓

$$2.43\text{mg} \times 24\text{hrs} = 58.46\text{mg/day (milligrams per hour} \times \text{hrs in a day)}$$

↓

$$58.46\text{mg}/23\text{kg} = 2.5\text{mg/kg/day (milligrams per day divided by patient weight to get mg/kg/day)}$$

This new dose can be compared to the therapeutic dose of metoclopramide and adjustments can be made as needed.

There are times when CRI requests are made and the patient has a partially empty bag of fluids running. The calculation should be made assuming a full liter for labeling purposes and then the amount specific for the partially empty bag calculated for the remaining fluids.

Pain management and blood pressure management medications are commonly administered as a constant rate infusion because in this format these drugs can easily be titrated according to patient

needs. For example, a patient needing orthopedic surgery for a fracture repair may require a higher dose of drug pre and intra-operatively but as they recover the dose can be decreased and the patient weaned off. Blood pressure support such as dopamine often requires fine tuning of titration up and down until the patient responds appropriately. In these situations, it is advantageous to create the CRI so that a fluid rate of 1ml/hr delivers a dose of 1mg/kg/hr to the patient (or, depending on the dosage, 1ml/hr delivers 1mcg/kg/hr or 1mcg/kg/min to the patient). This way, when doses need to be changed quickly, it simply involves a fluid rate change and not more math.

You will be monitoring anesthesia on a 12kg dog that sustained vehicular trauma and needs pain management. You are asked to create a fentanyl (50mcg/ml) CRI and start administering the dog 5mcg/kg/hr with the option to titrate up or down depending on patient response. You plan on making the CRI in a 100ml bag of 0.9%NaCl.

1ml/hr needs to deliver 12mcg/hr

↓

100ml bag = 100 hours of CRI (assuming 1ml/hr, since 1ml/hr = 1mcg/kg/hr)

↓

12mcg x 100hrs = 1200mcg (dose per hour x hours of CRI)

↓

1200mcg/50mcg per ml = 24ml of fentanyl needed (drug dose/drug concentration)

↓

Run the CRI at 5ml/hr to deliver 5mcg/kg/hr to your 12kg patient

Because this calculation used 100ml as the final volume of the CRI, you must first remove an equal amount of 0.9%NaCl from the bag before adding your fentanyl. In this case, remove 24ml of 0.9%NaCl and discard it before adding 24ml of fentanyl. The label should read "Fentanyl 1200mcg QS 100ml NaCl". The QS stands for quantity sufficient and means the total volume of the CRI is 100ml.

Look closely at drug doses as some CRIs are dosed as mcg/kg/min. Dopamine is a common example of such a drug. This drug dose is used within a range and may need frequent titration until an appropriate response is reached. Creating the CRI so that 1ml/hr delivers 1mcg/kg/min will make these changes easier on the nursing team.

A 17kg dog has been battling hypotension and you are asked to create a dopamine (40mg/ml) CRI and start administering 5mcg/kg/min to the dog. For ease of titrating, you will make the CRI so that 1ml/hr delivers 1mcg/kg/min. You will be making the CRI in a 250ml bag of NaCl.

1ml/hr needs to deliver 17mcg/min

↓

17mcg x 60 minutes = 1020mcg/hr (because the fluid rate is in ml/hr, you need to convert the dose to mcg/hr)

↓

1020mcg/1000 = 1.02mg/hr (because your drug concentration is in mg/ml you need to convert mcg to mg)

↓

250ml bag = 250hrs (assuming a rate of 1ml/hr)

↓

1.02mg x 250hrs = 255mg of dopamine needed for this CRI (the dose per hour x hours)

↓

255mg/ 40mg per ml = 6.34ml of dopamine needed (drug dose / drug concentration)

For this CRI, you need to add 6.34ml of dopamine to 234.7ml of 0.9%NaCl. To deliver 5mcg/kg/min to a 17kg dog, run the CRI at 5ml/hr. If the dose needs to increase to 7mcg/kg/min, simply increase the fluid rate to 7ml/hr. The label for this CRI should read “Dopamine 255mg QS 250ml NaCl”.

In smaller patients or those where there is concern for fluid overload, these titratable CRIs can be created so that 1ml/hr will deliver 2mcg/kg/hr (or 2mcg/kg/min) or even 5mcg/kg/hr (or 5mcg/kg/min). These concentrations require some basic calculations when a dose change is requested, but can still easily be performed. If you are asked to change the dose, divide the dose by the concentration and reduce the fluid rate.

If desired, some pain management constant rate infusions will have multiple drugs added to the same bag; an example is fentanyl, lidocaine, and ketamine. In this instance, calculate each drug separately using the appropriate formula. If you are creating a FLK CRI in a 250ml bag and you calculate needing to add 8ml of fentanyl, 27ml of lidocaine, and 0.7ml of ketamine, you would remove 35.7ml of saline (8+27+0.7) from the bag and then add all of your drugs. Take the math a step further to determine the mg/ml of each drug so you know what your patient is receiving if the CRI fluid rate changes.

Some drugs used in constant rate infusions require special care; it is the nurse’s responsibility to research these drugs and handle them appropriately. Some are light sensitive and must be covered. Insulin may be used in a CRI for patients and insulin will bind to the plastic used in IV tubing. In order to ensure the

patient receives the appropriate dose, add insulin to the bag of fluid, attach the dripset and then bleed 50ml of the insulin CRI through the IV line and discard it. This will allow for insulin to bind to the plastic in the line and subsequent drug will get to the patient. Insulin CRIs expire after 24 hours and must be changed out to ensure efficacy. Appropriate nursing rounds and team communication will ensure that constant rate infusions are created and managed appropriately.

Common drugs used in constant rate infusions:

- Fentanyl: 100 times more potent than morphine, only lasts ~30 minutes after IV injection
  - Loading dose of 2-5mcg/kg
  - 2-5mcg/kg/hr for pain management; 10-45mcg/kg/hr for anesthetic effects (ventilate)
- Ketamine: Helps strengthen response to opioids, helps with wind-up pain
  - Loading dose of 0.5mg/kg
  - 10mcg/kg/min for surgical stimulation, then 2mcg/kg/min post-operatively
- Lidocaine: Supplement to anesthesia (lowers inhalant requirements)
  - 25-50mcg/kg/min
- Dopamine: Increases cardiac output and causes vasoconstriction (dose-dependent)
  - 2-10mcg/kg/min
- Dobutamine: Increases cardiac contractility without increasing heart rate
  - 5-20mcg/kg/min in dogs; 2mcg/kg/min in cats
- Norepinephrine: Causes vasoconstriction, used for blood pressure management in sepsis
  - 0.5-2mcg/kg/min
- Metoclopramide: Gastrointestinal motility, used in ileus and post-operative GI surgery
  - 1-2mg/kg/day
- Potassium Chloride: Supplement in hypokalemia
  - Do not exceed 0.5mEq/kg/hr

Always double check calculations and clearly label constant rate infusions. If multiple drugs are added or the patient is already on fluid additives and/or other IV medications such as antibiotics, check a drug compatibility chart to ensure safety. Practice your calculations and you can greatly enhance your nursing capabilities with constant rate infusions.

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# The Art of Nursing

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As veterinary medicine advances and pet owners have come to expect specialty level care, the nursing care required for these patients has increased. Veterinary nurses and technicians are expected to have high-level understanding of physiology, pharmacology, anesthesia, hematology, and be comfortable performing advanced procedures. Veterinary patients are surviving critical diseases leading to challenging nursing requirements. High level specialty education conferences such as IVECCS provide an amazing resource to veterinary technicians when it comes to possessing cutting edge knowledge, but there is more to quality nursing care than medical knowledge. Excellent nurses must provide patient care beyond the treatment sheet, possess communication skills, think critically and challenge others to do the same, and take care of themselves so that they can provide care to others.

Patient enrichment, or care beyond the treatment sheet, means thinking about the mental well-being of the patients. We are all familiar with the nurse's role as patient advocate when it comes to pain management, but we also need to monitor anxiety in our patients. This is important because there are physiologic changes that happen in animals when they experience fear and stress. We can immediately detect an increase in heart rate, blood pressure, note panting or hissing, trembling, and vocalizing. While we may chalk those changes up to normal stress, in our debilitated patients these changes can delay healing. Their stress-fueled tachycardia and hypertension uses energy and requires increased oxygen. Nurses should not only be familiar and comfortable recognizing stress and fear, but should be adept at adjusting techniques to allay this fear.

Before interacting with any animal, fearful or not, it is important to survey the scene. Take control of your own emotions, as animals can pick up on our anxiety and frustration. Take a deep breath, pay attention to smells, sounds, space, surfaces, and body language and correct what you can before continuing. Everyone in the practice needs to be aware of these points and working as a team to improve conditions in the hospital. It is frustrating to both employee and patient to finally have that nervous dog ready for a blood draw and a cage door is slammed, undoing all of your hard work. Remind others to keep their voices low, resist playing loud music, and try to segregate loud animals away from fearful patients.

Even if the animal is not acting out and requiring special handling techniques, stress can delay the onset of eating and cause depression in our patients. Nurses must recognize that part of our daily duty is to not only provide top notch medical treatment, but also to do what we can to provide comfort and stimulation to ward off depression. A recumbent patient may need a trip outside and sit in the sun. A large cat may benefit from movement to a large dog run where he can stretch his legs. A terrified feline would benefit from a box to hide in. The anxious dog may feel better behind a blanket or under one. Some patients recovering from surgery are afraid to lie down, afraid of the pain they are used to experiencing when they

move. Gently assist them and they may quickly fall asleep. Some patients may benefit from one on one time to encourage eating. While many of us are overwhelmed with treatments, we should not neglect our patients and their little needs that will help them recover. If these techniques seem second nature to you, they are not to everyone. It is important to take the time to instruct coworkers on the importance of patient comfort and advocate for them.

Communication with both coworkers and clients is vital to the success of technicians. Working in close quarters for long shifts requires the ability to communicate clearly and efficiently. When communication does not go smoothly, teamwork, client care, and even patient care can suffer. When approaching any situation, it may be cliché but important to view the situation from the other person's perspective. Empathizing with someone else's stress, fear, and lack of knowledge can help one explain things more thoroughly, better set expectations, and save the task of having to repeat the same information over and over again. Always assume good intent of everyone. Be careful of negative assumptions, as they will impede the ability to remain open minded. Depending on your own experiences and communication style, it may be difficult to engage in any sort of confrontational communication. Confrontation does not always mean yelling; it can mean any conversation around a difficult topic. Sometimes, an easy two-minute chat for one can be a confrontational issue for another so be prepared for any sort of response. Waiting for the appropriate time to have a conversation with a coworker, even if it 'isn't a big deal' can make a big difference in the way the information is received.

Practice members need to place a premium on helping each other. While it is beneficial to foster comradery among small groups working together, do not let it turn into hostility for other teams. This can happen between shifts as well as between varying roles. Challenge employees to see each other as internal customers. Treat coworkers with as much respect as a client deserves. Encourage employees to participate in the training process. If a technician is having problems with a process that involves the front desk, a customer service representative can step in and help with the training process rather than waiting for the technician manager to respond. The technician team, rather than complain that the check in process is taking too long, can offer to help by taking phone calls. Cross-training between departments creates an opportunity not only for assisting with scheduling holes but also with empathy and understanding between employees. The goal of cross-training is not to have a group of people who can do every job in all areas of the hospital, but to create a group of coworkers who can help out in a busy time and have a working understanding of all areas.

Nursing also requires that technicians have good client communication skills. While clients are often a source of frustration and compassion fatigue, they are the reason we have patients to treat and are an important member of the medical team and should be viewed as such. In the emergency/ICU setting there is the added challenge of client anxiety and fear. Each interaction must begin with gaining the client's trust. This is tricky when the client is new to the practice, their pet is in an emergency situation, and there is minimal time to talk. Make eye contact, explain what you are doing (especially if the animal is there with you), listen to them, and show genuine concern for them and their pet. Empathizing gains trust, not talking over their head and treating them as an outsider.

It is also important to set client expectations. While hospital employees process multiple patients over the course of a day, think of each client as experiencing the practice for the first time. Let the client know what to expect next, who will be coming to talk to them next, and what is expected for them. If something changes, communicate that change with them. Do not make assumptions of understanding especially in emergency situations. Talk to clients at every step and ask multiple times if they have questions. People under stress are not able to absorb all of the information thrown at them during the course of an emergency appointment. Ensure they have written instructions and feel comfortable enough to call with any additional questions. What may feel like over-communication from the technician is often the minimal understanding for the client.

Critical thinking in veterinary technicians and nurses is one of the most important skills to have in the emergency setting. Critical thinking involves not just understanding the book knowledge and memorization taught in the classroom, but asking intelligent questions and using outside knowledge and past experiences to come to a conclusion. In a medical situation, critical thinking may allow for a technician to set up for a procedure that they know is coming in the future, or involve using their knowledge and experience to anticipate changes in their patients. Critical thinking allows technicians to become proactive, not reactive, in their job thereby making them an indispensable part of the team. Technicians should be empowered to think globally, plan ahead, anticipate need, and keep the hospital floor moving. This skill is often present in more senior technicians but can be taught and fostered in technicians at every level.

Potential new hires can be given a critical thinking interview; ask them medical scenario questions to gauge their ability to think ahead. Ask them to talk through a common phone triage scenario like a dog suffering from dystocia or a vomiting diabetic cat. Ask them to list reasons for tachycardia under anesthesia or pain management protocols they are familiar with. These types of questions can help hospitals discover critical thinking technicians and impress to new employees the importance on utilizing their skills and knowledge. Technicians should be asked scenario questions in the hospital about the patients they are treating. More experienced technicians and/or veterinarians should quiz medical staff on the reasons treatments are performed. Nursing means more than simply administering medications; nursing means understanding the why and how of those medications.

Technicians can test critical thinking skills by anticipating results. Look at the presenting complaint, medical history and physical exam results on a sick patient and anticipate their blood values. Anticipate what may be found on radiographs. Were you right? If not, why? Anticipate the results of vital signs every time treatments are performed on a hospitalized patient. If a post-operative gastrointestinal foreign body dog is sleeping soundly and breathing comfortably, one might anticipate a normal heart rate and blood pressure. If the heart rate is 140bpm and the blood pressure is decreased, something is wrong. Perhaps a different blood pressure cuff was used or a different machine than the last treatment time. Any abnormal vital signs should be reported to the veterinarian, but the nursing team must think through some

potential causes for the abnormalities and be confident in the results. Simply writing the results in the record and moving to the next patient is not utilizing skills and knowledge to their potential, as well as not benefiting the patient or the veterinarian.

Technicians may not have the authority to change orders, add medications to treatment plans, or make a diagnosis, but those limits do not mean that technicians should not educate themselves in all aspects of the treatment plan. When medical orders are made, ask yourself why? Why are we using this antibiotic over that one? Why is this patient having an arrhythmia now? Why is the blood pressure dropping in this situation and can I do anything about it? Why are we giving a fluid bolus now? As more is learned, technicians will be better about anticipating changes in the next patient and be prepared. This growth fosters teamwork and trust and allows for a higher standard of care.

Finally, the nursing team must place a priority on caring for themselves. Emergency and critical care work can place an enormous emotional load on all employees and a great deal of compassion is required to perform the job. Euthanasia can be a major trigger to burnout and even compassion fatigue. Depending on the patient population, some veterinary professionals are involved with euthanasia every single shift. While euthanasia can be a great gift for suffering animals, the grieving client also requires compassion. Sometimes an animal is euthanized for a correctable medical problem but the client lacks the funds. Other times, euthanasia is the appropriate choice but the client cannot bring themselves to authorize it. Animals are removed from the hospital to “die in peace” at home, leaving the staff in frustration. Even if a visit does not end in euthanasia some animals return home with minimal treatment due to the financial position of owners. While difficult, it is vital to the longevity of a technician’s career to not place our own knowledge and expectations onto pet owners. The constant disappointment as a result can easily bring down the strongest of veterinary professionals. Learn to let go of those expectations, assume good intent from pet owners, and make the priority offering the best care possible to the patient.

Many veterinary hospitals operate in a guilt culture, where staff members work long hours and extra shifts out of guilt to their coworkers and other patients. Lunch and breaks are not taken because there is always more work to do and more patients to care for. Staff members work through colds and fevers because staying home sick often means the team has to work twice as hard. Vacation hours go to waste because there is no one to fill the open shift. While caring for each other is important, nothing is as important as caring for yourself. In order to take care of others, we must take care of ourselves.

The tools to combat burnout are within us. First is to recognize changes in our attitude and outlook. Quick to anger, apathy, sadness, trouble sleeping, forced isolation, crying at work (or at the thought of work), and lack of hobbies outside of work are all signs. Are you simply going through the motions at work? Do you find yourself treating patients without caring who they are or why they are in your hospital? Do you lack empathy for your coworkers? Recognizing negative trends in your own behavior and thoughts can mean that you are suffering. There are many online tools that can help point to symptoms of burnout and fatigue and can help you with the next steps to take.

Taking time away from work, whether it be a few days at home watching movies, taking appropriate breaks, or leaving town for a few days can help to reset stressed employees. Discover hobbies and have something to look forward to outside of work. Eating better is necessary, and means that you have to take breaks at work to eat home cooked food. Exercise, as simple as walking the dog every night after work, can help ease anxiety and bad feelings. Many veterinary practices, invested in the long term health of their employees, are implementing self-care ideas in the practice. Working to change the guilt culture takes work and a progressive manager, but should be a goal in every hospital.

Even if you do not have symptoms of compassion fatigue, look out for problems in your coworkers. Look out for each other by ensuring everyone has appropriate breaks. Make it okay for everyone to discuss their feelings after a tough euthanasia. Plan activities outside of work for everyone to bond together in a non-stressful situation. Discuss the possibility of a wellness program at work. Reward healthy behavior, or have a central location for listing positive changes and encourage discussion. Create a small budget for buying healthy snacks for the team. Even small changes from leadership can show to the staff that mental and physical health is important and can help with compassion fatigue.

Compassion fatigue does not have to be a permanent situation. With recognition, self-care, and time it is possible to return to loving your job, your patients, and your clients. Evaluate yourself every few months and be honest with where you are in your career. What can you do to remain happy and ensure mental health in the future? Rely on your coworkers for support, and be a support to them. Take time to take care of yourself so that you can take care of others.

Good nursing care requires technicians to make learning a life-long pursuit. Medicine is ever evolving and clients are increasing their expectation of what medicine can do for their pets. The technician profession is evolving as well, and nursing teams are required to perform more advanced procedures and understand advanced disease processes. Make it a goal to increase not only your understanding of medicine, but also your understanding of communication, your ability to critically think, and make sure to take the time to provide enough care for yourself that you can spend many years advancing our great profession.

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# Anaphylaxis: How Why and What to Do

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An allergic reaction is defined as a systemic reaction in response to an antigen. These systemic responses can be divided into types of hypersensitivity reactions based on the cells involved; mast cells, antibody reactions, or T-cell responses. When treating anaphylaxis, patients are experiencing a Type 1 Hypersensitivity reaction which is facilitated through IgE bound to mast cells. Patients can experience symptoms from mild to severe and their treatment will vary greatly depending on their clinical signs and status upon arriving to the hospital.

An antigen is a substance such as insect venom or particles in a vaccine that will produce an immune response from the body. The patient is exposed to the antigen and Helper-T Cells and B cells communicate resulting in immunoglobulin (IgE) antibodies against the specific antigen released into circulation. The IgE bind to mast cells and basophils in the blood. This binding means the body is prepared to recognize the antigen if it is again introduced allowing an immediate reaction. Once the body recognizes the antigen, it is recognized and binds to the IgE causing degranulation of the mast cells and basophils. This degranulation causes a release of histamine and other inflammatory mediators into circulation.

Histamine is stored in mast cells and basophils and its results in the body are responsible for many of the clinical signs seen with anaphylaxis. Histamine is complex – it is responsible for at least 23 physiologic functions in multiple organ systems. There are at least four histamine receptors in the body, and the effects seen depend on which receptor is activated. The H1 receptor is most responsible for the clinical signs seen with anaphylactic reactions. Mast cells and basophils are mostly responsible for histamine manufacturing and release and can be activated by any number of allergens. The H1 receptor interacts with the endothelium and will cause vasodilation and an increase in vascular permeability. H1 receptors are found all over the body – in the brain, respiratory tract, the GI tract, heart, and vascular system. H2 receptors regulate the right side of the heart muscle and are also involved in gastric acid secretion.

When a patient encounters an antigen they may only have a mild response and reaction. As histamine is released, an increase in vascular permeability leads to localized edema and local reactions within the skin will result in urticaria. These mild responses should be examined by a veterinary team and treated. Treatment for simple allergic reactions is aimed at stopping the reaction to histamine release happening in the body. This is achieved by administering a histamine blocker – most commonly diphenhydramine (2-4mg/kg IM) immediately. A dose of steroids (anti-inflammatory) is also often administered as well (commonly dexamethasone sodium phosphate 0.125-0.5mg/kg IV). With many of these patients, a reduction in facial swelling and urticaria is often noted after 20-30 min and these patients can continue on oral anti-histamine for 24-48 hours and not see any long term negative effects.

With Type 1 hypersensitivity reactions, the histamine release is widespread and systemic results are noted. In dogs and cats the cardiovascular, respiratory, and gastrointestinal systems are most affected. Within the cardiovascular system, histamine is responsible for regulating cardiac function, vascular resistance, and circulating volume. When histamine concentration increases within the vascular system, arterioles dilate causing a sudden drop in both systolic and diastolic blood pressure. Capillary beds become more permeable leading to leakage of plasma and also contribute to severe hypotension. This hypotension quickly manifests itself in the common clinical signs of hypovolemic shock; most notably tachycardia, poor peripheral pulse quality, pale mucus membranes, hypotension, dull mentation, and cold limbs. Loss of blood and oxygen flow to tissues inhibits cells from maintaining normal functionality. Every cell in the body requires ATP to protect cell wall integrity and perform cellular functions. In a shock situation, because the body is no longer able to provide normal blood flow to tissues oxygen distribution is interrupted, ATP production decreases, and cells begin to die.

The body has baroreceptors to warn the body of decreased blood flow in an attempt to avoid this cell death. These receptors are located in the aorta and kidneys and in times of hypovolemia (for example) sense when blood flow is low and signal back to the body that blood flow is lacking. The sympathetic nervous system reacts with an attempt at vasoconstriction, increased cardiac contractility, and tachycardia. These adjustments will increase cardiac output in an attempt to maintain blood flow and oxygen flow as normal. The kidneys, via the renin-angiotensin-aldosterone system will begin to retain sodium and water in order to increase intravascular volume. These mechanisms are overwhelmed by the effects of histamine which continues to promote vasodilation and worsening of oxygen delivery to tissues.

In anaphylaxis, the release of histamine, prostaglandins and leukotrienes leads to congestion in the portal veins in dogs. Histamine also leads to smooth muscle contraction within the liver resulting in blood pooling both in the liver and intestines. As blood backs up in the portal veins, capillary injury in the intestines leads to bleeding contributing to hematochezia seen in anaphylaxis. Dogs will exhibit liver enzyme elevations on blood chemistry evaluation. Many canine patients may exhibit gall bladder wall edema which can be noted on aFAST scan if time and patient status allows for evaluation. In dogs that present collapsed to the hospital with unknown history, looking for liver abnormalities both on ultrasound and lab results can help point towards anaphylaxis but should not be examined in place of appropriate treatment for these patients. Priority in treatment for these patients lies in volume resuscitation and maintenance of blood and oxygen delivery.

Within the respiratory system, histamine release works on the smooth muscles of the airway and causes constriction, while also causing vasodilation. Vasodilation and increased capillary permeability leads to pulmonary edema and often respiratory distress. With histamine release there is also an increase in mucus secretion. Anaphylactic patients may exhibit tachypnea that continues longer than the initial exam period and immediate treatment. These patients should be administered supplemental oxygen and

monitored for hypoxia. Hypoxia can be a trigger event for more histamine release and inflammation and must therefore be avoided.

Cats will exhibit primary respiratory signs when suffering from anaphylaxis. They experience laryngeal edema and bronchoconstriction. Cats can also have localized skin reactions during anaphylaxis leading to pruritis often focused around the head and neck. Edema occurs in both the lungs and intestines and can progress to hemorrhage.

Histamine release also stimulates gastric acid secretion which can lead to stomach and duodenal ulceration. With the severe hypotension and hypoperfusion already happening, these patients (especially canines) will often experience hematochezia and hematemesis further complicating their status. The breakdown of the gut mucosal barrier can be a precursor to gut-derived sepsis and severely complicate the disease process. While an allergic reaction, even as severe as anaphylaxis is not itself an infectious process, severe hematochezia should be addressed with antibiotic therapy and close patient monitoring. Sepsis will complicate blood pressure management and hypoglycemia can lead to seizures and patient death. The nursing team must monitor patients closely for even minor decline in the vital signs of these patients. Gastroprotectants are important for anaphylaxis patients during hospitalization. Cimetidine, ranitidine and famotidine are all histamine (H<sub>2</sub>) blockers but their use has not been proven to be more beneficial in treating the gastrointestinal effects of anaphylaxis.

When a patient presents to the hospital suffering from anaphylaxis, speed and blood pressure support are vital for patient survival. Within one minute of detecting the insult, the body is flooded with histamine. H<sub>1</sub>, H<sub>2</sub> and H<sub>3</sub> receptors are activated; followed by bronchoconstriction, vasodilation, gastric acid secretion, and even inhibited norepinephrine release which prevents vasoconstriction, further compounding the hypovolemic shock the patient is experiencing. While there is no diagnostic test for anaphylaxis, these patients need symptomatic treatment which involves aggressive fluid therapy to restore and maintain appropriate blood pressure. IV access is obtained – a challenge in many of these cases – and crystalloid fluids administered. The shock does of crystalloids is commonly divided into 20ml/kg portions and administered quickly and repeated until an improvement in heart rate and blood pressure is noted. Oxygen support should also be administered during the resuscitation period and the patient monitored closely in case ongoing oxygen support is necessary.

Synthetic colloids remain controversial in veterinary medicine but may be utilized according to clinician discretion. In patients not responding to crystalloid treatment, a 5ml/kg bolus of synthetic colloids can be administered and repeated, not exceed a total dose of 20ml/kg/day. Nurses must be familiar in monitoring patients for fluid response and overload as these patients will receive large amounts of fluids in a short period of time. Monitoring blood lactate levels is beneficial in determining the fluid response as is blood pressure, heart rate, respiratory rate, skin turgor, and urine output. Quantifying fluid losses through vomiting and diarrhea is important as ongoing losses can be significant in these patients and must

be replaced. Urine output and urine specific gravity measurement can also be trended to determine fluid response.

Patients commonly require epinephrine administered IM (1:1000; 0.01mg/kg) and repeated to promote vessel constriction and cause smooth muscle relaxation. This will bring the benefit of vessel constriction to increase blood pressure and tissue perfusion, and smooth muscle relaxation can improve the patient's ability to breathe. Epinephrine as a CRI (0.05mcg/kg/min) may be more successful at restoring and maintaining perfusion than IM administration, but at this time there are no published studies to support this. Blood pressure should be monitored closely while on epinephrine, as well as heart rate and ECG. If the blood pressure does not respond to epinephrine other vasopressor drugs like dopamine, vasopressin, or norepinephrine may be required. It is important to note that these drugs should only be started after fluid resuscitation.

Antihistamines such as diphenhydramine can be utilized in true anaphylaxis but will not prevent the clinical signs and progression of disease and are not beneficial alone in a critical patient. Glucocorticoids are helpful in minor reactions, but take too much time in critical situations and can contribute to worsening gastric irritation and bleeding. Albuterol can be utilized in severe respiratory distress and bronchoconstriction.

Laboratory results in anaphylaxis patients may show increased ALT levels in dogs, increased lactate levels due to severe hypoperfusion and metabolic acidosis resulting from poor perfusion. Azotemia may also be present from decreased kidney perfusion and gastrointestinal bleeding can also cause an increase in BUN levels. Lactate levels can be utilized to monitor the success of fluid therapy alongside blood pressure and pulse quality serial measurements.

It is important to note that while rare, a biphasic anaphylaxis can occur in dogs and cats. They may respond to the initial treatment and resuscitation but can relapse into similar clinical signs as at admit and will require similar treatment. Response to fluids and medications is never a signal to decrease nursing care, as anaphylactic reactions can be biphasic or delayed.

As anaphylaxis is a disease initiated by inflammation, patients suffering from severe allergic reactions are at risk for developing Systemic Inflammatory Response Syndrome (SIRS). The development of SIRS can complicate treatment of anaphylaxis as it leads to a loss of vascular tone resulting in hypotension and poor organ perfusion. Dogs and cats with an inflammatory disease process and alterations in vital signs (see table) are at risk for developing SIRS and will require close monitoring after initial resuscitation. SIRS is often the final step before a patient develops DIC; while it cannot be avoided the nursing team must be aware.

## SIRS Diagnosis Criteria

	DOGS (2 of 4)	CATS (3 of 4)
Temperature	<100.6F or >102.6F (<38.1 >39.2)	<100F or >104F (<37.8 >40.0)
Heart Rate	>120bpm	<140 or >225bpm
Respiratory Rate	>20bpm	>40bpm
WBC (x10 <sup>9</sup> /L)	<6 or >16	<5 or >19

Inflammation is the major factor in a patient developing Disseminated Intravascular Coagulation, or DIC. The inflammatory process releases cytokines which then encourage tissue factor expression. Tissue factor is the trigger to the coagulation process and leads to fibrin formation, platelet activation, and thrombus formation. Endothelial tissue is also activated during inflammation causing platelets to bind to these cells creating micro-clots throughout the vasculature system. Antithrombin activity is decreased during inflammation; the liver will decrease production during systemic inflammation. This is unfortunate for the coagulation process because now not only is the body missing the ability to maintain normal blood flow around these inappropriate clots, but antithrombin also has an anti-inflammatory role in the body. By suppressing this natural anti-inflammatory, the body is pushed further into critical disease. The only cause of DIC in small animal patients is the cytokine activation of inflammation or the un-regulation of inflammation.

Understanding the risk factors for developing DIC is important to the nursing plan. Patients should be closely monitored for signs of bleeding such as hematuria, petechiae, hematemesis, hematochezia, and ecchymosis. Changes, even small changes, to blood pressure and heart rate must be taken seriously as that can indicate a worsening disease state. The nursing team responsible for these patients will need to closely monitor their organ systems for signs of early dysfunction or failure. The kidneys, lungs, and gastrointestinal tract are particularly susceptible to damage. Urine output and specific gravity are easily monitored and can be serially evaluated to watch both hydration and renal perfusion. Serum creatinine levels can also be monitored daily to track either progress or decline. As aggressive patient management is required with DIC, low urine output can quickly be addressed with an increase in fluid therapy or the addition of diuretic therapy. Any dyspnea or hypoxia can be addressed with the early administration of oxygen therapy. Pulmonary thromboembolism can occur in these patients and any changes in respiratory rate, effort, or pattern must be noted early. Nutritional support is required in any critically ill patient, but especially those at risk for decreased gut perfusion and the potential for gut-derived sepsis. Trickle feeding enteral nutrition can help enterocytes maintain a good gut mucosal barrier and the nursing team is instrumental in placing NG/NE tubes and advocating for early nutritional intervention. Knowledge of physiology, pharmacology, and advanced procedures such as feeding tube placement and care are necessary for the care of these patients. Animal and owner alike will need TLC as they navigate critical disease.

While challenging to treat both on emergency admission and as an ongoing critical patient, with aggressive supportive and intensive nursing care these patients can make a full recovery. Veterinary nurses must be familiar with the clinical signs of anaphylaxis as well as the immediate treatment. These cases require close monitoring as their status can change quickly but with appropriate nursing care can return home.

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# Emergency Care for the Seizure Patient

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A seizure is defined as a sudden excessive discharge of neurons which leads to altered consciousness and muscle movement. Some simple focal seizures may not involve altered consciousness, but the focus of this lecture will be the more complex seizures and their treatment. Focal seizures involve a small part of the body, most notably the face, engaging in bizarre and repetitive action that the animal is not controlling. Grand mal seizures involve a complete loss of consciousness and appear violent as the animal collapses onto their side and begins padding, urinating and/or defecating, may include barking, hypersalivation, and the muscle tremors involve the whole body.

Seizure activity can be divided into 3 phases. Many animals will have subtle behavior changes immediately pre-seizure. At home, this may include seeking out the owner or hiding. Then the seizure itself which can last from seconds to minutes or longer, followed by the post-ictal phase. Owners and veterinary staff alike must use caution when handling animals during a seizure and the unpredictable post-ictal phase as patients may unexpectedly bite and lash out. Use particular caution with cats during the seizure period.

Seizures can result from drug reactions, infectious diseases (such as distemper in dogs), nutritional deficits, traumatic brain injury, metabolic derangements, toxin exposure, brain tumors, or epilepsy. Diagnosis of the cause of seizures requires a complete medical history and the veterinary nursing team knowing the right questions to ask. Hypoglycemia can lead to seizures – is the patient a known diabetic? Is xylitol ingestion a possibility? Sodium disorders can also cause mentation changes and eventual seizures – has there been accidental sodium ingestion? Are the kidneys failing leading to a sodium change? Is the animal suffering from water toxicity? Depending on the area, mushrooms, molds, compost, and metaldehyde toxicity are common causes for tremors and seizures. Patients in the hospital for liver disease or failure can develop hepatic encephalopathy which can quickly lead to mentation changes and seizures. While immediate treatment of these patients is vital, so is a complete patient history important to a swift diagnosis and treatment.

Idiopathic epilepsy is a diagnosis of exclusion and requires at minimum a full neurologic exam and lab work to rule out any metabolic cause for seizures. Owners commonly mistake syncopal episodes with seizures so a complete physical exam is also necessary during the diagnostic process. Bile acids and fasting glucose levels may need to be performed to rule out liver disease causing neurologic problems. Generally, if a dog has its first seizure between 6 months and 5 years of age it is more likely to have idiopathic epilepsy. A first seizure at older than 7 years is more likely to have an acquired cause (such as a brain tumor). Male dogs are more commonly diagnosed with idiopathic epilepsy and a higher incidence is seen in beagles, German shepherds, dachshunds, golden retrievers, Labrador retrievers, boxers, and

poodles. Advanced imaging such as CT or MRI scans can be performed on these patients to look for abnormalities and intracranial reasons for seizure activity. Intracranial cause of epilepsy carries a grave prognosis. CSF examination can also be utilized to determine the cause of seizures.

When a patient has been suffering from continuous seizure activity or has two or more seizures and does not fully recover between seizures, they are suffering from status epilepticus. Not to be confused with cluster seizures (two or more seizures in a 24-hour period) which can progress to continuous seizures. This is a life threatening condition and must be treated as soon as possible. The longer a patient suffers from seizure activity, the seizures feed on themselves and become more resistant to the medications used to control them. Continuous seizure activity leads to neuronal necrosis and death. Cerebral blood flow is decreased during a seizure which can worsen neuronal death. Status epilepticus can also lead to other complications in the patient such as hyperthermia, hypoxemia, non-cardiogenic pulmonary edema, aspiration pneumonia, lactic acidosis, rhabdomyolysis and acute kidney failure, and hyperkalemia. The nursing team must not only be familiar with treating status epilepticus in the emergency phase, but also how to monitor and watch for complications as the patient recovers.

When a patient presents to the hospital in status epilepticus, the patient must be triaged immediately to the treatment area and IV access gained if at all possible. This is a difficult task in a constantly moving patient. Benzodiazepines are the first line of anti-seizure medications commonly used in emergency seizure cessation. These drugs are GABA receptor agonists which will inhibit neuronal firing. These drugs will readily cross the blood brain barrier and work quickly to stop the seizure. Diazepam can be irritating if accidentally administered outside a vein and will bind to plastic thereby reducing efficacy when administered through a fluid line. Midazolam negates these concerns and can also be administered into the muscle in those patients that are difficult to gain vascular access. If the seizure activity has been occurring less than 30 minutes, benzodiazepines are the drug of choice to stop the seizure:

- Diazepam: 0.5 – 1mg/kg IV (dogs), 0.25 – 0.5mg/kg (cats); 1 -2mg/kg rectally. If the patient is already on phenobarbital for seizure management, the dose may need to be increased by 25-50%
- Midazolam: 0.1 – 0.2mg/kg (dogs), 0.05 – 0.1mg/kg (cats) IV or IM. If the bolus ends the seizure activity but the patient seizes again, a CRI can be administered at 0.1 – 0.5mg/kg/hr.

Recent information is available on the use of intranasal benzodiazepines in dogs for the treatment of status epilepticus when intravenous access is not available. Rectal diazepam has historically been utilized as an at-home option for owners to treat emergency seizures, but therapeutic plasma concentrations of the drug may not be reached when it is administered rectally therefore making it a poor choice for emergency treatment. In humans, intranasal benzodiazepine use has been successful in the treatment of seizures. There is one published multi-center trial comparing the use of intranasal midazolam (0.2mg/kg) and intranasal diazepam (1mg/kg) for treatment of seizures lasting longer than 5 minutes in dogs. In this study, intranasal midazolam was more successful at stopping seizures and may be a method utilized more in the future for emergency seizure treatment.

A constant rate infusion of diazepam (0.1-2.0mg/kg/hr) can be utilized in patients that have a break between seizures but continue to seizure in the hospital. These animals are started at the low end of the dose and titrated up until seizures are controlled. This drug must be protected from light and as it binds to plastic, should not be drawn up more than a few hours at a time. When the patient is seizure free for more than 12 hours, the dose can be titrated down until discontinued.

It is important to obtain a full physical exam in these patients as soon as possible and provide treatment for a variety of complications. Oxygen therapy can assist patients that are not effectively ventilating due to prolonged seizure activity. Hypoventilation will increase patient CO<sub>2</sub> levels which can compound cerebral edema from prolonged seizure activity. Hyperthermia is common in these animals and if left untreated can quickly lead to dangerous heatstroke and multiple organ dysfunction. IV fluid therapy can assist with temperature control and ensure a stable blood pressure and heart rate. Patients that present with hyperthermia must be monitored for coagulopathy, acute kidney injury leading to azotemia, and monitored for pain.

If the animal has been seizing for longer than 30 minutes or the benzodiazepine has not been effective, the animal can be anesthetized with propofol (to effect) and kept anesthetized on a propofol CRI (6 – 10mg/kg/hr) then slowly recovered. The patient must be intubated and monitored as any other anesthetized patient during this time. If propofol supply and cost is not amenable to this treatment, anesthesia can be maintained with inhaled gas and lightened until awake as long as seizure activity has stopped. Ketamine can also be utilized in maintaining anesthesia in these patients. 5mg/kg can be given for induction and then a CRI at 5mg/kg/hr. Leviteracetam can also be administered to stop status epilepticus if benzodiazapines are not working. 30-60mg/kg should be infused IV over 2 minutes, and diazepam can potentiate the effects of the drug.

Phenobarbital is the most common drug used to treat seizure disorders and can be administered intravenously in the hospital. Because it takes at least 30 minutes to be effective and over an hour to reach peak levels, phenobarbital should not be used in status epilepticus as the only rescue drug. Phenobarbital loading, giving the patient an entire 24 hour dose of the drug within a relatively short time period, is often practiced to get therapeutic plasma levels. A 5mg/kg dose of phenobarbital is given every 30 minutes to the maximum 20mg/kg dose. It is important that the nursing team monitor patients closely during the loading period as patients can become profoundly sedate. Animals should be monitored for a gag reflex and appropriate response to stimulus prior to each loading dose of phenobarbital. If the patient cannot protect their airway they should be intubated and the dosing interval increased. Phenobarbital loading can be administered concurrently with the rescue medication as the effects of phenobarbital last longer than barbiturates. Phenobarbital loading can also be achieved with oral use of the drug if patients cannot be hospitalized; the nursing team must ensure that owners know how to monitor their pet for profound sedation and return to the hospital if the animal cannot protect its airway. Client education is important when their pet is taking phenobarbital as liver enzyme elevations can be seen with administration. Serum levels of the drug must also be tested to ensure therapeutic levels over time.

Patient monitoring can be intense and time-consuming for these patients as all of the anti-seizure medications will cause neurologic depression and patient mentation on these medications will range from mentally dull to anesthetized. The nursing team must closely monitor the patient's respiratory status and advocate for orotracheal intubation and possibly mechanical ventilation if severe respiratory depression occurs. Hypoventilation can lead to hypercapnia which will cause vasodilation and increased intracranial pressure. Heart rate, pulse quality, mucous membrane color and CRT should be monitored frequently. Fluid therapy may be required to maintain adequate perfusion, and nurses should monitor urine output and calculate fluid ins and outs as possible to allow for appropriate replacement. Body temperature can initially be dangerously high in seizure patients and heatstroke and the damaging sequela that comes with heatstroke must be avoided if at all possible. Activing cooling should be initiated and stopped when seizure activity stops to prevent hypothermia occurring when the medications cause profound sedation.

Blood pressure monitoring is a necessity with any neurologic patient, particularly patients with a history of seizures. Special care must be taken to maintain a normal mean arterial blood pressure (MAP) if at all possible. Cranial perfusion pressure relies on blood pressure to maintain oxygen delivery to brain tissues. In head trauma, a decrease in the MAP or increase in intracranial pressure will decrease cranial perfusion pressure. Less blood flow to the brain means less oxygen reaching brain tissue leading to an increase in CO<sub>2</sub> in brain tissue as it is not carried away. Increased CO<sub>2</sub> levels will cause vasodilation in an attempt to bring more blood flow, but this only increases the intracranial pressure. The sympathetic nervous system responds to the stress on the brain tissue by increasing the systemic blood pressure in an attempt to remedy the situation. It is common for these patients to develop a systolic blood pressure greater than 200mmHg. When the blood pressure is pushed this high, the animal experiences a reflex bradycardia with the heart rate dropping to 60 or 70bpm. When this, called the Cushing Reflex, occurs the animal is in imminent danger of herniating the brain and dying. This must be quickly recognized and treated to decrease the intracranial pressure. Hypertonic saline will improve cerebral blood flow and reduce neurotransmitter excitement. It can be combined with a synthetic colloid to increase the effects, as given alone it will only last for 30 minutes. 4ml/kg should be bolused if using 7.4% NaCl, 5.3ml/kg administered if using 3% NaCl. Mannitol can also be administered to combat Cushing's reflex given at 1.4g/kg IV. Any changes in blood pressure must be reported immediately and these patients closely watched for even minor mentation changes.

If at all possible head trauma patients should have their head elevated at a 30° angle (on a board, do not kink the neck with a towel) and never have any jugular catheters, bandages, collars, or any pressure around their neck. Oxygen support is recommended for these patients as either flow by or cage oxygen to supply as much oxygen as possible to the brain tissue. Nasal cannulas are not recommended in head trauma cases as the placement can be stressful and may cause sneezing. If these animals are profoundly sedate their eyes must be lubricated every 4 hours and monitored for signs of corneal ulceration. Oral care must also be performed, consisting of wiping the oral cavity with a wet gauze sponge to prevent bacterial buildup and potential pneumonia.

Recumbency changes should be performed every four to six hours using care to maintain the front half of the body in sternal recumbency to allow for appropriate chest excursion and prevent atelectasis. If the animal is minimally responsive, passive range of motion can be performed. Patients suffering from a seizure disorder and on anti-seizure medications are often polyphagic and may awake when offered food; if the animal is swallowing they should be offered food. As the patient begins to emerge from their sedation they can be vocal, ataxic, and unreasonable causing themselves injury. The nursing team may need to get creative to create adequate padding as these patients gradually wake up. TLC is important and can reduce anxiety as the animal slowly becomes more aware of their surroundings.

When the patient wakes up from the sedation of various anti-seizure medications, they will return home on maintenance medications.

- Phenobarbital is still a mainstay in seizure management and works on a wide variety of patients. 2-4mg/kg PO q12h in dogs and 2mg/kg PO q12h in cats can at first cause ataxia for the first 10-14 days. Liver values should be monitored for these patients, and phenobarbital blood levels should be measured (3-4 hours before the next scheduled dose) and the dose adjusted as needed to the lowest dose necessary to control seizures.
- Potassium Bromide can be administered at 20-30mg/kg PO q24h in dogs as an adjunct to phenobarbital or as a single drug for seizure control. It can take 16 weeks to reach therapeutic levels and must be given with a meal.
- Levetiracetam has a high safety margin but is cleared more quickly when given in conjunction with phenobarbital. Usually TID dosing (20mg/kg PO q8h) which is challenging for clients, but for patients >15kg there are now extended release tablets. This drug is not cleared by the liver and may be a good alternative drug for patients experience liver enzyme elevations on phenobarbital.
- Gabapentin is not a primary anti-convulsant, but can be added into a drug regimen to help treat refractory epilepsy at 10-15mg/kg PO q8h. The liquid formulation may contain xylitol and cannot be used in dogs or cats.
- Zonisamide can be added to phenobarbital or potassium bromide to treat refractory epilepsy but may cause hepatotoxicity. Liver enzymes must be monitored for patients on this drug. 10mg/kg PO q12h.
- Chlorazepate is a benzodiazepine and can be given between cluster seizures in an attempt to break the cycle of seizures at home or as an adjunct to phenobarbital or potassium bromide. The dose is 1-2mg/kg PO q12h, increasing the dose if the patient is on phenobarbital.

Client education is key with any seizure disorder. In most of these cases, the seizures will be a lifetime disorder to be managed and not cured. Owners should keep track of their pet's seizures – how often, how long they last, the character of the seizures, and the medication times. It can be helpful to veterinary staff if the owner can video the seizure or questionable activity to be shown in the hospital. Grand mal seizures are scary to witness and compassion must be exercised when consulting with these clients. Some seizure medications can cause temporary but profound mentation and mobility changes in animals. While these

effects are often short-lived, two weeks with an ataxic and mentally depressed pet can be stressful for owners. These owners should be warned of these changes and supported through this time. Expectations can be set and encouragement from the veterinary team may be important to some clients experiencing this for the first time. Education, compassion, and good nursing care can help many of these patients return to a relatively normal home life.

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# Canine Heatstroke: Keeping Your Cool

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Regardless of the climate, dogs run the risk of developing heatstroke. Even in cold and snowy regions, when temperatures begin to climb the cooling mechanisms of dogs may not be able to adapt appropriately and quickly enough to keep them from overheating. Heatstroke can be a deadly disease process that can affect all of the dog's major organ systems and requires quick recognition and aggressive treatment from the veterinary team. While cats can get heatstroke if trapped in a garage or attic, this discussion will be limited to dogs experiencing heatstroke as it is more commonly seen in canines.

Before beginning a discussion of heatstroke, it is important to note the difference between a physiologic fever (pyrexia) and heatstroke. Fever is the body's response to bacterial invasion or inflammatory injury and is necessary to the healing process. The hypothalamus in the brain controls thermoregulation by detecting when the animal needs to pant/seek shade or when the dog needs to start shivering to create heat. There is an internal set point in the hypothalamus at which these behaviors are triggered. In cases of infection or inflammation, the average body temperature set-point is increased, and the body will work to keep the temperature high despite external cooling efforts. Heatstroke is the result of external sources causing an increase in body temperature of which the dog cannot escape. Medical staff should be educated to the difference between heatstroke and fever and monitor and treat patients appropriately. In cases of actual fever, active cooling is discouraged; patients may be more comfortable with less bedding or by lying directly on the kennel floor.

Dogs dissipate heat through four main channels:

- Conduction – by laying on a cold surface
- Convection – air blows over their skin and cools the dog
- Radiation – they release heat into the atmosphere
- Evaporation – by panting they evaporate heat

In most situations, these methods are enough to keep the dog comfortable and functioning normally. If they can seek shade, they have access to water, their activity is limited in the heat of the day, or they can reside in an ambient temperature less than their body temperature, these dogs will remain comfortable and able to maintain an appropriate body temperature. Acclimatization will occur in dogs as temperatures warm with the season change. Their body will conserve higher quantities of water, retain salt; they will undergo plasma expansion, and increase cardiac output over a period of approximately 60 days. In humans, this process allows for an increase in sweat production and better cooling. This

acclimation period is essential to the body's ability to manage heat stress, and the reason why dogs might experience heatstroke in spring and early summer.

Heatstroke in dogs occurs when they can no longer dissipate heat efficiently, and their body becomes overwhelmed. If there are no cold surfaces, shade, wind, or available water the dog has no help with cooling. As the dog's body temperature rises, the mechanisms to actively cool require an increased metabolic rate and are creating more heat. Beginning with panting and quickly moving to vasodilation, increased cardiac output and increased mean arterial pressure the body attempts to remain at an acceptable temperature, but these mechanisms can also create metabolic heat. As the body temperature rises, heatstroke occurs, and without intervention, the dog can quickly progress to critical condition. In severe cases of heatstroke, all of the major organ systems are affected, and these patients need close monitoring and critical supportive care to survive. Core temperatures above 106°F can lead to permanent brain damage; above 109°F leads to organ damage. Body temperatures above 120°F can result in direct cellular damage.

Dogs experiencing heatstroke will have a history of heat exposure or extreme exercise in a warm environment, may have or are currently suffering from seizure activity, may be going through a laryngeal paralysis respiratory crisis, or experiencing increased muscle activity from hypocalcemia. These dogs will present to the hospital with a core body temperature >105°F (some as high as >110°F have been reported). Many of these patients will also experience decreased mentation, will have hyperemic mucous membranes, thick saliva, vomiting, and diarrhea, and even collapse and coma. Dogs more prone to heatstroke are the brachycephalic breeds, dogs with laryngeal paralysis, and those who have survived a heatstroke episode in the past.

When a dog is noted to be experiencing distress due to heat, active cooling should begin as soon as possible. Dogs need to be moved out of the sun, have water offered, and activity stopped. Owners at home should soak the dog with room temperature or even warm (never cold) water and run the air conditioner in the car on the way to the vet hospital. These early treatments will allow for evaporative and radiation cooling. Once at the hospital, cooling can continue by soaking the dog with room temperature water, ensuring that in long and double coated breeds the water reaches to the skin. A fan can be directed at the dog to assist in cooling, and the patient placed on a wet towel on a wet table or exam table. Do not cover the dog with wet towels as these towels can slow evaporative and radiative cooling. Never use ice packs against the skin of a dog when performing active cooling. Ice will cause local vasoconstriction and can delay the effect of conductive cooling. The practice of using alcohol on the pads of heatstroke patients is no longer a recommended treatment. Not only is the surface area of the paw pads relatively small when compared to the rest of the body, but used in large amounts can be toxic to the dog, and the nursing time and resources are better utilized performing more efficient and proven treatments.

It is important to stop active cooling once the dog's temperature reaches 103°F. At this point, dry the patient and cover them while continuing to monitor their body temperature. In severe heatstroke, the

hypothalamus is damaged and will not recognize when the patient's temperature is falling below normal, and will not signify shivering and other attempts to stay warm. It is not uncommon for heatstroke patients to experience hypothermia after cooling, and rely on the hospital staff to monitor their temperature and supply heat as needed.

Because all body systems are affected by thermal injury, the cooling process is only the beginning of treatment. A complete physical exam is performed on admission, and the patient monitored closely for decline or change. Baseline blood work including a CBC and full serum chemistry panel are conducted to assess organ function. The patient should be monitored for coagulation function and observed for changes in perfusion, mentation, comfort, and infection.

### **SHOCK:**

During thermal injury the body peripherally vasodilates, decentralizing blood flow to bring as much to the surface in an attempt to cool, and heatstroke patients can quickly experience hypovolemic shock. Evaporative cooling concurrently via panting can dehydrate the dog. Early in the process, cardiac output must increase to maintain blood pressure, but the dog cannot sustain this long term. As the heat situation becomes more critical, cardiac output continues to drop leading to decreased blood pressure and a further reduction in tissue perfusion. In other cases of hypovolemic shock, the dog will undergo vasoconstriction in an attempt to keep major organs perfused, but heatstroke patients remain vasodilated as they continue to try to dissipate heat. Every major organ system fails to receive appropriate blood flow and can suffer ischemic injury. As a result, we will see the clinical signs of shock in these heatstroke patients. Tachycardia, weak pulses, hyperemic mucous membranes and fast CRT (some may have progressed to pale mucous membranes), and reduced perfusion signal shock. Fluid therapy remains the mainstay of treatment for shock with crystalloids administered to clinical endpoints (decreasing tachycardia and increasing blood pressure) and the nursing team providing continued monitoring for patient improvement. Colloids may be used concurrently with crystalloids to maintain appropriate blood pressure and oncotic pressure. Hospitals with access to blood products may consider plasma and albumin products for life-threatening cases.

### **NEUROLOGIC SYSTEM:**

The intense heat experienced by heatstroke patients can cause cell rupture, at core temperatures of 120°F cellular necrosis occurs. Cellular death leads to edema, and cerebral edema manifests as mentation changes in the patient. Heatstroke patients often present collapsed, but they can be stumbling with ataxia, mentally inappropriate, or even present to the hospital with seizure activity. Reasons for mentation changes can range from poor cerebral perfusion, cerebral edema, direct thermal damage or hemorrhage. Decreased blood glucose levels can lead to seizure activity and should be checked to rule out hypoglycemia. Appropriate treatment with mannitol (0.5 – 1 gram/kg slow IV) considered if the dog's mentation fails to improve with treatment. Dogs that suffer from heatstroke should have their mentation evaluated often and close attention paid to declining changes.

## **GI SYSTEM and SEPSIS:**

A combination of direct thermal injury to tissues and reduced perfusion to the gut can cause GI ulceration and often vomiting and diarrhea. In many cases, evidence of intestinal sloughing is seen in diarrhea. Breakdown of the gut mucosal barrier can quickly lead to gut-derived sepsis. Broad spectrum antibiotics should be considered in patients that present with heatstroke and hematochezia and hematemesis to treat the onslaught of bacteria. Blood glucose levels should frequently be monitored, and hypoglycemia addressed as needed. With sepsis often comes hypotension requiring the nursing team to watch closely for hypotension.

## **SIRS:**

The increase in body temperature experienced in heatstroke triggers both a pro-inflammatory and anti-inflammatory reaction in the body. This response can increase gut permeability but also puts the patient at risk for developing SIRS (Systemic Inflammatory Response Syndrome) as the body's systemic response to an inflammatory focus. SIRS and sepsis in heatstroke patients can occur concurrently. Dogs with an inflammatory insult are at risk for developing SIRS if they have two or more of the following parameters:

Tachycardia (>120bpm)

Tachypnea (>20bpm)

Temperature (>103.5°F <100°F)

CBC (Neutrophils >18k <5k or >10% band cells)

SIRS leads to a loss of vascular tone creating blood pressure challenges and poor organ perfusion, disturbance of the endothelial permeability barrier and can stimulate inappropriate coagulation. Cytokine release can lead to coagulation in the microvasculature and contribute to organ failure. In severe heatstroke, SIRS and sepsis can overwhelm a dog very quickly, and the medical team needs to be aware of this and monitor blood glucose levels, supplement with IV dextrose as needed, provide antibiotics, and monitor WBC. Barrier nursing must be implemented in these patients to prevent secondary infections, and care taken with bedding and medical equipment used.

## **COAGULOPATHY:**

Coagulopathy is common in heatstroke patients. As mentioned previously, SIRS activates the clotting cascade, and clotting factors consumed. Hemorrhage can occur and can be catastrophic. Direct thermal injury to the endothelium and liver from extreme heat can also lead to inappropriate bleeding and the dog's inability to appropriately replace clotting factors. Once the clotting factors are consumed, it is common to see petechiation, ecchymosis, hematochezia, hematemesis, and bleeding from injection sites. Clotting times should be measured and monitored and the patient treated with plasma and PRBC as needed. Platelet counts should also be performed and rechecked as these will often drop as well.

## **HEPATIC SYSTEM:**

With body temperatures as high as are seen in severe heatstroke cases, the liver often suffers direct thermal damage. These dogs cannot rebound from clotting factor losses due to this damage. Liver enzyme elevations are common and should be monitored. Some patients may become icteric as total bilirubin levels rise due to red blood cell breakdown. In these cases, an abdominal ultrasound may be necessary to rule out any surgically correctable reasons for elevated total bilirubin (such as sloughing tissue causing gall bladder obstruction).

## **KIDNEYS:**

As blood shifts from the dog's core to the periphery to cool, the kidneys experience a decrease in perfusion. As the dog continues to dehydrate while attempting to cool and experiences severe hypovolemic shock, the kidneys begin to undergo reduced perfusion related damage. Azotemia will be evident in lab work. Inappropriate bleeding from lost clotting factors can also cause bleeding into the kidneys. Urine output is necessary to monitor in these patients, and measures taken to ensure that they can process the fluids they are receiving. These patients should be weighed multiple times per day to monitor for weight gain.

## **CARDIAC SYSTEM:**

Ventricular arrhythmias can occur in heatstroke patients as the result of hypovolemia, hypoxia, direct thermal injury, ischemia, or reperfusion injury. ECG monitoring should be a part of heatstroke patient management and arrhythmias treated as needed. If the patient is suffering from perfusion deficits or blood pressure changes due to the arrhythmias, treatment with lidocaine, procainamide, oxygen therapy, pain management, and electrolyte monitoring/supplementation can be considered.

## **NUTRITION:**

Because the GI tract takes such a hit in severe thermal injury, nutrition can be a challenge in these patients. Protein levels can drop leading to decreased blood pressure, peripheral edema, and increased gut permeability. Feeding the gut is an important part of the healing process and should be started early in the treatment. If possible, trickle feeding through an NG/NE tube should be instituted as soon as the patient is not vomiting. Due to reduced blood flow to the gastrointestinal tract, protective medications such as sucralfate, omeprazole, and pantoprazole should be considered.

## **PAIN:**

Pain management should not be neglected in the heatstroke patient. Sloughing gut, bleeding into organs, and prolonged recumbency are all sources of pain. These patients should be treated with opioids as needed to keep them comfortable during their hospital stay.

Heatstroke is a syndrome that requires quick and knowledgeable action from both the owner and the veterinary team. Multiple organ systems can be affected and need close monitoring. Nursing care is extensive and requires knowledge of what can occur, and open communication between teams is necessary. With excellent nursing care and supportive care, it is possible for these patients to recover and return to normal lives.

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# Emergency Anesthesia: Planning for the Unexpected

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Critical anesthesia can be a challenging time for the anesthetist. Depending on the procedure and the patient, the time can go smoothly or it can be the roughest 90 minutes of the day. Preparation and thinking ahead can save stress and help the team cope with anesthetic emergencies as they come up.

The first step to any anesthetic procedure is preparation. Even in emergency situations when the procedure needs to happen immediately, do not skimp on preparation. Start with the equipment. Take the time to make sure the correct anesthesia circuit is connected and that a leak test of the anesthesia machine has been performed.

As the anesthetist, understand the clinical effects of the medications given to the patient. The approach to bradycardia under dexmedetomidine will be different than a patient with bradycardia under diazepam/ketamine. Induction with propofol can lead to apnea and/or hypotension whereas induction with an opioid alone can have minimal effect on the cardiovascular system. Be aware of all medications administered and the reversal agents if necessary. Remember that pre-medication will also affect the patient's vital signs and must be considered when monitoring.

It is also important to make sure the patient is prepared. Obtain a full set of vital signs immediately before anesthesia induction. While this is happening, pre-oxygenate the patient. The intubation process is a hypoxic event for the patient; by pre-oxygenating the patient is better prepared for this process. Be aware of any complications that can/may occur during the procedure and discuss those potential complications with the team. Know doses of emergency drugs and fluid bolus doses. Will the patient need blood products? Will the patient need additional IV access? Thinking through potential problems and being prepared for them will save time when immediate action is needed in an emergency.

Checklists are utilized in the human medical world and can also be used in the veterinary surgery suite. While it may seem silly to run through a checklist on a puppy neuter, it is quite helpful to run that checklist with the team prior to an emergency splenectomy. By practicing and communicating on each and every procedure, mistakes are decreased. Take the checklist time to discuss potential complications and how the team wants to handle those. These checklists can also allow for a review of sterilization procedures, the care of instruments, and any problems with equipment.

## Heart Rate

Tachycardia (heart rate >140 in dogs; >200 in cats) can be the result of a variety of causes. The treatment for each cause will vary greatly, so it is important to quickly determine the cause so the correct treatment is applied. Is the patient tachycardic due to improper anesthetic depth? Surgical manipulation? Hypovolemia? Hyperthermia? Hypoxia? Anemia? Drugs administered? Quickly run through all of the potential causes for tachycardia and choose the correct treatment. To do this, check your equipment and then look at other vital signs. Remember that no vital sign measurement is in a vacuum; always look at the big picture. How is the blood pressure? The SpO<sub>2</sub>? Look at the patient – how well are they perfusing? What is the mucus membrane color and CRT? Talk to the surgeon – how do things look in the abdomen? Are the surfaces dry and tacky? Is there more blood than expected? If the cause of the tachycardia can quickly be determined it can be quickly corrected – with oxygen, blood products, a crystalloid fluid bolus (usually 5-10ml/kg depending on the patient and disease), adding a colloid, increasing the inhaled anesthesia, or administering pain medication. In many patients the problem can be corrected with a combination of factors. In some cases, administering an additional dose of pain medication will allow for less inhaled gas.

Bradycardia (heart rate < 60bpm in dogs; < 100bpm in cats) and can be the result of a deep plane of anesthesia, disease process, hypothermia, or drugs administered. It is important not to treat just a number, but to get the entire picture. How is the patient perfusing? Is their blood pressure maintaining? Bradycardia can be addressed with anticholinergics (atropine, glycopyrrolate) and monitored closely after those drugs have been administered. Atropine (faster onset, shorter acting) dose is 0.005-0.01mg/kg in dogs and 0.02-0.04mg/kg in cats. Glycopyrrolate (slower onset, longer lasting) dose is 0.005-0.01mg/kg in dogs and cats.

## ECG

Be familiar with the common arrhythmias seen and how to treat them. If you are working with a patient where the probability for arrhythmias is high, educate yourself on what they look like. Note when the arrhythmia started and watch the patient's heart rate, blood pressure, and perfusion status. Treatment may be necessary if the blood pressure or perfusion status becomes abnormal. Be familiar with which arrhythmias can occur with which surgical procedures and be ready with any necessary treatment.

## Respiratory

Tachypnea can be seen in patients mainly due to improper levels of anesthesia (too light), hyperthermia, and hypoxia. Look at other vital signs (such as heart rate and blood pressure) to help determine the cause. In most cases tachypnea can be corrected with a change in anesthetic depth or a dose of pain medication.

Bradypnea is often caused by anesthetic depth too deep, drugs given, patient status (obesity), patient position, disease process, hypothermia, and severe hypotension. Some of these problems the anesthetist will have no control over, but thinking ahead to what can be done to alleviate some of the problem can be helpful. Again, look at other vital signs and perfusion status when looking to correct this problem before making changes to anesthesia.

SpO<sub>2</sub> or pulse oximetry measures patient oxygenation. Hypoxemia is low oxygen content in arterial blood; hypoxia is low oxygen in tissues due to poor perfusion. Both can cause changes in the SpO<sub>2</sub> readings and need to be addressed immediately. SpO<sub>2</sub> measures the percentage of hemoglobin saturated with oxygen and requires a certain level of perfusion to read in the periphery. Looking at the oxygen hemoglobin dissociation curve, a SpO<sub>2</sub> in the low 90% range means a dramatic drop in PaO<sub>2</sub> readings, which means it is important not to let SpO<sub>2</sub> readings drop below 95%. The probe may need to be periodically moved to allow appropriate perfusion to the area. Check gum color, respiratory rate, and heart rate to help interpret readings.

End tidal CO<sub>2</sub> is the measurement of carbon dioxide in each exhaled breath. Carbon dioxide is the gas that drives respiration. Our patients inhale because the respiratory center in the brain detects higher than normal levels of carbon dioxide in the blood. If carbon dioxide levels get too high, the respiratory rate will increase to exhale more CO<sub>2</sub>. If levels get too low, the respiratory rate decreases to conserve CO<sub>2</sub>. In normal patients without lung disease or metabolic disease, this process is sufficient to keep CO<sub>2</sub> levels normal. When a patient is anesthetized, the drugs used can decrease the ventilatory drive in the brain and relax the intercostal muscles which can cause changes in ETCO<sub>2</sub>. Changes that, because they are anesthetized, the patient cannot change on their own. End tidal CO<sub>2</sub> is a measurement of patient ventilation and gives a more complete picture compared to using SpO<sub>2</sub> alone. Drugs administered, patient positioning, disease process and depth of anesthesia will all affect ETCO<sub>2</sub> reading. ETCO<sub>2</sub> is the result of not only ventilation, but also blood flow, cellular metabolism, and alveolar ventilation. In order for CO<sub>2</sub> to make it out of the lungs and into the capnometer, the patient must be perfusing cells and transporting CO<sub>2</sub> back to the lungs to be exhaled. ETCO<sub>2</sub> is reliant on ventilation and *perfusion*. It is also an instantaneous result, giving the anesthetist up to the minute results of what is happening with the patient. ETCO<sub>2</sub> is also a clue as to the patient's perfusion and circulation. Decreased cardiac output can lead to decreased ETCO<sub>2</sub>. The patient continues to ventilate, exhaling CO<sub>2</sub>, and if perfusion decreases there is less CO<sub>2</sub> being brought back to the lungs to be exhaled. A rapid drop in ETCO<sub>2</sub> is cause for alarm, as this can signify impending arrest. Changes in ETCO<sub>2</sub> readings may be related to problems beyond the respiratory system; be sure to examine other monitoring parameters when troubleshooting abnormalities.

- An elevated ETCO<sub>2</sub> (>45mmHg), or hypercapnia, signifies that the patient is hypoventilating. Common causes for this include: too deep a plane of anesthesia, an airway obstruction, pneumothorax, body position of the patient, and disease process (remember that obesity is a disease, especially when we place those patients in dorsal recumbency). To correct hypercapnia, increase the patient's respiratory rate until the ETCO<sub>2</sub> reaches a normal level, and adjust

anesthesia as needed. Troubleshooting the patient may be necessary if a pneumothorax is present or the patient is not responding as anticipated. If left untreated, hypercarbia can cause central nervous system depression and eventually acidemia.

- A decreased ETCO<sub>2</sub> (<35mmHG) or hypocapnia, signifies that the patient is hyperventilating. Common causes for this include: too light a plane of anesthesia, pain resulting in tachypnea, panting, pronounced hypothermia, decreased cardiac output, or excessive dead space in the anesthetic circuit. To correct hypocapnia, pain management or deeper anesthesia may be required to allow a lower respiratory rate, as well as monitoring other vital signs (such as temperature). Further troubleshooting may be necessary if the patient is not responding as anticipated.

## Blood Pressure

A blood pressure reading gives an idea of how well the body is circulating blood and perfusing organs. Blood flow equals oxygen flow, and oxygen is vital to life. Hypotension means the patient is not receiving adequate oxygen delivery to their organs, and if hypotension is persistent that equals cell death and eventually organ dysfunction. Shock is the result of inadequate oxygen delivery to tissues and your ability to recognize the signs of shock in your patient may alert you to a crisis before you get a low blood pressure reading. The causes of shock are numerous, but the common clinical signs are tachycardia, pale mucus membranes, prolonged capillary refill time, poor pulse quality, and cold extremities.

- Hypertension (systolic >140mmHg, MAP > 110mmHg) is usually a sign that anesthesia isn't deep enough; hypertension can be treated by increasing inhaled gas or providing more analgesia. It is important to think about the patient and whether or not they have a disease process that can be causing hypertension. Increased intracranial pressure (especially in trauma patients), chronic renal failure and metabolic diseases can cause hypertension.
- Hypotension (systolic < 80mmHg, MAP <60mmHg) is a common occurrence in anesthesia, as many of the drugs used to induce and maintain anesthesia will cause hypotension. Hypothermia can contribute to low blood pressure and is common in small animals under anesthesia. Look at blood pressure and heart rate together, as the heart rate will give clues as to why the blood pressure is abnormal. Determine the cause of the hypotension and quickly work to improve perfusion (look for reasons for decreased cardiac output, vasodilation, hypovolemia, hypothermia, or too deep under anesthesia) as the patient can suffer long term effects of prolonged hypotension. Appropriate pain management is important to allow for reduced gas anesthesia. The ability to increase or decrease a CRI of an opioid (or combination) can allow for fine tuning of inhaled anesthesia and better blood pressure readings.

When faced with a hypotensive patient it is important to begin treatment as soon as possible and search to find the underlying problem. The mainstay of hypovolemic (the most common) hypotension treatment is fluid therapy. Relatively easy to start and monitor, every veterinary clinic has the supplies necessary for treating hypotension with IV fluids. Goals are set (reduction in heart rate, increase in blood pressure) and fluids are administered until endpoints are reached. Crystalloids, in the form of balanced electrolyte

solutions, are administered first, generally starting with a 5ml/kg bolus. Crystalloids will provide a quick bump in intravascular volume, but remember that they will shift out of the intravascular space and into the interstitial space about 30 minutes after administration. Remember this when monitoring a hypotensive patient – one normal measurement does not equal fixed, if fluid shifts are occurring the blood pressure may drop again and needs to be monitored continuously. Colloids are made of larger molecules than crystalloids and will remain in the intravascular space longer than crystalloids. They will also help to draw fluids towards them thereby increasing intravascular volume. Blood products are also colloids. When dealing with normovolemic hypotensive patients colloid therapy becomes more common. Patients that are hypoproteinemic may also be on colloid therapy as a way to decrease edema. Colloids can be administered as a bolus (5ml/kg) with a maximum daily dose of 20ml/kg/day.

With some patients, their hypotension is not caused by a loss of fluid or blood, but by a systemic illness. Septic patients can be hypotensive and normovolemic, and correcting the hypotension with fluid challenges can be detrimental to their recovery. SIRS, anaphylaxis and cardiac disease patients can present the same challenge when treating their hypotension and their condition is important to keep in mind when treating.

## **Temperature**

Hyperthermia is uncommon under anesthesia but can exist due to infection, inflammation, and drugs given. Opioid administration in cats can cause dramatic (but transient) increases in body temperature. Malignant hyperthermia is a rare genetic disorder that causes muscle tremors and dangerously high temperatures. Hyperthermia can increase heart rate and cause vasodilation and hypovolemia if it goes untreated.

Hypothermia is probably the most common complication encountered with general anesthesia. Hypothermia can decrease the inhaled anesthetic needs of the patient; this should be top of mind in longer procedures where the patient continues to get colder. Hypothermia can also cause ECG abnormalities, decrease coagulation, inhibit platelet function, and eventually can be the cause of death. Upon recovery, hypothermia can prolong the recovery process and cause shivering which will increase the metabolic oxygen needs. Prevention is the best medicine for hypothermia and should remain on the list for the cause of intraoperative anesthetic complications. Wrapping feet in bubble wrap, warming the chest and head of the patient (assuming the surgery is abdominal), using warm abdominal lavage, and warming the IV fluids are all methods for warming or keeping patients warm. One of the more effective ways to accomplish this is to warm the inhaled air of the patient. Fresh oxygen is ice cold; placing a warm water bottle over the anesthesia circuit can be helpful.

## Pain Management

Pain management must be part of any anesthetic protocol and tailored for the specific patient's needs. Proper use of pain management can allow for lower doses of inhaled anesthetics leading to a more balanced anesthetic protocol. With the use of constant rate infusions, some critical patients can be maintained solely on injectable medication. Total Intravenous Anesthesia (TIVA) can be incorporated in critical patients that cannot tolerate inhaled gas anesthesia or in patients where inhaled anesthesia can be a danger to the surgical team (like a traumatic lung lobectomy). Utilizing constant rate infusions to maintain TIVA is preferable to intermittent bolus of medication to maintain a constant plane of anesthesia. Propofol and alfaxalone are common drugs utilized in total intravenous anesthesia and are used for both induction and maintenance. In critical patients, TIVA may be an option for a patient with hypotension or excessive depth of anesthesia on inhalant gas. Anesthetic depth changes happen more slowly with TIVA than with inhaled agents and drugs can accumulate in tissues over time which can prolong recovery.

Full mu agonist opioids (morphine, methadone, hydromorphone, oxymorphone, and fentanyl) are the strongest analgesic drugs available. Opioids have minimal effect on the cardiovascular status of the patient making them safe to use even in the most critical anesthetic patients. Fentanyl is popular in CRI form as it has a short half-life and rates can be adjusted quickly according to patient needs. CRI rates can range from 2-10mcg/kg/hr and can be increased up to rates as high as 30-100mcg/kg/hr as needed for total intravenous anesthesia. Respiratory depression will occur with high rates of opioid administration which can be managed with mechanical ventilation. These patients must be monitored closely on recovery as respiratory depression can last past reduction of drug rate.

Ketamine is a NMDA receptor antagonist and can be used for pain management especially in chronic pain, trauma pain, and orthopedic procedures. Patients experiencing breakthrough pain on an opioid CRI can experience relief with the addition of ketamine as a CRI. A loading dose of 0.5mg/kg IV should be administered and the CRI administered at a rate of 2-10mcg/kg/min. Ketamine is also used for TIVA protocols.

An emergency is defined as a serious, unexpected, occurrence requiring immediate action. As anesthetists, the best we can do is to remove the 'unexpected' from the definition. Think through the patient history, the disease process, and the procedure to determine the risks to the patient. Plan your response to potential emergencies by placing an additional IV catheter, having a unit of blood on hand, and knowing how to do a CRI calculation. There is no substitute for preparation and critical thinking, and with knowledge and planning comes successful anesthesia.

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## Lisa A. Yackel, CVPM, PHR, SHRM-CP

Over 40 years ago, Lisa started her career in the kennel. She went on to become a non-certified technician and then a Practice Manager. She became one of the first CVPMs in 1994 and a Professional in Human Resources in 2004. For 26 years, Lisa was the Hospital Administrator of Case Veterinary Hospital in Savannah, Georgia. She is now a part time consultant, speaker and online instructor writer. Lisa is passionate about helping those in this wonderful career grow and thrive. She was a CVPM Board member for fifteen years and still serves on committees for that organization.





## Stay & Grow

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Lisa A. Yackel, CVPM, PHR, SHRM-CP

For a new plant to grow, it must be planted in a “rich” environment to start with and then given water and fertilizer on a regular basis. If you feel you have landed in a good environment, it is worthwhile to take the time to allow “water and fertilizer” to strengthen your root system and allow you to thrive.

Most supervisors and managers in a veterinary hospital are very busy. Whether they are managing seven people or fifty people, they are in a constant balancing act to keep the practice profitable, the clients happy, and the team engaged. When a manager hires you, they see something in you that seemed right for the practice. They truly want you to succeed. In fact, they want you to thrive. But, you are only one of their many responsibilities and, too often, they don’t have the time to dedicate to you that they want to.

Here are some suggestions to keep you on their radar (in a good way!):

- Find out their schedule (days off, standard meetings they attend, etc.)
- Find out if they like to receive information by e-mail, the inter-office messaging system, by text, etc.
- Review the Organizational Chart if there is one in the Hospital Manual. If you have been assigned a trainer, ask for feedback from them after the first week. **First impressions do count.**
- Don’t wait too long to solicit feedback on how you are doing! Ask if you can have a scheduled 10 minute meeting at their convenience to review your progress. Do this on a weekly basis. This will equate to 12 short meetings before your actual 90 day Introductory Period Evaluation. The familiarity you will have established will be priceless.

### Evaluations:

Don’t view the evaluation process as a dreaded event. It has been proven that productivity increases when it is monitored. We don’t grow unless our limits are pushed and most of us resist attempts by anyone who tries to push us further than we want to push ourselves. These meetings are setting a foundation of trust and understanding between you and your boss. Utilize the time to really learn what is expected of you and, in turn, let him/her know what value you bring to the hospital. Employees need formal feedback just as people do in all relationships.

Whether for your first 90 Day Evaluation or for ongoing reviews, the following are some additional plans to utilize.

- Be on time (hopefully, it will already have been established that you are always prompt and reliable for your shifts)
- Turn your cell phone off or, better yet, leave it in your locker.

- Always bring a copy of the goals set at the last review and a summary of whether these have been accomplished. If for some reason, they have not been fully completed, have an answer for why and a plan for getting it done in a new time frame. Be sure that you do not “throw anyone under the bus” and that your reasons are factual and presented in a non-emotional way.
- Ask your supervisor/manager, “What can I do to help you (the hospital)?”
- Keep in mind, your awareness of your performance is more critical to your development and improvement than the manager’s awareness of your performance. You need to be in tune with where you are and how you are doing before you come to the evaluation. This is called “self-evaluation”. Not only is it critical to a successful evaluation session but it is a helpful technique to use in life in general. Those with high Emotional Intelligence know the value of self-analyzing.

There are other tips that will help you succeed. Set quarterly goals, check your progress monthly and make a plan for how you’re going to stay on top of them. While this won’t necessarily get your successes noticed, it will help you be more aware of them so you can know what to share with other people. A preferred method that has proven helpful is to lay out ahead of time what you want to achieve so you can measure your success against those goals. This will make sure you have clear benchmarks to track and milestones to celebrate.

Another tip is to start a Career Journal and at the end of every day, quickly jot down what went well and what you’re looking forward to tackling tomorrow. Capture your day-to-day successes, while also keeping your progress in the context of larger projects and how you’re helping your organization move forward. Becoming familiar with your job description is critical. For each duty, say to yourself, “I know I am doing this well because...” and list a specific example that illustrates your success. If you’re hoping to move up in the hospital, you can also do this exercise with the job description of the title you want.

Remember, there is no substitute for hard work. On the other hand, hard work won’t do you any good if your accomplishments are going unnoticed. Most of us want to believe that our work should speak for itself, but, unfortunately, that is not always the case. The idea of tooting your own horn may sound obnoxious and bragging, but it can be done in a constructive way that catches your boss’s recognition. Send a quick email to your supervisor or manager when you accomplish your first set of dental x-rays on your own, forward an e-mail from a client who compliments you, take a picture of how you reorganized the drug closet and e-mail it. It will be uncomfortable at first, but it will be very beneficial to keeping you in a positive light in your manager’s eyes.

## **12 Ways to Make Yourself Invaluable**

1. Be low maintenance
2. Treat your job like it’s your lifelong career, even if it is only a stepping stone
3. Cultivate a sense of ownership of the hospital
4. Become the most reliable person in the hospital
5. Own your mistakes-make accountability one of your own core values

6. Be a professional in all that you do
7. Take charge of your own destiny. Look for opportunities to take on
8. Become a problem solver-be part of the solution not the problem
9. Always be on a quest to learn
10. Practice always being courteous, respectful, and kind to team-members, clients, and the pets in your care.
11. Be Genuine
12. **Be Passionate in all that you do**
13. If you don't agree with something or if you think you have a better way of doing something—speak up! **But** do so assertively and politely, not loudly or disrespectfully. Employees who do this are sure to get recognized and rewarded. Employees who just do their job and nothing more, are telling the employer that is all they want. And most likely, that is all they'll get.
14. Many employees feel as though they are “above” a given task and voice their complaints. They fail to consider that if those tasks did not exist, they would not have jobs. Keep in mind that if your motto is, “That’s not my job,” you may soon find yourself *out of a job!*
15. Being successful and productive at your job doesn't only make you happier, but it also helps motivate others around you. While we may get annoyed at coworkers who seem to have everything go their way, often their success reflects their characteristics.
16. Be introspective. It can be easy to see the strengths and weaknesses of other people you work with, but it's always harder to critique ourselves. This is where that Accountability Partner and/or your mentors can really help.
17. Employees who are successful at what they do are more likely to have work-related and non-work related conversations with people around them.

**Become the “go to” person.** In every business, there always seems one person that everyone goes to if they have a question. This is especially true in a veterinary hospital. It may be the person who has the most longevity or it may be the “boss”. It doesn’t have to be either one. Those who avail themselves to helping others succeed and who make it a point to learn all areas of the hospital, truly are treasures. Here are some tips to help you become that person:

- It is human nature for all of us to want to do well when we feel appreciated. Be the person who says thank you often and with sincerity. Show gratitude when others help you and earn the reputation that you are happy to be able to work in a hospital that allows you to be doing something you love. Gratitude is contagious and makes others feel good. Who doesn’t want to be around someone who is joyous about what they do? (By the way, this works well with clients also😊)
- Dress for the job. Ok, how do you dress up scrubs? Even scrubs have levels of professionalism. Be sure they are neat and unwrinkled. Keep a change in your locker for those days when you get anal glands on you (your team will doubly appreciate not having to smell you all day). Emulate

your manager's attire when it comes to shoes, hair, jewelry, etc. This doesn't mean you have to become a clone, it just means you are taking cues on the image that the hospital wants to promote.

- Respect other's values and be sure not to prejudge or interrupt when they are sharing their thoughts.
- The veterinary industry is fast paced and ever changing. Those who do not make a conscientious habit of seeking continuing education will find themselves losing their value to the hospital very quickly. Many hospitals have formal programs that are assigned to you (this course may be one of those programs). Hospitals that understand the value of their employees as one of their most valuable assets, will often set a budget for each employee education. Take advantage of any opportunity you can. If you are a CSR or a kennel worker in your hospital, you may not be offered a stipend for CE. Don't let this stop you. Read the periodicals that are lying around the hospital (Trends, DVM360, etc). Go to the library and read up on customer service (for the CSR) or how to read a pet's body language (for the kennel worker). Show an interest in taking some of the many offerings within the veterinary industry on the internet. Any time you learn something of value, offer to share with your teammates at a staff meeting or through your hospital newsletter or other form internal communication. If you are comfortable writing, offer to write a blog for your hospital or post a tidbit on Facebook. If you learn a new skill on the computer, an easier way to take a dental x-ray, etc., be sure to share with your coworkers.
- The veterinary industry is one of constant change. The employee who excels in a veterinary hospital is one that embraces change and is always growing.
- In today's economy, if a business isn't learning, then they are going to fall behind. And a business learns as its people learn. You and your teammates are the ones that produce, refine, protect, deliver and manage your products or services every day, year in, year out. With the rapid pace of today's business changes, continual learning is critical to your hospital's continued success.
- A hospital that has created a learning culture begins by clearly communicating their expectations that employees should take the steps necessary to hone their skills to stay on top of their professions. These hospitals who aspire a true learning environment will have an edge against their competition and will be sure to support their efforts in this area by supplying the resources you need to accomplish this goal. Secondly, they will communicate to you the specific training needs and targeted results they wish to accomplish.
- If you are lucky enough to have landed in this environment, take advantage of every opportunity offered to you. The veterinary field can be an exhausting one but look around at those in the field who you see as being successful. It is almost a guarantee that they worked hard to get where they are by self-sacrifice of their precious personal time to make sure they attended seminars, read books, and took advantage of any continuing education they were offered.

## **Perpetual Growth**

The definition of perpetual growth as an adjective is as follows:

1. continuing or enduring forever; everlasting.

2. lasting an indefinitely long time

3. continuing or continued without intermission or interruption; ceaseless

Highly valued employees take responsibility for their own advancement and create the truest form of job security by becoming indispensable to the hospital. Seize opportunities when they present themselves and never complain that there are no opportunities out there. You and only you have the power to your destiny. In even the best run veterinary hospitals, there has been some project that has been abandoned, some opportunity that hasn't been developed, or a new technology or advancement that hasn't been pursued. Maybe the hospital's OSHA plan is outdated, or there is frustration because no one knows what is going on due to a lack of communication, or becoming a Cat Friendly Practice has been discussed but no one has volunteered to spearhead the job. Take the initiative to volunteer to organize the work to get these projects off the ground. Come up with a SMART plan (program handout) and present it to your supervisor and/or your Practice Manager. Be sure to include ways you can accomplish this without your primary job being compromised.

Be smart about what you choose. It should be something that will add value to the hospital (financially or with better systems to help the efficiency of the hospital). It should also be something that you can stay enthused about in the long run as your passion will be the motivation that sees this venture into success. Remember, in order to succeed, your desire for success should be greater than your fear of failure.

Some people will insist that it is not possible for them to make a difference. These are not the employees who will be successful in a veterinary hospital. These are not the employees who will stand out to their managers and who will advance their position in their job. You don't have to solve huge problems to make you valuable to the hospital. You just have to be willing to take ownership of a problem and try to come up with a solution.

In summary, the best way to create job security and to build a career is to remember that the driving force must come from you, not your employer. The surest way to achieve job security, advance in your position, and to make more money, is to become more valuable at work.

Stephen Covey's fifth habit of highly effective people is seeking to understand the other person before expecting him to understand you. From day one of employment, the relationship you develop with your supervisor/manager will be the most important relationship you will have on the job. Ensuring that communication is ongoing will help with any miscommunication and problems that may arise. Learning your supervisor's values is critical to helping you become a stand out team member. In a hospital where the culture is very developed, the hospital will have a written set of Core Values. Start with these values and observe how they are carried out by the supervisor and by your teammates.

Of course, there are occasions where you find your values and the hospital's values, clash. When this is the case, it will be very difficult to thrive in this type of environment. Certainly, this will be a discerning time to analyze if you truly are in an atmosphere that will allow you to grow and thrive.



# Is Management In Your Future?

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Lisa A. Yackel, CVPM, PHR, SHRM-CP

There is one question that is pivotal to ask yourself if you are a technician that wants to move into management. That question is- “Do you like encouraging, and teaching others?”

As veterinary hospitals have gotten larger and the demands on the practices have increased, the need to have layers of management and more delegation of duties has also increased. When an Organizational Chart is done correctly and there is strategic planning, hospitals benefit from empowering the right people to grow into positions of leadership and coaching. In fact, the role of supervisor truly becomes an integral part of the success of the hospital and the growth of the team.

Do you have what it takes? Below are some points to consider:

1. A skilled supervisor is an effective communicator, problem-solver and employee motivator.
2. To be a good leader and get the most out of all your employees, you need to have insight into their strengths and developmental needs.
3. You also need to have a clear set of expectations and goals.
4. Being a supervisor or manager is not necessarily the right fit for everyone.
5. Often those in management promote people because they are exceptionally good at what they do, a technician that has great technical skills, a CSR that is wonderful with clients, a kennel assistant who has phenomenal animal handling techniques.
6. While these skill sets are attributes that are of value, they don't necessarily mean that they make for good supervisory material.
7. Not only should a supervisor have great technical skills in their area, but they should have managerial skills, human skills, conceptual skills, problem solving skills, and political skills.

## So, what makes a good supervisor?

- **Has great communication skills:** As a supervisor one must communicate clearly and correctly to avoid misunderstandings and frustrations
- **Adapts to the changes:** Veterinary medicine as well as the business of running a veterinary practice is always changing. Being able to think outside the box is required as adjustments are ongoing to meet the needs of the organization.
- **Values the employees:** The team is any supervisor's real asset. They are ones running the business and the work. A good supervisor understands their worth and treats them accordingly.

- **Is a coacher/mentor:** A good supervisor shares her wisdom, knowledge and experience with the employees. Working alongside the team one is supervising and being their advocate strengthens the bond and the trust between them.
- **Understands that discipline is sometimes necessary:** A supervisor must set a behavioral code, often by example. There will be times when discipline is necessary.
- **Gives feedback/incentives:** Often a supervisor is the link in ensuring the individuals on the team are rewarded when they are deserving of a promotion, raise, feedback, etc. The supervisor is the ideal person to give accolades in a timely manner.
- **Leads by example:** Team members love a supervisor who works beside them and is hands on. They admire one who does not always delegate but often picks up some of the less attractive or uninteresting jobs. Setting an example to all the team about taking up challenges and getting the work done is important.
- **Is approachable:** The team should feel comfortable approaching the supervisor with their concerns and problems. An effective supervisor will make sure that there is enough trust and openness between her and the employees for the latter to come to her with their grievances.
- **Is considerate:** People are not just employees. They have families, friends and a life beyond work. A good supervisor will treat the team with fairness and try to ensure the workload is distributed appropriately so that there is balance.
- **Maintains a positive attitude:** A good supervisor sets the pace by maintaining an upbeat and encouraging demeanor. Always treat the team with respect and caring. Be polite, wish team members good morning, inquire about their families, and be generous in thanking them.
- **Can criticize constructively:** When mistakes happen, a good supervisor tries and understands the reasons behind the mishap. She criticizes or assesses the employee in proportion to the mistake. It is essential to the relationship to be able to give constructive feedback; showing them the right way to do things.

A common occurrence in a veterinary hospital is that many supervisors are promoted from the team and their former coworkers become subordinates. This can often be one of the most awkward situations you can encounter in your professional life. Bonds are often strong in veterinary hospitals between the employees as they are together long hours and in emotional situations. Going from being “one of the guys” to the “boss” can be one of the most difficult hurdles to overcome when getting a promotion to supervisor.

New supervisors often fall into the two extremes. They either try to remain buddies with the team or they allow the position title to wield power that is alienating and harmful to the team. You are no longer privy to the snarky jokes or inside gossip about what’s going on in the organization – you’re now *the subject* of said comments, jokes, and gossip.

Psychologist Abraham Maslow argued over 60 years ago that a sense of belonging is one of the most basic human needs, right after food, water and safety. It is safe to say that we need friends in our professional lives just as much as we do in our personal ones. In your new role, however, the status quo can no longer remain. Yes, you can still goof around with your team but you must establish that

fraternization line and no longer cross it. There can be a tendency to either be too hard or too lenient on your friends when you become the boss. Often, we see new supervisors give their friends more work (or projects that no one else seems to get done) with the assumption that their friend will understand and want to help.

As soon as you begin your new role, talk with those that are your friends about your promotion and how it may affect things. Be careful about creating a perception of favoritism. Also, be aware that there is a bigger risk than just the dynamics of your friendship, you could be exposing the hospital to discrimination lawsuits and, in general, cause morale issues.

Other things to think about as you make the transition:

- You will make mistakes.
- Be accountable for those mistakes and own them.
- Don't let the mistakes derail you. Believe in yourself and allow yourself to be human.
- Analyze what happened and learn from it. Discuss it with someone who is not on your team, either another supervisor who is a peer or your Practice Manager/Hospital Administrator.
- Your team will respect you more if they see you leading by example in all that you do. That includes how you handle your errors. Being humble and forthcoming about problems will build trust and admiration.
- Never let your newfound power go to your head to the point that you feel you can't allow your human side to show.
- Remember, your attitude is just as important as your actions.
- Be Respectful
- Be friendly and professional
- Be fair. Be firm. Be consistent. (A word of caution: Fair is not always equal.)
- Build up your team. One of the true advantages about having layers of management is that as a supervisor, you are working side by side with the team. This allows you to be able to give immediate praise and acknowledgement of a job well done. It allows you to see growth potential and areas where delegation is appropriate.
- Manage conflict. It is usually best to handle any problem immediately and not allow it to fester or think it will just go away.
- Be available.
- Be ever growing and learning.
- Build up your team: One of the true advantages about having layers of management is that as a supervisor, you are working side by side with the team. This allows you to be able to give immediate praise and acknowledgement of a job well done. It allows you to see growth potential and areas where delegation is appropriate.

- When having a discussion or writing up a disciplinary action, make it factual with the four “w’s”: What, When, Where, Witnesses.
- Say What Needs To Be Said

**Remember-What we permit, we promote.**

Supervisors are integral to the success of the hospital. It has been proven repeatedly that long term employees who are happy with their jobs often cite their relationship with their supervisor or manager as one of the main reasons they stay.

Other reasons why employees stay include:

- **Pride in the organization.** People want to work for well-managed hospitals.
- **Compatible supervisor.** People may stay just to work for a particular individual who is supportive of them.
- **Compensation.** People want to work for hospitals that offer fair compensation, including competitive wages and benefits as well as opportunities to learn and achieve.
- **Affiliation.** People want to continue working with team members they respect and like.
- **Meaningful work.** People want to work for hospitals that let them do work that appeals to their deepest, most passionate interests. Truly this is one area that veterinary hospitals have a leg up over other job.

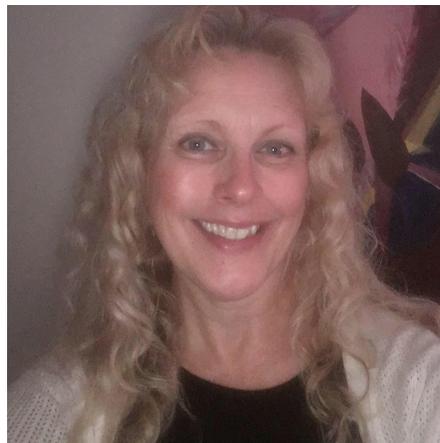
People are the most valuable resource in our hospitals and a supervisor’s role is of ultimate importance in preserving that wonderful resource for the greater good of the hospital.

## Victoria Elam, CVT VTS-LAM (Research Anesthesia)

Vicki graduated from Walter Reed Army Institute of Research as a Veterinary Animal Health Technician in 1978 and served in the Army as a veterinary technician until 1982.

Over her working career of 36 years, she has worked in private practice, Academia as an Instructional Assistant, Adjunct Instructor in local Veterinary Technician programs, as a surgical operating room technician at UW School of Veterinary Medicine, Surgery Anesthesia Technician at the National Primate Research Center and currently as a Research Specialist working with primates for the UW School of Medicine and Public Health in the Department of Psychiatry. Being involved in non-human primate research has allowed her to work with people from across the US, UK and travel to China to teach primate anesthesia and monitoring techniques.

She has been very active over the years in the WVTA as President and Fundraising Chair, a founding member and Secretary for the Society of Laboratory Animal Veterinary Technicians and a founding member of the VTS Academy of Laboratory Animal Veterinary Technicians and Nurses serving as the IT/Webmaster Chair.





# Practical Inhalant Anesthesia and Monitoring Techniques Used for Short Term, Non-invasive Cranial MRI/ $\mu$ Pet Imaging in Infant Macaques.

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Victoria Elam, CVT VTS-LAM (Research Anesthesia)

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## WHY WOULD RESEARCH USE INFANT MACAQUES?

Medical advances and interest in neuro-developmental and psychological disease, among various other central nervous system processes, has warranted an increased use of advanced imaging techniques of the neonatal macaque brain.

Stereotaxic brain microPET ( $\mu$ PET) and MRI scans have been used successfully for many years on adult and juvenile Rhesus macaques (*Macaca mulatta*) with technical differences for endotracheal placement and rebreathing circuits needing to be placed differently in the different devices available.

Using infant macaques for stereotactic imaging provides different challenges to consider in order to not only produce good images but to also provide adequate inhalation/analgesic practices to ensure a safe outcome of an anesthetic episode.

Limited space, poor lighting and challenging visualization of the laryngeal anatomy are all common challenges faced in these types of protocols. Nursing infants that can spasm or regurgitate may obstruct the airway and compromise the infant's well-being.

Taking into consideration a limited budget, this presentation will show anesthetic/analgesic monitoring in the magnified or radioactive environment using non-MRI compatible anesthesia machines and mutli/single parameter monitors that you may already have in-house and focusing on the difficulty of providing an adequate breathing system with intubation techniques for stereotactic  $\mu$ PET scans and MRI.

## WHAT ARE THE IMMEDIATE CHALLENGES TO CONSIDER??

- Using the correct sedation/analgesic doses of your drugs to give yourself the time needed to successfully intubate, position the animal into the stereotactic device and scan the subject.

- Selecting the correct endotracheal tubes that will not cause unnecessary trauma to the laryngeal folds, the trachea and provide an unobstructed airway.
- Minimize dead-space for optimal respiratory gas exchange
- Selecting an appropriate non-rebreathing system that can be used with a variety of stereotactic devices with minimal respiratory resistance for neonatal lungs.
- Choosing safe monitoring equipment and heating devices you will need for the different imaging environments.

Infants were separated from their mothers, weighed and transported to the imaging facility.

**Sedation/analgesia is as follows:**

- ❖ Ketamine 100mg/ml, 20mg/kg IM
- ❖ Atropine Sulfate 0.54mg/ml, 0.04mg/kg IM
- ❖ Ketoprofen 100mg/ml, 5mg/kg IM
- ❖ Isofurane 1-2%. Sevofurane 2-4%

Once sedated, a 24-22 gage saphenous catheter is placed to provide supplemental fluid administration, radioactive tracer administration for the  $\mu$ PET, and IV access in the event of an emergency .

Endotracheal tubes are un-cuffed 2.0mm-3.5mm with a dead space adapter for monitoring ETCO<sub>2</sub>.

Pediatric laryngeal handle and #0, #1 miller blade is used.

**Some of the non-rebreathing hoses available**

Vetamac “Safe sigh” with a manometer

Modified Jackson Rees

Bain with block

The non-rebreather anesthesia circuit we have found most appropriate for our studies was the Vetamac Safe sigh. It incorporates a longer breathing hose than a Modified Jackson Reese and it has an MRI compatible one touch valve that a manometer can fit onto along with a ¼ ltr. rebreathing bag.

Once the animal is intubated, carefully place the infant into the stereotactic device while making sure the non-rebreathing system and monitoring devices are not compromised.

The 90° microPET head angle positioning can cause the smaller uncuffed 2.0mm endotracheal tubes to bend which may obstruct the airway or add significant respiratory resistance for an infant in the first few weeks of it's life to appropriately exchange fresh and waste gases . Once the infants grow, the larger uncuffed ET tubes can be used, reducing the prior risk with a more rigid ETT.

Because these infants are so small (ranging from 0.360Kg – 1.5 kg) it is imperative that thermoregulation be addressed. Keeping their core body temperature in a safe range of 99-101.0° F can be tricky in cooled imaging rooms without adding weight on the patient which can effect respiratory tidal volumes. For the MRI, we have used small heated fluid bags wrapped in a towel alongside the infant and then covering with bubble wrap for further insulation. For the microPET, we use a Bair Hugger™ blanket laid over the towel wrapped infants. One can also use human infant socks on the hands and feet of the infants.

#### **Monitoring equipment kept outside the MRI**

- Surgivet v8401 handheld capnograph monitor with 30ft. of sampling line
- Nonin 7400 FO Pulse oximeter with 8000FI-30 - Infant/Pediatric Fiber Optic sensor, 30' cable\*\*
- Basic table top lab animal anesthesia machine with a 30' air/gas line

#### **Monitoring equipment used for the μPET Scanner**

- Stand small animal anesthesia machine
- Surgivet Advisor® Vital Signs Monitor V9204
- Covidien Nellcor PM10N Handheld Pulse Oximeter

- 3M Bair Hugger™ 505 Patient Warming Unit

Every facility will ultimately have different brands of equipment available to them.

Utilizing the equipment you have before you invest in MRI compatible equipment is feasible with modification and a little ingenuity.

Because of the amount of animals and scans we performed, we were able to divide up our equipment and purchase only the necessary pieces we needed for each facility.

Other monitoring suggestions that you might want to add is; NIBP and ECG. We have not been able to source conventional ECG cables that are MRI compatible and available for the veterinary patient.

#### **MORE FOCUSED ATTENTION SHOULD BE PAID TO:**

- ❖ **Always measure endotracheal tubes:**
- ❖ ETT's are the biggest culprit of dead space and respiratory resistance, however in this case we must take into consideration the need for some extra tube length to come around the head holder of the specific stereotactic device.
- ❖ **ETCO2 values and capnography tracing:**
- ❖ Staying within the normal limits of 35-45mmHg. ETCO2 correlates well to systemic perfusion of the animal. Capnography tracings are a first indicator of airway complications as placing the infants in the head holders and /or moving them into scanners may compromise ETT placement. The tube can be kinked during the  $\mu$ Pet procedures because of the position of the head.
- ❖ **Keep your patient warm.**
- ❖ Hypothermia can cause a delayed recovery and complicate the anesthetic event. The body will also burn glycogen stores quicker leading to hypoglycemia. It is prudent to check pre and post scan blood glucoses on neonate animals. Anything under 60 dl/mg, warrants oral corn syrup once recovered, with the swallow reflux regained. 50% dextrose PO and/or IV per order of your veterinarian can also be administered.

## Summary

- ▶ We found that out of the 400+ scans that were performed, approximately 3% of the infants had issues breathing in the  $\mu$ PET scanner head holder and most had their ET tubes adjusted and the scan was completed.
- ▶ Once efficiency is achieved with intubating infants in dorsal recumbency, becoming comfortable monitoring infants with their many nuances in physiology and responses compared to adults, the rest of the procedure should have minimal problems.
- ▶ The largest lesson learned during this project was patience, repetition and the ability to make adjustments in a timely manner!

## Acknowledgements for pictures and support

- ▶ Comeseeourworld.org
- ▶ © 2018 Americans For Medical Progress
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- ▶ UNC-Emroy
- ▶ Academy of Laboratory Animal Veterinary Technicians and Nurses (alavtn.org)
- ▶ Society of Laboratory Animal Veterinary Technicians (slavt.org)

**Happy Veterinary Technician Week**



## Kristin Luginbill, DVM, CCRT, cVMA

1997 graduate of UW Madison School of Veterinary Medicine. Certified in rehabilitation from Canine Rehabilitation Institute. Certified in acupuncture from Colorado State University. Work at Lakeshore Veterinary Specialists where practice is limited to rehabilitation and acupuncture.





# Science of Acupuncture

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Kristin Luginbill DVM, CCRT, cVMA

Acupuncture has been used for thousands of years to reduce pain, treat disease and improve health. There is evidence from a well-preserved prehistoric body that indicates that acupuncture or a similar kind of treatment was used in Europe in about 3300 BC. And there are acupuncture texts from China dating from around 200-100 BC. Certainly most of the medical treatments from such a long time ago have completely fallen from favor with advancement in medical and scientific knowledge, but acupuncture has persisted and remains relevant today. How does it work? Traditional Chinese medicine explains the effects of acupuncture by discussing meridians located throughout the body, through which “qi” energy flows and that acupuncture needles placed along the meridian in specific points will influence the qi flow to affect pain and health. While not necessarily untrue, this explanation of the effects of acupuncture cannot be validated by today’s science. We cannot identify meridians or qi in dissections or fluid analysis. But there are many measurable and validated ways to scientifically explain the positive effects of acupuncture experienced by both people and animals.

The process of acupuncture involves inserting a fine gauge, solid core needle into precise anatomical points located throughout the body, carefully selected to achieve certain therapeutic goals. Different point selection, needle selection and needle numbers will be considered with each patient. The basic technique involves identifying the correct position, inserting the needle through the skin, advancing the needle to the desired depth, then manipulating the needle briefly and allowing it to stay in position for the desired amount of time. Acupuncture is very safe with very few undesirable side effects. It is also tolerated well by most companion animals.

## **Local Effects:**

Acupuncture stimulates sensory nerves located in the skin and muscle tissue into which the needle is placed. When the nerve is stimulated it initiates an action potential, an electrical response to stimulation. The action potential spreads locally and various physiologic responses occur as a result. One of these responses is the release of a vasodilator called Calcitonin Gene Related Peptide. As a result of its release, local blood flow increases. It also stimulates the formation of new blood vessels, which can promote local tissue repair if there is injury. The local release of neuropeptides such as CGRP and others such as Nerve Growth Factor, Vasointestinal active Peptide and neuropeptide Y can also affect other local structures. For example, placing needles in the area of a salivary gland can increase the production of saliva in patients with dry mouth post radiation therapy.

### **Segmental Pain Relief:**

After an action potential is initiated, in addition to spreading through nerve fibers in the local area, it also travels up the sensory nerve to the spinal cord. In particular, the dorsal horn of the spinal cord segment specific for the nerve. This results in a depression of activity in that segment of the spinal cord through the release of neuromodulators (peptides that have a longer duration of action and modify the activity of a cell over time) like the opioid peptide *enkephalin*. This depression of activity results in a decrease in transmission of pain signaling from the innervated area. We can place needles and stimulate nerves in the same spinal cord segment as the origin of the pain (a spinal cord segment supplies both the joint and the muscles that move the joint) to reduce pain in the area.

There are other beneficial “side effects” of this segmental pain relief. For example, pain in a joint can create muscle guarding and tension in associated muscles. This tension can decrease mobility and become its own source of pain. With the reduction in pain of the area, there will be reduced muscle guarding possibly resulting in increased movement in the joint and decreased muscle related pain.

When dealing with abdominal pain, we use this segmental response since we cannot directly needle internal organs.

### **Extrasegmental Pain Relief:**

After arriving at the dorsal horn of the spinal cord, a nerve impulse will then proceed up the spinal cord to the brain. Remember that the opioid peptide *enkephalin* is released in the spinal cord in response to acupuncture. In addition, another opioid called *beta-endorphin* is released and can be measured as increased in the brain as a result of acupuncture. Studies (Han and Terenius 1982, Pomeranz and Chiu 1976, Mayer et al 1977) have shown that some of the pain-relieving effects of acupuncture can be reversed with *naloxone*, the same drug we would use to reverse a morphine or heroin overdose, indicating that this release of our natural opioids is contributing to the pain relief experienced. *Beta-endorphins* activate our descending inhibitory pain control systems. This is a system of nerves that go from the brain down through the spinal cord and can release neurotransmitters (a chemical that can stimulate the activity of a nerve- often produced by another local nerve) at each segment throughout the spinal cord. One of these neurotransmitters is *serotonin*, which acts on cells in the dorsal horn of the spinal cord to produce other neurotransmitters and neuromodulators. Another measurable substance released is *noradrenaline*, which inhibits transmission cells. These substances therefore both modify (decrease) the transmission of pain signals when released.

### **Other Effects:**

Human patients often report feeling calmer and more relaxed and sleeping better following acupuncture. These are also described by dog and cat owners after acupuncture treatments. These responses can possibly be partially explained by imaging studies that show that the limbic system of the brain is activated. The limbic system is a group of structure in the brain that process pain and emotions. This activation seems not even to need the needle, but just activation of tactile system in the body. Acupuncture has been shown repeatedly to improve nausea and vomiting symptoms of various causes in

studies but the mechanism of action for this is not well defined. We can also influence the autonomic nervous system with acupuncture. The sympathetic tone can be increased locally but have a widespread, longer acting decrease in tone. We may also be able to influence the immune system and endocrine systems with acupuncture, but these responses are not as well understood or as well researched.



# Understanding Pain

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Kristin Luginbill DVM, CCRT, cVMA

The International Association for the Study of Pain defines pain as “an unpleasant sensory and emotional **experience** associated with actual or potential tissue damage”. Untreated pain can significantly influence the quality of life in our veterinary patients and is a significant concern for pet owners. Understanding that pain is a complex **experience** and that experiencing pain is the result of complex physiologic process can help us better plan our pain treatment plans and ensure that our patients are happier and more comfortable.

The “Pain Pathway” was once thought to be a relatively simple pathway in which a series of nerves would convey information to the brain but extensive on-going research continuously proves that the process is much, much more complex.

Nociception is the process by which a noxious (meaning potentially harmful, unpleasant) stimulus arrives in the central nervous system.

A nociceptor is a type of receptor at the end of a sensory neuron found in our skin and tissues that responds to damaging or potentially damaging stimuli by sending warning signals to the spinal cord and brain. Nociceptors respond to specific types of stimulus – such as chemical (inflammatory mediators), mechanical (pressure) or thermal (hot or cold). When the nociceptor is activated, the information is converted into electrical energy, which can travel through a nerve. This is called *Transduction*.

The movement of the electrical energy from the site of the stimulation to the spinal cord and brain is called *Transmission*.

*Modulation* of the stimulus occurs through inhibition or facilitation in the spinal cord. Inhibition can decrease the sensation of pain and comes from opioids, norepinephrine, serotonin that are produced by the central nervous system and descend through the spinal cord. Facilitation will increase the experience of pain. Fear or unpleasant memories can actually facilitate pain and make the pain worse.

The stimulus is then moved up to the brain, which is called *projection*.

The processing of this electrical information in the brain is called *Perception*. *If the brain does not perceive the information, there is no pain experience regardless of the amount of tissue damage.*

Because of all of the processing and modulation that occurs in the spinal cord and brain, the pain experience will be very different for different animals based on their physiology, their past experiences their personalities, etc. Just because something doesn't seem to cause pain in one animal, doesn't mean that it isn't genuinely experienced as painful to another. *Because of the processing and modulation that occurs in the spinal cord and brain, pain can be experienced even in the absence of an obvious ongoing noxious cause.* An extreme example of this would be phantom pain.

**Pain can be classified in many different ways.**

Acute pain is the result of a noxious stimulus. It is generally short acting and duration is proportionate to the extent of the stimulus. It is usually a relatively easier type of pain to treat.

Chronic pain is described as pain that lasts longer than the period of tissue healing. The pain may not correlate well to the amount of pathology present. The duration may be days, months or even years past the inciting cause and it is no longer serving a biological function to protect tissues. It is more difficult to treat and may have emotional consequences for the patient, such as depression or fear.

Adaptive or Physiologic Pain is pain that serves a purpose. The purpose is usually to protect tissues from damage. For example, the pain that you feel when you burn your finger on a hot stove protects you by causing you to pull away and avoid tissue damage from prolonged exposure to the hot surface.

Maladaptive Pain does not serve a purpose. It is a malfunction of neurologic processing. The pain itself is now the disease.

Somatic pain is a generally well localized pain that results from the activation of peripheral nociceptors originating in skin, ligaments, muscles, bones or joints. It can be superficial or deep. Deep somatic pain arises from joints, bones, ligaments where superficial arises from the skin or mucous membranes.

Visceral Pain is a poorly localized aching or cramping sensation resulting from activation of nociceptors of the thoracic, pelvic or abdominal viscera. Visceral pain can be referred to another site- such as when a patient experiencing a heart attack feels pain in the neck or arm.

Nociceptive pain is the result of stimulation of a sensory nerve by a noxious stimulus, which can be inflammatory, mechanical, thermal or stretch related.

Neuropathic pain is due to a primary lesion or dysfunction of the nervous system, either peripheral or central. It is often described as a shooting or burning type of pain.

These classifications are not all encompassing and overlap definitely exists. For example, you can have chronic neuropathic pain or acute somatic pain.

With all of these classifications, you can see why there is no one “pain medication” and why different animals will respond differently to the same pain medication.

Understanding the process of pain and the different types of pain that can be experienced will be critical in picking the right pain control plan.

Let’s look at the example of osteoarthritis. It begins with abnormal forces acting on a joint (from dysplasia or a ruptured cruciate, for example). This will create inflammation. Inflammatory mediators will act on chemical sensory receptors and activate the nerve to fire. The information will travel along the nerve and reach the spinal cord where it will be processed and modulated, projected to the brain and perceived as pain. At this stage an anti-inflammatory may have a very positive effect as it blocks the inflammatory mediators that cause the nerve to fire in the first place.

### **Why do anti-inflammatories not help every animal with arthritis?**

One reason is that when there is persistent stimulation of receptors, they can become “**sensitized**” both in the periphery and centrally in the spinal cord. There are molecular, cellular and CNS processing changes that occur to cause this centralization and pain may become maladaptive. Normally, central sensitization (or “wind up” pain) is transient, but with long-term or severe stimulus, it can persist indefinitely.

In addition, in the case of arthritis, with time you will have additional sources of pain associated with disease process, such as an increase in intraosseous pressure, neuropathic pain and secondary muscular pain associated with movement compensation strategies. The source of these experiences of pain are not inflammatory. Therefore, anti-inflammatories may no longer work as well as they once did and other pain interventions may become necessary.

Recognizing pain is challenging in veterinary patients for many reasons, not the least of which is that we don't speak the same language. In addition, because of all the factors discussed already that make pain a personal experience, it is always hard to interpret someone else's pain. Crying and whining are rare indicators of pain in our patients unless pain is severe or sudden. Animals will tend to hide pain because of their predator/prey status and because they are pleasers.

There are many pain recognition scales that can be helpful but all have their limitations. The most important thing is to pick a scale that you think can be used consistently within your clinic and over time. There are acute and chronic pain scales. A good validated acute pain scale is the Glasgow Composite Measure Pain Scale. There are feline and canine versions of this assessment. Other examples of pain scales for chronic pain are the Cincinnati Orthopedic Disability Index (CODI) and the Health Related Quality of Life Scale (HRQLS). Colorado State University has both acute and chronic pain scales that can be used to help you think about what pain an animal may be experiencing.

Questioning the owner and going over home behavior is very important in assessing pain as well. Behavior changes that can indicate pain in animals include limping, not wanting to be pet, change in behavior towards owners/people/other pets in household (grouchy, not greeting at the door, not jumping up), change in sleeping/resting habits, change in play or walking habits, change in bathroom/litterbox habits, change in posture or body position. Changes in any of these could be an indication of pain.

Treatment of pain should consider the patient's specific type of pain, the degree of pain and the duration of pain. Treatment can be *pharmacological* (ex. opioids, NSAIDs, Tylenol, local anesthetics, NMDA-receptor antagonists, anti-depressants, bisphosphonates, cannabinoids, corticosteroids) and *non-pharmacologic* (ex. nutrition, supplements, intra-articular injections, PRP, stem cells, PEMF, physical medicine (laser, acupuncture, massage, therapeutic exercise)).

Using pain recognition techniques and an understanding of the experience of pain can help us better treat pain in our patients and help them live happier, more comfortable lives.

## Christopher Snyder, DVM, DAVDC

Dr. Snyder is a Diplomat of the American Veterinary Dental College and Clinical Associate Professor at the UW School of Veterinary Medicine. He prides himself on the dental education he provides to students at the University of Wisconsin and the residents he has successfully mentored in the specialty.





# Periodontal Disease: The Silent Killer. Identifying Disease, and Why Treatment Matters

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Christopher Snyder, DVM, Dipl. AVDC

Any source of infection in the body can be sufficient to stimulate a reaction resulting in inflammation. The process generates chemical and physiologic inflammatory mediators that result in the body's perception as pain. Periodontal disease has repeatedly been reported to impact up to 80% of dogs and 70% of cats over 3 years of age. Identification of both the gross evidence of disease as well as picking up on cues in the client's history can lead to client education, justification for further investigation and ultimately treatment and improvement in the pet's quality of life.

The most obvious signs of periodontal disease result in gingivitis, calculus accumulation and, in severe cases, gingival bleeding, gingival recession and root exposure. In less obvious cases, identifying unilateral (asymmetric) calculus accumulation or asymmetric gingivitis may be helpful at identifying subtle signs of disease. The absence of mechanical disruption of plaque by preferentially chewing on only one side permits the time needed for plaque to mineralize into calculus.

The success associated with the treatment of pathologic periodontal pockets rests on first identifying the underlying cause for the condition. Recognizing the increased depth of the sulcus (periodontal pocket) and acknowledging that the host's defense mechanisms against the bacteria in those pockets are designed to flush out the pocket and keep it clean, and then modifications to that local environment must occur. These modifications include A) making the pocket less hospitable for gram negative, anaerobic bacteria, as well as B) creating an environment conducive to reversing the periodontal attachment loss. The greatest prognostic determinant influencing the success of periodontal treatment is the client's willingness and ability to institute home dental care.

## Periodontal Disease Staging

Periodontal disease is categorized by stages because it is a progressive disease process that is likely to continue to worsen over time in the absence of treatment or preventative measures.

Stage 0: Healthy periodontium (normal) and is the absence of attachment loss or inflammation. This is the desired state that we should strive to maintain in the pet's mouth. The normal sulcus depth of a dog is 1-3mm and <0.5-1mm in cats.

Stage 1: Gingivitis only. This is the only reversible stage of periodontal disease and exists with inflammation and an absence of attachment loss. Gingivitis is graded by stages (stage 1-inflammation only, stage 2- bleeding on probing, stage 3- spontaneous bleeding)

Stage 2: Mild Periodontitis can be defined by gingivitis and attachment loss commonly progressing to loss of supporting alveolar bone. The extent of attachment loss can be up to <25% the length of the root.

Stage 3: Moderate periodontitis is defined by the presence of gingivitis and 25-50% attachment loss of the length of the root which frequently involves exposure of the furcation of multi rooted teeth.

Stage 4: Advanced periodontitis includes the presence of gingivitis and >50% bone loss. Multirooted teeth typically exhibit furcation exposure and without extensive efforts to rehabilitate the periodontal attachment, these teeth carry a very poor prognosis long term. Any multirooted tooth demonstrating furcation exposure is considered to have Stage 4 periodontal disease. Without heroic treatment, all stage 4 teeth should be extracted.

### **Root Planing and Subgingival Curettage**

Root planing is the process of cleaning a pathologic periodontal pocket on both the soft tissue (gingival side) and hard tissue (cementum) surfaces. With targeted removal of the subgingival calculus, necrotic cementum, long junctional epithelium and leaving a healthy root surface, residual periodontal ligament cells and nonkeratinized epithelium have a chance to reattach.

Instruments designed for subgingival curettage include universal subgingival curettes, Gracey subgingival curettes (these instruments have an extra angle in the working end of the instrument to facilitate debridement) and dental hoes. Subgingival curettes are the most commonly used instruments used for this purpose when treating periodontal disease. Familiarity with the instruments is important for successfully executing treatment procedures. Curettes have a rounded toe (tip) and, when closely looking at the working surface, the cutting surface is either angled perpendicular or 70° to the shank. Instruments with a cutting surface angled perpendicular to the flat surface are known as Universal curettes. When the cutting surface is angled 70° to the shank, the instrument is termed a Gracey curette. Knowing the orientation of the cutting surface of your instruments is important to successfully position the instrument to accurately prepare the tooth and soft tissue surfaces.

Closed root planning is the act of *non-surgically* treating the tooth root by removing subgingival calculus, accumulated organic debris and necrotic cementum. This is reserved for treating periodontal pockets that are no deeper than 5-6mm in total depth in the canine and <2mm in the cat. The curette should be delivered into the base of the sulcus with the cutting edge parallel to the root surface until it is positioned at the bottom of the sulcus. The handle of the curette is then orientated parallel with the long axis of the tooth crown- this will engage the cutting surface onto the root surface. The active motion of the universal curette is during the pull stroke where the cutting edge engages the root surface and debrides pathologic tissues.

Subgingival curettage is the act of removing the epithelial lining of the periodontal pocket in attempt to regain healthy periodontal attachment. Once the root surfaced has been prepared, the working end of the curette should be used where the cutting edge is placed away from the root to clean the soft tissue surface of the periodontal pocket. Successful removal of long junctional epithelium is necessary for new epithelial attachment and subsequent periodontal ligament rehabilitation.

Open root planing involves surgically creating a mucogingival flap that allows definitive access to the root surface needing to be cleaned. Flaps greater than 6mm should be surgically accessed to provide more reliable root preparation (through visualization) and soft tissue surface debridement. Vertical releasing incisions should be created along the line angles of the tooth root (as if a surgical extraction was going to

be performed) to facilitate flap repositioning. The root surface can be debrided with a curette, hand scaler, ultrasonic scaler or diamond bur on a water-cooled, high-speed hand piece (the pedal should need to be depressed less than 50%). Once root and flap debridement has taken place, the flap should be repositioned into the original location and sutured with 4-0 or 5-0 monofilament delayed absorbable sutures.

Periodontal pocket medicaments labeled for veterinary use are limited. Doxycycline is the most commonly used antibiotic with a carrier designed for periodontal pocket treatment. The goal of antibiotic placement into the pathologic pocket is to maintain an environment conducive (reduced load of pathologic periodontal pathogens) for periodontal ligament cell reattachment and epithelial attachment at a normal depth. The tetracycline family of drugs is frequently chosen for treatment in periodontal pockets.

Doxirobe (doxycycline in a gel labeled for veterinary use) and Arestin (minocycline in a powder form labeled for use in humans) are the most common formulations used by veterinary dentists. Doxirobe is a gel (two-part carrier/doxycycline mixture) that is delivered into the periodontal pocket with an administration cannula. The mixture begins setting up once in contact with fluid (water or crevicular fluid). Clindoral is another periodontal dressing delivered into the sulcus and provides 10 days of slow release clindamycin. Clindoral's shorter duration of activity is not a true periodontal pocket treatment and does not have an equivalent indication for use to Doxirobe gel.

Frustrations working with Doxirobe usually arise from efforts to deliver and place the gel into the periodontal pocket. Once the unset material flows out of the sulcus, efforts to knead the Doxirobe gel into the sulcus usually results in the gel sticking to placement instruments. To combat these difficulties, Doxirobe administration may require two sets of hands (help from an assistant). *It is important to remember that the gel should only occupy the "pathologic" portion of the pocket. Filling the gel to the surface may result in the body treating the gel as a foreign body and reestablishment of the periodontal pocket.*

Diligent efforts to subgingivally condition the root surface and opposing soft tissues, coupled with compliant efforts by the client to institute home care, provide a periodontal environment that retains a potential for healing. While substantial periodontal attachment gains can occur as the result of a single treatment, it is more predictable to prepare clients for a 50% reduction in the pathologic depth of the periodontal pocket at each treatment. The deeper the periodontal pocket, the more likely that a repeated treatment may be necessary to rehabilitate the tooth's periodontal attachment. Periodontal reevaluation (ideally) should be performed in 3-4 months under general anesthesia and should involve periodontal probing and repeat radiographic reevaluation.

### **Guided Tissue Regeneration**

Teeth are designed to be supported by the periodontal ligament (provides flexibility and reduces fracture) and alveolar bone (the anchorage point for the periodontal ligament to span to from cementum). Instances of attachment loss below the gum line, and in the presence of alveolar bone, are ideal to regain attachment resulting in that normal cementum-periodontal ligament-alveolar bone relationship with root planing and subgingival curettage. When alveolar bone has resorbed secondary to the presence of inflammation, periodontal rehabilitation of a deep pocket will likely result in establishment of long junctional epithelium attaching to the root surface. Upon recheck evaluation these teeth probe normally

however the long-term prognosis of healthy attachment is less than excellent due to the tooth's inability to be adequately supported- *it wants to be supported by bone!* In these situations, performing an advanced procedure where bone is encouraged to form in addition to regenerating periodontal ligament is called *guided tissue regeneration (GTR)*. GTR is typically only successful when treating vertical bony defects. Think of it this way- osteoblasts only like to lay down new bone by migrating out of existing bone in a horizontal direction. Reversing horizontal bone loss in the mandible or maxilla is the most hotly researched topic in human dentistry because it just doesn't work. The greater the number of bony walls that a periodontal pocket has, the more surface area for osteoblasts to move in and regenerate bone. The hallmarks of guided tissue regeneration are to clean the pocket of infection and inflammatory byproducts (against the bone, in the pocket and on the root surface) and to place an osteoconductive or osteoinductive material into the pocket and cover it appropriately.

Maintaining a healthy oral cavity is pivotal to the success of this procedure. A client's diligent efforts with periodontal homecare is key to creating an environment where bone regrows and periodontal ligament reestablishes. Recheck of this procedure should take place at 4 months post-treatment. Normal periodontal probing depths and radiographic reestablishment of normal alveolar bone with a visible periodontal ligament space are signs of success.

## **Home Dental Care**

Numerous studies with various levels of evidence have been published regarding the benefits of various oral health products, techniques and medications. These evaluations are rarely compiled together in a way that a logical approach to developing a home care "plan" can be formed for patients. I like to think of these different products or techniques as having positives and negatives and along with that, different efficacies for preventing periodontal disease. When considering these materials' functionality and efficacy as proven by the veterinary and human literature, a general tiered system can be used. Constructing a home care regimen is individualized for each patient.

### Tier 1

Daily brushing remains the gold standard for the prevention of periodontal disease and maintenance of good oral health. Since veterinary patients are being brushed to prevent periodontal disease, rather than prevent cavity formation, brushing once daily is sufficient. Different studies show reduction of plaque or tartar based on various frequencies of brushing. Having the pet become accustomed to the toothbrush gradually can improve client and pet compliance. Pairing the brushing with a positive daily "reward" will create a pet who expects or seeks the tooth brushing activity. I recommend brushing once daily before feeding. By disrupting the subgingival bacterial matrix once daily, an environment which predisposes the gram negative anaerobic bacteria to overpopulate is reduced. Human toothpaste should never be used in pets since the amount of fluoride contained in these products is not meant to be swallowed and may cause fluorosis of the kidneys. Veterinary research suggests that brushing with water versus veterinary toothpaste demonstrates no significant difference in the amount of plaque and tartar accumulation. Veterinary toothpaste tastes good to dogs and cats and functions more as a reward than a primary cause for plaque reduction.

## Tier 2

Regarding food, in most situations dry food results in less plaque and tartar accumulation. Veterinary prescription diets such as T/D and Royal Canin incorporate technology which results in the fiber within the food to be orientated in a manner that predictably affects the way the kibble fragments when chewed. Repeated chewing creates a mechanical disruption of plaque before it can mineralize into calculus. Eukanuba/lams and Royal Canin also make pet food products containing polyphosphates. Polyphosphates are responsible for chelating calcium found in saliva. When the salivary calcium is rendered unavailable to plaque, the process of plaque mineralization and subsequent calculus formation slows. Polyphosphates are bound to sodium in the dry form. The molecules dissociate in saliva. Prescription diets designed to be low in sodium (renal and cardiac diets) will not be labeled as having polyphosphates in the ingredient list. It has also been shown that a 50% increase in kibble size results in a 42% decrease in calculus formation in dogs. Increasing kibble size in cats results in a decrease in gingivitis.

## Tier 3

Dogs who are heavy chewers are prone to fracturing the major chewing teeth (upper 4th premolars) when gnawing on hard objects. Careful selection of chew treats and toys should be made to avoid tooth fracture. A good rule of thumb for clients is- *“if you can hit yourself in the knee with it and it hurts, it’s probably too hard for them to chew on.”* Chew treats and toys offer a limited mechanism of periodontal disease prevention. While mechanical disruption of plaque may be helpful, the chew objects do not reach below the gum line where periodontal disease occurs. The Veterinary Oral Health Council website ([www.VOHC.org](http://www.VOHC.org)) is a great source for clients and veterinarians to find reliable information about which oral health products work. Barrier sealants like Oravet have also been shown to reduce plaque and tartar buildup. While Oravet is not a replacement for brushing, it can be used in patients where daily tooth brushing is taking place.

## Tier 4

The use of water additives, oral rinses and prescription antibiotics should be used carefully (and sparingly) in veterinary oral health management. An ingredient in some water additives include xylitol, which despite having antibacterial properties, also has a very narrow therapeutic margin and puts canine patients at great risks of xylitol toxicity. Oral rinses typically contain chlorhexidine or ascorbic acid which both functionally serve to have antibacterial properties. The ultimate problem with rinses and water additives, even when used properly, is that they do not penetrate into the targeted area where periodontal disease develops- subgingivally.

## **Conclusion**

Early identification of periodontal disease and appropriate client selection can make for a rewarding experience when treating pathologic pockets associated with periodontal disease. Tooth preservation offers the benefit of quicker recovery than surgery, maintenance of tooth function, and improved general health from the perspective of fewer bacteria associated with intermittent bacteremia and inflammatory mediators in circulation. Commitment to home dental care efforts help support the body’s ability to heal and should be emphasized as such when counseling clients.

## **Recommended reading:**

1. Zetner K, Rothmueller G. Treatment of periodontal pockets with doxycycline in beagles. *Vet Ther.* 2002; 3(4):441-52
2. Holmstrom SE, Fitch PF, Eisner ER. Periodontal therapy and surgery in *Veterinary dental techniques for the small animal practitioner*, 3<sup>rd</sup> edition. Saunders, Philadelphia PA, 2004:233-43
3. Lobprise HB. Root planing and periodontal pocket therapy in *Blackwell's Five-Minute Veterinary Consult Clinical Companion Small Animal Dentistry*, Lobprise HB ed. Blackwell Publishing, Ames IA, 2007:61-69
4. Roudebush P, Logan E, Hale FA. Evidence-based veterinary dentistry: a systematic review of homecare for prevention of periodontal disease in dogs and cats. *J Vet Dent.* 2005 Mar;22(1):6-15
5. Hale FA. The owner-animal-environment triad in the treatment of canine periodontal disease. *J Vet Dent.* 2003 Jun;20(2):118-22
6. [www.vohc.org](http://www.vohc.org)
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# Intraoral Radiographs- What Am I Looking At? More Importantly, Did I Get It?

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Intraoral radiography has evolved a great deal over the past 10 years. Initially it was only available through dental specialists and people with a comprehensive interest in dentistry. Now it has become readily affordable for the average practitioner and is commonly available in practice. The indications for dental radiography are variable and many but being able to understand and interpret the results are paramount to identifying pathology and recommending the appropriate treatment.

## **Indications for Radiography**

Missing, mobile, fractured, discolored teeth and teeth with periodontal pockets are all teeth that should undergo radiographic evaluation to determine treatment options. Teeth that are planned for extraction, for whatever reason, should be radiographed to aid in treatment planning, correct estimate creation and to appropriately counsel clients about potential complications. Some individuals even suggest that radiographs prior to and after a procedure may even serve as a protection against clients who may question the quality of a procedure.

## **Marketing Radiography to Clients**

The information that intraoral radiography can provide is invaluable for correctly and accurately identifying abnormalities and recommending treatments that will improve the veterinary patient's quality of life. With  $\frac{1}{2}$  to  $\frac{2}{3}$  of the normally erupted tooth anatomically being located below the gum line, educating the client about the health of the subgingival periodontal attachment as well as the vitality of the endodontic system can only be impressed through the demonstration of a radiograph. The use of acrylic models or "smile" books displaying "normals" can be helpful at conveying to clients this valuable service.

Once radiographs have been taken, printing the radiograph images and including them with discharge instructions provide the client with something tangible which can help to validate the service, justify the charges and provides redundancy of the information in the medical record. In my experience, clients typically appreciate the images and discussing the treatment and its justification is more easily understood.

## Digital Intraoral Radiography Options

Advantages of digital intraoral radiography in today's practice lies both in the easy access of computers, ease of sharing for referral evaluation, and an improved learning curve with even quicker attainment of radiographic images. Digital radiographic mediums require significantly less radiation exposure, which makes them safer compared to traditional film when considering operator safety. Generally speaking, there are two technologies that make up digital dental radiography today. Both technologies provide radiographic images significantly faster than traditional film radiography and digital offers the ability to easily magnify and manipulate certain qualities of the image.

*Direct digital radiography*- this technology utilizes hardware capable of generating an image while being "directly" connected to the computer. The sensors of these units are only available as size 2 or size 0 (because these sizes are most frequently used in human dentistry) and these sensors are rigid and several millimeters thick. Direct digital units offer the advantage that their image creation and display on the computer screen is virtually instantaneous. With the instant gratification of image display, an incorrect radiographic positioning relationship can be corrected without disturbing sensor and generator tube head. Damage to the sensor results in being unable to continue to collect information without replacing the sensor as well as the sizable investment of the sensor which may cost an upwards of \$8,000-10,000 or more.

*Indirect digital radiography*- involves technology of exposing phosphor plates that are then fed into a device which scans the plates, erases the plates and sends the image data to the computer. An advantage of this system are that the plates are thin and flexible, so they may accommodate the mouths of patients more comfortably, and include a variety of sizes. Dental specific readers can accept plates as large as about 5" x 7" which may be helpful for imaging exotics. Larger tabletop scanners have the capacity to accept large films typically use with tabletop radiograph units. Disadvantages of this setup includes the necessary step of removing the plate from the patient's mouth, which increase image acquisition time and makes modification of positioning to rectify improper images more challenging. Phosphor plates are somewhat fragile and shouldn't be folded, bent or scratched. In the event they need to be replaced, most cost less than \$100.

## Positioning

Before radiographic films can be properly interpreted, they need to be properly taken. The fundamental principal of the parallel positioning technique is exactly the same as radiographic exposure of other parts of the body- place the anatomy to be radiographed against a film so that both structures are parallel to each other and then aim the radiation source perpendicular to the structure and the film. This approach is only successful when dealing with the caudal mandible because of the flexibility afforded by the soft tissue structures between the mandibles.

The bisecting angle technique enables the operator to generate anatomically appropriate images without excessive artifacts such as foreshortening or elongation. The bisecting angle technique is necessary to properly radiograph the entire maxilla and the rostral mandible. This technique works best if the operator has a preconceived idea of what the proper film should look like. By visualizing the long axis of the tooth structure, and visualizing the long axis of the radiographic film, evenly splitting the angle in half provides us with the bisecting angle. By positioning the generator tube head perpendicular to this bisecting angle, a representative image will be created.

The most common reasons for disposing of film or retaking films are 1) missing the area of interest, 2) foreshortening of the image or 3) elongating the image. Foreshortening or elongation can be easily remedied by altering the angle of the x-ray generator. When these difficulties are encountered using the bisecting angle technique, modifications to the x-ray generator can be made in a manner analogous to looking at a person's shadow when on the beach. When standing on the beach, the person represents the tooth and the sand represents the x-ray film. If the sun is in a position in the sky where the sun's rays are perpendicular to the bisecting angle between the person and the sand, a 1:1 scale shadow is made. If the sun is too much towards the horizon, the shadow becomes elongated. If the sun is directly overhead, the shadow appears short and squatty (foreshortened). Thinking of the x-ray generator as the sun, and looking at an image that is either foreshortened or elongated, the operator should have an easier time reasoning through which direction the x-ray generator needs to be moved to acquire the desired image.

Radiographing the maxillary fourth premolar correctly is a common point of frustration for many people. To assess all three roots of this tooth, two views are needed. A lateral view will enable visualization of the distal root. In this view the mesial buccal and palatal roots will appear superimposed. To effectively separate these roots, the x-ray generator needs to be oriented in either a caudorostral or rostrocaudal direction. The "SLOB" rule has been coined to help decipher which root is represented in which location on the film/image. SLOB stands for Same Lingual Opposite Buccal. The same/opposite are in relation to the direction the x-ray generator moves. If the x-ray generator is swung rostrally to shoot in a rostrocaudal direction, the resultant two mesial roots will be able to be interpreted as follows: the root moving in the "Same" direction on the film as the x-ray generator (rostrally) is the Lingual root. The root moving in the "Opposite" direction of the x-ray generator (generator went rostrally, so the root moving caudally on the film) is the Buccal root. This technique takes practice and, in general, a hospital should decide how to position for these radiographs where the roots are separated, and everyone use the same positioning, or the films must be labeled.

### **Positioning for Feline Radiographs**

Maxillary fourth premolar teeth are particularly challenging to image in cats due to superimposition of the zygomatic arch. This superimposition can be overcome by using the *Extraoral Technique* of laying the sensor on the table, against the maxilla and outside the cat's mouth. By exposing the film through the cat's mouth propped open, an image is created that removes the zygomatic arch and more clearly depicts the cheek teeth. *Reminder:* using this technique creates "backwards images" (right side looks like the

left.) This occurs because the convention of labial mounting is based on the sensor being in the mouth and radiation originating from extraoral. Remember to relabel these images so people don't get confused!

## Normal Anatomy

Enamel and dentin are comprised of a greater percentage of mineral than bone, so they appear more radiodense. The periodontal ligament is primarily comprised of collagen so it should show as a distinct radiolucent line. The periodontal ligament should be thin, well demarcated, and of consistent width throughout the outline of the tooth. The pulp chamber is located in the center of the tooth root and should be of uniform diameter throughout. Whenever a radiographic abnormality is suspected, radiographic comparison is recommended of the contralateral or comparable tooth. Remember that as the tooth matures, the pulp chamber narrows as secondary dentin is produced. If a widened pulp chamber is identified compared to neighboring or contralateral teeth, chronicity of the lesion is demonstrated. The younger the patient (the wider the pulp chamber) the more profound the difference will be in the amount of secondary dentin laid down in six months. Mature patients can have very little change in pulp chamber diameter from year to year.

## Pathology

*Bone loss:* Loss of mineral opacity is typically seen in vertical or horizontal orientations. If there is regionalized horizontal bone loss, a chronic inflammatory condition likely exists in that area or there are multiple neighboring teeth severely affected by periodontal disease. **Bone loss does not equal attachment loss, it just guides prognosis for periodontal disease treatment.** So, if there is there is periodontal attachment at the level of the cemento-enamel junction and signs of underlying vertical bone loss, those teeth are not as periodontally "sound" long-term because of the quality of the soft tissue attachment holding the root. In the most extreme cases of bone loss involving mandibular teeth, dental radiographs can impress upon the client the risk of jaw fracture while performing the extraction.

*Periapical radiolucency:* Periapical radiolucencies are typically what veterinarians are concerned with when evaluating for tooth vitality. Widening of the periapical periodontal ligament space can be suggestive of pathology. Triangular shaped radiolucencies extending from the apices of canine teeth and mandibular first molar teeth are referred to as "chevron" signs. This can be a normal finding on these particular teeth and are not indicative of tooth nonvitality. If one is unsure about a radiographic finding, radiographing the contralateral tooth is recommended. Treatment for all these conditions would still involve root canal therapy or extraction- especially since our patients cannot definitively tell us what type of pain they are in.

*Root pathology:* Radiographic evaluation of tooth roots provide a tremendous insight into the difficulty or involvement of an extraction. Root fractures, extra roots and unusual root shapes (root dilacerations) are all conditions that directly influence the time and likely success of tooth extraction.

*Root resorption/ankylosis:* Presence of a periodontal ligament reassures veterinarians that a soft tissue separation exists between the tooth and alveolar bone which will serve as some “wiggle room” for placement and action of the dental elevator or luxator. There are many causes for root resorption with inflammation being the most common. Instances where resorption has occurred but a periodontal ligament remains visible may demonstrate the tooth is weakened and may fracture during elevation. Instances of root resorption with ankylosis (bony fusion between cementum and alveolar bone) should prepare the veterinarian for a more complicated extraction that will likely take longer. Older dogs with a history of being heavy or aggressive chewers will frequently exhibit ankylosis in dental radiographs. Extraction of these teeth typically take longer and may involve additional steps necessary for creating space for elevator placement. A practitioner should feel comfortable estimating a greater cost to the client to extract teeth that will be difficult.

*Felines- Types of Tooth Resorption:* Tooth resorption is a process very commonly noted in cats. Radiographic evaluation of the affected teeth is necessary to guide appropriate treatment recommendations. Since the process is a mixed destructive and proliferative condition, loss or involvement of the tooth crown may or may not be indicative of the extent of root involvement. Type I tooth resorption is defined as a radiographically present periodontal ligament structure. These teeth are treated with extraction since leaving remaining root structure behind to undergo replacement resorption is believed to require inflammatory mediators which could contribute to discomfort. Type II resorption is defined as a tooth undergoing resorption where the periodontal ligament is no longer visible, indicative of replacement resorption having taken place. Crown amputation of these teeth are deemed acceptable (as long as other criteria are not met) since the root is believed to have already undergone replacement resorption and the act of trying to extract these teeth could cause more discomfort. Type III resorption is defined as a multi-rooted tooth demonstrating one root with a periodontal ligament present while another root demonstrates replacement resorption. The root with the periodontal ligament present should be extracted while the root undergone replacement resorption should be crown amputated.

*Pulp Chamber Abnormalities:* Occasionally radiographic evaluation of a tooth may demonstrate various abnormalities of the pulp chamber. Pulp chambers can be noted to be too large (indicative of tooth non-vitality and arrested dentin production), too small (pulp canal obliteration and accelerated dentin production may occur as a result of chronic inflammation or pulpitis) or contain mineralized material. Internal root resorption can sometimes also be found on radiographic evaluation. Odontoclastic differentiation within the pulp chamber can result in dentin resorption within the pulp cavity. These lesions should be differentiated from external root resorption by comparing multiple oblique angles of the same tooth. If the resorbing area does not move from the central location within the canal system, internal resorption is confirmed. External root resorption will result in the resorbing area being cast to the edge of the tooth root on oblique views.

*Bony Changes Associated with Benign and Malignant Oral Tumors:* There are really not many rules associated with the radiographic interpretation of oral masses and the correlation between the process being malignant or benign. Generally speaking, benign processes can be destructive or productive when

considering bone, however these processes rarely cause destruction to tooth structure. It is not uncommon for benign processes such as peripheral odontogenic fibromas (+/- ossifying type) and acanthomatous ameloblastomas to displace tooth structure while in bone. Squamous cell carcinoma, sarcoma family of tumors and malignant melanoma can all vary in the degree of bony involvement they display radiographically. At initial evaluation, radiographs should help direct the selection of appropriate tissue to be submitted for histologic evaluation. Any sort of mass causing bony change should include that aberrant bony architecture in the histologic submission. Radiographic evaluation should also help guide decision making for definitive surgical treatment of oral masses if computed tomography is not available.

### **Post-Procedural Radiographs**

There are many circumstances where dental radiographs following extraction are helpful and provide the practitioner with reassurance that the job is complete. My recommendation is that a radiograph should always be taken at ANY time a root is suspected to have fractured during extraction and always taking post extraction radiographs is best practice. Gaining confidence in gross appearance of root structures after extraction and being able to compare them to pre/post-operative films helps to build confidence. Post extraction radiographs may also be beneficial for sharing with clients at discharge to help justify procedural cost and objectify the pathology and the treatment. Printed dental radiographs provide the client with tangible information that can sometimes soothe concerns over client-perceived feelings that “unneeded procedures” were performed (you know, those clients who say their animal’s mouth isn’t painful but your exam and radiographs show otherwise!)

Occasionally you may have patients who have received advanced dental procedures (root canal, vital pulp therapy/pulp capping, crown placement.) Dental radiographs should always be taken of these teeth out of convenience if the animal is not undergoing a prescribed anesthesia for the purpose of dental treatment monitoring. Comparing immediate post treatment radiographs to present day films are usually necessary to comment on the success of the procedure. Be sure to share these films with whoever performed the original procedure and ask to be shown how to evaluate success.

#### Recommended Reading:

1. Atlas of Dental Radiography in Dogs and Cats. Authors: Gregg DuPont and Linda DeBowes 2008
2. Veterinary Dentistry: Principles and Practice. Robert Wiggs and Heidi Lobprise. 1997
3. To see an objective, independent evaluation of multiple image sensors and associated images, visit [www.vetdentalrad.com](http://www.vetdentalrad.com). A free CD with sample images can be requested which may help determine which system is right for your practice.

# Ouch, That Hurts! What Are True Dental Emergencies and How to Treat Them?

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How often are do people really call in with “dental” emergencies? How often does a particular scenario sound like an emergency/urgent situation to you, where the owner on the other end of the phone call doesn’t share your sense of urgency? Certain aspects of veterinary medicine are clear-cut. When considering oral emergencies, they are not so clear-cut. Hopefully after this lecture you’ll better be able to manage those phone calls and understand what is truly an emergency and what isn’t.

## **Actual Emergencies**

In veterinary dentistry, there are very few true emergencies. True oral emergencies would include: fractured teeth (especially in young animals), jaw fractures, teeth knocked out of the socket and TMJ luxation. Even within that list, some of these are more urgent than others. It is the goal of the veterinary team to triage those emergent complaints and address them in a timely fashion.

### *Jaw Fracture*

Jaw fractures are ultimately a form of broken bones. When broken bones are considered “open,” they are exposed to outside the body or, more commonly, into the oral cavity. Sufficient evidence exists in humans suggesting that the longer a jaw fracture is exposed to the oral environment, the more at-risk the fracture is to infection. Whenever a client calls about a jaw fracture it should ultimately be treated as an emergency because of the pain associated with the condition. Typically, if the patient is stable for anesthesia, we will not want to wait more than 24-48 hours to fix an open jaw fracture. When waiting for optimal time for repair, a loose fitting nylon or custom made tape muzzle can be applied to help reduce fracture fragments and resultant inflammation. The patient should be maintained on appropriate antibiotics and pain medications. Broad spectrum antibiotics capable of handling oral bacteria will be sufficient to reduce the likelihood of the most common post-surgical infections. Any use of antibiotics should be continued for a total of 10-14 days.

When evaluating oral trauma patients, fractures of the upper or lower jaws may result. Fractures involving the maxilla are likely to be associated with swelling of the muzzle, swelling around the eyes, nasal bleeding and possibly malocclusion. Not all maxillary and midface injuries require fixation or treatment. In some cases, these fracture fragments may be well reduced and 6-8 weeks of softened food may result in acceptable bony healing. If soft tissue lacerations are present, they should be closed to prevent formation of an oronasal fistula.

Fractures in the mandible tend to occur in the horizontal mandibular body- the area of the mandible that constitutes this includes the canine tooth to the third molar. In both dogs and humans this area may be

somewhat an area of vulnerability. Remember that teeth are held in place by periodontal ligament which is made up of collagen. The size of the mandibular first molar tooth and the enormity of its roots contribute to this vulnerability. Small dogs being predisposed to dental crowding and periodontal disease only increase their risks of pathologic fracture in this area of the mandible.

### *Tooth Fracture*

Teeth develop and the inside of the tooth matures in a similar way to how trees lay down concentric rings. Trees grow by laying down rings of cambium, which results in the outside circumference getting larger. Teeth are similar but deposit mineralized material (dentin) in completely the opposite direction- from the periphery working toward the center. The newly erupted tooth is comprised of mostly soft tissue (pulp) internally which is comprised of arteries, veins, nerves, lymphatics and connective tissues. The large pulp chamber is lined with odontoblasts that lay down additional dentin as the tooth ages. This results in the internal pulp diameter (soft tissue) narrowing as additional dentin is laid down (hard tissue). No matter what intervention a veterinarian (or veterinary dentist) involves, a tooth will never be as strong as what nature intended. Performing a procedure designed to replace the pulpal contents with something foreign (root canal therapy) results in a tooth that is structurally weaker than a natural tooth but at least it is not a source of infection and pain.

Any time deciduous (primary) teeth are damaged, the correct treatment is always extraction. Permanent teeth on the other hand are worth preserving. If a permanent tooth in a patient less than 18 months old is fractured, a treatment known as vital pulp therapy should be performed to create a new barrier against the outside world. The goal is to use specific materials that will resist infection/pain while maintain the health of the odontoblasts required for additional dentin to be deposited. Performing a root canal on a tooth in a patient younger than 18 months of age is challenging because files aren't wide enough to clean all of the canal walls simultaneously. Even if files were large enough, the remaining dentin may not be strong/ durable enough to withstand a life span's use and abuse.

The take home message: fractured deciduous teeth should be extracted (sooner the better because of the approximation of the deciduous tooth root and the developing tooth bud). Fractured permanent teeth in dogs and cats younger than 18 months of age should be addressed quickly- this gives the patient the best chance of receiving a treatment that will enable them to maintain a comfortable, living tooth. The sooner the vital pulp therapy is performed, the higher the likelihood success. Vital pulp therapy performed during the first 48 hours stand the best chance for success. Success has been reported to be approximately 25% for fractured teeth exposed to the mouth for up to 2 weeks.

### *Tooth Avulsion and Luxation*

Luxated teeth are described as teeth that are displaced within their socket, while avulsed teeth have been displaced out from the mouth altogether. **These are emergencies!** If the client's intent is to keep those teeth, they ideally should alert you to the injury before it even happens! What we know from human literature is that teeth avulsed from the socket for more than 30 minutes have a diminished success rate for re-implantation. Obviously, the expense and work associated with replacing these teeth is sizable, so unless a patient is a show animal or working animal (police dog), resituating the teeth into the socket is probably unnecessary for the animal to have a functional, comfortable bite. The option to salvage the tooth should always be given. For clients who do want to preserve the tooth, the recommendation should be to place the tooth in a "tooth-friendly" medium. Milk, Hank's Balanced Salt solution or saliva

are all fluids with appropriate osmolality to maintain the periodontal ligament cells on the surface of the tooth root in order to improve the chance for successful replacement without resorption. These cases invariably require specialty attention since the materials necessary to replace the tooth and situate it into the alveolus are expensive and technique sensitive. If a tooth is described over the phone as avulsed or luxated and the client expresses interest in preserving the tooth, coordinating a straight referral is the most efficient way to manage these patients. If the tooth is luxated or avulsed, it would be in the patient's best interest from a comfort perspective to anesthetize the patient and close the socket.

### *TMJ Luxation*

The temporomandibular is a joint (TMJ), stabilized by ligamentous soft tissue just as any other joint in the body. Although this joint is not weight bearing, similar stretching of the supporting ligaments occurs when the joint is dislocated. TMJ luxation typically present with dogs and cats unable to close their mouths. Close evaluation reveals the dentition is maloccluded. Frequently these animals may be distressed and the situation typically results from a traumatic event. Reduction of TMJ luxations can occasionally take place simply by opening the mouth but commonly require brief general anesthesia. Reduction of the luxation can be as easy as placing a pencil or pen in the caudal oral cavity (between caudal molar teeth) and closing the mouth. Patients with luxation are at greater likelihood to re-luxate and placing the patient into a loose-fitting muzzle, orthodontic elastics or maxillomandibular fixation (bonding of the canine teeth) for 3 weeks will hopefully give the soft tissues a chance to heal and return to the more restrictive nature.

### **Not Quite Emergencies**

"Not quite emergencies" can encompass a wide variety of complaints and conditions. Ultimately, if the client feels their patient has an emergency, it should be triaged as such. No one wants to turn a client away from a perceived emergency that results in the pet suffering a critical condition.

Feline patients who are not eating are rarely primarily affected by oral disease. Although tooth resorption and feline chronic gingivostomatitis (FCGS) can present as quite painful, neither condition typically results in acute anorexia. Seeing these patients quickly can be important to properly assess them and to make sure there is no underlying severe condition. Seeing these patients also serve to keep the client's emotions in check.

### **Fractured Teeth**

Anyone who has ever fractured a tooth can attest to the intense pain associated with the injury. The nerves present in the pulp chamber and root canal system are directly exposed to the oral cavity and easily triggered to propagate nerve impulses to the spinal cord and brain. The most stoic of veterinary patients may mask the intense pain of pulp exposure. Owners of dogs or cats who have a pulp exposed fractured tooth, typically can be interviewed in a manner which elucidates that the fracture likely occurred due to a specific occurrence. The incident typically results in a change in chewing food, reluctance to play with toys, or increased time required for eating. These animals should be assessed so that the veterinarian can properly inventory the extent of the injuries and properly schedule the patient

for further treatment. It is important to remember that traumatic injury resulting in pulp chamber exposure will always result in a dead pulp and subsequent tooth root abscess. Pulp lacks the regenerative capacity to spontaneously heal over with dentin. Pulp exposure is also where the acute pain and stimulation arise. The only exception to this phenomenon of progressive pulp disease and death is pulp exposure associated with tooth resorption. For an unknown reason, pulp exposure is a predictable result of worsening tooth resorption. Despite pulp exposure during this condition, it rarely develops periapical disease reflective of pulp cavity death.

As stated above, dogs and cats over 18 months of age typically have root canal diameters capable of being appropriately cleaned and shaped if root canal therapy is the client's intention. At this age, the pulp chamber has typically narrowed sufficiently for files to appropriately clean all the walls simultaneously; thus the greatest chance for appropriate cleaning and pulp chamber debridement can occur. Just because root canal therapy does not occur during off hours or the weekend, doesn't mean the patient's symptoms shouldn't be addressed. These patients should be minimally treated with pain medications +/- antibiotics until either root canal or extraction is performed.

Other non-emergencies include noticing discolored teeth or newly bleeding gums. Discolored teeth usually represent teeth that have suffered from pulpitis. Red blood cell lysis results in hemoglobin being released into dentinal tubules and, as it moves towards the dentinoenamel junction, the tooth begins to appear pink/purple. Our best understanding of this condition suggests that over 90% of these discolored teeth are nonvital and either root canal therapy or extraction is indicated. This condition, although believed to be painful, is not an emergency.

Bleeding gums is usually indicative of extremely inflamed gums (severe gingivitis). When severe enough, the gingivitis may result in bleeding with routine brushing, chewing food or playing with toys. If the bleeding is episodic or self-limiting, the patient does not need to be seen urgently, but the condition probably shouldn't be allowed to continue much longer without being seen by a veterinarian. Use of antibiotics and anti-inflammatories can be helpful at providing temporary pain relief to these patients as well making the tissues less friable and healthier for surgical manipulation.

### **Dealing with Clients Who Think the Non-Emergency Is an Emergency**

Often clients may seem to either drastically overestimate or underestimate the pain they feel their pet is in. On one hand, the client is the one living with the pet on a daily basis and should be best at identifying concerning behaviors. On the other, we have been trained as medical professionals to recognize life-threatening and painful conditions. The client is ultimately responsible for paying the bill and maintaining a good word of mouth reputation for the veterinary clinic. Therefore, acknowledging and affirming the client's concerns are important. Minimally, a pet's change in eating habits, playing with toys or nonspecific display of oral pain would benefit from oral pain medications. It is always safe to triage a client's perception of an emergency and, at a minimum, manage the patient conservatively with appropriate medication. Encourage your practice to see these clients as a technician appointment so that a veterinarian can perform a physical exam away from the client if necessary. This may help to reduce the impact that dental emergencies may have on the routine of the practice.

## Summary

Managing dentistry emergencies is a fine balance of listening to the client and properly understanding the nature of the situation. It will never be wrong to encourage that the animal be seen, but certain conditions like tooth avulsion or tooth fracture in young patients and TMJ luxation are conditions that may be time sensitive when considering successful treatment. Take clients' concerns seriously and encourage oral emergencies be seen by your practice to expedite treatment for the truly urgent, and keep comfortable the ones that aren't quite so urgent.

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# How to Do It Right- Local Block Techniques for Dentistry Patients

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Christopher J. Snyder, DVM, Dip. AVDC

Regional anesthesia and pain management are fundamentally important skills to successfully leverage for the benefit of our dentistry patients. Despite these patients being under general anesthesia while undergoing procedures, there are inherent benefits to practicing techniques that will allow for the reduction of inhalant anesthesia, improve recovery and improve the patient's comfort at the time of discharge. Anatomy of the face and mouth is complex. There are many locations and combination of locations where local anesthetics can be administered which will result in regional anesthesia.

Regional anesthesia can offer many benefits by reducing the animal's response to painful stimuli during the procedure as well as provide postoperative analgesia. Local anesthetics work by inhibiting transmission through their effects on Na<sup>+</sup> channels. Effective local blocks are not a replacement for safe, effective general anesthesia. The addition of local blocks to the anesthesia and analgesia protocol will provide the benefits of polypharmacy which can be recognized as threefold. These drugs: (1) prevent peripheral and central sensitization, (2) reduce the adverse effects associated with larger doses of medication and (3) provide better postoperative pain management to smooth out the recovery of the patient.

When given as a local anesthetic, the lidocaine family of drugs provides a variety of options with different onsets of action and different durations of action. Doses should not exceed 5mg/kg in dogs and 2mg/kg in cats. Lidocaine is commonly used in human regional and local anesthesia because a quick onset and short duration of action is desirable. Compliance with human patients for taking oral medications is quite good and return to function (frequently the workplace) is important. In canine and feline dentistry, bupivacaine is a popular medication used off label because of its longer duration of action. Depending on placement the duration of action may be 6 to 10 hours. Recent study information suggests that duration of action may vary by individual dog, but that bupivacaine may last 1-3 days. Time to onset of action is longer with bupivacaine than lidocaine, some texts referring to a 20-minute period necessary before the nerve impulses are effectively blocked. Lidocaine is labeled for veterinary use while bupivacaine does not carry that label for veterinary use. The most common concentration of bupivacaine is 0.5% (5mg/mL) while lidocaine is 2% (20mg/mL). Mixing these drugs should be discouraged until substantiated research is performed determining that the drug remains active and what the concentration of active drug is. A recent publication demonstrated that mixing lidocaine and bupivacaine worked longer than lidocaine alone.

Using the techniques covered in this presentation, it has been the experience of the author that small dosages are sufficient to achieve the desired result of local blockade. Using the techniques discussed, the entire mouth can be anesthetized through the administration of local anesthetic in only four locations.

## Bupivacaine

0.1-0.15mL per site (cat or small dog)

0.2mL per site (medium dog)

0.3mL per site (large dog)

The various blocking locations are listed below.

### Infraorbital Block

*Location:* immediately within the infraorbital canal

*What it blocks:* maxillary incisors, canine tooth, premolars 1-2, +/- 3, buccal mucosa, ipsilateral lip, ipsilateral soft tissue of that side of the face

*What it won't block:* palatal mucosa, PM4 (commonly extracted), may not completely anesthetize for extraction of the central incisors due to crossover innervation

### Caudal Maxillary Block

*Location:* advance the needle parallel with the hard palate trough the infraorbital canal to approximately half the length of the most lateral surface of the zygomatic arch

*What it blocks:* all the maxillary teeth in that quadrant, ipsilateral lip, ipsilateral hard/soft palatal mucosa, ipsilateral soft tissues on that side of the face

*What it won't block:* may not completely anesthetize the central incisors

### Middle Mental Block

*Location:* ventral to the mesial root of the 2nd premolar (dog). Enter in through the mesial aspect of the labial frenulum and place the needle against periosteum half the height of the mandible and centered over the tip of mesial root of the second premolar

*What it blocks:* ipsilateral lip and rostral soft tissues, incisors? and canine tooth?

*What it won't block:* Questionable coverage for the ipsilateral mandibular incisors and canine tooth (probably due to diffusion into the mandibular canal)

## Caudal Mandibular Block (Inferior Alveolar Block)

*Location:* two main approaches

1. Intraoral: half the distance between the angular process and the mucosa immediately caudal to the third molar (lingual side of the mandible)
2. Extraoral: palpate the ventral notch of the mandible, half the distance of the length of the notch, place needle perpendicular to the notch and immediately on the lingual surface, advance needle ½ to 1 cm

*What it blocks:* all ipsilateral mandibular teeth, rostral mandibular soft tissues

*What it won't block:* questionable coverage for caudal mandibular soft tissues, if applied correctly, should not risk anesthetizing the tissues of the tongue

*Note:* It has been shown that intraoral administration of the caudal mandibular block is more accurate than the extraoral approach- this may be useful in helping to reduce the risk of inadvertent blocking of the sensory innervation to the tongue.

Once the needle has been placed, it is important to aspirate, and re-aspirate, while rotating the needle 90° along its long axis to ensure the injection is not given intravascular. Medication should be administered with the needle being placed on periosteum for the middle mental, and caudal mandibular blocks. Even if the bevel is not directly over the nerve, by being deposited on periosteum, the local will cover more surface area and increase the chance that the nerve will be coated. Once the local has been administered, the needle should be withdrawn and digital pressure should be placed for 1 minute to provide adequate time to prevent hematoma formation.

There is reasonable expectation that the addition of opioids to a local block may improve postoperative analgesia long after the effects of the sodium channel blockade wear off. In a study performed in dogs comparing bupivacaine versus bupivacaine + buprenorphine (15mcg) it was shown that 3 of 8 dogs with the combination demonstrated analgesia 72-hours post administration while 2 of 8 dogs experienced analgesia 5 days following administration. It has been well established that  $\mu$  receptors exist in the peripheral nervous system and are up-regulated when exposed to chronic noxious stimulation. Dentistry patients undergoing procedures for acute injuries, such as tooth fracture, are less likely to demonstrate the benefits of opioids in their local blocks as compared to cats with stomatitis or tooth resorption. Chronic conditions may make some drugs work better or last longer.

There are several situations where long term desensitization of a surgery site may be undesirable. Patients suffering from an oronasal fistula already have a loss of bone and a communication between the oral and nasal cavities. Repairing these defects and having the surgery site be completely numb may result in the animal becoming preoccupied with feeling the sutures on their tongue and subsequently tongue thrusting through the surgery site up into their nasal cavity. Similar potential situations exist with maxillectomy patients. It is this author's experience and recommendation that using short acting local anesthetics like lidocaine followed by aggressive post-operative pain management will result in a

comfortable patient after surgery with decreased risk of tongue thrusting. Procedures involving the tongue should never receive local block administration because these patients will be at very high risk of self-trauma and risk “chewing their tongue off.” Use of large volumes when performing local blocks has also been anecdotally reported in resulting in this form of self-mutilation. Sticking with the small volumes and accurate placement afford good results with decreased risk.

Whenever there is potential for the local block needle to traverse through an area of possible tumor, the local block should not be performed. Seeding tumor cells through the infraorbital canal may extremely complicate treatment options available for a maxillary tumor. Using a 25 gauge 1 inch to 27 gauge 1.5-inch needle helps reduce possible nerve injury.

### **Complications**

Complications with local anesthetic blocks have been reported in the literature. Paraesthesia, altered sensation and motor changes are occasionally reported anecdotally from practitioners. It is unclear as to where the origin of nerve injury associated with local anesthesia comes from. While histologic nerve changes associated with local anesthetic administration are reported in veterinary patients (Correspondence: J Anthony), true clinical significance should be considered since similar blocks have been performed in humans for decades with a low incidence of true complications. Peripheral nerve paraesthesia is a rare complication reported in humans. One human dental textbook states it reportedly occurs 1 case in 1 million injections. Peripheral nerve paraesthesia and subsequent self-mutilation of the veterinary patients’ tongue has been only anecdotally reported. The technique for proper needle placement for local anesthetic placement is different than it is for venipuncture. After initial needle penetration, the needle should be guided into position for local administration. When these needles are guided through foramen (as in the infraorbital or caudal maxillary blocks) the needle should be advanced slowly and in most situations the needle bevel with help to displace the neurovascular bundle as the bevel is advanced. Nerves penetrated by needle placement can have variable effects- from no change to permanent sensory or motor dysfunction.

There is a school of thought that nerve injury associated with local blocks may not be directly related to physical damage by needle placement. Peripheral nerve ischemia associated with the addition of epinephrine to a local block may also be associated with nerve injury. The addition of epinephrine to long acting local blocks has therefore been recommended against for that very reason. Beyond the delayed absorption of local anesthetics by the vasoconstriction associated with epinephrine, it has been shown that this catecholamine has some alpha-2 agonist analgesic activity.

The use of small doses in regional anesthesia and aspiration immediately after needle placement can help avoid inadvertent intravascular injection. The most common complications with intravascular injections of local anesthetics include seizures and cardiac toxicity. Bupivacaine has a high affinity for cardiac

sodium channels and can cause brady-dysrhythmias as well as ventricular tachycardia and ventricular fibrillation in humans.

The complications of inadvertent anesthesia of the tongue and iatrogenic globe penetration with the needle while performing the maxillary nerve block should both be effectively prevented by close attention to careful needle placement. Iatrogenic perforation of the globe by a needle during local anesthetic placement has a high mortality rate to the eye.

## Conclusions

Effective local blocks are not a replacement for safe, effective general anesthesia or multimodal postoperative pain management. Use of local anesthetic agents helps to reduce the amount of inhalant general anesthesia required to keep a veterinary patient anesthetized. The unwanted, most frequently seen complications associated with general anesthesia in veterinary patients who are anesthetized for any reason are hypotension, cardiac dysrhythmias, hypercapnea and hypoxemia. Multimodal analgesia anesthesia can help reduce these unwanted side effects by reducing the amount of gas required to keep the patient anesthetized.

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# Malocclusions- Why They Matter and What to Do About Them

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## What are malocclusions?

An animal's occlusion, at the most basic level, is the relationship between the upper and lower arcades of teeth. By design, canines and felines have an anisognathic jaw relationship. This means the upper jaw is wider than the lower jaw and the lower jaw is slightly shorter than the upper jaw. Since canines and felines are carnivores, most of their teeth are designed to exhibit a shearing force on food. The head is a very complicated and dynamic growing structure with very few growth plates serving as growth centers. Appositional bone growth is the type of growth that occurs involving the maxilla while the mandible has growth plates at the TMJ and the coronoid processes.

The maxilla's and mandible's growth, even from the right to left sides, are all independently controlled, however the interdigitation of teeth serve as a checks and balances system to guide synchronous development. Most malocclusions can be traced to either an underlying genetic cause (most common) or may result subsequent to a traumatic event prior to reaching skeletal maturity. Trauma does not always have to result in fractures of bone, the formation of scar tissue and inflammation can be enough to disrupt or restrict appositional bone growth. Pet owners of juvenile animals experiencing head trauma should always be counseled for the unknowns about how a seemingly innocuous traumatic event may result in profound facial asymmetries later.

Any patient with a malocclusion, whether it be a malpositioned tooth in an otherwise normal mouth, or a skeletal malocclusion resulting in a jaw length discrepancy, any treatment should include appropriate genetic counseling. Since most situations of malocclusion are genetic in origin, it should be made clear to the pet owner that breeding of the pet is discouraged and that the pet should *not* be shown. It may be wise to have the client sign a release form releasing liability of the clinic for altering the patient and that counseling against showing or breeding was discussed. Above all, the American Veterinary Dental College's position statement says that animals are entitled to a functional, comfortable bite.

## Classification of occlusions

*Normocclusion:* The normal relationship between the maxilla and mandible should result with the maxillary teeth being buccal to the mandibular teeth, and the mandibular incisors resting on the back (cingulum) of the maxillary incisors. The mandibular canine teeth should rest in the space (diastema) between the maxillary canine and third incisor teeth.

*Class 1 Malocclusion:* This class of malocclusion includes normal jaw length relationship with malpositioning of individual teeth (sometimes referred to as a dental malocclusion.) The most commonly noticed manifestation of this malocclusion is “base narrow” (linguoversion) of the mandibular canine teeth. The abnormal position results in the palatal trauma from the mandibular canines which can manifest itself as a head shy cat or dog. If not treated, the palatal trauma can progress to ulceration, inflammation, bone resorption and development of an oronasal fistula. Having a head shy puppy or kitten is counter to the desirable handling skills we encourage pet owners to establish with their pet at a young age.

Crossbites and mesioverted mandibular canine teeth are both examples of Class 1 malocclusions. A crossbite refers to the buccal-lingual relationship of occluding teeth being reversed from normal. An example of an anterior crossbite would be where one or more mandibular incisor teeth are tipped forward relative to the occluding maxillary incisors. Since the normal self-cleaning mechanisms are absent with this relationship, consideration for orthodontic movement into a normal position could be considered. Patients with this malocclusion frequently have a functional and comfortable bite so intervention is less important. Mesioverted maxillary canine teeth are referred to as “lanced canines” since the canine teeth erupt pointed forward instead of erupting down and most commonly noted in Shetland Sheepdogs or pure-bred cats. This condition isn’t noted until eruption of permanent dentition. The forward position of the maxillary canine teeth results in unerupted tooth crown which forms a periodontal pocket predisposing periodontal disease. Oriented this way, these teeth exhibit little function and should be extracted or orthodontically moved into normal position.

*Class 2 Malocclusion:* The occlusal relationship between the jaws for this malocclusion results in the maxilla being relatively longer than the mandible. This may be due to an excessively long maxilla (maxillary prognathism) or a shortened mandible (mandibular brachygnathism.) Due to the anisognathic relationship with the narrow mandible and wide maxillary width, the discrepancy does not have to be very severe before the mandibular canine teeth start causing palatal trauma. Treatment to alleviate the traumatic contact is recommended.

*Class 3 Malocclusion:* The occlusal relationship with these patients includes the mandible being longer than the maxilla. This can be due to maxillary brachygnathism or mandibular prognathism depending on which jaw is abnormal. This class of malocclusion is normal for certain breeds (Boxers, Pugs, Shih-tzus, Boston Terriers, Burmese cats, etc.) Patients with this occlusion should be evaluated for whether mandibular incisor teeth are causing soft tissue trauma to the lingual mucosa/mandibular incisors but otherwise usually do not involve intervention.

*Class 4 Malocclusion:* Frequently referred to as asymmetrical skeletal malocclusions, this class of malocclusion typically results following a traumatic event. Examples of this malocclusion can manifest as asymmetry in a rostrocaudal, right-to-left, or dorsoventral direction. Animals with this class of malocclusion should be screened for traumatic contact between occluding teeth as well as the extent of

aberrant function resulting from the malocclusion. Treatment for these conditions could be considered in effort to improve the self-cleaning relationship between occluding teeth.

## **Malocclusion Treatments**

*Interceptive orthodontics* refers to the treatment of the deciduous teeth associated with a malocclusion. Very young patients with a Class 2 malocclusion may demonstrate palatal trauma from the deciduous mandibular canine teeth. These patients may be unthrifty, very head shy or demonstrate challenges nursing. All symptoms stem from their pain in the oral cavity. A very practical rule of thumb is that any time a deciduous tooth is creating a problem, the correct treatment is extraction. Radiographic evaluation of the deciduous tooth being extracted should always be performed to ensure it is well known the extent of the root to be removed as well as to document the development of the forming permanent tooth. Extreme care must be taken to remove deciduous teeth to avoid damaging the developing permanent tooth and clients should always be warned (and it documented in the medical record) that careful extraction of the deciduous tooth was performed and if there was any damage to the permanent tooth, it won't be known until eruption. Interceptive orthodontics is typically only necessary for base narrow mandibular canine teeth or Class 2 malocclusion patients demonstrating palatal trauma. If deciduous mandibular canine teeth do not appropriate exfoliate during eruption, the permanent canine teeth will erupt lingual resulting in palatal trauma.

*Crown reduction* and endodontic treatment alleviates issues with traumatic contact by removing a portion of the tooth crown. Removing a portion of the tooth maintains function of the tooth while alleviating the need for an extensive recovery from surgical extraction. Palatal trauma from mandibular canine teeth is a common clinical situation where this is performed. Reduction of crown height exposes the pulp chamber and warrants endodontic treatment. Typically, orthodontic treatment is performed in young patients with wide pulp chambers. Efforts to maintain pulp vitality is achieved by partial pulpectomy and vital pulp therapy being performed. The goal of therapy is to remove a portion of the pulp, place a medicament on the pulp to encourage continued dentin deposition, followed by restorative materials to resist bacterial penetration. Vital pulp therapy may be less invasive than extraction but does require multiple anesthetized radiographic evaluations which incurs cost.

*Active force orthodontic tooth movement* involves movement of the tooth itself, usually with a tipping force. Use of orthodontic elastics, anchored on other teeth, anchored on transmucosal screws or use of springs or other fabricated orthodontic devices provide the anchorage or the force that facilitates movement. Care must be taken to exert light constant forces when orthodontic elastics are used to prevent inflammation and risk root resorption or ankylosis. This type of treatment device is typically used with mesioverted maxillary canine teeth or to correct crossbites. General anesthesia may be necessary to collect stone models for fabrication of the device at a prosthodontic laboratory, to install the device and for device removal.

*Passive force orthodontic tooth movement* utilizes typically custom fabricated appliances formed and trimmed chairside or in the patient's mouth. Cold cure, bis-acryl composite is a popular class of materials applied to the oral cavity to create tipping forces on target teeth. The tipping force of passive devices is generated when the animal closes its mouth and it is the interaction between the target tooth and the composite or anatomy. An example of this type of device include crown extensions. Mildly base narrow mandibular canine teeth are an example of a clinically useful situation where crown extensions would be beneficial. The divergent orientation of the mandibular canine teeth lends themselves to building up composite on the cusp tips to direct the crowns into the diastema with a lateral tipping force during eruption. In complicated orthodontic movements, application of crown extensions may be necessary to provide extra height to serve as a retainer after ending up in the desired location.

The most commonly utilized example of a passive force orthodontic device includes inclined planes, sometimes referred to as a "bite plate." Fabrication of composite material to the roof of the mouth allows ramps to be created in the composite to guide the contacting teeth into a desired position. The composite material is brittle and patient compliance is key to not fracturing the device. Anesthesia is necessary for placement, +/- modification and removal of the device. Most inclined planes will successfully tip teeth in young patients within 8-12 weeks. Once teeth are moved into the desired location, either the inclined plane should remain in place for 3-4 weeks as a retainer, or once the device is removed the new position of the tipped teeth must be self-retained in position.

*Ball therapy* is sometimes mentioned as a low risk, low yield method of achieving orthodontic movement. This technique is probably most useful for mildly linguovered mandibular canine teeth and requires the patient to hold an appropriately sized ball in the front of the mouth for 5-10 minutes, 2-3 times per day. The force of biting down on the ball generates tipping forces in the buccal direction and in actively growing animals, this may be sufficient if slight movement is necessary. Biting on the knotted end of a rope toy may also provide similar application of forces.

When teeth occlude as nature intended, they comfortably optimize functional chewing while simultaneously self-cleaning. Early identification of abnormal occlusions affords the veterinary team the opportunity to intervene and provide the best opportunity for a functional comfortable bite. Counseling clients as to the underlying cause of the malocclusion is important to either anticipate the development of a painful condition or reduce the likelihood of perpetuation of the condition.

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# An Overview of Endodontic Therapy- Who Is the Right Candidate and Why?

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Damage to the tooth structure is not only a source of discomfort for the patient, but may also serve as a source of local infection or inflammation around the root apex. Depending on concurrent injury involving this area, the presence of infection or inflammatory mediators may inhibit bone healing. Disruption of the blood supply to the tooth would predictably result in ischemia and tooth death. The pulp chamber receives blood supply from a variety of locations including direct blood supply from the superior and inferior alveolar arteries, the periodontal ligament and gingiva. It is the contributions by the collateral sources of blood supply that result in some teeth maintaining vitality following major artery transection due to trauma or oncologic surgery. In situations where the tooth is compromised, knowing appropriate treatment options is important.

## Indications for Endodontic Treatment

The tooth is made up of enamel, dentin, cementum and pulp (soft tissue structures including arteries, veins, nerves and connective tissues). Once erupted, the tooth's hard tissues have limited capacity for healing following injury. Enamel is of epithelial origin and is formed from ameloblasts prior to tooth eruption. Damage to enamel following eruption results in irreversible loss of structure. Loss of enamel results in exposure of dentin, a tubular structure radiating out from the dental pulp (arteries, veins, nerves). Dentinal tubules contain free fluid responsible for nourishment of odontoblastic processes extending down the tubules from the odontoblasts, which line the periphery of the pulp chamber. Exposure of dentinal tubules results in dentinal fluid shifts which is perceived by tooth sensitivity or pain. While dentinal tubules are small enough to prevent the passage of red blood cells out to the surface, the tubules are large enough for bacteria to percolate through the tubules and infect or incite an inflammatory response into the surrounding periapical tissues.

The purpose of endodontic treatment is to relieve the patient from sources of chronic pain or infection. Depending on the severity and underlying cause of tooth injury, treatment goals of endodontic therapy are to either maintain/preserve the vital tooth (maintain pulp vitality) or to remove exposed or infected pulp and apply treatment necessary to preserve tooth function in a non-vital tooth.

The most common indications for endodontic treatment include: tooth fracture, tooth discoloration (intrinsic staining), evidence of periapical radiolucency and evidence of pulp chamber widening. Discolored tooth crowns demonstrate blood or blood byproducts becoming exposed close to the dentinoenamel junction. One study has suggested 92.2% of discolored teeth are non-vital. Radiographic

evaluation is necessary both for appropriate diagnosis, and also for documenting and evaluating endodontic treatment.

## Types of Endodontic Treatment

### Root Canal Therapy

The most commonly performed endodontic therapies recommended to clients involve vital pulp therapy (direct or indirect pulp capping) and root canal therapy. Less commonly performed endodontic procedures are specifically aimed at supporting the body to continue development of the root apex (apexification, apexogenesis) for the purpose of performing a standard root canal at a later time.

Root canal therapy can be broken down into three fundamental steps: *instrumentation*, *sterilization* and *obturation*. *Instrumentation* is centered on accessing the root canal system with as straight-line access to the root apex as possible. Upon gaining access to the root canal system, a series of endodontic files are placed into the canal to mechanically debride: pulp, debris and the smear layer. This step is particularly important since the mechanical removal of root canal contents take place in a pulp chamber that is typically oval or ribbon shaped and the use of serially larger endodontic files results in preparation of the canal into a round column. Instrumentation is initially performed using a dental high-speed hand piece and carbide or diamond bur. Initial stages of canal preparation are performed with short files designed to create a 'funnel' shape at the access site. This initial canal preparation serves to direct longer files to the root apex and to serve as a reservoir for chemical lubricants or sterilizing agents. A variety of files and instruments exist in clinical practice to facilitate treatment of the entire length of the canal. Hand files and rotary file systems are two of the most commonly used systems available. Hand files have a working segment of the file and are standardized by International Organization for Standardization (ISO) sizes. K-file and H-file (Hedstrom file) shapes are machined and used slightly differently. File choice is typically based on operator's preference. Several rotary systems exist using nickel titanium (Ni-Ti) files. These files may have similar tapered 16mm of cutting surface on each file, or otherwise contain 3-4mm of a cutting portion on the working end. Ni-Ti files demonstrate greater flexibility than hand files and rotary systems demonstrate reduced operator fatigue. Following root canal preparation, the tooth is inherently weaker due to loss of tooth structure. When considering the removal of tooth structure, mechanical treatment of the tooth creates an inherently weakened tooth. Rotary systems offer the advantage that the prepared root canal system is cylindrical and uniform the length of the preparation.

The *sterilization* step involves the use of sterilizing agents are used to aid in the passage of the endodontic files into the root canal. Some sterilizing agents also serve as canal lubricants to facilitate file passage and prevent binding. Extensive research has been performed evaluating the effectiveness of various irrigants for the purpose of sterilization. Various strengths and temperatures of bleach are commonly used, however chlorhexidine has been proposed as an alternate irrigant if patency of the apex is of concern (chlorhexidine digluconate is less irritating to periradicular tissues.) Following mechanical and chemical debridement of the canal, the canal is dried using ISO sized paper points.

*Obturation* is the step involving placing a sealer cement into the prepared canal followed by gutta percha points. Gutta percha is a rubber-based material that does not undergo volumetric expansion or contraction. Regardless of the instrumentation technique, the goal of obturating the canal is to maximize the amount of gutta percha and minimize the amount of sealer cement. Many sealer cements contract during curing which creates space or voids which can be occupied by opportunistic bacteria. After the initial gutta percha plug (master cone) is placed snugly into the canal assuring a tight fit in the root apex, the canal is backfilled with additional gutta percha cones. The access opening is then filled with a composite restoration. Amalgam restorations have fallen out of favor compared to light curable composites.

In instances of file separation during treatment or normograde root canal failure, surgical endodontics may be a treatment option. In *surgical endodontics*, an incision is made in mucosa and an alveoloplasty is performed exposing the root apex which is removed and a restoration performed. In teeth demonstrating failed root canal therapy, surgical endodontics offers the advantage of removing the diseased root apex and apical delta where residual bacteria may reside.

#### Direct/Indirect Pulp Capping

Direct and indirect pulp capping refers to providing endodontic therapy to a tooth with the goal of maintaining long-term survivability of the tooth. The advantage of these procedures is to encourage the tooth to continue to develop secondary dentin, thus strengthening the tooth as compared to filling the tooth with sealer cement and gutta percha.

*Direct pulp capping* is performed in conjunction with vital pulp therapy. This procedure is performed following deliberate (controlled) removal of tooth structure, thus exposing the pulp or is performed in traumatically fractured teeth resulting in pulp exposure. Direct pulp capping should be performed within hours of injury to provide the best opportunity for treatment success. Steps involved in this procedure include removal of 6mm of coronal pulp. Following coronal pulpectomy hemostasis may be achieved with cold saline and sterile paper points. Two millimeters of pulpal dressing is applied onto the pulp surface. Mineral Trioxide Aggregate (MTA) and calcium hydroxide are commonly used materials. Following radiographic success of MTA/calcium hydroxide placement, an intermediate layer of glass ionomer is placed and light cured. The cavity preparation is performed leaving 2 mm of composite restoration at the access site.

*Indirect pulp capping* is performed in teeth undergoing acute loss of tooth structure without obvious pulp exposure. Similar treatment is performed without the removal of pulpal tissue or direct exposure of the pulp. Thinner layers of materials are placed for indirect pulp capping, however follow up of both procedures remains the same. Careful consideration for whether indirect pulp capping or just a dentinal sealant is indicated must be evaluated. Depending on the proximity to pulp exposure, dentinal bonding agents and filled composites are indicated most of the time.

Follow up for direct/indirect pulp capping is centered on visual and radiographic evaluation of the treated tooth. Tooth discoloration, draining tracts, facial swelling, periapical radiolucency or failed continued development of pulp chamber (signified by continued pulp chamber narrowing) or lack of dentinal bridge formation are all indications for a failed pulp capping procedure. If the tooth is sufficiently mature, standard root canal therapy can be an alternative to extraction. Radiographic evaluation should take place every 6 months for 2 years and then yearly thereafter.

### **Treatment Decision Making**

The age of the patient should be a consideration when determining which of the aforementioned endodontic treatments should be performed. Root canal treated teeth will likely never be as strong as a healthy tooth and teeth receiving vital pulp therapy will require a lifetime of follow up. Injured teeth in young animals have less secondary dentin and are structurally weak. Opportunities, when appropriate in these patients, should be taken to provide treatments that err on the side of maintaining tooth vitality in order for additional secondary dentin to be deposited. Older patients demonstrating pulp exposure will deposit less secondary dentin during their remaining lifespan a smaller pulp chamber may benefit from standard root canal therapy.

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## Cecilia Robot DVM, DACVIM (Oncology)

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# Top 10 Signs of Cancer in Dogs

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Cecilia Robat DVM DACVIM (Onc)

The facts:

- 1) 1/4 to 1/3 dogs will develop cancer
- 2) 1/2 dogs over the age of 10 years will die of cancer and 1/3 younger dogs will die of cancer
- 3) The earlier cancer is diagnosed, the earlier treatment can be initiated and the better the outcome (usually!)

Breed predispositions:

- 1) 1/5 Golden Retrievers will be diagnosed with hemangiosarcoma
- 2) 1/8 Golden Retrievers will be diagnosed with lymphoma
- 3) 1/4 Bernese Mountain dogs (BMDs) will die of histiocytic sarcoma (HS). BMDs are 225 times more likely to develop HS than any other breed.
- 4) 55.1% of Bernese mountain dogs and 63.8% of Flat-coated retrievers die of malignant tumors

Treating cancer in pets:

The most important element to always remember is that the goal of therapy in dogs and cats is very different than the goal of therapy in humans with cancer. In the veterinary world it's all about quality of life, not quantity of life. It's not because we *can* treat that we *should* treat. Pet owners should be provided with the facts, to be able to make an educated decision to treat or not to treat, and if they decide to treat, which route they want to pursue. During an Oncology appointment clients are provided with all treatment options, from least aggressive to most aggressive. Less than 20% dogs develop side effects because we treat with lower doses of chemotherapy and administer treatment less frequently.

Top 10 signs of cancer in dogs

## 1) Lump or bump

It's IMPOSSIBLE to tell if a mass on a dog is benign or malignant by simply looking at it or feeling it!! Masses can feel just like lipomas (benign fatty tumors), yet be a malignant tumor such as a soft tissue sarcoma or mast cell tumor. Any mass on a dog greater than the size of a pea should be investigated. If the mass has been growing fast, bothers the dog, is ulcerated or changing in consistency it should be sampled sooner rather than later. When a mass is found on a dog, it should be mapped (ie measured and

its location noted in the chart). Ideally a mass should be sampled, starting with a fine needle aspirate. These are easy to perform, cheap, do not require anesthesia, and are associated with very little morbidity for the dog. In addition, an answer can be obtained quickly, within 24 hours, when sent out to a clinical pathologist. When slides are sent out to the laboratory, always check them first to make sure the sample is adequate quality for the pathologist to be able to make a diagnosis. Some tumors do not exfoliate well, meaning that a fine needle aspirate will not provide an adequate sample. In these cases, a biopsy should be performed. Anytime a mass is removed it should be submitted for analysis.

#### a) Mast cell tumors

These are common skin tumors in dogs. Certain breeds are predisposed, such as Boxers, Rhodesian Ridgebacks, Pitbulls, Bulldogs and Pugs. They are situated on or under the skin and can look and feel like anything! Any dog diagnosed with a mast cell tumor should be placed on antihistamine drugs. Surgery is the treatment of choice for MCT. The need for further staging (ie how far has the cancer spread) or chemotherapy will depend on several factors, most importantly the grade of the tumor. This can only be obtained after histologic analysis of the tumor. There are 2 grading systems in dog (Patnaik: 1, 2, 3, and Kiupel: low/high). Pathologists should be reporting both grades until further information is obtained on the accuracy of these grades in predicting clinical behavior of MCTs. Prognosis for most low grade mast cell tumors is excellent. It is guarded for high grade tumors.

#### b) Lipomas

These benign tumors usually do not increase in size. They are subcutaneous and usually soft, but can be firm as well. They can only be diagnosed by fine needle aspiration and cytology.

Lipomas do not generally need to be removed surgically unless they are in a location causing discomfort or become ulcerated (usually ventral thoracic/abdominal lipomas, due to pressure when the patient lies down).

#### c) Soft tissue sarcomas

This is a group of diverse tumors arising from the skin and subcutaneous connective tissues, such as fibrous tissue, muscle, fat, cells along nerves etc. These tumors are grouped under the umbrella term "soft tissue sarcoma" (STS) because they all behave the same, are treated the same and have the same prognosis. Examples of common STSs are peripheral nerve sheath tumor and fibrosarcoma. STS are very locally invasive. This makes it very difficult to remove STSs surgically (beware of what the pathologist calls "clean" – you want to make sure all margins are at least 5mm). Early diagnosis is vital with these tumors. Metastases are uncommon with STSs. There are 3 grades of STSs (determined after removal and histopathology analysis). Grade is associated with behavior and outcome. Surgery is the treatment of choice. If margins are incomplete, further therapy should be considered such as a second surgery (if possible) radiation therapy or metronomic chemotherapy. Overall prognosis is excellent for soft tissue sarcomas treated surgically.

## 2) Changes in urination

### a) Increased urination

Polyuria and polydipsia (PU/PD) can occur secondary to high calcium values in dogs with certain neoplasias, the most common being lymphoma, anal sac gland adenocarcinoma and multiple myeloma. One of the mechanisms by which this occurs is the production of a “fake” parathyroid hormone by the cancer cells (parathyroid hormone related protein – PTHrp). When hypercalcemia is diagnosed, further testing can be performed through Michigan State University – indeed the hypercalcemia panel will dose ionized calcium (metabolically active form of calcium) as well as PTH and PTHrp to help orient the diagnosis. Hypercalcemia may lead to GI upset and kidney damage and should be addressed promptly. Effectively treating the tumor will result in decreased calcium values. In cases of severe hypercalcemia hospitalization is necessary.

### i) Lymphoma

Lymphoma is the most common cancer diagnosed in dogs, usually after a pet owner has found a mass in the dog’s neck area (mandibular lymph node). Lymphoma can occur at any age, and is more common in certain breeds (Golden Retriever, Boxer, Beagle, Bulldog). There are 2 common types of lymphoma recognized in dogs: B cell and T cell. B cell lymphoma tend to carry a better prognosis with treatment than T cell lymphoma. Lymphoma is very treatable with chemotherapy, but rarely curable. Outcome is reported in “median survival times” which means that 50% patients do better and 50% do worse. With standard of care protocols (“CHOP” aka “UW Madison lymphoma protocol), average survival times are 10-12 months for B cell lymphoma and 6-8 months for T cell lymphoma. Palliation with steroids (prednisone) results in a median survival time of 6-8 weeks.

### b) Decreased urination

Decreased overall volumes of urine or multiple small micturitions can be present in patients with cancer. Bladder, prostate or urethral cancer will result in pollakiuria, stranguria and hematuria. Anal sac tumors may lead to difficulty urinating or defecating.

### i) Transitional cell carcinoma (TCC)

This is the most common bladder cancer in dogs. It can also develop in the urethra or prostate. Certain breeds are overrepresented such as Scottish Terriers, Shelties, Beagles and Westies.

Most dogs present with a history of urinary tract infections. Patients with recurrent urinary tract infections should be screened for underlying diseases such as bladder stones, neoplasia or metabolic diseases. Definitive diagnosis of TCC can be obtained by exfoliative catheterization or biopsy via cystoscopy. A cystocentesis should never be performed in a dog with a possible TCC as this can lead to seeding of the needle tract by cancer cells. Treatment is rarely surgical and usually includes a combination of non-steroidal anti-inflammatory drugs and chemotherapy. Median survival times range from 3-12 months.

### 3) Changes in defecation

Chronic diarrhea, especially when associated with weight loss, or blood in the stool (frank blood or melena) can be signs of intestinal neoplasia. Most common intestinal cancers in dogs are lymphoma, adenocarcinoma (cancer of the lining cells of glands) and leiomyosarcoma (cancer of smooth muscle)

Straining to defecate can occur when a mass is present, compressing the colon/rectum or narrowing the anal opening. This mostly occurs with prostatic tumors, enlarged metastatic lymph nodes or with anal sac tumors.

#### i) Anal sac gland adenocarcinoma (ASGACA)

ASGACA is a relatively common cancer in dogs. Cocker Spaniels and German Shepherds are overrepresented. Early diagnosis is key with this disease and outcome can be very good if the tumor is caught early and treated appropriately. In order to achieve this, a rectal examination should be part of every physical examination! Up to 50% dogs with anal sac tumors may present with hypercalcemia of malignancy. ASGACA will metastasize to local lymph nodes in the pelvic canal, as well as to the liver, spleen and lungs. Metastatic rate is around 50-80%. Treatment of choice is surgery. Chemotherapy and radiation therapy are recommended in select cases. Prognosis depends on extent of disease and treatment and ranges from 3 months to several years.

### 4) Bad smell

When a dog presents with the complaint of bad breath, a thorough (sedated) oral and pharyngeal examination should be performed. Oral tumors are common in dogs. They can arise from anywhere in the mouth. Most common oral tumors are melanoma, squamous cell carcinoma, fibrosarcoma, osteosarcoma and benign epulides.

#### i) Melanoma

Malignant melanoma is the most common oral tumor in dogs. Chow Chows, Golden Retrievers and Cocker Spaniels are overrepresented. These are highly metastatic tumors. Treatment of choice is surgical excision +/- radiation therapy followed by chemotherapy and/or immunotherapy (Melanoma Vaccine), although these modalities are only associated with an approximately 25% response rate. Prognosis ranges from 2 months to 18 months on average.

#### ii) Fibrosarcoma (FSA)

These tumors are very locally invasive with a low metastatic rate. There is a subtype of oral FSA known as “high-low”. These occur most commonly on the maxilla of Golden Retrievers. They are biologically high grade (grow fast, recur and spread readily) but histologically low grade (read out as low grade FSA on histopathology, or even as benign fibromas or scar tissue). Average survival time for oral FSA is around 1-1.5 years with treatment.

## 5) Weight loss

It’s important to regularly monitor a patient’s weight. Undesired weight loss could be the first sign of cancer. Focal weight loss (ie muscle wasting) should also be considered suspicious as tumors of nerves or affecting nerves can lead to rapid denervation of muscles.

## 6) Lethargy

Lethargy, especially if sudden or lasting more than a couple of days, could be a sign of cancer.

Hemangiosarcoma is a common cancer in dogs. It most commonly arises in the spleen of older dogs, with German Shepherd dogs and Golden Retrievers overrepresented. The most common clinical presentation is acute lethargy with pale gums +/- a distended abdomen due to tumor rupture. Treatment of choice is surgery followed by chemotherapy. Other therapies such as Yunnan Baiyao (Chinese herb that may help reduce bleeding) or I’m Yunity (coriolus mushroom, may have immune stimulating properties) may be beneficial. Median survival time ranges from 1 months to 10 months with therapy.

## 7) Decreased appetite

The first and most important question is whether the patient *can’t* eat or *doesn’t want to* eat. Patients who can’t eat may have oral pain (tumor, dental disease ...) or facial pain. Owners may report that the patient is eating normally when offered “people food” but will not eat his/her regular kibble. That qualifies as inappetence and should not be considered normal.

## 8) Respiratory problems

Owners may report that their pet is not able to go on normal walks, has less energy for normal games, pants excellively, has raspy breathing, a cough or nasal/ocular discharge. These clinical signs could be occurring secondary to a nasal tumor, throat/tonsil tumor, lung tumor or thyroid tumor.

### i) Nasal tumors

These are more common in dolicocephalic dogs. These tumors have a low to moderate metastatic rate. Clinical signs include recurrent/chronic nasal discharge, epistaxis, facial swelling, eye bulging/discharge,

snoring. Computed tomography is the imaging modality of choice to investigate the nasal cavity. A definitive diagnosis can be obtained on biopsies of the nasal mass. If facial swelling is present, or if the mass extends into the nasal cavity, a fine needle aspirate may be diagnostic. Treatment of choice is radiation therapy. Median survival time is 12-18 months with therapy.

ii) Thyroid tumors

These are most common in Boxers, Beagles and Huskies. It is VERY important to always feel a dog's neck during a physical examination. Prognosis is excellent when caught early and removed surgically. These tumors readily grow into surrounding structures and once this occurs, surgery is no longer an option. Palliation includes radiation therapy and chemotherapy.

**9) Discharge from anywhere**

Discharge from any location, especially when chronic or recurrent, should be investigated as it could be the sign of neoplasia. For example, nasal tumors will result in chronic mucous/mucopurulent or hemorrhagic discharge. Retro-orbital tumors (can be nasal in origin) may lead to exophthalmia and ocular discharge.

**10) Pain**

Osteosarcoma is the most common bone tumor in dogs. It can occur in very young (1.5 year old on average) and older dogs. Large and giant breed dogs, especially Rottweilers and Greyhounds are overrepresented. Lameness, pain or swelling that does not improve rapidly with rest/NSAIDs should be investigated with a radiograph. Standard of care therapy includes amputation and chemotherapy. Median survival times are around 10-12 months with treatment.

# Chemotherapy: How it Works and Chemotherapy Safety

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Cecilia Robat DVM DACVIM (Onc)

## 1) Chemotherapy

Chemotherapy is a systemic treatment. It is used to prevent metastasis, treat systemic disease and in some cases to shrink a tumor prior to surgery. It kills rapidly dividing cells which are cancer cells as well as normal rapidly dividing cells. This “collateral damage” on normal cells results in side effects of chemotherapy. Fewer than 20% veterinary patients experience side effects.

Gastrointestinal side effects can occur 3-5 days post treatment. Damage to the bone marrow, resulting in low bloodcounts usually happens 1 week post treatment. The most common finding is neutropenia. It's vital to perform a complete bloodcount 1 week post chemotherapy (10-14 days with carboplatin) to monitor neutrophil counts. Antibiotics may be needed if the neutrophil count is very low. Alopecia rarely occurs in veterinary patients.

## 2) Chemotherapy safety: why all these precautions?

Chemotherapy agents (aka hazardous drugs) are toxic. They can cause cancer, rashes, gastrointestinal upset, genetic mutations, reproductive abnormalities. The big problem is that these agents are usually odorless and aerosol is invisible. Historically, before safety procedures were implemented, chemotherapy residues were found in lots of hospitals, on medical workers. Several studies have been performed in veterinary medicine. In a 2010 study of American veterinary teaching institutions, surface contamination was found in 10% of swabs in preparation areas. It's likely that the increasing use of biological safety cabinets and closed system devices has contributed to the decrease in environmental contamination by hazardous drugs.

More and more veterinarians give chemotherapy. In the UK, 71% practices reportedly administer chemotherapy. In a survey of Canadian clinics, 30% general practitioners reported using chemotherapy. Many general practitioners and their staff do not have adequate training for the handling and administration of chemotherapy. In addition, more and more pets receive chemotherapy, including chemotherapy orally at home. Residues are present in urine/feces/saliva and vomitus. The Center for Disease Control (CDC)'s National Institute for Occupational Safety and Health (NIOSH) published a document in 2010: Safe Handling of Hazardous Drugs for Veterinary Healthcare Workers. In 2018, a consensus document on the Safe Use of Chemotherapy in Practice was published in the Journal of Veterinary Internal Medicine (available for free online). The major focus of this document was the safety of people involved in chemotherapy handling.

Routes of exposure to chemotherapy are multiple. For staff, preparation, administration of chemotherapy as well as contact with pets the day of hospitalization for treatment or when in the hospital for treatment of side effects. Clients administering oral chemotherapy at home and any pet parent of a dog/cat having received chemotherapy in the hospital are at risk of exposure to chemotherapy agents, either directly (oral at home administration) or via residues in urine, feces, saliva and vomitus. Routes of exposure include oral, aerosolization and through skin.

### 3) Training

Training of personnel involved in handling of chemotherapy will be key to minimize environmental and staff contamination. It's well known that people are more likely to be compliant with recommendations when the risk is well understood. Several states now mandate following NIOSH guidelines. In addition, new guidelines will soon be implemented. The USP is a nonprofit scientific organization that develops and disseminates guidelines for medicines, setting professional standards for pharmacy and drug manufacturers. USP800 applies to hazardous antineoplastic drugs. It will be implemented in 2019 and sets stricter rules to further protect workers in contact with hazardous drugs.

Training includes providing adequate documents to staff such as safety data sheets, drug insert information, as well as information on personal protective equipment, closed system devices and cleaning.

### 4) Storage

The risk of contamination starts at delivery of the hazardous drugs. Several studies report 0-100% surface contamination of chemotherapy vials and bottles. Vials/bottles should be labeled clearly and stored in a separate cabinet or refrigerator and in a dedicated and identified room. An inventory should be kept including lot numbers. Personal protective equipment (PPE) should always be worn when handling chemotherapy vials/bottles.

### 5) Preparation

No food or drinks should be allowed in the preparation area. The chemotherapy room should ideally be a negative pressure room (USP800). Surfaces should be cleaned with a deactivating, decontaminant agent such as dilute bleach- most common cleaning products contain bleach. This should not be sprayed directly on to the surface due to risk of chemotherapy aerosolizing but rather sprayed on paper towels first. Drugs should be drawn up under a hood (biological safety cabinet – BSC).

When preparing injectable chemotherapy personal protective equipment (PPE) should be worn (gowns, head, hair, and shoe covers, and two pairs of chemotherapy gloves are required). For administering injectable chemotherapy, two pairs of chemotherapy gloves and a gown are required.

Closed system devices (CSDs) minimize the risk of contamination. They close the main route of contamination which is the connection between the chemotherapy-containing syringe and the catheter. Many types of CSDs are available. They are relatively cheap and easy to use.

Chemotherapy spills are rare but if they do happen, it's vital to be prepared. The most common spill occurs after a vial breaks. Spill kits should contain an extra set of PPE, absorbing pads, bleach, two chemotherapy safe waste bags, a disposable scoop and puncture-resistant container to pick up and dispose of glass fragments.

Individual chemotherapy doses should be drawn up under a hood. The syringe should be labeled and placed in a ziplock bag labelled with the patients' name, drug, dose and placed in a dedicated plastic bin (identified as containing chemotherapy) or refrigerator until use. The preparation area should be cleaned immediately and waste disposed up in a dedicated chemotherapy container. Hands should be washed with soap. The same precautions should be taken with oral drugs. Tablets should not be crushed or cut and capsules should never be opened. A prescription should be written by the veterinarian in charge for each patient, and checked by another person. The prescription should contain the date, patient's name, weight (FROM THE DAY OF THE VISIT!) in kilograms, body surface area, bloodwork results, drug, dosage, dose, route of administration, pharmacy details. The prescription/chemotherapy treatment sheet should always be in the patient's record.

## 6) Administration

The 5 "R"s of administration are: right patient, right drug, right dosage, right dose, right route of administration. If the answer is "yes" to all of these, then it's ok to proceed. Supplies should be prepared in advance (chucks, catheter, tape, wrap, flush, chemotherapy dose in zip-lock bag, bandage). Injectable chemotherapy should always be administered through a perfectly placed catheter, and never in a bag of fluids, through a butterfly needle or as a direct intravenous injection. PPE should be worn, even for oral and subcutaneous chemotherapy. Ideally, 2 people should hold the patient and also wear PPE. A catheter should be placed, using a regular aseptic method. If the placement is not perfect, then the catheter should be removed and placed elsewhere. The catheter placement should be checked by flushing with saline, and observing a "flash" of blood when you draw back. Chemotherapy is then administered following guidelines for the individual drug, and then the catheter should be flushed before being pulled. Saline containing heparin should not be used as some chemotherapy agents will precipitate when in contact with heparin. In addition, there is a higher risk of bleeding and spilling of chemotherapy. Administration should always be documented in the patient's chart with the drug administered, limb used and person administering the treatment. Disposal of all materials post chemotherapy administration should be performed in a dedicated waste bin, labeled, leak-proof and covered. Patients should be housed in a low-traffic area and their cage labelled appropriately.

**a) Doxorubicin**

Doxorubicin should be administered slowly (1ml per minute maximum) due to the risk of acute arrhythmias and allergic reaction. Every precaution should be taken to avoid extravasation as it will cause severe tissue destruction that will progress up the limb and can, in severe cases, necessitate amputation of the affected limb. If extravasation occurs, it should be contained. Doxorubicin can also cause cardiotoxicity – this is usually cumulative but can occur after one dose in dogs with preexisting heart disease. Breeds at risk such as Dobermans, Great Danes, Boxers, Newfoundlands, Irish Wolfhounds, Cocker Spaniels should have a cardiac assessment prior to starting treatment with Doxorubicin. In cats, doxorubicin is nephrotoxic.

**b) Vincristine**

Vincristine is administered as a slow bolus. Extravasation of vincristine can cause severe sloughing of the tissues. If extravasation occurs, the drug should be spread (unlike Doxorubicin). Rarely, vincristine can cause a peripheral neuropathy and dogs can present with hindlimb weakness, cats with ileus/constipation.

**c) Cyclophosphamide**

Cyclophosphamide (“Cytoxan”) can cause sterile hemorrhagic cystitis (SHC). Cyclophosphamide is an inactive drug, metabolized in the liver into active metabolites and inactive metabolites, one of which can irritate the bladder wall. This metabolite is called Acrolein. SHC occurs in 1% patients of maximum tolerated dose of cyclophosphamide, when administered concurrently with a diuretic, and in up to 20% patients on metronomic chemotherapy. Symptoms of SHC are similar to those of a urinary tract infection. A urinalysis performed when symptoms occur will show presence of red blood cells but no to rare white blood cells and no bacteria. Cyclophosphamide should be discontinued immediately if this occurs. Treatment is mostly symptomatic.

**d) Carboplatin**

Carboplatin has a delayed and double nadir, meaning that neutrophil counts will drop around 10-14 days and then again around 21-23 days. This means that it’s not uncommon that patients come in 3 weeks after treatment, and can’t receive the next dose. We can also see a cumulative neutropenia with carboplatin.

**e) L-Asparaginase**

This is an enzyme, used in dogs with lymphoma. It depletes lymphoma cells of an “ingredient” they need to divide. Because it’s a foreign protein, it can cause an allergic reaction after more than one dose is

given. It's important to premedicate dogs with diphenhydramine (antihistamine) prior to treatment. Lspar is usually administered subcutaneously.

**f) Lomustine**

Lomustine is more myelosuppressive than most other chemotherapy agents. It can cause severe neutropenia, and a cumulative, possibly irreversible, thrombocytopenia. It's important to base the decision to treat or not, not only on the neutrophil count but also on the platelet count. Hepatotoxicity is also seen in patients treated with lomustine. That's why all patients are placed on liver protective medication (Denamarin) as this has been shown to reduce the risk of liver damage.



# Diagnosing Cancer: Aspirates, Biopsies and Other Testing

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Cecilia Robat DVM DACVIM (Onc)

## 1) Fine needle aspirates

Fine needle aspirates (FNAs) are used to obtain a diagnosis after a mass is found. The advantages of performing a FNA are that they are easy to perform, cheap, and results are obtained quickly. A disadvantage is that they are lower yield than a biopsy, and will not provide information on the grade of the tumor. Obtaining a diagnosis via FNA will help make an appropriate treatment plan and help with surgical planning.

Almost everything can be sampled via FNA on a patient except a bladder mass as there is a risk of seeing the tumor (transitional cell carcinomas may spread along the needle tract, other tumors do not typically seed), and a cavitated splenic mass, as this could cause the mass to rupture, creating a hemoabdomen. Contrary to popular belief, thyroid masses can readily be sampled- because thyroid tumors bleed readily, it's important to keep the aspirate short, quick and place a finger on the hub of the needle to avoid blood contamination.

Few supplies are needed for a FNA: needles (it's recommended to start with a small gauge such as 23G, and only try a bigger gauge if the aspirate yields no material), syringes (6-10cc), slides (ideally with a frosted end to facilitate identification), stain (Diff Quick) and the patient! Sedation is rarely necessary to obtain a FNA. Cytology samples are most routinely collected by FNA but can also be obtained by impression smear (for example of a biopsy sample), or direct smear/centrifugation of blood or effusions. Both non-aspiration and aspiration techniques can be performed. Non-aspiration should be favored due to decreased blood contamination and cell rupturing. If this technique fails, then the non-aspiration technique can be used. FNAs are not always successful in yielding an adequate sample for diagnosis. Ruptured cells are common with either harsh technique or when sampling neoplastic cells (especially lymphoma cells), sometimes the sample is too thick, or the staining is inadequate. Sometimes the wrong tissue/organ is sampled (the most common example is sampling a salivary gland instead of a mandibular lymph node). It's very important to check a slide to make sure the sample is of adequate quality prior to sending it to the clinical pathologist.

A common finding on cytology is inflammation. This can be neutrophilic/suppurative/purulent (neutrophils), mixed (e.g pyogranulomatous (neutrophils and macrophages) or macrophagic/granulomatous (macrophages), eosinophilic. Neutrophilic inflammation can be septic if bacteria are present. You can have a septic inflammation without seeing the bacteria on the slide. Ruling it out can only be done with a culture. Common neoplasia diagnosed are round cell tumors (e.g lymphoma, mast cell tumor, plasma cell tumor, transmissible venereal tumor, histiocytoma), carcinomas (tumors of epithelial tissue) and sarcomas (tumors of mesenchymal origin). Criteria of malignancy include

macrocytosis, anisokaryosis, multinucleated cells, mitoses, nucleoli, increased nuclear/cytoplasmic ratio, increased cellularity. If in doubt after assessing a cytology sample, a biopsy is recommended.

## 2) Biopsies

The advantage of a biopsy over a FNA is the higher likelihood of obtaining a definitive diagnosis. It may also provide the grade of a tumor, although small biopsy samples may not be representative of the whole tumor- that's why pathologists may be reluctant to grade a tumor on a biopsy sample. Biopsies are usually fairly easily obtained, and sedation and a local anesthetic block are often sufficient to obtain incisional biopsies. Excisional biopsies usually require a larger surgical procedure and general anesthesia may be necessary. The disadvantages of a biopsy are the higher cost, invasiveness of the procedure (compared to a FNA), possible need for general anesthesia, and the delay in obtaining results. Any mass that is removed or biopsied should be sent for analysis. Indeed, it's vital to obtain a definitive diagnosis on a removed mass, know the grade of the tumor and whether margins are clean or not. Recurrent masses act more aggressively, are more likely to spread and less likely to be easily removed. Clients who decline to have a mass analyzed usually do not understand the implications of this decision.

The most common ways to obtain a biopsy sample are either using a scalpel and incising directly into the mass, using a Jamshidi instrument (to penetrate bone), a punch instrument (to incise into a superficial mass), or a TruCut instrument (to obtain a sample of a more deeply seated soft mass).

## 3) BRAF test

Transitional cell carcinoma (TCC) is the most common bladder tumor diagnosed in dogs. A recurrent bladder infection, especially if it's in an older (male!) dog should be further investigated, as there is likely an underlying cause (stones, tumor, or a metabolic illness predisposing to the development of urinary tract infections (UTIs) such as diabetes mellitus or Cushing's disease). Overrepresented breeds are Scottish Terriers, Shelties, Westies, Beagles. TCC is most commonly found in the trigone of the bladder. It is a highly locally invasive tumor that has usually seeded the rest of the bladder by the time of diagnosis. Metastasis are uncommon, but can occur to lymph nodes, liver, spleen, lungs and bone. Local disease is usually the cause of the patient's demise. A tentative diagnosis of a bladder tumor can be made when a mass is noted in the bladder on imaging, however other conditions, both benign and malignant, can cause a mass to develop in the bladder. A definitive diagnosis requires a biopsy sample (obtained via cystoscopy or surgical biopsy). Urine cytology is rarely diagnostic, however a sample obtained from an exfoliative catheterization may be diagnostic on cytology and is usually the go-to diagnostic test for this cancer. Recently a test called the CADET BRAF mutation test has become available. This test detects a common genetic mutation in dogs with transitional cell carcinoma or urothelial carcinoma. This test is performed on urine (30ml). A positive result is highly specific for a diagnosis of transitional cell or urothelial carcinoma. The test can detect as few as 10 mutation-bearing cells in a urine sample and so is able to detect the presence of a developing tumors, often several months before any advanced clinical signs associated with the cancer become evident. It can therefore be used not only as a diagnostic test but also as a screening test for breeds at risk. Results are available 2-3 days after the sample is received by the

laboratory. Treatment of choice for TCC is a non-steroidal anti-inflammatory drug, chemotherapy +/- surgery. Recent studies suggest that prognosis is improved if surgery can be performed (non-trigonal tumors).

#### 4) Molecular testing for lymphoma

Lymphoma is the most common cancer diagnosed in dogs. It occurs in dogs of any age, any breed with Golden Retrievers, Boxers, Bulldogs, Beagles and several other breeds being overrepresented. The most common presentation is the finding of enlarged peripheral lymph nodes (multicentric lymphoma) although it can occur in other sites (gastrointestinal, (muco-) cutaneous), other. The most common type of lymphoma is known as "intermediate to large cell" based on the size of cells, compared to a neutrophil, on cytology. The finding of greater than 50% large lymphocytes on the slide is diagnostic for lymphoma. Lymphoma is usually readily diagnosed on a FNA sample of lymph node/organ, but sometimes, further diagnostics are required. Indeed, common situations where this may be necessary are: if there are just about 50% large lymphocytes but not quite enough to call lymphoma (a reactive node will also have an increased number of large lymphocytes on cytology), or if there is a suspicion that this may be an uncommon form of small cell lymphoma (small lymphocytes are normally the major population in a lymph node, but in the face of enlarged lymph nodes, this finding may indicate small cell lymphoma). Another way to help obtain a definitive diagnosis is to biopsy the affected lymph node.

Flow cytometry is the sorting of live cells based on size and granularity as they are hit by a laser beam. Cells can be identified using fluorescent markers. Normal and abnormal expression of cell surface proteins can be detected using fluorescent antibodies. Flow cytometry is usually used to help distinguish between reactive and cancerous lymphocytes, help diagnose leukemia in dogs with lymphocytosis, distinguish between lymphoid and myeloid malignancies. Samples submitted for flow cytometry are usually FNA samples of enlarged lymph node or abnormal organs that on cytology are suspicious for lymphoma, blood or bone marrow, or effusions.

PCR for antigen receptor rearrangement (PARR) is used to look for clonality. Clonality is one of the hallmarks of cancer. If all the cells are identical, then a diagnosis of cancer can be made. PCR amplifies very small amounts of DNA. Neoplastic cells have the same sequence at the variable region of the T or B cell receptor genes but this region of the receptor will be slightly different in non-neoplastic cells. This test is particularly used to diagnose lymphoma vs a population of reactive lymphocytes.



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# On Your Last Nerve: Regional Anesthesia For Veterinary Professionals

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Pain is a complex sensory and emotional experience that can be associated with actual or potential tissue damage. It is our duty as veterinary professionals to alleviate pain in our patients. An effective way to do this is through the use of local and regional anesthesia.

## Why treat pain?

Pain suppresses the immune response, promotes inflammation which delays wound healing and predisposes the patient to infection and additional medical care. Pain increases patient risk under anesthesia because more drugs are needed to maintain a stable plane. Pain also increases morbidity and the cost of patient care. Pain should be treated in all circumstances which will in turn produce a general feeling of satisfaction among staff and owners because patients are not needlessly suffering.

## Understanding the pain pathway

Painful sensations follow a specific pathway through the central nervous system (CNS). Painful stimuli are recognized and transformed into a signal that the nervous system can understand (*transduction*). These electrical signals are transmitted via the peripheral nervous system (PNS) to the spinal cord (*transmission*) where they are changed (*modulation*) before being relayed to the brain for final processing and ultimately awareness (*perception*).

Analgesic drugs work at various levels of the pain pathway by blocking or changing the pain signal. Understanding what drugs work at which level of the pathway can help us treat pain more completely and provide better patient care.

### Transduction

- Drugs that work 'on site'
  - Local anesthetics (lidocaine, bupivacaine), NSAIDs, Steroids, Intra-articular opioids

### Transmission

- Drugs that stop nerve impulses from being transmitted and drugs that alter the signal peripherally
  - Local anesthetics, Alpha-2 agonists

## Modulation

- Drugs that change the pain signal and make it more tolerable
  - Opioids, NSAIDS, Alpha-2 agonists, Local anesthetics, NMDA antagonists (Ketamine, Methadone), Tricyclic antidepressants

## Perception

- Drugs that provide unconsciousness
  - General anesthetics (inhalants, induction drugs like propofol, barbiturates although these drugs are not considered analgesic), Opioids, Alpha-2 agonists

## The importance of treating pain appropriately

The memory of a painful event is shaped by a number of factors specific to an individual including behavior patterns, the environment and expectations. Neuroplasticity is the ability of the nervous system to change or adapt its biochemical and physiological functions in response to both internal and external stimuli- it is how we navigate our environment. The idea is that multiple minor painful events or a single significant painful event can change the way the nervous system responds to stimuli in general. Patients who have a considerable memory of pain or have experienced unrelenting pain for 12-24 hours are less responsive to analgesic therapy.

On a similar note, intense pain that is not treated appropriately or at all activates specific receptors (AMPA, NMDA) which have the ability to alter gene expression and increase the central nervous system's response to further input. This is referred to as central sensitization or wind-up.

## Why locals?

Local/regional anesthesia is an effective, inexpensive and fun way to treat pain. This modality prevents stimuli from reaching the CNS and reduces the need for systemic drugs. Locals are relatively safe, they provide operative and post-operative pain control and prevent the formation of central sensitization. Pain on injection, decreased effectiveness in inflamed tissue, muscle weakness/paralysis, potential allergic reaction, nerve damage/hematoma/infection and the potential for toxicity are among the disadvantages.

## Regional vs. local

Regional anesthesia is the loss of sensation to a part of the body by interrupting the sensory nerves conducting impulses from a specific region. Regional block include epidurals, brachial plexus blocks and ring blocks. Local anesthesia is the loss of sensation to a defined area SQ and includes dorsal line blocks, incisional blocks and infiltrative blocks.

## Toxicity

There are two types of potential toxicity when using local anesthetics; neurotoxicity and cardiotoxicity. Toxicity is very drug, dose and route dependent.

*Neurotoxicity* is characterized by shivering that progresses to muscle twitching and seizures. The patient may go from being depressed to unconsciousness and if not treated coma and respiratory arrest. These signs may be masked by general anesthesia.

*Cardiotoxicity* can be detected more easily in the appropriately monitored anesthetized patient. Signs include a decrease in contractility, decreased rate of conduction, alterations in vascular tone, rapid progression from normo- to bradycardia, decreased cardiac output, cardiovascular collapse and death.

To avoid toxicity, always aspirate before injecting, do not exceed the calculated dose, divide said dose among each site, avoid bupivacaine in highly vascular areas (NEVER give it IV) and monitor for allergic reactions. Do not shy away from volume as long as it is within the dosage range. Adequate volume is essential to successful local anesthesia.

Treatment is often supportive in nature and may include IV fluids, positive inotropes, diazepam, general anesthesia, oxygen/intubation/ventilation and the monitoring and treatment of any arrhythmias.

## Techniques and specific blocks

Blind injections of local anesthetic will work, but nerve location will work better because concentration of anesthetic at the desired site is more important than volume.

### *Regional anesthesia of the head*

#### **Maxillary nerve block**

- Located within the infraorbital canal in the rostral orbit
- Innervates bone, maxillary teeth, soft tissues and palate unilaterally
- Effective for dental cleanings of severely inflamed tissue, extractions of maxillary teeth, maxillectomy if margins are rostral to the orbit, hard palate surgery (recommend palatine block also)

- Approach: open mouth, palpate zygomatic arch and the edge of the hard palate just medial to the last cheek tooth on the desired side, aspirate and deliver calculated dose.

#### **Deep maxillary nerve block**

- May also reach the floor of the nasal cavity
- Good option for rhinoscopy and nasal biopsy
- Approach: Measure IV catheter tip to medial canthus and mark cannula, insert catheter and stylet into infraorbital canal. Deploy catheter off of stylet and insert to pre-measured mark. Aspirate and deliver adequate volume.

#### **Infraorbital nerve block**

- Nerve located outside of the infraorbital canal
- Innervates lateral maxillary teeth from the canines rostral and associated soft tissues
- Effective for extractions of maxillary canines and maxillary incisors
- Approach: palpate foramen rostral to the 4<sup>th</sup> premolar in dogs and the caudal border of the 2<sup>nd</sup> premolar in cats. Deposit local anesthetic beneath the tissue using a 25 g needle rostral to the foramen or just inside of it using a small volume (less than 1 ml).
- Note: Too much volume or inserting the needle into the foramen may result in a maxillary nerve block

#### **Mandibular nerve block**

- Located on the medial aspect of the ramus of the mandible
- Innervates mandible and associated structures
- Effective for anesthesia of the ipsilateral mandibular teeth, skin and mucosa of the lower lip and chin
- Approach: intraoral- palpate and strum the nerve, pass a 25 g needle to the site staying close to the bone, aspirate and inject. Extraoral- palpate notch on ventral ramus, insert 22-25 g needle staying close to the bone, aspirate and inject small volume (usually less than 1 ml)

#### **Mental nerve block**

- Nerve exits the mental foramen on the lateral aspect of the mandible just caudal to the canine tooth
- Innervates lower lip and incisors
- Effective for procedures involving the lower lip and extractions of the incisors, bilateral blocks are recommended due to crossover

- Approach: palpate foramen, insert 25 g needle into foramen, aspirate and inject a small amount of local

### **Palatine nerve block**

- Located on the caudal border of the hard palate midway between the mesial maxillary carnassials and palatal midline bilaterally
- Innervates maxillary incisors, canines, premolars and oral side of hard palate and possibly the nasal floor
- Effective for procedures involving those structures and rhinoscopy, hard palate surgery in conjunction with maxillary nerve block
- Approach: insert 25 g needle SQ at the level of the palatine nerve, aspirate and inject

### ***Forelimb Blocks***

#### **Brachial Plexus block**

- Know your anatomy! This block is challenging because the brachial vein and artery run right alongside the nerve. Block is more effective if using a nerve locator or ultrasound.
- Provides analgesia distal to the elbow
  - More proximal if ultrasound guided
- Limitations: large volume of local required, technically challenging (recommend use of ultrasound or nerve locator), risk of hemorrhage (axillary artery and nerve run alongside the nerve), incomplete blocks are common, can only perform unilaterally, not effective for the humerus and shoulder, risk of pneumothorax if needle ends up medial to the 1<sup>st</sup> rib
- Approach: enter at point of shoulder, do not pass 1<sup>st</sup> rib, use a 20-22 g insulated needle that is 4-10 cm in length if using a nerve stimulator, spinal needle if not, aspirate and deliver

#### **Ring block**

- Distal nerves are more superficial, there is less soft tissue surrounding the nerve making SQ local blocks effective
- Provides analgesia for fractures distal to the block, wounds, declaws, digit amputations
- Be generous with the drug but stay below the toxic dose!
- A digit block is just a ring block of the toe
  - Great for broken nails

#### **Epidural**

- 12-24 hours of good-excellent analgesia, decreases the need for intraoperative anesthetics, decreases the need for post-op systemic drugs, better respiratory function

- Effective for analgesia of the hind limbs, pelvis, abdomen, perineum and perianal area, may also help treat thoracic pain and forelimb procedures as well (catheter is better)
- Contraindicated in coagulopathy, infection/neoplasia at the site of injection, possible meningitis or encephalitis, SIRS or sepsis, preexisting urinary or motor dysfunction, lesions or fracture that changes spinal anatomy
- The spinal cord usually ends at L5 in dogs so the risk of cerebral spinal fluid CSF is low. In cats the cord ends more caudally (L7) so the chances of getting CSF are greater.
- Effective for: lumbosacral (L6-L7 or L7-S1) for hind limbs, abdomen +/- thorax, sacrococcygeal (S3-Co or Co1-Co2) for perineum/perianal, blocked cats, vulvoplasty, tail sx, anal sac sx
- Approach:
  - Place patient in lateral or sternal recumbency
  - Clip and aseptically prep a large area over the injection site (L7-S1 etc)
  - Wearing sterile gloves and using a sterile drape, locate the space by placing the thumb and pinky finger on top of the wings of the ilium, on midline between the wings you will feel a significant depression; this is L6-L7. Just caudal to that is a smaller depression that is L7-S1; this is where most epidurals are performed. You can also walk up from the sacral vertebrae.
  - Place the needle perpendicular to the skin on midline in the middle of the space. Sometimes angling the needle top slightly cranially is beneficial
  - Insert the needle slowly. Often, resistance can be felt as the needle passes through the ligamentum flavum at the top of the vertebral canal. At this point, the needle is in the epidural space.
  - Remove the stylet from the needle. If blood comes from the needle, the needle should be removed, if CSF is encountered, cut the dose of local in half, opioid doses, in general do not need to be altered.
  - Inject 1 ml of air using a loss of resistance syringe to verify placement. If the air stays in place without pushing the syringe plunger back, you are likely in the right spot, if there is resistance and push back, you are not in the epidural space.
  - An alternative method involves a hanging drop of sterile saline. This works best when the patient is in sternal recumbency.
    - Once the needle is passed through the skin and before reaching the ligamentum flavum (LF), the stylet is removed and the needle hub is filled with sterile saline until a convex meniscus is formed.
    - The needle is then slowly advanced until the saline is sucked into the needle indicating that it has entered the epidural space.

- Sometimes the needle gets plugged with tissue and the 'suck' does not occur. If you feel that you have popped through the LF but no negative pressure is present, try gently injecting a small amount of saline. There should be no resistance.
- Drugs should inject easily with no resistance.
- Tips: use a slip tip syringe, stabilize the needle by resting your little finger on the patient, know your anatomy!

### Testicular block

- The idea is that the local anesthetic moves up the spermatic cord providing analgesia/anesthesia for neuters
- It takes 10 minutes for this block to take effect
- Approach: prep testes and insert a 22-25 g needle into the caudal pole and direct it cranially. Aspirate and inject the local as you slowly withdraw the needle until the teste is turgid in the scrotum.
- Alternative approach is to grab the band of tissue cranial to the teste and insert the needle within the center of the bundle (containing spermatic cord, blood vessels), aspirate and inject.

### *Other blocks of interest*

#### Intercostal block

- Indicated for rib fx, pre/post thoracotomy, chest tubes
- Block 1-2 sites cranial and 1-2 sites caudal to the incision
- Approach: sterile prep of area, place a 22-25 g needle just caudal to the rib (1-2 spaces cranial and 1-2 spaces caudal to the incision), aspirate for blood and inject.

#### Interpleural block

- Indicated for thoracic and cranial abdominal pain especially pain related to pancreatitis
- Approach: aseptically prepare the site. Place a 22 g butterfly catheter or 20 g long IV catheter (a chest tube already in place can also be used) in the 9<sup>th</sup> intercostals space. Aspirate and if clear of blood, inject **LIDOCAINE FIRST** (1.5 mg/kg). The patient may vocalize momentarily because lidocaine stings, but once the drug begins to work (a few seconds) the patient should calm down. Inject bupivacaine **SECOND** (1.5 mg/kg). If the bupivacaine is injected first the patient will thrash and vocalize for 15-25 minutes as the block takes effect.
- You can roll the patient into dorsal so that the local flows into the paravertebral gutters and block the nerves before they enter the spinal cord or position them with the affected or unaffected side down. If the block doesn't seem to work, alter the position to change the distribution of local anesthetic

- This block can be repeated every 4-6 hours

## General information on local anesthesia

### *Infiltration block*

- **When to use:** laceration repairs, mass excisions and as a line block before or after a surgical incision is made.
- **Technique:** The total volume of local anesthetic is deposited in subcutaneous injections around the area to be anesthetized.
- **Tips and Tricks:** Local anesthetics tend to be painful when injected so take care to use small needles and make subsequent injections through previously desensitized skin.

### *Splash block*

- **What is it?** The splash block involves direct application of local anesthetic on the site of interest e.g. abdominal body wall closure, ovarian ligaments, spermatic cord, peritoneum etc.
- **Dosage:** 2 mg/kg lidocaine or bupivacaine is dripped from a sterile syringe onto the area to be blocked after any abdominal flushing/suctioning.

### *Diffusion catheter*

- **What is it?** A diffusion catheter is a fenestrated catheter with a closed tip that is sterilely placed into a painful site for the continuous or intermittent delivery of local anesthesia.
- **When should I use it?** This type of catheter is easily placed during a surgical procedure like limb amputations, large tumor resections or total ear canal ablation. They can also be positioned non-surgically at the lesion site when necessary.
- **Side effects:** Limited literature exists on the use of diffusion catheters in veterinary medicine but this technique is used extensively in human medicine. Few serious adverse effects can be attributed to this technique and complications can include infection, hematoma, inadvertent IV injection and cardiovascular collapse, intraneural injection resulting in nerve damage. Strict aseptic technique and good nursing care should be employed when taking advantage of this technique.
- **Placement:** A diffusion catheter is similar to intra-operative placement of a wound drain. The catheter should be placed in the deepest part of the wound and all fenestrations should be within the muscle or subcutaneous tissue layers. Minimal tacking can be done with absorbable suture however it is important that the catheter be able to slide out easily when removal is necessary. If a drain is to be placed at the same time, the drain should be placed ventral to the diffusion catheter. The diffusion catheter can be tacked to the skin with suture to keep it in place and then bandaged to provide additional security.

- **Dosages and delivery:** Elastomeric pumps exist for the continuous infusion of local anesthetic and syringe pumps can be used in a similar fashion. Drugs like lidocaine and mepivacaine are the safest for continuous infusion and can be delivered at 1-2 mg/kg/hr. Intermittent delivery of bupivacaine can be used at 1-2 mg/kg every 4-5 hours. Diffusion catheters are typically left in place for one to three days but can be maintained longer if strict aseptic technique is used.

### Local anesthesia basics

- Base all dosages on lean body weight
- Calculate the maximum safe dose to avoid toxicity. Typical dosages are 2 mg/kg or either lidocaine or bupivacaine.
- Consider reducing dosages in debilitated, geriatric and neonatal patients
- Local anesthetic over dose may present as agitation, muscular twitching, seizures, unconsciousness, coma, respiratory arrest, cardiac depression, dysrhythmias, hypotension and death. Many of these signs are masked by general anesthesia.
- Cetacaine spray should be avoided in cats as it can lead to methemoglobinemia. A suitable and less expensive alternative involves dripping a total of 0.2 ml of lidocaine onto the aretenoid cartilage of cats prior to intubation.
- Strict aseptic technique is essential when performing local blocks to avoid the complication of infection. It is imperative to surgically prep the skin, wear sterile gloves and use only sterile needles and syringes and catheters. Sterile skin prep (shaving etc) is not necessary for digital nerve blocks and testicular blocks and sterile gloves are optional.
- Always aspirate prior to injection to avoid inadvertent venous or arterial injection.
- The addition of buprenorphine has been anecdotally shown to extend the duration of analgesia significantly compared to local anesthesia alone.

**In a nutshell... Find a nerve and figure out what it does for a living. Know who lives around it and then reach it with a needle. Anticipate complications and do the math!**

Reference for all techniques:

Gaynor, J.S. & Mama, K.R. (2009). Local and regional anesthetic techniques for alleviation of perioperative pain. In Gaynor, J.S. & Muir, W.W. (Eds.), Handbook of veterinary pain management (pp.277-300). St. Louis: Elsevier.



# BEHIND THE EIGHT BALL: Playing catch-up with a painful patient

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## Introduction and physiology

Pain is a complex process involving the peripheral nervous system (PNS) and the central nervous system (CNS). It activates changes in the nervous system that increase stress but the nervous system isn't the only part of the body that is affected by pain. Painful patients cannot heal as effectively due to high levels of circulating hormones like cortisol, along with other metabolic responses that can have a negative effect on immune function. Pain increases the activity of the sympathetic nervous system leading to an increase in heart rate and blood pressure and may cause cardiac arrhythmias.

**PNS review:** The peripheral nervous system consists of nerves and ganglia outside of the brain and spinal cord. Their main function is to provide a means of communication between the limbs and organs and the CNS. The PNS is not protected by bone or the blood brain barrier like the CNS is so it is prone to insults from toxins and injury. The PNS is divided into the somatic and autonomic nervous systems. The somatic nervous system is associated with voluntary control of body movements and consists of afferent and efferent nerves. The autonomic nervous system unconsciously regulates the functions of the internal organs and some muscles in the body. It is further divided into the sympathetic and parasympathetic nervous system. The sympathetic nervous system is responsible for responding to perceived threats by getting the body ready to fight or flee. The parasympathetic nervous system controls

homeostasis and readies the body for rest and repair.

**CNS review:** The central nervous system consists of the brain and spinal cord. Both organs are encased in bone, insulated with cerebral spinal fluid (CSF) and the brain is protected from toxic insults via the blood-brain barrier; a filtering mechanism of capillaries that block the passage of certain substances. The CNS is responsible for integrating sensory information and responding accordingly. The spinal cord is the conduit for signals between the brain and the rest of the body and it also controls musculoskeletal reflexes. The brain is responsible for integrating most sensory information and coordinating body function both consciously and unconsciously. Complex function like thinking and feeling as well as homeostatic regulation take place in the brain.

Physiologic pain results from inflammation or injury and has an adaptive function; to warn when external stimuli may induce tissue injury or threaten life. It enables healing by hypersensitizing surrounding tissues to increase sensitivity and encourage the animal to leave the area alone. Following damage to tissue, a torrent of hyperexcitable events occurs in the nervous system. This physiologic wind up starts at the skin and is potentiated along the peripheral nerves and climaxes in a hypersensitivity response from the spinal cord.

Inflammatory mediators like cytokines and histamine are released to start the process of healing. However, these substances are also irritating and have the potential to change the properties of the primary sensory neurons around the area of tissue damage. These substances are responsible for activating the high-threshold silent nociceptors leading to peripheral sensitization. At this point, physiologic pain becomes pathologic pain because it has taken a step beyond being adaptive.

### **Peripheral and central sensitization**

Peripheral sensitization is a reduction in the threshold and an increase in responsiveness of the peripheral nociceptors. Normally, high-threshold nociceptors, A-delta and C fibers are active in response to noxious stimuli. Damaged cells release chemical mediators in response to tissue injury and inflammation. These substances have direct effects on the excitability and sensitizing of sensory nerve fibers. They promote vasodilation and recruit inflammatory cells, macrophages, lymphocytes, platelets and the substances implicated in inflammatory soup. This effectively lowers the threshold for A-delta and C fiber activation. Silent nociceptors are exquisitely sensitive to the effects of the inflammatory soup and go from benign unmyelinated polymodal c fibers to vigorously firing c fibers.

Central sensitization can happen as a result of peripheral sensitization and is the indirect consequence of tissue trauma and inflammation. Pain sensations spread beyond the site of insult to nearby undamaged tissue. Central sensitization can result from unrelenting stimulation to the peripheral nociceptors, which lead to sustained release of glutamate and other neurotransmitters from primary afferent nerves. The liberation of these substances activates receptors in the dorsal horn of the spinal cord that will then lead to an increase in the excitability of the dorsal horn neurons projecting up to the brain. These substances are *N*-methyl-D-aspartate (NMDA) and  $\alpha$ -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) and their release can lead to a 'wind up' of the CNS, the inciting cause of central sensitization leading to an exaggerated response to subsequent nociceptive and non-nociceptive input.

NMDA and AMPA are both activated by glutamate, a prominent amino acid and excitatory neurotransmitter. Noxious stimulation causes a release of glutamate that binds to both NMDA and AMPA receptors. Weak stimulation activates AMPA resulting in slight cell depolarization by making the postsynaptic neuron permeable to sodium and potassium, generating nerve impulses. The NMDA receptor is normally blocked by magnesium so it doesn't allow ions to freely pass through to generate an impulse. During weak stimulation, excitatory signals are mediated entirely by AMPA receptors. When greater stimulation occurs, AMPA receptors can depolarize the membrane with enough strength to dislodge magnesium from the NMDA receptor allowing it to actively respond to glutamate. When activated, the NMDA receptor allows large amounts of calcium to pass through activating several intracellular signaling cascades and ultimately leading to increased nerve transmission and heightened nerve excitability. As such, the process of peripheral and central sensitization is maintained through the excitatory neurotransmitter, glutamate, which is released when tissues are damaged.

Cyclo-oxygenase (COX) also plays a role in peripheral and central sensitization. COX-2 is one of the enzymes that is created during the inflammatory process. COX-2 converts arachidonic acid to prostaglandins that then increase the sensitivity of the peripheral nociceptors. COX-2 is also released into the CNS, and it is thought that signals from the periphery are responsible for this upregulation. COX-2 has the ability to increase neurotransmitter release and produce direct depolarizations as well as inhibit the action of the inhibitory neurotransmitter, glycine.

A neurotransmitter can influence a neuron's electrical excitability in one of two ways, exciting it or inhibiting it. It does this by influencing trans-membrane ion flow to either increase (excite) or decrease (inhibit) the probability that the cell with which it comes into contact with will produce a nerve impulse or action potential.

Substance P, glutamate, gamma-aminobutyric acid (GABA), endogenous opioids, serotonin and norepinephrine are the key players in signal management. For example, the excitatory neurotransmitter glutamate can amplify the pain signal whereas GABA can inhibit it. Serotonin and norepinephrine can inhibit excitatory neurotransmitters like glutamate and facilitate inhibitory neurotransmitters like GABA, helping to augment and decrease the pain signal before it reaches the brain.

## Treating pain

Peripheral sensitization can lead to central sensitization in patients who have experienced severe or unrelenting pain or pain that has been un- or under managed. Once this has been established, larger doses of analgesics are required to treat this response. Larger drug dosages are associated with greater side effects and those side effects (think vasodilation, hypotension) can increase anesthetic risk for surgical patients. The use of one class of analgesic, even in large doses, may not be enough for many patients. Using a multimodal approach to analgesia helps to minimize post-operative pain. Pre-emptive or preventative analgesia has been shown to reduce the impact that all of these stimuli have on the CNS and allows for easier treatment of post-operative or continuing pain during healing. A pre-operative dose of an opioid may reduce the post-operative dose necessary to maintain comfort significantly.

Like pre-emptive opioids, pre-emptive NSAIDS, can contribute to a reduction in the necessary dose of post-operative opioids and other analgesics. NSAIDS are not benign drugs and patient comorbidities should be considered before use. Blocking nerve transmission with epidural analgesia or locoregional blocks using local anesthetics have been shown to prevent the development of central sensitization. NMDA receptor antagonists like ketamine and methadone given pre-operatively have also been shown to reduce the need for post-operative opioids.

The ideal pre-operative, intraoperative and postoperative treatment regime includes NSAIDS to reduce the activation of nociceptors, locoregional techniques to block sensory influx and sensory acting drugs like opioids.

## Application of knowledge

How does this translate into the treatment of a patient with pre-existing major tissue damage, pain, peripheral and central sensitization?

The goals are to make the patient comfortable by using lower drug dosages to achieve the desired effect without increasing anesthetic risk and to allow the patient to return to normal function without lasting pathologic pain.

One important step is to avoid lapses in analgesic therapy. Do not wait for a patient to demonstrate that they are painful before giving analgesics. Give drugs regularly, on schedule and don't skip any doses in the first 24 hours post-operatively even if the animal is not 'acting' painful. After 24 hours (48 in some severe cases) the pain plan can be reassessed and altered.

Use a multimodal approach to analgesia. Combining different drug classes and techniques to treat pain at as many points along the pain pathway as possible has been shown to reduce central sensitization. Utilize locoregional blocks to help quell pain e.g. epidurals, ring blocks, nerve blocks to stop the signal from reaching the CNS. Both techniques will stop continuing signals from being transmitted making central sensitization worse. Even if a painful patient is not surgical, locoregional techniques should still be considered.

Consider using NSAIDS if appropriate for the patient. NSAIDS block the COX enzyme (specifically the COX-2 enzyme depending on the drug chosen) that plays a role in both peripheral and central sensitization. A large percentage of pain is a result of inflammation and the inflammatory process. Treating inflammation is paramount to treating most pain.

When activated, the NMDA receptor is responsible for central sensitization. Utilize drugs that block this receptor and calm down the CNS to allow for other analgesics to work more effectively. Ketamine is an inexpensive NMDA receptor antagonist and a drug that should be utilized when central sensitization is suspected. Subanesthetic dose constant rate infusions of ketamine are beneficial when given pre-operatively in the patient that presents with wind-up pain. Ideally, infusions should be given for a few hours prior to surgery and then continued intra-operatively. The CRI can be continued post-operatively as well.

Other NMDA receptor antagonists include amantadine, methadone, dextromethorphan and tiletamine. Each substance has a different level of NMDA antagonism. Amantadine can be given orally once per day for treatment of chronic pain or central sensitization outside of the hospital. Methadone is a pure mu opioid with NMDA antagonist activity that can be employed as part of the analgesic plan in hospital due to its poor oral bioavailability. Dextromethorphan may not be as effective in veterinary patients as it is in humans as dogs, in particular, do not make the active metabolite of this drug when given orally.

Gabapentin binds to specific subunits of voltage gated calcium channels and that reduces the release of excitatory neurotransmitters like glutamate. It is through these mechanisms that gabapentin is a great choice as an adjunctive medication in an analgesic cocktail for painful patients. Side effects are few and dissipate with continued use.

Treat stress and anxiety along side pain. Drugs like trazadone, subanesthetic doses of dexmedetomidine and even low dose acepromazine can augment an analgesic protocol to keep patients comfortable. Non-pharmacologic means of pain management should also be considered. Good nursing care, comfortable bedding, dimmable lights, white noise, essential oils, good food, attention, acupuncture, massage, an empty bladder and a dry, clean body can do wonders for hospitalized patients.

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Saturday  
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## Michael C Petty, DVM, CVPP, DVMA, CCRT, CAAPM

Michael Petty is a 1980 graduate of the veterinary school at Michigan State University and has been in private practice since then. He is the past president of the International Veterinary Academy of Pain Management, a co-author of the AAHA/AAFP 2015 Pain Guidelines, and the author of Dr. Petty's Pain Relief for Dogs.





# Laser and Massage: How you can make them fit into your practice

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Michael C Petty, DVM

## Basic Properties of Lasers

1. Why laser therapy?
  - a. 20% of veterinary practices have lasers
  - b. Client driven-they often ask for alternative therapies
  - c. Used in Rehabilitation
  - d. Much more research supporting use
  - e. Better products on market
2. Too many clinics don't use them at all or underutilize them
3. Terminology
  - a. L\_A\_S\_E\_R = light amplification by stimulated emission of radiation
  - b. Photobiomodulation Therapy (PBMT) is becoming the more popular term
    - i. Includes LED therapy, but that is a different lecture
  - c. Old terms
    - i. Low level laser
    - ii. Cold laser
    - iii. High Intensity laser
4. Laser Tissue Interactions
  - a. Photothermal
    - i. Long pulses biological effect due to heating
    - ii. Hair removal
    - iii. Surgical lasers
  - b. Photomechanical/sometimes called photoacoustic

- i. Short pulsed lasers cause ablation
  - ii. Tattoo removal
  - iii. Photorefractive keratectomy
- c. Photobiochemical
  - i. The non-surgical lasers generally used in veterinary practice
  - ii. Pain reduction
  - iii. PBMT

### **Mechanism of Action of Lasers**

1. What does PBMT do?
  - a. Relieve Pain
  - b. Increase microcirculation
  - c. Modulate the inflammatory response
  - d. Accelerate healing
2. Mechanism of action: Cellular level
  - a. Chromophores
    - i. Components of various cells and sub-cellular organelles which absorb light.
      1. Mitochondria
      2. Cell membrane
  - b. Restoration of energy balance
    - i. When enough mitochondria are engaged by CCO absorption, the organelles mediate a series of biochemical reactions that lead to therapeutic clinical outcomes
      1. Increased activity of Cytochrome C Oxidase
      2. Increased mitochondrial function
      3. Increased Oxygen consumption
      4. Increased ATP production

- ii. This leads to
  - 1. Reduction in pain
  - 2. Modulation of inflammation
  - 3. Increased microcirculation
- 3. How does laser affect pain?
  - a. Serotonin increases
  - b. Endorphins released
  - c. Acetylcholine increases
  - d. Bradykinins decrease
  - e. Direct analgesic effect on nerves
- 4. Tissue Interactions
  - a. Physics
    - i. Power = Energy over time
    - ii. 1 watt = 1 Joule second
      - 1. Joule is unit of energy
    - iii. Laser dose is listed in Joules/cm<sup>2</sup>
    - iv. The wavelength governs the depth of penetration into the tissue
  - b. Tissue interactions with laser
    - i. Can:
      - 1. Reflect
      - 2. Scatter
      - 3. Transmit through
      - 4. Absorb
    - ii. Absorption is what we want
      - 1. Chromophores are exposed to laser light
      - 2. Causes alteration in cellular function

3. Increases healing
- c. Clinical effects
  - i. Angiogenesis
  - ii. Neovascularization
  - iii. Increased collagen production
    1. Reduces scars and improve elasticity
  - iv. Increased muscle generation and decreases muscle atrophy
    1. Activates satellite cells responsible for muscle regeneration
  - v. Increases bone formation by causing proliferation of osteoblasts
  - vi. Increases cartilage production
    1. Increased chondrocyte productivity

### **Current Evidence**

1. What do we currently know about lasers
  - a. Electromagnetic spectrum
    - i. Infrared to near red. The closer to infrared the further it penetrates
  - b. Classification
    - i. Based on danger posed to eyes
    - ii. Class 1 and 2 are visible lasers. Safe for accidental viewing
      1. Printers
      2. Scanners
      3. Dvd and CD players
    - iii. Class 3R
      1. 1-5 mW
      2. Laser pointers, light shows
        - a. OK if you don't stare at them. But potentially hazardous
    - iv. Class 3B

1. 5-500 mW
2. Therapeutic laser
3. Eye danger

v. Class 4

1. >500 mW
2. Eye danger
  - a. Direct viewing
  - b. Diffuse reflection

2. Penetration

- a. Determined by
  - i. Wavelength
  - ii. Power
  - iii. Dose
- b. Proper penetration
  - i. Avoid scattering
  - ii. Surface absorption
  - iii. Absorption by unwanted chromophores
- c. Wavelength between 650 and 1000 nm is best for this purpose
- d. Power
  - i. Determines the number of photons at the depth of absorption/saturation of tissue
- e. Dosage
  - i. Need to determine appropriate number of treatments
  - ii. Use good technique

3. Emission

- a. Continuous wave

- i. Saturates the tissue with photons faster than a laser emitting in a pulsed mode
  - b. Pulsed
    - i. Maybe better/safer when treating over an open wound or a painfully sensitive area
- 4. Comparison of lasers
  - a. Example:
    - i. Treatment area 300 cm<sup>2</sup>
    - ii. Dosage 10 Joules/cm<sup>2</sup>
    - iii. Target energy delivered 3000 Joules
  - b. 5 mW laser 10,000 minutes
  - c. 500 mW laser 100 minutes
  - d. 3 W laser 16 minutes
  - e. 10 W laser 5 minutes
- 5. Laser Safety
  - a. Whether class 3 or 4, use eye protection
  - b. Class 4 requires constant movement and adjustment for hair and skin pigmentation because of heat

## **Use Rehab**

- 1. Intervertebral disc disease
  - a. Benefits
    - i. Reduce cytokines and radicals that infiltrate the spinal cord
    - ii. Stimulate neuronal sprouting and regrowth of severed axons
  - b. Protocol
    - i. 2-8 J/cm<sup>2</sup> daily until ambulation
    - ii. Dorsolateral approach toward the intervertebral foramen and directly on spinous processes
    - iii. Treat trigger points in muscles

## 2. Peripheral Nerve Injuries

- a. Benefits
  - i. Peripheral nerve regeneration
  - ii. Increased motor neuron survival
- b. Protocol
  - i. 1-5J/cm<sup>2</sup> daily for three days
  - ii. Approach is like spinal cord injury
  - iii. Then q2-3 days until resolution
  - iv. Warn owners that this is minimum of two months...varies with distance from spinal cord, etc.

### **Other uses of Laser: Pain and chronic conditions**

1. Osteoarthritis
  - a. Benefits
    - i. Pain management
    - ii. Reduction of Interleukin 1
    - iii. Regrowth and replication of chondroblasts
    - iv. Increased NO levels
2. Other chronic issues to consider
  - a. Post op pain management
  - b. Aural hematomas
  - c. Otitis
  - d. Lick granulomas
  - e. Soft tissue injuries
  - f. Wound healing
  - g. Anal gland sacculitis
  - h. Hot spots, dermatological issues

- i. Gingival stomatitis
  - j. Idiopathic cystitis
3. Goal of treatment
- a. Applied until condition is manageable or goal is reached
  - b. Administration protocol
    - i. Aggressive phase: every other day (or even daily) for at least three therapy sessions
    - ii. Transitional phase: gradual reduction in frequency of treatments
      - 1. Watch for relapse
    - iii. Maintenance phase: Therapy as needed
4. Dosage for OA and other conditions
- a. 8-10 J/cm<sup>2</sup>
  - b. Trick for determining dose:
    - i. A CD/DVD is 100 cm<sup>2</sup>
    - ii. Size of CD needs 800 to 1000 Joules total

### **Massage**

- 1. Many applications for anxiety and neurologic issues as well as pain
- 2. Three types of massage
  - a. Effleurage
    - i. A circular stroking motion. Used to let the animal know something is about to happen. Useful prior to more vigorous massage techniques. Helps calm anxious animals.
  - b. Tapotement
    - i. Rapid tapping or light striking of the body. Especially helpful for neurologic deficits.
  - c. Petrissage
    - i. Muscle kneading. Especially helpful for myofascial Pain

3. Resource: *Canine Medical Massage* by Narda Robinson



# Pain Management in Cats

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Michael C Petty, DVM

## Acute Pain

1. Unique needs of cats
  - a. Difficulties in treatment once they leave the clinic
    - i. Caregivers often unable to give oral medications
    - ii. Caregivers often unwilling to give oral medications
  - b. For some cats, each physical interaction causes a progressive worsening in their willingness to cooperate.
  - c. Danger to staff administering oral medications or multiple injections to fractious cats.
    - i. Cat bites are the #1 worker's comp claim in veterinary clinics
  - d. Hands-off approach to treating pain is the best
    - i. CRI's +/- diffusion catheters can present problems
      1. Twisted iv lines require a lot of baby-sitting
      2. Catheters get chewed out
2. NSAIDS
  - a. Both NSAIDS licensed for use in cats have an injectable form and are very effective at treating pain and inflammation of surgery
  - b. However, limited dosing options, especially with Metacam.
  - c. Problems may be using in cats with renal disease and dehydration
    - i. Many clinics only use them after surgery for this reason...but it is better to have on board prior to surgical insult
3. Sodium Channel Blockers: Bupivacaine and lidocaine
  - a. Local anesthetics should be used insofar as possible for the prevention of pain
  - b. Only class of drug that can stop 100% of pain

- c. Side effects are rare and usually involve overdose: Too much area to treat with a safe dose
  - d. Ineffective in areas of infection
  - e. Many possible routes
    - i. Local
    - ii. Regional
    - iii. Epidural
    - iv. Pleural
    - v. Abdominal
    - vi. Etc
4. Ketamine
- a. A neurolept analgesic with NMDA suppression
  - b. Makes an OK analgesic when used by itself
  - c. Makes great analgesic when used in conjunction with opioids, alpha-2's and locals
5. Opioids
- a. Morphine
  - b. Hydromorphone
  - c. Methadone
  - d. Buprenorphine
    - i. Simbadol
  - e. Can all be used parenterally watch out for dysphoria and hyperthermia
6. Alpha-2's
- a. Very useful in cats
  - b. Pain and sedation
  - c. Best used in combination with opioids and or ketamine
  - d. Higher doses when used as stand-alone drug...greater potential for side effects

- i. Even so, adverse events are uncommon
- ii. Reversible

## 7. New-ish drugs

- a. Onsior tablets and injectable – already discussed earlier
- b. Dexmedetomidine – already discussed earlier
  - i. New DexDomitor 0.1 makes is easier to give to small animals like cats.
  - ii. Less room for error with low strength drug
- c. Simbadol
  - i. High concentration buprenorphine for sub-q use
  - ii. One injection 1 hour prior to surgical procedure
  - iii. Lasts for 24 hours
  - iv. Excellent pain control
  - v. FDA approved....no other buprenorphine products are
  - vi. Can be used in combination with
    - 1. Locals
    - 2. Dexmedetomidine
    - 3. Ketamine
    - 4. Nsaids
    - 5. Always give simbadol first, wait one hour prior to other meds
  - vii. Can be given three days in a row: many owners would rather come back than give a pill
  - viii. Side effects
    - 1. Happy cats, euphoric and loopy, but warn the owners
    - 2. Dilated pupils
- d. Nocita
  - i. Off label in cats

- ii. Three day bupivacaine

## Chronic Pain

### 1. NSAIDS

- a. Mode of action in cats
  - i. As in dogs, NSAIDs have their action by the inhibition of Cox 2
    - 1. Cox 2 ultimately produces prostaglandins which then cause pain
  - ii. As in dogs, big limitation in use is the potential for adverse events
    - 1. GI, Renal
  - iii. Unlike in dogs, largest limitation is regulatory
- b. Meloxicam
  - i. Metabolized by oxidation
  - ii. Plasma half life varies from cat to cat, about at 24 hours +/-
- c. Robenacoxib
  - i. Degrades to form  $\gamma$ -lactam
  - ii. Plasma half life 1 ½ hours
    - 1. Persists 24 hours in tissue
- d. Approved use
  - i. Meloxicam one single injection
  - ii. Robenacoxib 3 days
- e. Off label use with long term administration
  - i. Meloxicam labeled dose is not appropriate for more than a single injection
  - ii. Half life of meloxicam can lead to accumulation in plasma, reaching toxic doses with daily administration
    - 1. Have to give smaller than labeled doses on a daily basis
    - 2. Has been shown to be safe and effective given long term at doses approved in the EU

- iii. Renal disease is not as big of an issue in cats as it is in dogs. In dogs, most renal disease is glomerular. In cats most renal disease is nephritis: can be helped by NSAID

## 2. Alternative Pain Medications

- a. Amantadine for suppression of the NMDA pathway, hyperalgesia and allodynia
  - i. Not many people are using it in cats
  - ii. No studies I know of
- b. Gabapentin
- c. Tramadol
  - i. No efficacy studies in cats, high incidence of serotonin syndrome

## 3. Diet and Nutraceuticals

- a. Glucosamine and chondroitin sulfate supplements
- b. Mobility diets
- c. Soybean Avocado Unsaponifiables
- d. Weight loss

## 4. Acupuncture

- a. Acupuncture in cats is easier than it sounds
- b. Physical Therapy
  - i. Hands on mobilizations and massage
  - ii. Strengthening exercises
  - iii. Laser therapy
  - iv. Underwater treadmill

## 5. Home makeover

- a. Increase ability to move around and provide environmental enrichment
  - i. Looking out windows
  - ii. Increased social interactions with humans and other animals in household

- b. Litter box on each floor
  - i. Geriatric cat litter

# Pain Assessments in Dogs: Acute and Chronic

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Michael C Petty, DVM

1. Causes of Chronic Pain in Dogs
  - a. Osteoarthritis
  - b. Cancer
    - i. Usually osteoarthritis but any type of cancer can cause pain
  - c. Neurological issues
    - i. Intervertebral Disc Disease
    - ii. Cauda Equina Syndrome/Lumbo Sacral Disease
    - iii. Syringomyelia
    - iv. Neuropathic pain
      1. E.g. diabetes, chronic OA pain
    - v. Fibrocartilagenous Embolism
      1. Pain is usually fleeting although paresis issues stay
  - d. Neuropathy Causes
    - i. Trauma
    - ii. Surgery
    - iii. L-S lesions
    - iv. Spinal cord injury
    - v. IVDD
    - vi. FCE
    - vii. Discospondylitis
    - viii. CNS tumors
    - ix. Pancreatitis

- x. Inflammatory Bowel Disease
- xi. Saddle Thrombus
- xii. Polyradiculoneuritis
- xiii. Diabetic Neuropath
- xiv. Brachial plexus avulsion
- xv. Fractures
- xvi. Osteosarcoma
- xvii. Nerve sheath tumors
- xviii. Any cancer
- xix. Chemotherapy
- xx. Untreated acute pain
- xxi. Osteoarthritis

## 2. Chronic pain of OA

- a. Serves no real purpose
  - i. Exercise and activity actually improves function
    - 1. Maintain muscle mass
    - 2. Maintain ambulation
- b. Considered a maladaptive pain
- c. Evaluation tools for OA
  - i. Best evaluated by the owner
    - 1. In drug studies, things like force-plate analysis are being phased out
    - 2. Veterinary evaluations don't carry the weight of owner evaluations
  - ii. But! When reported owner observations don't match with what you are seeing
    - 1. Realize many owners are optimistic about how well their dog is doing
    - 2. May be psychological/social/financial reasons for not wanting to report it
- d. Canine Brief Pain Inventory (CBPI)

- i. One of the main go-to evaluation tools in FDA studies
- ii. Adapted from a human tool
- iii. Validated
- iv. Can be use for initial evaluation of OA and for response to treatment
- v. Three parts
  - 1. 4 pain severity score questions (0 no problem to 10 as worst)
    - a. Pain at its worst in the last 7 days
    - b. Pain at its least in the last seven days
    - c. Pain at its average in the last 7 days
    - d. Pain as it is right now
  - 2. 6 pain interference score questions (0 no problem to 10 as worst)
    - a. General activity
    - b. Enjoyment of life
    - c. Ability to rise to standing from lying down
    - d. Ability to walk
    - e. Ability to run
    - f. Ability to climb
  - 3. 1 overall quality of life question
    - a. Overall quality of life in the last 7 days
      - i. Poor-fair-good-very good-excellent
- e. Client Specific Outcome measures
  - i. Several out there
  - ii. Cincinnati Orthopedic Disability Index is one example
  - iii. You can make up your own questions if you like.
    - 1. As the client to think of activities, especially ones that were no problem at one point, that their dog has issues with now

2. Scored from
  - a. No problem-a little-quite a bit-severe-impossible
3. CODI contains standard orthopedic questionnaire as well
  - a. Walking
  - b. Running
  - c. Jumping
  - d. Getting up
  - e. Lying down
  - f. Climbing stairs
  - g. Descending stairs

- f. Things to keep in mind
  - i. I always ask clients to think back and compare their dog today as to what he was like a few years ago
    1. Too many people think problems are age related
  - ii. Ask clients to be honest: describe how it is instead of what they want
- g. Postural abnormalities you should look out for in the clinic
  - i. How the dog walks into the clinic
  - ii. How they stand
  - iii. How they stand up
  - iv. Watch for a complete body shake...partial is abnormal
  - v. Listen for clicking or dragging of feet

### 3. Quality of Life Scale

- a. Common topic for dogs with chronic pain issues
  - i. Many people see pain issues in their dogs, and don't say anything until the problem affects THEIR quality of life: carrying dog in and out of house, soiling issues, etc.
  - ii. Euthanasia should always be the last resort

- b. Vetmetrica
  - i. Web-based HRQL instrument for the dog
  - ii. 22 item structured questionnaire completed by the owner in about 5 minutes
  - iii. Online data capture and computation of scores
    - 1. 4 domains of QOL
      - a. Energy
      - b. Happiness
      - c. Comfort
      - d. Calmness
  - iv. Looks at QOL on both a physical and emotional level
  - v. Vetmetrica.com
- 4. Acute Pain Assessments in Dogs
  - a. Glasgow Short Form Composite Pain Scale
    - i. Distant observation
    - ii. Take dog out of kennel and walk it
    - iii. Palpation of wound
    - iv. Overall assessment of disposition
    - v. <http://www.newmetrica.com/acute-pain-measurement/download-short-form-pain-questionnaire-for-dogs/>
  - b. Colorado State University Acute Pain Scale
    - i. Not validated
    - ii. Distant and interactive evaluation
    - iii. [https://www.researchgate.net/figure/Colorado-State-University-Canine-Acute-Pain-Assessment-teaching-tool\\_fig1\\_49661913](https://www.researchgate.net/figure/Colorado-State-University-Canine-Acute-Pain-Assessment-teaching-tool_fig1_49661913)
- 5. Dysphoria
  - a. Often an issue with dogs and opioids

- b. You can get an idea from palpation if it is dysphoria v. pain
- c. Not sure, intervene with some dexmedetomidine
  - i. 5 micrograms/kg IV push
  - ii. Not enough to sedate, but enough to stop dysphoric behavior

# Pain Assessments in Cats: Acute and Chronic

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Michael C Petty, DVM

1. Acute Pain Myths
  - a. Animals do not feel the degree of pain that humans do
  - b. It keeps them quiet post-op; this is a good thing
  - c. OHE', neuters, minor procedures don't' need medication at home
  - d. Analgesics cause adverse events
  - e. Pet owners won't pay for pain control
2. Pain Scales in general-guide for use
  - a. Intervals of assessment should be determined by anticipated pain from the procedure as well as the health of the animal and expected onset and duration of the drugs used.
    - i. Hourly for the first 4 hours post-surgery
  - b. Start after recovery from anesthesia: usually as they are trying to go into sternal recumbency
  - c. Allow animals to sleep
    - i. Be aware that cat-napping is not real sleep
  - d. Continuous and distant observations are best, utilizing specific pain scale interactive observations periodically
3. Acute pain and validated scales
  - a. Pain scales were developed for the assessment of pain from surgery
    - i. Can probably be used for assessment of acute traumatic pain as well.
  - b. Two pain scales discussed in this lecture
    - i. Scotland: Newmetrica/Glasgow Feline Composite Pain Scale
    - ii. Brazil: UNESO-Botucatu-MCPS
    - iii. There are other pain scales out there.

- c. Botucatu
  - i. Multidimensional Composite Pain Scale
  - ii. Looks at a combination of physiological/behavioral/body position factors
  - iii. Several pages long. Difficult to use in clinical setting.
  - iv. Well-validated for postoperative pain.
- d. Newmetrica Pain Scale
  - i. Can be downloaded for free for non-commercial use at [newmetrica.com](http://newmetrica.com)
  - ii. Called the Glasgow Feline Composite Pain Scale
  - iii. Utilizes
    - 1. Reaction to palpation and interaction. You must get this as a baseline prior to the surgical procedure
    - 2. Grimace scale
      - a. Muzzle position
      - b. Ear position
  - iv. Suggested to intervene when post-op score, less the baseline, is >7
  - v. Various questions with different scores possible
    - 1. Watching cat in cage
    - 2. Interacting with cat
    - 3. Palpating the wound
- 4. Acute pain and common sense
  - a. Best to use a validated scale
    - i. But as you walk past the cage...good for distant observation as discussed above
  - b. Posture
    - i. Cats can try to hide
    - ii. But a hunched back is a way cats withdraw into themselves and try to protect a painful region

- c. Recline
    - i. Most normal cats will lie on their side, with all 4 paws close together, generally facing the front of the cage
    - ii. Most painful cats will
      - 1. Turn their backs to the front of the cage
      - 2. They may lay flat out
  - d. Eyes
    - i. Normal cats have a round or almond shaped eye opening
      - 1. As you draw a line from the lateral canthus to the medial canthus of each eye, the lines usually meet on or near the bridge of the nose
    - ii. Painful cats have a squint, with the above mentioned line meeting further down the nose.
  - e. Normal behaviors
    - i. Be aware of normal stretching behaviors: Cats do downward dog position even better than dogs
    - ii. Grooming, usually starts at back end, and works their way up...not paying too much attention to any one area of body
5. Practice with your staff
- a. A good source of videos is available at [animalpain.com](http://animalpain.com) It is a Brazilian site but they have PDF scores in English for each of the videos. This is a free site.
  - b. <http://www.animalpain.com.br/en-us/avalie-sua-habilidade.php>
6. Chronic Pain
- a. Feline Musculoskeletal Pain Index
    - i. Developed at NC State University
    - ii. Download for free after filling out questionnaire
    - iii. <https://cvm.ncsu.edu/research/labs/clinical-sciences/comparative-pain-research/labs-comparative-pain-research-clinical-metrology-instruments-feline-musculoskeletal-pain-index/>
    - iv. A variety of questions

1. Each activity ranges from normal to impossible
  2. Read instructions each and every time
  3. Don't let clients see prior score or answers
- v. The higher the score, the less impairment there is
- vi. Some questions will not be answered as they may not be applicable
1. Directions on how to get score as a percentage of questions answered

b. Behavioral changes in chronic pain states

- i. No validation
- ii. Some clients find it easier to answer
- iii. Best to compare to how cat used to act a few years ago
- iv. Samples
  1. Ability to groom...cat may be dirty or matted in certain areas
  2. Using objects to jump up on things. For example using a chair or ottoman to jump up onto a window sill or table
  3. Using objects to jump down from things. For example jumping down from a table using a chair
  4. Sliding down a cabinet or piece of furniture as far as possible prior to jumping.
  5. Inappropriate elimination
    - a. Especially going right next to a litter box. Or only getting partway into a litter box because of height of sides or difficulty walking on litter substrate
  6. Difficulty going up or down stairs
  7. Does not initiate or engage in play

7. Final points

- a. Always try to use validated scales
- b. Be proactive whether it is acute or chronic pain

- c. Acute pain is hard to chase down without overmedicating.
  - i. Importance of early detection and intervention



# Cannabidiol for Pain

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Michael C Petty, DVM

1. Hemp v Marijuana
  - a. Marihuana
    - i. High THC (5-30+%), low CBD
    - ii. No known use in veterinary medicine
  - b. Hemp
    - i. Low THC, (<0.3%) high CBD, other cannabinoids and terpenes
    - ii. Suggested uses in veterinary medicine
2. Both Marijuana and Hemp
  - a. Medicinal, rope, cloth, canvas, paper and traditionally used for caulking ships
3. Marijuana
  - a. Schedule I
  - b. Can't prescribe in veterinary medicine
  - c. Research largely prohibited
    - i. Federal regulation
4. History
  - a. China
    - i. Evidence of use as long ago as 5000 years
  - b. Book of Exodus 30:23
    - i. Moses was instructed by God to use hemp
  - c. Egyptians
    - i. Evidence of use 3500 years ago
  - d. Indians (Atharvaveda)

- i. 3000 years

## 5. Mechanism of Action

- a. Endocannabinoid system THC
  - i. Receptors in the brain and PNS
- b. Physiological effects
  - i. Appetite
  - ii. Pain
  - iii. Nausea
  - iv. Mood
  - v. Inflammation
- c. Endocannabinoid Receptors CBD
  - i. Mostly in the PNS
  - ii. Especially immune system
  - iii. In CNS as well
- d. THC and CBD
  - i. Deeply involved in communication or neurotransmission
  - ii. Act as modulator; telling some transmissions to speed up and others to slow down
  - iii. Purpose it to return the body to a normal state

## 6. So why CBD?

- a. Was probably the active ingredient in marijuana that made it work
- b. Not psychoactive
- c. Higher safety levels than THC
- d. Many attributes given to CBD

## 7. Client Attitudes

- a. Are they using it?

- i. Many people that visited the Canna-Pet website bought it
- b. Why do they use it?
  - i. Pain
  - ii. Sleep aid
  - iii. Anxiety
  - iv. Nervous system support
  - v. Reduce inflammation
  - vi. Seizures
  - vii. Nausea
- c. Perceived Problems with use
  - i. Increased appetite
  - ii. Lack of Energy
  - iii. Panic Reactions
  - iv. Dry mouth
  - v. Sedation
  - vi. Nausea
  - vii. Increased Seizures
- d. How did it stack up compared to conventional medications?
  - i. Better than any 19.3%
  - ii. Better than most 24.7
  - iii. Better than some 18.4%
  - iv. As well as some 20.8%
  - v. As well as most 9.3%
  - vi. Worse than many 2.8%
  - vii. Worse than any 2.6%
  - viii. Worse than most 2%

## 8. Safety

- a. Over 1000 research papers on CBD
  - i. Most all are human
- b. CBD found to be non-toxic
- c. Rare side effects
  - i. Possible interference with cytochrome P450
  - ii. Ivermectin? Discontinue CBD for 2-3 days
- d. Not addictive, actually anti-addictive
- e. In humans can be used as adjunct treatments to addictions such as tobacco, alcohol, opiates

## 9. Evidence

- a. Mostly from human research
- b. Anecdotal in animals
- c. Anxiety
- d. Stress areas of brain (e.g. amygdala) are rich in CBD receptors
- e. Noise aversion
- f. Separation anxiety
- g. Fear of strangers
- h. Cognitive Dysfunction

## 10. CBD's in particular

- a. Cognitive Dysfunction
- b. Neuroprotective
- c. Anti-inflammatory
- d. Antioxidants
- e. Regenerate new neurons in the part of the brain responsible for memory and can improve memory.

- f. Autoimmune disorders
  - i. Autoimmune thyroiditis
  - ii. Immune Mediated Hemolytic Anemia
  - iii. Immune Mediated Thrombocytopenia
  - iv. Pemphigus
  - v. Lupus
- g. Bone Health
- h. Helps Heal Fractures
- i. Cancer
  - i. Manage signs of cancer
  - ii. pain
  - iii. nausea
  - iv. Reduce inflammation
  - v. Induce cancer cells to die
  - vi. Slow cancer growth
  - vii. Inhibit neovascularization of tumors
  - viii. Protect non-cancerous cells
- j. Inflammatory Bowel Syndrome
  - i. Reduces mobility and inflammation
- k. Degenerative Myelopathy
  - i. No evidence in dogs
    - 1. Works well in ALS
- l. Glaucoma
  - i. Actual cat study!
    - 1. Unfortunately they used CBG cannabigerol
    - 2. Relieves pressure

- m. Degenerative Joint Disease
    - i. Reduces inflammation and pain
    - ii. Inhibits release of TNF
  - n. Inflammation is the underlying basis of a number of diseases
    - i. Pain suppression through attaching to receptors in parts of the brain responsible for pain reception
  - o. Reduction of neuropathy
11. Endocannabinoid Deficiency Syndrome
- a. For some animals, there may be a problem where the endocannabinoids fail to do their job
  - b. Supplementation may help
  - c. Acupuncture has been shown to help as well
12. Sources of CBD
- a. Hemp plants absorb heavy metals, toxins and radiation from the soil at a high rate
  - b. China produces 1/4 of the worlds hemp
    - i. Very high percentage has heavy metal contamination
  - c. US hemp is high quality but not allowed to be used commercially
  - d. European hemp is the best and safest option
13. Production of Hemp
- a. Cold Press extraction produces low CBD
  - b. CO2 extraction is best
    - i. Most effective and safest extraction method
    - ii. Similar to decaffeinating coffee
    - iii. Expensive method

# Care of the Surgical Patient: Before, During and After the Visit

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Michael C Petty, DVM

## INTRODUCTION

As a profession we have a duty to care for every aspect of an animal that has been entrusted to our care. Never is this truer than when an animal is brought to us for a surgical procedure. The owner trusts that we will do our best when an animal is in our care and out of their sight. This discussion is to outline those kinds of things that should be happening in our hospitals and clinics. We should also be counseling pet owners on how to prepare for their pet's visit and the steps necessary to keep that animal comfortable at home.

In the United Kingdom, the Farm Animal Welfare Council created a set of "rules" called The Five Freedoms. <http://www.fawc.org.uk/freedoms.htm> These rules or guidelines were designed to provide for the humane treatment and care of farm animals. Although designed for farm animals, these Five Freedoms are applicable to our domesticated pets as well. They are as follows:

- Freedom from hunger and thirst
- Freedom from discomfort
- Freedom from pain, injury and disease
- Freedom to express normal behavior
- Freedom from fear and distress

The remainder of this discussion will talk about how best to implement these ideals for our patients. But as these animals' caretakers, we often don't think beyond a comfortable place in the cage and the drugs necessary to control pain. This is a part of it, but other aspects, outlined above need to be considered. We will discuss:

- Comfort
  - Emotional support
  - Nausea and vomiting
  - Low stress handling
  - Physical environment

- Nursing care
- Post-operative nutrition

The needs of dogs and cats will be discussed together where appropriate and separately where necessary. Additionally, we will discuss how some dog breeds have special considerations.

## **ACUTE PAIN**

The 3 A's of pain should always be kept in mind: Anticipate-Assess-Alleviate. In order to treat pain properly, we need to expect when and how much a procedure is going to hurt. We need to then measure that pain to know if we are doing a proper job. And finally, we need to respond to our assessment and change our treatments to avoid both undertreating and over treating the pain. From the patients point of view, pain impacts them three different ways. First there is how the pain feels, or the physical discomfort they perceive. Then there is the psychological effect of the pain, often causing depression and leading to the third issue, how it makes them feel. The pain, the feelings of social isolation and subsequent depression and in the end their emotional state is all closely tied together as both pain and emotion arise from the same part of the brain.

Another thing to think about is the genetics of pain. Often times, different dogs are labeled as "crybabies" because of how they react to pain compared to other breeds. Think of some breeds such as Beagles and Shelties: often reacting strongly to even seemingly minor procedures. Then there are vocal breeds such as many of the Northern breeds such as Huskies who react quite strongly and vociferously to many procedures. In addition, this breed has a disproportionate number of dysphoric reactions to opioids. But, pain is what an animal tells us it is. And these animals really do feel pain more acutely than some breeds. There is a human equivalent in red haired and blue-eyed people. People with red hair suffer from gene variants of Catechol-O-methyltransferase (COMT)

Melanocortin-1 receptor (MC1R) gene variants. These people have a lower thermal threshold, need higher levels of anesthetics and don't always respond to the effects of lidocaine like other people do. I suspect that some animals have the same issue.

Finally, we need to consider the differences in animals with age and comorbidities. Our approach, both pharmacologically and socially needs to adapt to each animal group's needs. And just as we choose an antibiotic based on type of infection and length of treatment, we need to use the same reasoning when we choose our pain treatments; considering the degree of pain, the type of pain, and the individual being treated.

## **THE PLAN STARTS AT HOME**

Two things that are out of our immediate control are the emotional aspects of the ride into the clinic as well as the nausea that may accompany that trip in. Although most dogs and few cats seem to enjoy car rides, we need to consider what we can do to alleviate the stress in those animals that don't. Additionally, nausea on the car ride in can set the tone for the entire hospital stay.

Cats are most stressed by the car ride in. And before the car ride even starts, there is the stress of being chased down and stuffed into a carrier. The carrier can and should be a safe-haven for the cat. Although this can be accomplished at any age it is best to start out when they are just kittens. Using the carrier to hold a soft bed, keep toys in and give treats in will acclimate cats to that carrier, making it a home-away-from-home on the trip to the veterinarian. For those cats that are still stressed, or have not been acclimated to the carrier, they can be treated with medications prior to the ride in. My favorite method is to give them 100 mg of gabapentin. This can be given about 90 minutes prior to the ride in either by mouth, or by withholding food and then mixing it with a tablespoon of their favorite wet food. Most cats will respond to this method of sedation and be much more comfortable for both the car ride and the initial examination at the clinic. Just warn the owners that the cat may be a bit woozy. Feliway wipes can also be dispensed to help with reducing stress in cats. I often offer cat owners this pheromone at the time they come in to pick up their gabapentin

Dogs are less likely to have the same degree of stress for car rides and veterinary visits, but for those dogs that do trazodone can be administered at a dose of 2-3 mg/kg or gabapentin at 5-10 mg/kg. As with cats, warn the owners of sedation. In both dogs and cats, neither of these drugs seems to have a significant effect on the doses of anesthetic medications administered.

Nausea is a big problem for many animals. Although the brand name drug Cerenia is indicated for the control of vomiting, recent studies show that if it is given both the day before and the day of travel and anesthesia, that it reduces feelings of nausea as well. In one human survey given post operatively, people said that the feelings of nausea were much worse than the pain that they felt. Looking at some of our animals, I would agree that it is true of them as well.

## **THE PLAN CONTINUES AT THE HOSPITAL**

And the plan starts with emotional support and a quiet and calm environment. This includes caging and recovery areas that are quiet, separated by species and have low lighting. Remember, as we discussed earlier, the emotional and pain centers are handled by the same areas of the brain. Many clinics have

separate cat and dog wards, but the recovery and intensive care areas are often shared. Try to keep cats separated from dogs as much as possible. This includes keeping them on the higher levels of cages.

Give your patients emotional support, allowing your nursing staff to spend the time to socially interact with all animals, but especially those that are more obviously frightened and elderly animals that might not be as used to changes in their environment. Encourage owners to bring favorite toys, stuffed animals or blankets in with them. Keep a supply of stuffed animals at your clinic to put in cages with your patients.

You may want to consider training your staff and becoming either an AAFP Feline Friendly Practice or a Fear Free practice. In any case, you need to make sure that the following needs are being met:

- Emotional and physical support
- Assess pain and anxiety
- Assess the environment
  - Light
  - Noise
  - Smells

## **HOSPITAL DESIGN**

Slippery floors are scary to dogs, especially older dogs, dogs with degenerative joint disease and dogs with neurologic conditions. Consider in the design or remodel of any hospital to use rubber flooring. Although slightly harder to keep clean of fur, it is non-slip and hygienic. In lieu of changing the flooring, yoga mats can be bought inexpensively in 30-foot rolls and used to help dogs negotiate hallways.

Certain types of music have been shown to be calming to both dogs and cats. Playing this music in the reception area and in the animal wards can have a profound effect on their emotional state.

Cages should be another consideration. In an effort to economize, many recovery areas and animal wards are made with the smallest cages possible. Cats especially suffer; with litter box next to food and bedding. Newer cage setups include a separate area for the litter box and an elevated private area for sleeping. Older cages can be modified by putting in a platform with a curtain for those cats that feel safer when out of view. Even something as simple as domed cat bed added to the cage can give the cat needed privacy and separates the litter boxes from the feeding area. Stressed cats have elevated stress hormones, which lead to anorexia, hyper vigilance increased level of arousal and lack of sleep, all

contributors to pain. Both dogs and cats need a sufficient area to stretch out as much as they would like and on comfortable padded bedding.

Patient positioning and transport falls under both hospital design and patient handling. Too many times, large dogs are moved inappropriately or are made to lie on tables too small. All tables should be warm, padded and of an appropriate size. Consider the addition of a power gurney to lift and transport large dogs.

Patient positioning during surgery is another source of pain for both dogs and cats. In human medicine, medical staff is often trained to consider their patient's positions by getting in those same positions themselves and holding them for a period of time. We can't do the same for our patients, but we need to take a hard look and consider what it would feel like. And we also need to consider all of the rope restraints that are commonly used. If an animal is under anesthesia or sedation with good pain control, the only restraint usually needed is a V-trough.

## **ANESTHETIC CONSIDERATIONS**

Nausea and vomiting was discussed as starting at home, but we need to consider it in our hospitals as well. But remember some drugs cause nausea more than others. Morphine, hydromorphone and alpha-2 adrenergic drugs can all cause nausea and subsequent vomiting. Consider the addition of Cerenia or even acepromazine to reduce the incidents of vomiting. Cerenia also has the added benefit of decreasing MAC anesthetic gases.

Warmth, especially during surgery and recovery, and especially with the use of an alpha-2 adrenergic that prohibits shivering, is very important to the comfort and well being of an animal. Warming devices for veterinary patients are available. Even the simple act of warming towels or blankets in a dryer can help with the comfort of recovering patients. And the next time you walk through a pet store, look in the discount bin for coats and sweaters for both dogs and cats.

Nutrition is an important part of an animal's recovery. Timely nutrition can sharply reduce the recovery period of both dogs and cats. The following guideline should be considered when starting a postoperative patient on food:

- Dogs = 30% of calories from fat and at least 27% of calories from protein. Carbohydrates in nutritional support diets should not include maize, wheat or, especially, soy.
- Cats = 30% of calories from both fat and protein.
- Providing nutrition via a functional digestive system is the preferred route of feeding
- Provide small amounts at first

Prolonged anorexia can lead to:

- Generalized wasting
- Delayed wound healing
- Impaired immune function
- Altered drug metabolism
- Increased morbidity and mortality in human patients, so probably in our patients as well

For those animals that do not want to eat, just remember that what is appealing to one animal may not be so appealing to the next. Try a variety of foods and always try to get the animal to eat voluntarily. Consider possible inflammation from intubation as a source of pain. There are appetite stimulants available including

- Benzodiazepines
- Cyproheptadine
- Mirtazapine
- Entyce

For those animals that still refuse to eat, other causes must be considered. The first set of considerations fall under pseudo-inappetence and include:

- Oral disease
- Food aversions
- Unpalatable food
- Neuromuscular disease

True inappetence may be secondary to systemic disease or reactions to treatments such as:

- Systemic illness

- Renal failure, cancer
- Pain
- Nausea
- Drug related
  - Opioids, chemotherapy
- Heart failure

### **THE IMPORTANCE OF RECOVERY IN THE POST OPERATIVE PERIOD**

The greatest risk of death occurs during the immediate post anesthetic period. In cats 61% and in dogs 47% of anesthetic related deaths occur mostly within the first 3 hours. This makes it important that patients recovering from anesthesia are closely monitored and managed.

### **OVERLOOKED SOURCES OF PAIN AND DISCOMFORT**

There are many possible “incidental” sources of pain in our patients. Some to consider are:

- Venous catheter placement
- Urinary catheter placement
- Cystocentesis
- Thoracocentesis
- Clipper burn
- “Minor” biopsies
- Skin scrapings
- Drains
- Feeding tubes
- Bandage changes

Careful clipping and treatment of even minor procedures or the pain of those procedures can add to the overall comfort of a patient. Consider the use of topical lidocaine patches or lidocaine creams for areas of skin pain.

## GOING HOME

When should the animals be allowed to go home? What analgesics should be used? What other treatments should be considered? Consider the following when choosing at-home treatments:

- What works at home?
  - What can owners deal with?
  - Compliance
  - What is efficacious?
  - Monitoring?
  - Liability issues?

Cold compression may seem very “old school” to some, but it is inexpensive and easy to use. The reader can look up cold compression techniques, but the equipment is basic; a tea towel, an ice source and an ace bandage. Local application of cold causes temporary decreases in:

- blood flow to the area
- edema formation
- hemorrhage
- histamine release
- local metabolism
- muscle spindle activity
- nerve conduction velocity (NCV)
- Pain
  - 1 or 2 -15 minute applications can have benefits up to 24 hours

Where appropriate, certain drugs should be considered as well.

NSAIDs have long been considered the gold standard of both acute and chronic at home pain relief. They should always be considered whenever appropriate based on the surgical procedure and any comorbidities the animal might have.

The new piperidine drug, grapiprant should be considered for use in dogs where NSAIDs are not appropriate for that patient. Although only approved for the treatment of chronic pain due to degenerative joint disease, there are acute pain studies in rats that show they are effective. This has also been my experience as well.

Simbadol injectable buprenorphine for cats lasts 24 hours and can be given for up to three days. It is not appropriate to send home with an owner, but many clients will happily bring their cat in for additional injections. Oral transmucosal buprenorphine can be used as well, although sending home an injectable opioid does carry with it some regulatory risk.

Oral opioids, besides buprenorphine, have little to no availability. They should not be relied upon for postoperative pain control.

Tramadol is an effective pain reliever in cats, but studies show that has poor bioavailability in dogs. Even when it is detected in the plasma, it often lasts no more than minutes. In cats, the opioid effect of tramadol when given at 2-4 mg/kg lasted up to 260 minutes. However, in both dogs and cats there were reports of toxicity.

Gabapentin has been shown to help with postoperative pain in cats. No good studies have been shown to have the same effect in dogs, but it still might be a consideration given its anti-neuropathic pain properties.

## RESOURCES

- Pain tools
  - Glasgow CMPS-SF – dog
  - Glasgow CMPS – feline
- Feline friendly handling and nursing guidelines
  - [www.catvets.com](http://www.catvets.com)
- 2015 AAHA/AAFP Pain Management Guidelines
- 2015 AAHA Canine and Feline Behavior Management Guidelines

## April J Bays CVT, VTS (ECC)

April has worked in veterinary medicine since 2001, becoming a certified technician in 2007. After working relief at an emergency clinic she found her true calling, and earned her Veterinary Technician Specialty in Emergency and Critical Care in 2013. Her interests include pain management, metabolic, endocrine and respiratory emergencies. More than anything, April enjoys mentoring technicians and motivating them to push themselves to their full potential.





# Sugar, We're Going Down

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April Bays CVT, VTS (ECC)

Diabetic ketoacidosis constitutes a severe life-threatening complication of diabetes, requiring prompt and aggressive therapy. Most patients presenting to the hospital with DKA are undiagnosed diabetics. Symptoms such as polyuria, polydipsia, polyphagia, and weight loss may initially be subtle, going unnoticed which can result in an animal in crisis seemingly overnight.

## What Happens?

First, let's review what is supposed to happen. Every part of the body is made up of cells, each with a myriad of tasks and functions. In order to produce adenosine triphosphate, or ATP (the energy that almost all cells use to function), cells need fuel in the form of sugars and fats from an outside source. When these carbohydrates are consumed, they are broken down into smaller molecules and finally, glucose. When introduced intracellularly via insulin, glucose is further metabolized through the pathways called glycolysis and the Krebs cycle, ultimately producing ATP. When glucose demands are met and no longer needed for energy by the body, it is stored away as a fuel reserve known as glycogen. Glycogen is a multi-branched chain of stored glucose that can easily be broken down and released in the blood. The brain can only use glucose and ketones for energy (preferring glucose) but it only stores a very small amount of glycogen, requiring a steady supply of glucose.

Insulin helps to regulate blood glucose levels and works by attaching to receptors on cell walls. Together they act as a 'lock and key' type mechanism. This action allows the uptake of glucose into the cell, which then can be utilized within the mitochondria, and voila, we have ATP! Insulin's effect can also be seen at the sodium/potassium ATPase pump ( $\text{Na}^+/\text{K}^+\text{ATPase}$ ), the function of which is to maintain electroneutrality between the inside and the outside of the cell membrane. It does this with two major cations (positively charged ions), sodium and potassium. The majority of sodium, about 60%, resides outside of the cell in the extracellular fluid, whereas potassium is 98% intracellular. Insulin acts on this pump to facilitate the movement of potassium into the cell in exchange for sodium molecules, keeping it balanced at all times. Without insulin, potassium levels in the serum may appear normal or even elevated, but this is due to the lack of movement into cells (later exacerbated by the acidosis, shifting potassium out of cells as well) and therefore, misrepresent the total body numbers.

## **The Pancreas**

The pancreas sits adjacent to the stomach in the cranial abdomen. Its functions include hormone synthesis and secretion, as well as producing enzymes that aid in digestion. These enzymes include trypsin, amylase, and lipase. The digestive enzymes are actually in an inactive form in the pancreas and do not activate until later after leaving the pancreas.

Trypsinogen, the inactive precursor of trypsin, is activated to trypsin in the small intestine. Trypsin is responsible for activation of the other pancreatic enzymes that aid in digestion. Premature activation of trypsinogen to trypsin within the pancreas results in autodigestion of pancreatic cells and causes acute inflammation of the pancreas. Once autodigestion begins, inflammatory mediators perpetuate the progression of pancreatitis. These mediators directly injure pancreatic tissue further, resulting in more damage, and a vicious cycle ensues. The underlying etiology remains under speculation; however, for dogs and cats it is most commonly idiopathic.

The pancreas contains areas of specialized cell clusters called the islets of Langerhans. Within the islets are alpha cells and beta cells. Beta cells secrete insulin in response to high serum blood sugar. Conversely, alpha cells secrete glucagon in response to low serum blood sugar. Together they are mainly responsible for glucose homeostasis. Due to the degeneration or destruction of these islets, the pancreas can no longer produce enough insulin, which is the main cause of insulin-dependent diabetes mellitus.

## **Insulin**

Diabetics can have a relative or complete lack of insulin production. As discussed, lack of insulin prevents glucose from entering cells to be used, resulting in a hyperglycemia. So, even though these patients do not have a shortage of fuel, without insulin the cells are essentially starving.

This perceived low serum glucose stimulates the secretion of glucagon to raise blood sugar. Glucagon then triggers the liver to reverse the process by which glucose was stored as glycogen. You'll remember excess glucose is synthesized into chains and stored away as backup fuel in the form of glycogen.

Glycogenolysis is the process of breaking down (-lysis) those chains of glycogen, back into simple sugars to be released into the blood. Meanwhile, instead of metabolizing carbohydrates, the body will also resort to fat oxidation to produce energy.

Serum glucose levels continue to rise in the blood. Eventually, if glucose reaches a threshold (around 250mg/dl), glucose will begin spilling into the urine. Glucose is a molecule that draws water to it by osmosis, otherwise known as osmotic pull. This keeps the fluid portion of blood within the vessel walls, rather than leaking out. When glucose floods into urine, so does water. This is known as osmotic diuresis.

In other words, patients end up losing excessive amounts of water. This explains the frequent urination and excessive thirst seen in diabetics.

The lack of insulin and elevated glucagon levels complicate things even further. They both inhibit glycolysis, and stimulate gluconeogenesis (discussed below) as well as decreasing the utilization of glucose at peripheral tissues. Decreased glucose utilization combined with increased gluconeogenesis has a significant effect on serum glucose levels, producing profound hyperglycemia and osmotic diuresis. This results in hyperosmolarity, a higher concentration of solutes in plasma, leaving the patient extremely dehydrated. Electrolytes are lost at increased rate because of the osmotic diuresis, and so to replenish the intravascular electrolytes, these electrolytes leave the intracellular space to the vascular space. This eventually leads to a total body depletion of sodium, potassium, phosphorus and magnesium.

### **Gluconeogenesis, Lipolysis, Proteolysis, and Ketones**

Gluconeogenesis primarily takes place in the liver. Gluco (sugar), neo (new), genesis (creation of). Essentially it is simply the creation of new glucose, and more specifically, it is synthesized from the usual non-carbohydrate sources. Gluconeogenesis is the opposite pathway of glycolysis. Instead of breaking down sugars into tiny molecular pieces, it uses new pieces, along with other contributions from fat and muscle breakdown and reverses the process to create sugars. Though not as efficient, it is a dependable alternative to a desperate need for fuel.

Those pieces come from multiple sources. Glucagon induces proteolysis, providing amino acids, and lipolysis providing free fatty acids. The body will begin to break down muscle (proteolysis) into amino acids. Amino acids will take part in gluconeogenesis in the liver. Glucagon induces breakdown of triglycerides from adipose tissue. Triglycerides are further metabolized into glycerol, which is carried to the liver for gluconeogenesis, and free fatty acids.

In the liver, these free fatty acids are oxidized for ketogenesis (the creation of ketones). Ketones break down further into three ketone bodies;  $\beta$ -hydroxybutyrate acids, acetoacetone and acetone. The liver will then excrete these ketone bodies into the blood. Ketones can enter and be used in cells freely, unlike glucose which requires insulin. This offers cells fuel. The patient can breathe off the acetone which has a sweet smell, explaining the "ketone breath" that can sometimes be appreciated in diabetic patients. Large amounts of ketones will eventually exceed the renal threshold and also spill into the urine. This is known as ketonuria. As acids that must be buffered to be excreted, ketones will bind sodium and potassium. This draws with it even more water, and further exacerbates the osmotic diuresis as well as electrolyte loss.

## Acidosis

Now amino acids and ketones are circulating, both contributing to a severe metabolic acidosis. Acidosis is a condition where a systemic buildup of acids lowers the blood pH, offsetting the balance between acids and buffers leading to an increase in circulating acids. The body works hard to maintain a pH in a narrow window between 7.35-7.45, and does so for good reason. Derangements on either side of that window can have deleterious effects. A patient with a pH of <7.35 is considered acidotic, and a pH of >7.45 is considered alkalotic. The causes can be further broken down into metabolic, respiratory, or mixed. Acidosis begins to alter the way many enzymes work, particularly in the brain, which can lead to coma or death. Hydrogen ions (H<sup>+</sup>) that accumulate in the blood can be exchanged for potassium cations from the cells. An efflux of potassium from the intracellular to extracellular space takes place causing a total body loss, as excessive potassium is then lost in the urine with osmotic diuresis. Patients with a severe acidosis may also have altered respirations, known as Kussmaul respirations (more rapid, deep breaths), to compensate for acidemia. Remember, one way the body rids itself of acids is to breathe them off, such as in the case of acetone.

## Presentation and Diagnostics

Patients can present with varying degrees of altered mentation, obtundation, severe dehydration, weakness, tachypnea, acute onset of abdominal pain, acetone breath, and Kussmaul respirations.

Diagnostics should start with full blood work, including a complete blood count, chemistry analysis, and electrolytes, including a venous pH for acid/base evaluation. Also a spec cPL to test for pancreatitis. A complete urinalysis is necessary to look for urinary tract infections, which are common in diabetics, as well as glucosuria and ketonuria. Urine culture may be warranted and therefore cystocentesis is ideal for collection. Serum ketones can be tested on the urine dipstick. The urine reagent strips only detect acetoacetate, but do not measure  $\beta$ -hydroxybutyrate. Adding one to two drops of hydrogen peroxide to the urine will convert  $\beta$ -hydroxybutyrate to acetoacetate and acetone, which can be measured on the ketone pad.

Patients with DKA will have an elevated anion gap. An elevated anion gap indicates that there are unmeasured anions (negatively charged ions) in the blood, and is determined by calculating the difference between the measured cations and anions. The primary measured cations are sodium (Na<sup>+</sup>) and potassium (K<sup>+</sup>); and the primary measured anions are chloride (Cl<sup>-</sup>) and bicarbonate (HCO<sub>3</sub><sup>-</sup>). In order to calculate the gap we add together the number of Cl<sup>-</sup> and HCO<sub>3</sub><sup>-</sup> anions and subtract from the number of Na<sup>+</sup> and K<sup>+</sup> cations. Therefore;  $AG = (Na^+ + K^+) - (Cl^- + HCO_3^-)$ . The gap simply occurs because laboratory equipment doesn't routinely measure all ions. Albumin, the major protein in plasma, contributes to most

of the anion gap, so some gap is normal. It is when there is an excess that we need to track down the culprit. Causes of an increased anion gap metabolic acidosis are; lactate, urea, ethylene glycol, and ketones.

## **Treatment**

So what do we need to treat? Remember we have more than hyperglycemia. We have a hyperketonemia, hypokalemia, severe dehydration, and metabolic acidosis. Treatment is aimed at correction of the metabolic acidosis most acutely. This is achieved by replacing fluid loss via intravenous fluids, restoring electrolyte balance, and then treatment with insulin. It is essential to start with replacing the volume depletion with IV fluids such as 0.9% sodium chloride with no additives initially. Rehydrating the patient will begin the process of restoring electrolyte balance and begin lowering glucose levels, but no more than 50-100mg/dl/hr

A peripheral intravenous catheter (IVC) should be placed to begin fluid volume replacement. Ideally, a long term central catheter would be placed, such as a peripherally inserted central catheter (PICC), or a central line placed catheter in the jugular vein. These long term catheters are ideal for rapid fluid administration, which our dehydrated patients desperately require. The dramatic fluctuations in glucose and shifting of electrolytes demands diligent monitoring, often every two hours. These large bore catheters facilitate easy, painless blood sampling, without stress to the patient and trauma to multiple vessels.

Large vessels are ideal for hypertonic solutions. Hyperosmolar (or hypertonic) solutions are osmotically active with a higher concentration of osmoles (or molecules) than the plasma. Remember we learned glucose has osmotic pull, thus a dextrose infusion with its high concentration of glucose is considered a hypertonic solution. Infusion of these solutions in small peripheral veins can result in irritation and discomfort as it effectively dehydrates the surrounding tissue leading to a chemically induced phlebitis.

The jugular catheter sits in the cranial vena cava and can be used to monitor central venous pressures (CVP). As long as the PICC line ends in the caudal vena cava, you may use it to measure CVPs as well. In a hemodynamically unstable DKA patient, these pressures are quite valuable in monitoring progress and guiding fluid therapy.

As serum glucose reaches 250mg/dl, your doctor may instruct you to begin an insulin constant rate infusion (CRI). This is done by adding 2.2U/kg for dogs and 1.1U/kg of regular insulin in a 250ml bag of

0.9% saline solution. Regular insulin is very short-acting, unlike the long term maintenance insulin the owners will be giving at home, and is therefore ideal for frequent adjustments and to avoid overdosing. When setting up your CRI, remember that INSULIN BINDS TO PLASTIC, so you must connect the set through all the tubing that the insulin will run through and run 50ml through it. Once you've run the line, you'll begin a slow infusion of the insulin solution. It is important to also give the body some sugars to prevent a sudden hypoglycemia, so a dextrose CRI will be started simultaneously.

As blood glucose levels fluctuate, there will be adjustments made to the insulin CRI and the dextrose CRI. The best way to account for the frequently changing concentration of the dextrose solution is to use a buretrol, or burette. The burette is attached to the liter bag of fluids like the usual drip set. There is a separate 150mL chamber that allows you to make adjustments to 150ml aliquots rather than changing out a whole bag. Dextrose is added to a smaller chamber instead of the liter bag, allowing for easier emptying and changing of concentrations. Your doctor may use the insulin/glucose table at this time to make adjustments while monitoring glucose closely. At this time you may very well have a euglycemic (normal glucose level) patient, but they are still very ketotic, and therefore, acidotic. Glucose levels can return to normal in 12 hours, while it takes 24-48 hours to eliminate the built up ketones that remain in the body. This is why we continue insulin therapy until the anion gap and excess ketones are eliminated.

You'll also recall how insulin was responsible for some of the uptake of potassium intracellularly. The lack of insulin and the resulting ketoacidosis causes a transcellular shift. A large amount of the shifted extracellular potassium is lost in urine due to osmotic diuresis. Serum levels may not accurately represent total body stores and depletion is a common consequence, even though blood work may show normal to high levels.

The administration of isotonic fluids is the first step to beginning to improve the acid base balance of this patient. Once the insulin is initiated, glucose and potassium both move intracellularly, combined with diuresis and the resolution of acidosis, you will usually see such a dramatic flux of potassium that it'll be necessary to supplement at this point, to prevent hypokalemia. The same effect is observed with phosphorus. Phosphate will return to the intracellular compartments with the introduction of insulin, revealing a hypophosphatemia. Monitoring of these values can help prevent extreme shifts in either direction. Potassium chloride (kcl), or both potassium/phosphorus (Kphos), can be added to the IV fluids to supplement. Potassium should not be increased more quickly than 0.5mg/kg/hr.

Keeping in mind that there's usually an inciting stressor involved, our job here is not done. It is essential to identify any underlying conditions such as, concurrent disease process, infection, inflammation,

hyperadrenocorticism (Cushing's disease), or congestive heart failure. Treatment will depend on diagnosis, but may include antibiotics, dietary supplementation, and analgesics. For example, pancreatitis, a common concurrent condition, is known to be extremely painful. Do not underestimate the importance of treating our patients' pain to the best of our ability. Not only is pain uncomfortable for our patients, but it also is proven that providing analgesia improves healing, circulation, and oxygenation, as well as more promptly returning to proper nutrition.

When serum glucose levels maintain balance, we rehydrate our patients, and our pH normalizes, we will switch to a longer acting insulin.

### **Nursing care**

These patients are highly susceptible to infection, and strict aseptic technique should be practiced. Gloves should be worn with these patients and injection ports wiped with alcohol before use. Pay special attention to patient comfort and cleanliness. If the patient is non-ambulatory care should be taken to provide adequate bedding, and rotate recumbency every two to four hours. Lubricate eyes and mucous membranes if the patient isn't accomplishing this on its own. Giving large volumes of fluids necessitates frequent walks and bed checks. If the patient soils itself, clean thoroughly to prevent skin break down and infection. To assure adequate renal function a urinary catheter may be placed to measure urine output. Remember, sterility is of utmost importance, especially in terms of urine collection systems. Sterile technique should be practiced for placement of the urinary catheter and it too, should be cleaned.

A vital component of nursing care is being aware of changes in your patient. Electrolyte abnormalities can cause electrical disturbances including arrhythmias leading to death. Observing the patient for alterations in heart rate and rhythm, respirations, muscle weakness and mentation, is critical to early and prompt intervention.

Overzealous fluid therapy can lead to fluid overload, and signs such as chemosis (edematous conjunctiva), pulmonary edema, and peripheral edema may be observed. It is of critical importance for the technician to be aware of the signs of complications, from phlebitis to neurologic signs of cerebral edema.

References available upon request



# Learn To Love Your Curves

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April Bays CVT, VTS (ECC)

RBC's carry the critical protein hemoglobin (Hgb), within which is the iron containing heme group that oxygen ultimately binds to. Other substances are also capable of binding to Hgb which will affect the affinity that hgb has for oxygen. We will discuss those factors and what role they play in gas exchange and the delivery of oxygen in critical illness.

## **PO<sub>2</sub> and Hemoglobin Affinity**

Oxygen molecules travel either bound or unbound in the blood. The majority of oxygen being bound, the rest is considered unbound, and dissolved in the plasma. Oxygen levels are measured as a *partial pressure* (PO<sub>2</sub>), or PaO<sub>2</sub>, to signify arterial oxygen partial pressure. Essentially this translates to the oxygen molecules' collective exerted force on its environment. This number represents the amount of oxygen, therefore the more oxygen molecules in the blood, the higher the partial pressure.

Partial pressure will determine the binding affinity to Hgb. One could think of affinity as how tightly Hgb holds on to the oxygen molecule. The higher the affinity, the more easily oxygen will bind, and the more difficult it will be to offload the oxygen. A lower affinity will mean that it is more difficult to load the oxygen, but easier to offload.

## **The Curve**

The oxyhemoglobin dissociation curve represents the carrying capacity of Hgb. You'll see by the sigmoid shape, that from the zero point, the percentage of saturated Hgb is zero because no O<sub>2</sub> is available (Fig. 1). The Hgb doesn't begin the upswing of taking on appreciable numbers of O<sub>2</sub> until the PO<sub>2</sub> reaches a significant amount. The characteristic curve represents cooperativity, or cooperative binding. Cooperative binding means that once oxygen starts binding to hgb it encourages the binding of more oxygen. So the binding of one oxygen molecule, will in turn, make the hgb more likely to pick up even more oxygen, until the point where most of the hgb is full. This is the level at which the curve begins to plateau. The upper regions of the curve shows that once Hgb is around 97% saturated, even a large change in PO<sub>2</sub> will not significantly alter the bound Hgb percentage. Conversely, on the lower portion, a sharp slope demonstrates that even a small change in oxygen concentration, results in a dramatic change of saturation.

The curve is dynamic, in motion, to meet the needs of the body. This allows Hgb to bind as much oxygen as possible when it circulates to the lungs, by having a very high affinity. And once it returns to the tissues, where oxygen needs to be easily released, it will have a lower affinity.

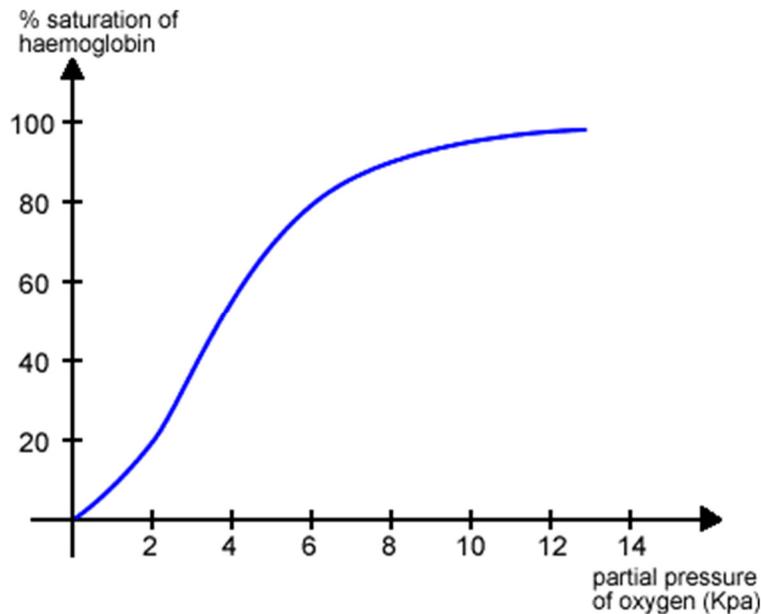


Fig 1. The X-axis represents arterial partial pressure of oxygen, which is measured in mmHg.

The Y-axis represents the saturation of Hgb by oxygen, measured as a percentage.

As tissues serve their function they utilize oxygen and respire  $\text{CO}_2$ . The arrival of saturated Hgb to an area of low  $\text{O}_2$  results in the diffusion of  $\text{O}_2$  in exchange for  $\text{CO}_2$ . As one oxygen molecule is released to the tissue in an area of low concentration, it relaxes the binding of Hgb, allowing more offloading to occur. Once again, when the RBC returns to the lungs, where the alveolar concentration of oxygen is high, Hgb grabs onto an oxygen molecule, and releases a carbon dioxide molecule. The Hgb will eventually become fully saturated, holding on tightly to the oxygen molecules, until the RBC moves along in circulation, reaching an area fitting the criteria to allow offloading.

The point at which 50% of Hgb is saturated is referred to as P50. This point is important because we consider it to be where the shift originates from. From this point, we can understand the affinity for oxygen at any given concentration or condition.

## Factors

Factors affecting the affinity of Hgb are; temperature, concentration of CO<sub>2</sub>, pH, and 2,3 diphosphoglycerate (2,3 DPG) - an intermediate of glycolysis, present in RBCs. Each of these provoke the movement of the curve either to the left, contributing to a higher affinity, or to the right, contributing to a lower affinity. You can also imagine that to the left of the curve the blood is in the lungs, where Hgb needs to grab onto as much oxygen as possible. And to the right, blood is in the tissues, where Hgb should be relaxed, and willing to offload the bound oxygen molecules.

## Shifts To The left

A “shift to the left” means the curve will keep the same shape, just moved left. This is where the P50 is utilized to help illustrate the change. Now, P50 will require less PO<sub>2</sub> for Hgb to become saturated to 50%. In other words, it takes a lower concentration of O<sub>2</sub> to reach that 50% saturation. For example in the lungs. This is an area that would benefit from a higher affinity. The air being inspired will be cooler than the body, there is a lower concentration of CO<sub>2</sub> due to ventilation, lower concentration of 2,3 DPG, and a higher pH. Which means that with a slight increase in PO<sub>2</sub>, that more binding will take place.

## Shifts To The Right

The opposite produces a “shift the the right”. You’ll recall that a right shift is typical in an area of actively respiring cells, such as muscles, where tissues need to receive oxygen on demand. Thus, an elevated CO<sub>2</sub>, elevated temperature, increased 2,3 DPG, and low pH are the physiological conditions that cause a right shift, allowing Hgb to offload oxygen.

## In The Critically Ill

What we care about in our patients is how much O<sub>2</sub> the tissues are receiving. And we’ve discussed how Hgb saturation should represent oxygenation, which is dependant on PO<sub>2</sub>. How do we assess Hgb saturation? The pulse oximeter (SpO<sub>2</sub>) is the easiest way to measure Hgb saturation bedside. The gold standard would be arterial blood gas (ABG) measurement, however due to the complexity of the procedure and materials required, ABG testing is not readily available. So with the tools we have available, and the concept of the curve, the SpO<sub>2</sub> can be correlated to an approximate PO<sub>2</sub>. Keeping in

mind that a normal  $PO_2$  should be between 80-100mmHg (at sea level), one can quickly surmise that an  $SpO_2$  of 90%, which correlates with a  $PO_2$  of only 60mmHg, demonstrates severe hypoxia for your patient.

Now you can predict, and treat your patient's oxygenation through utilization of these skills. Understanding how the consequences of illness, such as fever, or shock states, acid/base balance etc, can factor into the overall well-being of our patients, is critical in nursing your patients.

References available upon request

# Unintended Heavy Breathing

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April Bays CVT, VTS (ECC)

Oxygen delivery is the single most life sustaining element for all animals. It is dependent on the functioning of both respiratory and cardiovascular systems. Oxygen must get into the lungs, but equally important, it must be delivered to reach the demands of the body. Dysfunction in either of these systems results in detrimental consequences.

## Clinical Signs

Increased respiratory rate is a classic sign of dyspnea, though not always. Excitement, stress, and pain can all cause an increased RR, thus evaluating effort is equally important. Notice that an animal with increased effort is also focused on breathing and is not easily distracted. Catecholamine release in stress can cause vasoconstriction leading to a pale mucous membranes. They may have increased abdominal effort, and an abnormal posture. Orthopnea is a posture exhibited in severe distress where an animal has an extended neck, and front legs are spread, to maximize the movement of air. Cyanosis represents an SpO<sub>2</sub> of 73-78%. Considering that hypoxemia is defined at oxygen saturation less than 95%, cyanosis is a sign of extreme hypoxia and require immediate intervention. It also reminds us that pink mucous membranes or tongue, are not representative of adequate oxygenation.

Dyspneic cats in particular are extremely fragile. They are experiencing severe stress and should be considered on the verge of collapse. The three most likely causes for respiratory distress in cats are cardiogenic pulmonary edema, asthma, and pleural disease. These cats should be placed in an oxygen cage and LEFT ALONE. Do not underestimate how easily these cats can be pushed over the edge. This means taking many breaks between diagnostics and treatments. You may only be able to take one D/V xray, and that's it. Put the cat back in O<sub>2</sub> immediately and give it a break. If your patient is so stressed that it could go into respiratory arrest, you may not get a chance to complete any diagnostics. Don't be afraid to tell your doctor that you are not comfortable stressing the cat further. That is why you are there! For cats that are simply too distressed to examine, one might consider treating for the most likely causes by giving lasix, dexamethasone and terbutaline. And most importantly, provide oxygen and no handling.

## A-a Gradient

The difference between the alveolar partial pressure of oxygen (PAO<sub>2</sub>) and arterial partial pressure of oxygen (PaO<sub>2</sub>) is known as the A-a gradient. Because we cannot measure the amount of oxygen in the alveoli, we use the alveolar gas equation to calculate it. (See end for equation).

$$\text{A-a gradient} = \text{PAO}_2 \text{ (calculated)} - \text{PaO}_2 \text{ (measured)}$$

Measuring this difference in the blood gas is useful in determining the origin of hypoxemia. *Hypoxemia* is defined as a low oxygen level (PO<sub>2</sub>) in the blood. *Hypoxia* is the result of inadequate oxygen delivery to the tissues. There are five basic conditions that result in hypoxemia;

*Low FiO<sub>2</sub>* (fractional concentration of inspired oxygen) can develop into hypoxemia in a patient at high elevation or with an upper airway obstruction, such as laryngeal paralysis, where a patient is unable to inspire enough oxygen. These conditions cause both a low PAO<sub>2</sub> and PaO<sub>2</sub>, therefore the A-a gradient would not be elevated. Oxygen supplementation is beneficial and CO<sub>2</sub> levels are normal or even low if ventilation is increased in an attempt to improve oxygenation.

*Hypoventilation* can result from failure of the respiratory center in the brainstem, drug induced/anesthesia, defects in the chest wall, or an obstruction. It is manifested by an elevated PCO<sub>2</sub>, typical of hypoventilated patients as CO<sub>2</sub> builds in the absence of appropriate ventilation. A-a gradient is typically normal and there is a good response to oxygen therapy in these patients.

*V/Q mismatch* is the most common cause for hypoxemia in small animal medicine. The “V” represents ventilation and “Q” represents perfusion. Therefore V/Q mismatch occurs when the oxygen level in the lungs does not match tissue oxygenation. Pneumonia, pulmonary edema, emboli, and contusions can cause alveoli to partially or fully collapse, reducing the oxygen loading into the blood. Usually this occurs due to accumulation of fluid, preventing appropriate ventilation in these areas. In these cases, oxygenation does improve with supplemental oxygen. A-a gradient would be elevated and PCO<sub>2</sub> elevated.

*Shunting* is the most severe type of V/Q mismatch. Severe atelectasis with completely collapsed alveoli or with severe disease produces area/s where blood passes by without gas exchange. Examples include

severe pneumonia, pulmonary edema, acute respiratory distress syndrome (ARDS), and alveolar collapse. In the case of complete intrapulmonary shunting, the blood from the right side of the heart enters the left side without entering the lungs and taking part in gas exchange. Also called right-to-left shunting, there are three types; anatomic, capillary (i.e. submersion victim who aspirated H<sub>2</sub>O into alveoli, causing surfactant washout and atelectasis), and venous admixture (i.e. blood traverses pulmonary capillaries and respire with alveoli that have low O<sub>2</sub> tension). Because of the shunted circulation, they do not typically improve with oxygen therapy, there is an elevated A-a gradient, and normal PCO<sub>2</sub>.

*Diffusion impairments* occur when there is an impairment of oxygen crossing the alveolocapillary membrane due to thickened areas between the alveoli and capillary. Pulmonary fibrosis, edema or hemorrhage that hasn't reached the alveoli but is in the interstitium can all cause diffusion impairments, although these are less common causes of hypoxia in veterinary species. Patients will benefit from oxygen therapy, have an elevated (A-a) gradient, and a normal PCO<sub>2</sub>.

	Responds To Oxygen?	A-a Gradient	PCO <sub>2</sub>
Low FiO <sub>2</sub>	Yes	Normal	Normal
Hypoventilation	Yes	Normal	Elevated
V/Q Mismatch	Yes	Elevated	Elevated
Shunting	No	Elevated	Normal
Diffusion Impairment	Yes	Elevated	Normal

### 8 Types of Respiratory Emergencies

With an understanding of how hypoxia occurs, these are the main types of conditions that lead to respiratory distress due to hypoxia or hypoxemia.

### Upper Airway

Conditions originating from the upper airway typically result in noise such as stridor, or stertor. Stridorous noise is described as a laryngeal obstruction type sound, whereas stertor is more nasal, like a snorting sound. There tends to be more inspiratory effort, however both inspiratory and expiratory noise is possible. Stabilization begins with sedation to reduce the stress and tension. If this is not sufficient the focus is then aimed at relieving the obstruction (ie laryngeal paralysis, airway sx, tracheostomy).

### Lower Airway

The bronchi in these patients is full of fluid, inflamed, or constricted, causing high pitched wheezing sounds on auscultation. Wheezes are the result of narrowed airways and are typically expiratory in nature. In cats, the classic condition associated is asthma. Chronic bronchitis with a cough is more commonly found in dogs. Treatment includes bronchodilators, possibly steroids and oxygen supplementation.

### Pulmonary Parenchyma

Inspiratory crackles are the hallmark sign of a parenchymal disease. The alveoli are full of fluid and have collapsed during exhalation. Crackles are the sounds of them popping back open. The cause can be alveolar or interstitial preventing oxygen diffusion from the alveoli into the blood. The condition associated depend on the fluid accumulated in the lungs. Pulmonary edema may be cardiogenic or noncardiogenic (NCPE), i.e. choking, strangulation, seizure, near drowning, chronic upper airway disease. Pus may accumulate from pneumonia or trauma leading to infection. Blood may also accumulate in the event of trauma, presence of contusions, or a coagulopathy. Increased permeability in the pulmonary capillaries are responsible for the NCPE, and these patients should be treated with lasix, rest and oxygen therapy as that is their primary problem. A diuretic will reduce the fluid in the lungs, while vasodilator therapy decreases the pressure and stress on the respiratory and cardiovascular systems. For infectious causes, patients would benefit from breaking up the pus and material, therefore treating with nebulization and antibiotics. Coupage remains of controversial benefit.

### Pleural Space

The space between the body wall and lungs can accumulate fluid resulting in increased pressure on the outside of the lungs. Without the ability to fully expand, oxygenation and ventilation becomes increasingly difficult. Dull lung sounds are common on auscultation due to the increased dead space. These patients present with rapid, shallow respirations known as a "restrictive pattern". The cause of distress may be from air (pneumothorax), blood (hemothorax), pus (pyothorax), chyle (chylothorax),

other exudates (FIP) that have accumulated in the pleural space. Thoracocentesis is an important diagnostic AND therapeutic tool. Tap that! One important note, check for petechiae/ecchymoses before a tap, in case of coagulopathy. A chest tube with suction would be warranted for repeated thoracentesis.

### Chest Wall

Chest wall injuries may result from vehicular accidents, bite wounds, etc, resulting in a defect in the sturdy chest wall such as with a “flail” chest. This occurs when a segment of the rib cage is fractured, leaving the area unattached and not moving with the breath. On inhalation, the section of chest wall will collapse leaving the lungs incapable of fully expanding due to the negative pressure and poor chest wall compliance. Patients in lateral recumbency may benefit from lying injured side down to optimize the functioning lung fields. Treatment involves oxygen therapy, analgesia, and potentially surgical repair of flail segment if severe.

### Pulmonary Thromboembolism

PTE is an obstruction from a clot lodged in a pulmonary vessel. The blood is no longer able to reach the oxygenated areas resulting in a type of shunt and hypoxemia. Hypercoagulable conditions (i.e. sepsis, SIRS, cardiac dz, heat stroke etc) are quite common, however thankfully, PTE aren't. Treatment consists of supplemental oxygen, and supportive care. Anticoagulants will not break down clots but prevent the formation of new clots. Thrombolytic therapy is required to break down clots.

### Abdominal Distention

A distended abdomen putting pressure on the diaphragm reduces the space the lungs can expand resulting in hypoventilation. The most common acute causes are hemoabdomen from a ruptured mass or trauma. Other causes are chronic in nature such as liver disease or right-sided heart failure resulting in ascites. Organomegaly, neoplasia, and even fat accumulation can have the same effect. Relieve pressure with an abdominocentesis to determine the inciting cause. Treatment involves ultrasound and medical or surgical intervention depending on the source.

### Look Alikes

These are animals that appear to be in respiratory distress, but do not have a primary respiratory problem. After ruling out a primary respiratory cause, look for other origins like behavioral as patients in

stress, with extreme fear or pain, may exhibit similar signs. Other causes such as metabolic acidosis can result in increased respirations in an effort to blow off CO<sub>2</sub>. Kussmaul respirations for instance are deep and slow breaths as a result of severe metabolic acidosis, seen in cases such as DKA or kidney failure. An anemic animal with hypoxia due to low Hgb may increase respirations in an attempt to improve oxygenation.

## Case Examples

References available upon request

Alveolar gas equation:

$$PAO_2 = F_{iO_2} \times (P_B - 47) - (0.8 \times P_{aCO_2})$$

F<sub>iO<sub>2</sub></sub> in room air is 0.21, under anesthesia (100%) O<sub>2</sub> is 1

P<sub>b</sub> is the barometric pressure (760 mmHg at sea level)

47 represents the water vapor pressure, in mmHg

P<sub>aCO<sub>2</sub></sub> is the alveolar carbon dioxide, assumed to be equal to arterial PCO<sub>2</sub>.

R is the respiratory quotient and is approximately 0.8 at steady state on standard diet.

# Coagulability. Believe The Hype

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April Bays CVT, VTS (ECC)

Coagulation is equally dangerous if your patient has too much, or too little of it. This lecture will cover hyper and hypocoagulable states along with the many related diseases and disorders. We'll discuss tools to recognize the risks and signs associated with derangements and their implications in your patients.

## Coagulation

In healthy animals, normal coagulation and fibrinolysis occur only as needed and follow a cause and effect model: clots are produced on demand and are broken down when no longer useful. Hemostasis is a tightly regulated process divided into primary and secondary phases. Primary hemostasis involves interactions between the vessel wall and platelets and results in the formation of a platelet plug. This platelet plug provides a physical barrier to inhibit loss of blood and provides membrane surfaces as binding sites for the formation of thrombin in secondary hemostasis. Secondary hemostasis involves the formation of fibrin in/around the primary plug, the stabilization of the platelet clot through the generation of thrombin, and the activation of the fibrinolytic system. This is known as the coagulation cascade.

- Primary hemostasis—Activated platelets form a platelet plug (minutes)
- Secondary hemostasis—Reinforcement of the frail platelet plug with fibrin strands (hours)
- Fibrinolysis—Clot dissolves after vascular wall repair (days)

The new cell based model to thrombosis incorporates the role of cells. This model suggests overlapping systems rather than the distinctly separate traditional coagulation cascade. It is comprised of 3 phases; initiation, amplification and propagation.

Initiation phase occurs on the surface of the perivascular tissue factor (TF) bearing cell. Importantly, factor VII is the only coagulation factor that circulates in its active form. Once factor VII comes in contact with exposed tissue factor, thrombin is generated which activates platelets and initiates coagulation. The complex formed between TF and active factor VII, further activates factors IX and X. Meanwhile, circulating antithrombin (AT) will inactivate clotting enzymes downstream from the site of injury, to ensure that coagulation does not occur elsewhere. It's the small quantity of activated thrombin, and

activated factor IXa that goes on to the amplification phase. The amplification phase occurs from the thrombin produced that is now free to activate platelets and results in the cleavage of vWF, further activating additional factors. The increase in platelet numbers signals to even more platelets and the amplification is under way, compared to the initiation phase which resulted in more of a priming quantity. The propagation phase occurs on the platelet surface resulting in spilling of the granules from within the platelet and resultant recruitment of additional platelets. Additionally, the propagation phase will allow the level of thrombin to be amplified and it's this thrombin which takes fibrinogen, converts it to fibrin to be combined with the platelet clump which in turn becomes the meshwork that creates a more stable clot.

To summarize, the four forces are: antithrombotics (prevent clots from forming), fibrinolysis (anticoagulating mechanism for removing existing clots), work together to keep vessels open and free of obstruction. Thrombotics (forms the clot) and antifibrinolytics (prevent premature clot removal) both promote coagulation.

The clinical signs of bleeding indicate both primary and secondary hemostatic defects. Clinical signs of primary hemostatic dysfunction include petechia or ecchymosis, signaling platelet dysfunction or loss. Signs of organ failure, such as oliguria or dyspnea, occur as a consequence of obstruction of the microcirculation by thrombi (e.g., PTE). Defects in clotting factors and secondary hemostasis results in deep bruising, delayed bleeding and hemorrhage.

### **Hypercoagulable states**

The formation of thrombi, and eventually emboli, occur because of a disruption in the balance described above. A patient that is said to be in a *hypercoagulable state* carries a higher risk of developing a clot. Traditional in-house coagulation tests are useful in identifying specific component alterations or activation of factors. But they cannot detect a hypercoagulable state. Understanding the physiologic process behind why these conditions predispose patients to hypercoagulability is crucial in preventing complications from thromboembolic disease.

### **Pathophysiology Of Thromboembolism.**

Virchow's Triad, also known as the triad of death, is comprised of three elements found to be factors that contribute to the formation of clots, or promote thromboembolism (TE) formation; alterations in blood flow, (blood stasis allows increased contact time with coagulation factors and platelets), and endothelial

abnormalities (endothelium maintains antithrombotic properties, damaged endothelium shifts to a prothrombotic state), and hypercoagulability.

Any condition inducing inflammation activates inflammatory mediators, all of which can lead to endothelial damage. Once the damage has occurred, activation of the coagulation cascade follows, to the potential development of thrombi. Inflammation causes an “upregulation” of procoagulant factors, resulting in increased thrombin generation, systemic fibrin formation, downregulation of natural anticoagulants, and delayed fibrin removal as a consequence of inadequate fibrinolysis. Through several mechanisms, inflammation triggers the coagulation cascade and is associated with increased risk for TE. Patients with immune-mediated hemolytic anemia (IMHA) suffer from hemolysis, the lysis of RBC’s, and the subsequent release of cytokines (inflammatory mediators) which contribute to the associated hypercoagulability.

Thyroid hormone plays many roles on the vasculature, cardiovascular health, and endothelial integrity. Hyperthyroid cats are at increased risk for TE. In dogs with hypothyroidism, atherosclerosis (arterial plaque deposits) is a rare complication of the hypercholesterolemia and lipid abnormalities that can lead to thrombosis and multiorgan dysfunction.

Immune-mediated thrombocytopenia (ITP). The immune system makes antibodies that destroy platelets, and patients may present with signs associated with platelet dysfunction; bruising and spontaneous bleeding from mucosal surfaces, epistaxis, hematuria, melena. They may also have no signs at all.

Cats and dogs with Cushing’s disease are known to be at increased risk for thromboembolic disease though the mechanism isn’t entirely understood, there are multiple factors at play. Decreased AT activity, increase in circulating coagulation factors, and an increased PCV, predispose these patients to develop blood stasis and risk for TE. Hypercoagulability also occurs in animals that have been exposed to high levels of exogenous steroids. Decreased AT activity can also be due to renal disease, resulting in poor production as well as loss through urine. Glomerular disease is also associated with increased platelet aggregability, a risk for TE.

In patients with diabetes melitus, oxidative stress contributes to increased platelet aggregation and a tendency for endothelial adhering. Diabetic patients are found to have a propensity toward increased blood viscosity, additionally putting them at risk. Hyperglycemia’s hypertonicity has an irritating effect on

endothelial tissues which will incite and promote coagulation. Endothelial damage can even be caused in a similar way by irritating solutions such as those containing dextrose, causing phlebitis.

Cancer cells have the ability to activate the coagulation system. Cancer patients have increased platelet activation, promoting the initiation phase, increased expression of TF, and decreased clearance of coagulation factors.

### **Conditions Associated With The Presence Of Thromboembolism**

The aortic trifurcation is the most common location for sudden TE seen in cats, this is known as Feline Aortic Thromboembolism (FATE). Most commonly found in older cats with concurrent cardiac disease, these cats present with the same signs associated with arterial TE of a limb: pain, pulselessness, pallor, paresis, and poikilothermy. Cats experiencing FATE may have some degree of difference between the glucose of the suspected affected limb and a normal limb. Without the use of angiography to confirm, TE can be diagnostically challenging, shifting the diagnosis to clinical signs, history and basic testing.

In canines, pulmonary and portal TE are more common. Risk factors will have a common theme of involvement in Virchow's triad. Acute pulmonary thromboembolism presentation is dramatic though underdiagnosed due to the lack of diagnostic availability in the majority of veterinary hospitals. Acute onset of respiratory distress, hypoxia, tachycardia and collapse are exhibited in these patients. Patients experiencing a TE may or may not have an associated thrombocytopenia.

### **Disseminated Intravascular Coagulation (DIC)**

DIC is a syndrome in which excessive intravascular coagulation leads to coagulopathy, multiple organ microthrombosis, and subsequent multiple organ failure. DIC causes uncontrollable bleeding due to the inactivation or inappropriate consumption of platelets and clotting factors secondary to enhanced fibrinolysis. In DIC, there is an imbalance between the prothrombotic and antithrombotic activities of coagulation. Excessive thrombin is generated, fibrin and plasmin activated systemically, and normal anticoagulation mechanisms suppressed. Fibrin removal is delayed as a consequence of impaired fibrinolysis. During this excessive intravascular coagulation phase, platelets and coagulation factors are consumed resulting in thrombocytopenia, impaired thrombocyte function, and depletion of coagulation factors.

The most common form of DIC is fulminant DIC found in dogs after heat stroke, trauma, acute pancreatitis, envenomation, or certain toxicities.

Treatment of the patient experiencing a coagulopathy is focused on minimizing activity and stress. Oxygen therapy, coupled with strict rest is aimed at decreasing the demand of the cardiopulmonary system. Fluid therapy prevents blood stasis and improves oxygen delivery but must be administered with extreme caution for the risk of fluid overload in patients with right heart dysfunction. Anticoagulant therapy is ineffective against existing clots but will aid in the prevention of developing clots. Thrombolytics would have to be used to dissolve formed clots through the use of fibrinolytic agents. These patients would then be at risk for reperfusion injury once perfused.

## **Hemostatic Disorders**

*Inherited coagulopathies*, while rare, do occur. Most commonly these conditions are realized after their first surgery or bleeding episodes in otherwise healthy animals. Von Willebrand disease is the most common and would predispose an animal to hemorrhage by disrupting the primary hemostasis phase.

Acquired bleeding disorders occur in far more cases with older animals. Probably the most common *acquired coagulopathy* is found with ingestion of a vitamin K antagonist, better known as rat bait. The enzyme that activates vitamin K is blocked, leading to the consumption of more and more coagulation factors as the active form of vitamin K becomes depleted. In simply existing in the world, the active coagulations factors will end up being utilized leading to a depletion. The vitamin K dependant factors are II, VII, IX and X (factors measured with PT). Treatment would involve supplemental vitamin K while the body reestablishes this pathway. The actively bleeding patient may be coughing up blood, in respiratory distress, or bleeding from any orifice.

The liver is responsible for producing proteins involved in coagulation, including procoagulants and anticoagulants. In turn, hepatopathy results in a reduction in the amount of proteins being synthesized, leaving the body at risk for bleeding. Therefore, liver function should be assessed in patients presenting with coagulopathies for this reason.

## **Dilutional Coagulopathies.**

Even in situations where a patient is receiving life saving care, there is a small risk of inducing a coagulopathy by providing medical treatment, referred to as a *dilutional coagulopathy*. In the event of trauma or acute injury resulting in hemorrhage, there will be some loss of procoagulants (hemostatic proteins, platelets) through blood loss. At the same time there is significant effort exerted at the site of injury which will consume resources as well. The administration of fluids will further decrease the amount of procoagulants available in the blood. If these patients end up being transfused, the transfusion would

contain citrate as an anticoagulant, contributing to these complications. In addition, if the transfusion product is hastily prepared and not appropriately warmed, it would expose the patient to hypothermia, which interferes with the activity of coagulation factors.

### **Trauma induced Coagulopathy**

Coagulopathy in the trauma patient is a unique and dynamic process that has historically been attributed to a combination of hemodilution, acidosis and hypothermia, known as the “lethal triad”. These syndromes are thought to be manifested by a combination of factors that develop in the post traumatic injury patient, resulting in coagulopathy, and a higher incidence of early death. Low volume, or permissive hypotensive resuscitation may be warranted in these patients in their initial stabilization efforts. Crystalloid fluid administration to a target systolic pressure of 80-90 mmHg is recommended. Aimed at preventing dislodgement of any hemorrhage controlling clots, and hemodilution, which is a factor leading to the lethal triad. In addition, attempts should be made to prevent hypoperfusion which contributes to lactic acid buildup and acidosis. A delicate balance must be maintained in efforts to treat without worsening symptoms in these fragile patients. Other considerations in polytrauma patients include maximizing oxygen delivery through oxygen therapy (prevention of shock, hypoventilation leading to increased CO<sub>2</sub> levels, contributing to acidosis), direct pressure and bandaging to control hemorrhage (further contributing to hypoperfusion), treatment with whole blood transfusion (including autotransfusion), and fresh frozen plasma may be warranted. Monitoring blood pressure, preventing hypothermia, and ensuring attention to the treatment of pain, are integral to nursing trauma patients. This attention will aid in the early detection of a coagulopathy and ameliorate risk of consequences.

### **Diagnostics**

Coagulation profiles commonly consist of a basic CBC with platelet count, and coagulation tests including both a prothrombin time (PT) and partial thromboplastin (PTT). PT is associated with factors V, VII, X, II, or I. These factors are associated with the extrinsic pathway of the coagulation cascade. The PTT test is associated with factors XII, XI, IX, VIII, X, V, II, or I, which are similarly associated with the intrinsic pathway.

Other simple in-house testing options available for detecting coagulopathies are activated clotting time (ACT) and buccal mucosal bleeding time (BMBT). ACT involves filling an incubated tube containing diatomaceous earth with blood, following the testing process, and measuring the time for clumping to begin. Normal time for clot activation for canines is <120 seconds, and <90 seconds for felines.

Confirmation would verify the activation of factor XII, and in turn, the function of intrinsic and common pathways. Therefore, this quick test can evaluate prolongation of factors similarly to the PTT. BMBT involves creating a small incision on the inner lip mucosa and recording the time for bleeding to stop. This test measures the endothelial and platelet function only, which is associated with the primary hemostatic plug. Prolongation with this test would indicate dysfunction of platelets, or a thrombocytopenia. Normal times are between one to three minutes for canines and felines.

### **Transfusion Therapy**

The transfusion therapy of choice is based on the need of the patient. *Fresh whole blood* will contain most of the components to replace whole blood loss (hemorrhage) including red blood cells, platelets, leukocytes, and plasma. It will also contribute to the colloid osmotic pressure, improving blood pressure. *Packed red blood cells* (pRBC) are produced from separating whole blood into RBCs from the other components. By replacing RBC's, we can increase the oxygen carrying capacity, when there is a life threatening anemia. Remember, RBCs carry a significant effect on volume, so the normovolemic anemic, or those with cardiovascular disease may become volume overloaded rapidly. *Plasma* comes in variety of forms, most commonly utilized is fresh frozen plasma (FFP) and frozen plasma (FP). FFP would remain "fresh" for one year after collection. After which, it is considered frozen plasma and still has a freezer shelf life of up to 4 more years. FFP contains all of the clotting factors and FP will contain all but reduced numbers of the labile factors (factors V and VIII). They are not always ideal for volume replacement or hypoalbuminemic patients as they do not have sufficient amounts of albumin to increase levels.

References available upon request



# Becoming A RAAS Badass

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April Bays CVT, VTS (ECC)

The function of this endocrine system is to maintain blood volume and pressure. One way this system is activated is by a reduction in renal perfusion and they respond by releasing renin. How do they do that and how does it affect blood pressure?

## **Baroreceptors, Chemoreceptors**

Every nephron contains the means for measuring and responding to alterations in hemodynamics. This physiologic process involves a baroreceptor mechanism - pressure sensing, and a chemoreceptor mechanism - sodium sensing.

Baroreceptors located in the carotid sinus via the sympathetic nervous mechanism extrinsically, and via the renal system intrinsically. Macula densa mechanism – chemoreceptors.

## **Renin**

You'll recall that each kidney contains millions of nephrons. Each nephron has an afferent arteriole, delivering blood, and an efferent arteriole, returning the filtered plasma to circulation. The arterioles have the ability to stretch and recoil, thereby protecting the kidney from hypertensive injury or by increasing blood pressure entering the nephron, improving filtration capacity.

If we follow blood into the kidney, it would enter the nephron via the afferent arteriole and enter the glomerulus for filtration. The filtration rate is dependant on the pressure of the blood entering it. The resultant filtrate empties into Bowman's capsule, enters the proximal convoluted tubule, into the loop of Henle and eventually, the distal convoluted tubule and collecting duct to produce urine.

The distal convoluted tubule is situated in close proximity to the afferent arteriole, and this placement is important. Located on the lining of the walls of the afferent arterioles are endothelial cells. They act as baroreceptors, detecting pressure directly from the blood volume entering each nephron. Just beyond the endothelial cells are smooth muscle cells and juxtaglomerular cells. It is from the juxtaglomerular cells

that the enzyme, renin, is released. Renin then enters and exits the glomerulus, bypassing filtration to be released via the efferent arterioles to enter into systemic circulation.

At the distal convoluted tubule, there is another set of specialized cells called the macula densa, and they, too, are constantly sampling the blood. They are responsible for the secondary renin trigger, and they do so by measuring the sodium content. As you can imagine, if the volume of blood entering the nephron is low, then the filtrate entering the tubules would contain less fluid, and flow at a slower rate.

The result is an extraordinary amount of sodium being extracted from the filtrate due to the extended time in the tubule. Therefore, low sodium detected by the macula densa indicates that the filtration pressure in the glomerulus (and therefore arterial blood) is low. This is the chemoreceptor mechanism. The macula densa respond by releasing prostaglandins. Due to the proximity to the juxtaglomerular cells, the message is received directly and renin is released.

A drop in blood volume or pressure is also detected by baroreceptors in the vagal system, which activates the sympathetic outflow, to send a signal to the juxtaglomerular system to release renin. These baroreceptor and chemoreceptor mechanisms will similarly signal the stop of renin release in response to appropriate or high blood volume and pressure.

### **Angiotensinogen, Angiotensin I, and Angiotensin II**

Meanwhile, liver cells are synthesizing most of the proteins for the body, including angiotensinogen. Angiotensinogen is always present in circulation in its inactive form. When renin comes in contact with angiotensinogen it is activated, converting angiotensinogen to angiotensin I. Circulating angiotensin I ultimately enters the lungs where it undergoes another change.

Present on the surface of endothelial cells in the pulmonary capillaries is an enzyme known as Angiotensin-converting Enzyme (ACE). The function of ACE is to convert the inactive precursor angiotensin I to the vasoactive peptide angiotensin II. Angiotensin II is a potent vasoconstrictor with receptors present on arterial and venous smooth muscle. ACE is also responsible for the breakdown and deactivation of bradykinin. Bradykinin acts as a vasodilator. Therefore, by blocking the action of bradykinin, it effectively inhibits vasodilation while promoting vasoconstriction.

Importantly, angiotensin II preferentially affects renal efferent arterioles, and to a lesser degree, the afferent arterioles. This preserves high pressure in the glomerulus. As one of the final products of this

pathway, angiotensin II has then increased renal perfusion. Angiotensin II can also stimulate sympathetic nerve activation through central mechanisms. This stimulates norepinephrine release. Norepinephrine contributes to additional vasoconstriction. Juxtaglomerular cells respond to sympathetic nerve stimulation by releasing renin.

### **Aldosterone**

In addition, angiotensin II stimulates the release of aldosterone. The zona glomerulosa cells of the adrenal cortex detects angiotensin II, which triggers the release of the steroid, aldosterone. Aldosterone enters circulation and binds with principal cells. Principal cells, or p-cells, are a type of epithelial cell present on the luminal membrane of the distal convoluted tubules and collecting tubules. The opposing wall of the p-cells is a basolateral membrane against which capillaries run. When aldosterone enters p-cells, the aldosterone receptor complex binds, producing a few different proteins with various actions. Na-channels and K-channels are planted on the luminal membrane the p-cells. With the addition of Na-channels on this surface, Na that would normally be excreted in the urine instead passes through the channel to enter the p-cell. In exchange, K moves out of the cell, into the lumen (urine).

In addition, planted on the basolateral membrane of the p-cells (between the p-cells and capillaries) are Na-K-ATPase pumps. They facilitate the active exchange of Na and K across the cell's membrane. Thus, under the influence of aldosterone, K leaks out into the lumen to be excreted and Na is reabsorbed into the capillaries. With Na goes water, effectively increasing the circulating blood volume.

### **Antidiuretic Hormone**

In response to angiotensin II, the hypothalamus stimulates production of antidiuretic hormone (ADH), also known as vasopressin, from the pituitary gland. This system also stimulates receptors in the thirst system, increasing water intake. ADH's role is to trigger the cells of the last part of the nephron to become more permeable to water. The effect is to retain water in the medullary interstitium of the kidney, which is hyperosmolar.

Because this area is hyperosmolar, and solutes cannot cross the nephron's luminal wall, water will instead be pulled from the lumen (an area that is hypo-osmolar), to an area that is hyperosmolar. Water will rush through the cells to the interstitium so that it is not lost in the urine. ADH thereby inhibits water diuresis by causing active water resorption in the renal collecting tubules. This water will contribute to blood volume and pressure.

These processes serve to preserve vascular volume. As systemic pressure increases, so does the venous return to the heart. Cardiac filling is then increased and the more filling occurs, the more contraction the heart is capable of, leading to increased stroke volume. In the end, this means increased cardiac output, with an end result of increased systolic pressure.

## **Dysfunction**

Conditions resulting in poor cardiac output such as heart failure, results in chronic sympathetic activation of the renin-angiotensin-aldosterone system (RAAS). In the early stages of congestive heart failure (CHF), these systems service to restore cardiac output and maintain circulatory volume and perfusion. This is of vital importance as systolic blood pressure depends mainly on cardiac output.

However, as the disease advances, vasoconstriction causes an increased afterload (resistance for the heart to contract against) and increased volume causes an increase in preload (filling pressure during diastole). These mechanisms can overwhelm a failing heart, leading the congestion known in CHF. Pulmonary edema and pulmonary arterial hypertension may result in cases with chronic left-sided heart failure, and pleural effusion and ascites often accompany right-sided heart failure.

## **Therapeutic Options With RAAS Affecting Agents**

If angiotensin II and aldosterone are chronically elevated, they also affect the myocardial cells directly by stimulating growth factor that lead to morphological changes, like pathological hypertrophy. These alterations cause cardiac remodelling, which results in progressive cardiac failure. Therefore treatment for heart failure aims to correct these maladaptive activations and their effect on the body.

ACE inhibitors are used to negate the effects of angiotensin II by preventing the conversion of angiotensin I to angiotensin II. ACE inhibitors currently used in veterinary medicine include benazapril, enalapril, imidapril and ramipril. The efficacy in dogs with CHF, especially those with chronic valvular disease has been convincingly demonstrated with improved hemodynamics, clinical signs, and survival time. In cats with cardiovascular disease, little information is available except for reports of some benefit in cats with hypertrophic cardiomyopathy.

ACE inhibitors have also been used for the medical management of chronic kidney disease. Via their mild to moderate hypotension effect, they decrease the glomerular capillary pressure, have antiproteinuric

effects, and tend to delay the progression of chronic renal failure. ACE inhibitors also have vasodilator effects.

Diuretics aim to decrease the circulating volume, preload to the heart, and thus cardiac load. There are different types of diuretics based on their area of action, including loop diuretics such as furosemide, thiazide diuretics such as hydrochlorothiazide, and potassium-sparing diuretics such as spironolactone.

Positive inotropes are agents that increase the force in the cardiac contraction. These include digoxin, milrinone, and dobutamine. Pimobendin, an inodilator (has both inotropic and vasodilator effects), promotes positive inotropy and reduces afterload.

Angiotensin II receptor antagonists are used as an alternative to ACE inhibitors in humans that don't tolerate ACE inhibitors well. However since dogs do typically tolerate ACE inhibitors, and reportedly do not produce the active metabolite, they are rarely used in veterinary patients.

The multiple therapeutic options for treatment of patients with common heart disorders require careful consideration for the particular disorder and progression of the disease. Many veterinarians will also recommend exercise restriction, moderation of dietary sodium intake and close attention to caloric intake to combat the weight loss that can attend advanced heart disease.

References available upon request



# Crisis Management. Let's Get Salty, K?

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Canine hypoadrenocorticism, also known as Addison's disease results from inadequate production of adrenal hormones. An Addisonian crisis is a life threatening complication requiring aggressive treatment. Termed "the great pretender," due to the way it mimics symptoms commonly seen with other conditions, it has been known to be underdiagnosed. The better technicians can familiarize themselves with the disease processes and symptoms, the better they can anticipate the needs of these critical patients. Hypoadrenocorticism is uncommon in dogs but rare in cats. For the purpose of this lecture, I will focus on dogs primarily.

## Adrenals

The adrenal glands sit atop each kidney. Within the capsule of each adrenal gland lies the cortex (the outermost portion) and medulla (innermost portion). The cortex consists of three functionally distinct zones that release hormones, most importantly for this lecture mineralocorticoids and glucocorticoids. The medulla is primarily responsible for catecholamines such as norepinephrine and epinephrine. Adrenal medullary function is usually remained in hypoadrenocorticism dogs. (Adrenal medullary function is usually maintained in hypoadrenocorticism dogs)

The first zone of the cortex is the zona glomerulosa, which is primarily responsible for the synthesis and secretion of mineralocorticoids. The most significant of which is aldosterone, which regulates electrolyte balance. Next, the zona fasciculata synthesizes and secretes glucocorticoids, which regulate glucose, fat, and protein metabolism. The most important glucocorticoid, cortisol, plays a major role in physiologic homeostasis. The innermost zone of the cortex is known as the zona reticularis, which primarily secretes androgens, the sex hormones.

## Causes

Immune-mediated destruction of the adrenal cortex remains the most common cause of *primary hypoadrenocorticism* in dogs. A small number of dogs suffer from iatrogenic primary Addison's disease. *Secondary hypoadrenocorticism* occurs when the pituitary fails to secrete ACTH, so these patients are deficient only in cortisol. Iatrogenic secondary hypoadrenocorticism can result from administration of exogenous glucocorticoids. ACTH secretion from the pituitary is suppressed, leading to atrophy of the

zona fasciculata and the zona reticularis. Glucocorticoids should be tapered and never withdrawn abruptly for this reason.

Hypoadrenocorticism tends to occur in young to middle-aged dogs. Studies have shown an overrepresentation of females in some breeds. Breeds with higher incidents include great danes, poodles (all types), west highland terriers, Portuguese water dogs, bearded collies, rottweilers, soft-coated wheaten terrier, springer spaniel, basset hound, and St Bernards.

### **Mineralocorticoids**

As the name suggests, the mineralocorticoids regulate the mineral balance, or in other words, electrolyte regulation. Specifically, aldosterone is integral in the handling of sodium, chloride and water resorption, as well as potassium excretion. Clinical signs of mineralocorticoid deficiency consequently are polyuria/polydipsia, hypovolemia, cardiac arrhythmia, bradycardia, dehydration, mental depression, nausea, hypotension, and weakness.

### **Glucocorticoids**

Glucocorticoids are essential for times of stress and survival in a starvation situation. Specifically, cortisol promotes gluconeogenesis and glycogenolysis, and antagonizes insulin's glucose lowering effect (increases insulin resistance). Clinical signs of glucocorticoid deficiency include weight loss, lethargy, vomiting, diarrhea, impaired mentation, anorexia, weakness, and trembling.

### **Understanding The Consequences Of Deficiency**

Aldosterone is synthesized from cholesterol in the blood, triggered by the presence of angiotensin II or hyperkalemia. When a body is deficient in aldosterone, it leads to a diminished ability to retain sodium, and therefore water, resulting in massive total body water loss and an accumulation of dangerous levels of potassium.

Aldosterone works in three ways in the principal cells (P cell) of the last part of the nephron. In the distal convoluted tubule and in the collecting duct, the P cell sits between the luminal membrane and the basolateral membrane adjacent to the capillaries. Aldosterone stimulates the addition of Na channels on the luminal membrane, which ultimately removes Na from the filtrate, and moves it into the p cell. Furthermore, it enhances the action of Na<sup>+</sup>/K<sup>+</sup>ATPase pumps on the basolateral membrane (between the

P cell and capillaries), the net effect resulting in resorption of Na and water. Additionally, aldosterone increases the number of open K channels.

The lack of aldosterone will interfere with this system and total body stores of Na become depleted quickly, resulting in medullary washout. Serum chloride levels often parallel sodium loss and hypochloremia is commonly found as well. Further compromised by gastrointestinal losses, this leads to a total body loss in circulating volume and pressure. The loss of extracellular fluid compromises cardiac output leading to decreased renal perfusion. Prerenal azotemia ensues with inappropriately dilute urine.

Aldosterone also enhances the renal excretion of hydrogen ions ( $H^+$ ). A buildup of hydrogen will lower the blood pH, leading to a metabolic acidosis. Aggravated by the progression of hypoperfusion, metabolic acidosis can further exacerbate hyperkalemia because  $H^+$  will move intracellularly in exchange for K in an attempt to lower the concentration of  $H^+$  in the plasma. Hypoperfusion will contribute to a build-up of lactic acid as lactate is a product of anaerobic metabolism, further contributing to the acidosis. Severe metabolic acidosis can have deleterious effects on cardiovascular system, hepatic and renal functioning, making it a major threat to survival. The loss of bicarbonate ( $HCO_3^-$ ) through small bowel diarrhea can also contribute to the development of metabolic acidosis.

Cortisol is also synthesized from cholesterol. The pituitary secretes corticotropin-releasing hormone (CRH) which stimulates the release of ACTH. In response, the adrenals release cortisol. Cortisol is one of the most important substances, aside from ATP and oxygen, that maintain physiologic homeostasis. Cortisol has multiple effects on vascular integrity and also stimulates erythropoiesis. This manifests in some Addisonian patients as "lack of a stress leukogram", with eosinophilia, anemia (attributed to lack of erythropoiesis and sometimes GI losses), hypocholesterolemia (due to lack of production), and hypoglycemia. Cortisol also promotes normal gastrointestinal function.

## **Presentations**

The dog with chronic Addison's will commonly have waxing and waning gastrointestinal symptoms with a history of anorexia and periodic lethargy. These patients may present with any number of physical signs from bright, otherwise healthful looking, to an obtunded, flat, shocky dog. The majority of Addisonian patients won't have all of the classic signs, but rather variable nonspecific symptoms, hence the importance of understanding the disease.

In contrast, dogs presenting in an acute Addisonian crisis paint a different picture, and are far more evidently ill. Presentations are associated with hypovolemic shock including severe dehydration, weak

pulses, prolonged capillary refill time, pale mucous membranes, and with severe hyperkalemia, bradycardia.

Addisonian patients generally will have a deficiency of both mineralocorticoids and glucocorticoids. But just like so many variabilities with this disease, there's also a subset of dogs that are considered "atypical" and they exhibit only signs associated with glucocorticoid deficiency. These patients are even more difficult to detect as they are able to maintain normal electrolyte balance. These patients will typically present with signs associated with the chronic form.

### **Diagnosis**

A low sodium:potassium ratio of less than 27:1 is the hallmark of this disease, however there is only one definitive diagnostic test for Addison's disease. Testing involves measuring the serum cortisol level before, and one hour after a synthetic adrenocorticotrophic hormone (ACTH) (cosyntropin, cortrosyn) injection. The introduction of ACTH should stimulate a cortisol release in a dog with functioning adrenal glands. An inadequate adrenal response will result in a post injection cortisol level that has not deviated far from the baseline level.

Exogenous glucocorticoids will mimic the physiologic response and are contraindicated prior to testing with the exception of dexamethasone, which will not cross-react with the test. If your clinic is unable to perform an ACTH stimulation test and your patient would benefit from the use of steroids, dexamethasone would be ideal for this reason as it would not delay treatment.

### **Treating The Addisonian Crisis**

Survival of a patient in crisis depends on a quick diagnosis, expedient treatment, and the ability to respond to the effects of electrolyte derangements. Aimed at the careful correction of the hypovolemia and hypotension, emergency stabilization involves restoring adequate circulating volume and tissue perfusion.

Placement of an IV catheter, from which the blood should be drawn. Utilizing this opportunity will be much easier than a subsequent blood draw due to the severity of the compromised vascular volume. Correction of the metabolic acidosis will begin almost immediately as fluid therapy is initiated through

dilution and diuresis. Blood pressure should be evaluated every 2-6 hours, throughout treatment. Watch for weakness, signs of hypotension, pale mucous membrane, delayed CRT, weak pulses, bradycardia, collapse, shaking and seizure, as these are signs of hypovolemic shock.

Hyponatremia results in hypo-osmolality of plasma which will induce a shift of water from the extracellular space, to the intracellular space. In other words, because there is an imbalance of sodium between the plasma and inside the cells, and solutes are unable to freely move in and out of cells, the easiest way to balance the sodium concentration is through the movement of water. Therefore, water will shift intracellularly in an attempt to maintain balance. In severe cases, this can lead to cell swelling and lysis. In the central nervous system (CNS) this can mean cerebral edema.

The severity of signs, as well as the rate of correction is determined by the onset of the derangement. Patients with acute development of hyponatremia should be treated with intravenous administration of balanced electrolyte solution, such as normal saline. It contains adequate sodium, but a lower amount of K than other solutions. Profound or chronic hyponatremia has the potential to be markedly more detrimental, though these patients may have no signs because the CNS has been compensating as the brain's tonicity slowly dropped. Too rapid elevation of sodium can result in cerebral myelinosis, a condition that causes damage to areas of the brain due to the way the cells adapt to prevent brain swelling. Therefore increases should be no faster than 0.5mEq/L/hr. This may be achieved through the use of a 0.45% saline solution. Failure to allow sufficient time for the brain to respond can put the brain at risk for osmotic demyelination, a syndrome characterized by sudden deterioration, days after therapy.

Hyperkalemia warrants immediate electrocardiogram evaluation. The most deleterious effect of hyperkalemia is observed on the myocyte. Increased plasma K concentrations will affect myocardial excitability by way of shifting the *resting membrane potential* closer to the *threshold potential*. Normally, the myocyte's membrane potential (measured as voltage) rests at -90mV. A rapid influx of cations causes a positive change in voltage and once the threshold potential is reached at -75mV, depolarization is achieved. Repolarization occurs when the membrane returns to a negative voltage again. However, when hyperkalemia shifts the resting membrane potential to a *less* negative value, the degree of concentration gradient diminishes. Additionally, reduced time between depolarizations produces a decrease in strength of the contraction. With severe hyperkalemia, the resting potential may become so much less negatively charged that it is unable to depolarize and bradycardia develops. This will ultimately slow conduction and result in fatal cardiac arrhythmias. Mild hyperkalemia causes increased positive or negatively deflected T wave amplitude (peaked T waves) while moderate hyperkalemia causes bradycardia and a flattening of the P wave, also known as atrial standstill. Severe hyperkalemia is associated with prolongation of the PR interval and bradycardia.

Remember, fluid therapy will initiate the resolution of the elevated K levels. When this is not enough to prevent arrhythmias, treatment with calcium gluconate will help protect the heart. Calcium gluconate has no effect on the K level directly, instead it increases the threshold potential, thereby creating an appropriate gradient. Correction of hyperkalemia can also be achieved through the administration of insulin. Insulin drives K intracellularly through its effect on the Na<sup>+</sup>/K<sup>+</sup>ATPase pump. A dextrose CRI may be warranted until they are able to regulate their glucose appropriately. It is extremely important to assess the patient for hypoglycemia prior to administering insulin. For this reason, dextrose administration may also be a chosen therapy, utilized to trigger the secretion of insulin, leading to the movement of K back to the intracellular space.

Hyperphosphatemia is common in these patients as phosphate ions shift similarly to potassium. Serum phosphorus levels can rise due to redistribution from intracellular to extracellular sites. Due to decreased renal excretion secondary to hypovolemia and renal hypoperfusion, phosphorus levels should be monitored closely. As with most electrolyte derangements, resolution is generally achieved through correction of the underlying cause, in this case, hypoperfusion, decreased glomerular filtration rate and metabolic acidosis.

Fluid resuscitation will generally resolve acid/base derangements on its own. Sodium bicarbonate administration for the treatment of acidosis is controversial and generally trending out of popularity in favor of judicial resolution through other means. Therefore bicarbonate therapy is usually reserved for those cases in which the correction may take time and the acidemia may be affecting cardiac function (pH < 7.2). It should never be administered if the patient cannot adequately ventilate on its own, as CO<sub>2</sub> is eliminated through the lungs. The use of a buffered solution such as Normosol-R or LRS may be considered to aid in resolution of acidosis if it is safe to do so.

Nutrition can be overlooked in the ER setting but this is another way that technicians shine. Antiemetics would be of benefit to prevent vomiting and nausea to get these dogs eating again.

With the help of glucocorticoid replacement and mineralocorticoid supplementation for the duration of their lives, they can return to health and lead a full life.

References available upon request