Tech Tip

**Double Gloving: Sx Sepsis**

Double Gloving" for orthopedic surgery is something we have adhered to for over 30 years. Wearing a size larger than your regular glove initially seemed to be a reasonable choice, however we quickly learned they restricted blood flow to our fingers. We then went to gas sterilized exam room gloves and found them to be much more comfortable. In the last 10 years the commercial surgical gloves are of better quality, a little thicker, thus providing more protection. Now I double glove when handling K-wires, cerclage wire, and when doing open fracture reduction.

This Abstract was presented at a recent ACVS meeting. It is objective data that fortifies what we subjectively assumed for many years.

**INVESTIGATION OF INCIDENCE AND RISK FACTORS FOR SURGICAL GLOVE PERFORATION IN SMALL ANIMAL VETERINARY SURGERY**

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**Introduction:** To identify incidence and risk factors for surgical glove perforation in small animal surgery.

**Materials and Methods:** Observational cohort study conducted at a veterinary teaching hospital. 2132 surgical gloves worn over 363 surgical procedures. All gloves worn by operative team members were assessed for perforation at end-procedure using the water leak test. Information was recorded on putative risk factors by a surgical team member. Associations between risk factors and perforation were assessed using multivariable multi-level random-effects logistic regression models to control for hierarchical data structure.

**Results:** At least one glove perforation occurred in 26.2% of surgical procedures. Identified risk factors for glove perforation included increased surgical duration (surgery>1 hour OR=1.79, 95%CI= 1.12-2.86), performing orthopedic procedures (OR=1.88; 95% CI=1.23-2.88), any procedure using powered instruments (OR=1.93; 95%CI=1.21-3.09) or surgical wire (OR=3.02; 95%CI=1.50-6.05), use of polyisoprene as a glove material (OR=1.59, 95%CI=1.05-2.39), and operative role as primary surgeon (OR=2.01; 95%CI=1.35-2.98). The ability of the wearer to detect perforations intra-operatively was poor, with a sensitivity of 30.8%.

**Discussion/Conclusion:** There is a high incidence of unrecognized glove perforations in small animal surgery. Double gloving should be considered when performing invasive procedures on small animals. Double gloving may be indicated for all procedures, particularly when surgical duration is over one hour in length, when orthopedic procedures are performed, or when powered instruments or surgical wire are used. Acknowledgments: Pet Trust Fund, Ontario Veterinary College, University of Guelph
The first consideration of fluid therapy is based on patient status as each patient is an individual with specific needs. What is the patient's current physical condition based on physical exam and evaluation of lab work? What is the scheduled procedure? What is the speed of your surgeon?

The goal of fluid administration should be the support of oxygen delivery, systemic blood pressure whether due to hypotension or hypovolemia, prevention of, or correction of electrolyte imbalances, metabolic or acid-base disorders.

As we all know, total body fluid composition is divided into extracellular fluid and intracellular fluid. Approximately 1/3 of the body's fluid is distributed into the extracellular space and the remaining 2/3 considered to be intracellular fluid. Of the extracellular fluid this is further divided between the interstitial fluid which contains 3/4 of the extracellular fluid and plasma containing the remaining 1/4 of the extracellular fluid. To put this in a different perspective, approximately 605 of the patient's body weight consists of fluid with 20% of the body weight being extracellular fluid and the remaining 40% of the body weight being intracellular fluid.

Going back to our patient status, evaluate hydration, electrolyte balance, renal and hepatic function. What are we working with and what do we have in our armory to effect correction? Fluid therapy is critically important during the perioperative period. The most important goal is to maintain hemodynamic stability and protect vital organs from hypoperfusion (heart, liver, brain, kidneys). All sources of fluid losses must be accounted for. Good fluid management goes a long way toward preventing problems.

- Conventional Crystalloids
- Colloids
- Hypertonic Solutions
- Blood/blood products and blood substitutes

Conventional crystalloid is combinations of water and electrolytes. Combination of water and electrolytes. These are balanced salt solution with electrolyte composition and osmolality similar to plasma. The most commonly used crystalloids are lactated Ringers, Plasmalyte, and Normosol. They have a short intravascular retention as the fluids equilibrate with intracellular and interstitial compartments. They contain a base source (Na\(^{+}\)CO\(_3\)^-) : lactate: liver metabolism acetate: muscle metabolism and gluconate: metabolism in most body tissue. Crystalloids are comprised of small molecules. These fluids are good for volume expansion. However, both water and electrolytes will cross a semi-permeable membrane into the interstitial space and achieve equilibrium in 2-3 hours. It is important to remember: 3mL of isotonic crystalloid solution are needed to replace 1mL of patient blood. This is because approximately 2/3rds of the solution will leave the vascular space in approximately 1 hour or less. A major disadvantage is that it takes approximately 2-3 times the volume of a crystalloid to cause the same intravascular expansion as a single volume of colloid. Commonly calculated crystalloid rate of administration for surgical patients are 5 ml/kg for the first hour for anticipated procedures without significant blood loss and decreasing by ½ for each subsequent hour. If significant blood loss or extension surgical time is anticipated, this may be raised to 10/kg for the first hour and decreasing to ½ after the first hour.

Colloids are large molecular weight solutions (nominally MW > 30,000 Daltons) these solutes are macromolecular substances made of gelatinous solutions which have particles suspended in solution and do NOT readily cross semi-permeable membranes or form sediments. Because of their high osmolarities, these are important in capillary fluid dynamics because they are the only constituents which are effective at exerting an osmotic force.
across the wall of the capillaries. These work well in reducing edema because they draw fluid from the interstitial and intracellular compartments into the vascular compartments. Initially these fluids stay almost entirely in the intravascular space for a prolonged period of time compared to crystalloids. These will leak out of the intravascular space when the capillary permeability is deranged or leaky. Albumin solutions are available for use as colloids for volume expansion in the setting of CHF however albumin is in short supply right now. There are other solutions containing artificial colloids available. The general problems with colloid solutions are:

- Much higher cost than crystalloid solutions
- Small but significant incidence of adverse reactions
- Because of gelatinous properties, these can cause platelet dysfunction and interfere with fibrinolysis and coagulation factors thus possibly causing coagulopathies in large volumes.
- These fluids can cause dramatic fluid shifts which can be dangerous if they are not administered in a controlled setting.

Common rates of administration for the canine patient are 3-5 ml/kg/hr with a daily total volume to remain within a 20-30ml/kg range. The feline rates are lower and may be calculated at 1-3 ml/kg/hr with a daily total volume of 20 ml/day. Should colloids be used in conjunction with IV crystalloid therapy, the rate of administration of the crystalloid may be reduced by up to 50%.

Hypertonic solutions are those containing sodium concentrations greater than normal saline. They are available in 1.8%, 3%, 5%, 7.5%, 10% solutions. Hyperosmolarity creates a gradient that draws water out of cells; therefore, cellular dehydration is a potential problem. These solutions are often used in veterinary medicine as a quick "band aid" for refractory hypotension until other interventions are made available. The most common calculated dose is 3-7 ml/kg IV bolus given over time up to 15 minutes. With the elevated sodium content, the patient must first be euolemic prior to administration. It is recommended that only a single dose of hypertonic saline be administered due to the potential for cellular dehydration.

The decision to administer blood products preoperatively is often based on the packed cell volume and hemoglobin concentration. In veterinary medicine a packed cell volume of 20% is often considered the transfusion trigger. Whole blood may need to be administered in volumes of 10 to 30 ml/kg, depending on the magnitude of anemia and hypovolemia (cats: 5 to 15 ml/kg). These volumes should be halved if packed red blood cell products are used. The rate of administration depends upon the magnitude of the hypovolemia. The amount of blood to administer can also be calculated: (desired PCV - current PCV) x body weight (kg) x 2 ml whole blood (assumes a PCV of about 40%) (Or 1 ml packed red blood cells [assumes a PCV of about 80%]). Intraoperatively, the decision is based on the amount of acute blood loss with the initial packed cell volume being taken into consideration. Typically at TAMU, if there is significant observed blood loss and the packed cell volume has decreased by at least 25%, blood products are prepared for delivery. Bearing in mind that the entire patient status should be considered regarding the ability to deliver oxygen to the cells. Having said that, what is the blood pressure, what is the heart rate, has it raised as a compensatory response to a change in volume status, and has the end tidal CO2 decreased as a result of volume loss? Remember that the oxygen saturation estimated by the pulse Oximeter only tells you the percent of hemoglobin saturation which is not helpful in blood loss situation. Bottom line - if the hemoglobin has dropped to an unreadable level due to blood loss, the pulse Oximeter can still give you excellent % saturation readings. Look globally at the patient!
Tech Tip

Easier to breathe on my back!

BRACHYCEPHALIC AIRWAY SYNDROME (BAS)

Brachycephalic breeds have become more popular in recent years. We now recognize early intervention surgically, at a young age, will avoid the more difficult and expensive surgical procedures when these dogs are older. The initiating abnormality is stenotic nares. This is followed by elongation of the soft palate and everted laryngeal sacculles. We now do much more upper airway surgery than we did in the past. Early in the disease surgically removing the tissue narrowing the nasal openings and later in the disease when the classic triad is seen: Stenotic nares, Elongated soft palate, and Everted laryngeal sacculles.

Technicians play a key role in facilitating effective treatments to patients suffering from Brachycephalic airway syndrome (BAS). As exam room technicians you will begin to recognize the very narrowed nasal openings in young puppies when receiving their early vaccinations. As surgical technicians you are a vital part of the anesthetic and surgical management of the disease. BAS is a condition affecting short-headed dogs and cats. These patients may suffer from stenotic nares (narrowed nostrils), elongated soft palate, everted laryngeal sacculles, and hereditary hypoplastic tracheas. Pug, Pekinese, Maltese, Boston Terriers, Shih Tzu, French Bulldogs, and English Bulldogs are common canine breeds affected, and the Persian and Himalyan are among the cats. The symptoms are classic of many upper respiratory conditions, including inspiratory stridor and stertorous breathing, cyanosis, hyperthermia, exercise intolerance, excitability, leading to collapse in severely affected patients. Owner’s may also report coughing, gagging, and vomiting.

Sedatives, such as acepromazine are often recommended to help relieve anxiety and excitement, as well as reduce the incidence of regurgitation. A complete physical exam, including auscultation of the chest and tracheal sounds, along with tracheal palpation for abnormalities is done on all patients. Right and left lateral, and ventrodorsal chest radiographs are taken to check for evidence of aspiration pneumonia or heart disease. Lateral cervical radiographs should be taken to determine tracheal diameter there as well, as they can have both cervical and thoracic hypoplastic tracheas. Radiographs can be taken with
the patient under light sedation, such as butorphanol (0.2-0.4 mg/kg) and acepromazine (0.01-0.03 mg/kg) given IM or IV and flow by oxygen delivered via face mask. Because there is a risk of vagal stimulation with many of these patients, an anticholinergic, such as atropine or glycopyrrolate (0.1 mg/kg IM), is given intramuscularly (IM) as a premedication to prevent bradycardia. Metoclopramide (Reglan) can be used to help reduce the incidence of regurgitation.

Other considerations for technicians is to always use a laryngoscope during oral exams and intubations. Just because you are capable of intubating without the aid of a laryngoscope, it does not mean you should. Light is necessary in recognizing potential irregularity or irritations of the oral cavity, that may be missed in the dark. Always have oxygen and a variety of endotracheal tubes (ETT) available when administering sedation to patients affected by airway disease, often the ETT size is over estimated for patients with hypoplastic tracheas. It is helpful to have a rigid stylet, such as a polypropylene urinary catheter, to aid in the intubations of cats or small dogs. Many of these patients are administered steroids, so nonsteroidal anti-inflammatory drugs, NSAIDS, should be avoided due to the risk of GI ulceration that can lead to GI perforation.

When the airway is obstructed by stenotic nares and the amount of air required by the lung is not achieved, the pressure on the area is increased. The increase in pressure acts like a vacuum and pulls on the soft palate and surrounding tissues. Stenotic nares greatly reduces the amount of air the patient can breathe. Surgical treatment is required to resolve the clinical signs. The surgery option available for stenotic nares varies but the ultimate result is the same, a larger nasal passage. Surgical repair is recommended at 3-4 months of age, but can be done as early as 9 weeks in clinically affected patients. The sooner stenotic nares are fixed, the less likely the patient will have to be treated for elongated soft palate and everted laryngeal saccules. An alar fold (obstructive nasal folds) resection can be performed on very young dogs. Because the alar folds are too small to allow primary wedge removal and closure with sutures we no longer suture the tissue. Following removal of the nasal folds, at any age, they heal well without suturing. Laser can be used, however the owner should be warned the nares will be white afterwards but will turn back to the original color (usually black) within 2-4 months.

Dogs with elongated soft palates will suck the soft palate back during inspiration, covering the larynx. A computed topography (CT) evaluation of the soft palates of brachycephalic breeds were shown to be thicker than non-brachycephalic breeds. The soft palate is considered too long if it hangs down 1-3mm below the level of the epiglottis. During a soft palate resection surgery the patient is intubated, positioned in sternal recumbancy and the head is elevated so the mandible can hang open. Another method to keep the mouth open during pharyngeal/laryngeal surgery is to place two equal size mouth gags on the canine teeth to hold the mouth open. These can be held by the surgical tech to position the head so the surgeon can see the pharynx and larynx well during surgery. A bright, narrow focus light source is necessary for good visualization by the surgeon. The redundant soft palate tissue is excised, traditionally, by a cut and sutures technique, and a 3-0 or 4-0 monofilament absorbable suture (PDS) is placed to approximate the wound and control hemorrhage. Laser and radiofrequency cautery are both acceptable alternatives, often much faster than the traditional method and have similar clinical outcomes.

Laryngeal saccules are located behind the arytenoid cartilages in the larynx and when everted they block the opening of the larynx. They are lateral to each vocal cord and "bulge" or "balloon" out obstructing the larynx. The surgeon may elect to remove the saccules if they are significantly blocking the airway. Often the patient needs to be extubated for this procedure, so IV anesthetics (i.e. Propofol) should be
available during this procedure. The surgeon can simply remove the saccules with long scissors or cup-forceps. There is no surgical treatment for hypoplastic trachea.

BAS patients are at risk for aspiration pneumonia when heavily sedated. In dogs the aspiration can be silent, so a rapid recovery and late ETT extubation is recommended. If there are any concerns, the patient's neck is shaved and prepped in case an emergency tracheostomy is needed. If the soft palate has been shortened a soft food diet is recommended for 10-14 days post-op. Steroids; prednisone (0.5-1.0 mg/kg PO) or dexamethesone (1 mg/kg IV) is given to decrease edema and inflammation after surgery. Antibiotics are recommended prophylactically for an appropriate period of time. The outcome is favorable in young dogs when treated early for stenotic nares. If nares surgery is not done at a young age then it is often necessary to correct the nares, soft palate and laryngeal saccule protrusion in adulthood. At either age the results are often dramatic. They snore much less, can exercise more easily, become more tolerant of warm summer days, thus enjoying life more.
Tech Tip

Breathe Safe Respiratory Apnea Monitor

This respiratory monitor is a new version of old methods. It is an additional monitoring device that our anesthetic/surgical techs have tested and find very helpful. They like the apnea alert even though they are monitoring HR, BP, Temp., PO2, PCO2, EKG and RR by visually monitor the anesthetic/O2 bag the 45 sec apnea alert always gets their attention and the surgeons.

![Breathe Safe Respiration Monitor](image)

**Breathe Safe Respiration Monitor**
Item#: JOR166RM
Vendor: Jorgensen Laboratories
Price:$205.00

This tiny-smaller than a cigarette pack-monitor makes a great apnea alert monitor. The small microprocessor beeps with every breath with a very high sensitivity.

- Easy to use: just connect between endotracheal tube and anesthesia circuit
- No adjustments: a sophisticated algorithm recognizes
- and indicates every breath with a beep, working
- from a 1 lb. kitten to a 200 lb. St. Bernard.
- Apnea alert; a distinctive alert sounds if patient fails to
- breathe for 45 seconds
- Auto shut-off
- A high density long-life lithium battery provides years of service
- One-year warranty
Tech Tip

This abstract of a paper presented at the ACVS Symposium Technician Program - Small Animal in 2011 is very helpful in understanding the cardiac effects of the commonly used anesthetic agents we use today.

CARDIAC EFFECTS OF ANESTHETIC AGENTS
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Autonomic nervous system controls involuntary functions i.e. regulation of the heart, blood vessels, smooth muscles and many glands. It is composed of two parts - sympathetic and parasympathetic.

Sympathetic activity is controlled by mediation or release and uptake of the hormones epinephrine, norepinephrine and related adrenergic hormones. (Fight or flight) The sympathetic nervous system often called the adrenergic nervous system. Direct-acting adrenergic receptor agonists (sympathomimetic agents) interact with and activate adrenergic receptors:
Alpha adrenergic receptors- α1 stimulation causes the blood vessels to constrict
Beta (β) adrenergic receptors- β2 stimulation causes the blood vessels to dilate
Dopamine receptors

Anticholinergic agents - Atropine/glycopyrrolate: will cause an increase in heart rate, contractility, cardiac output and myocardial oxygen consumption. Often there will be no change in blood pressure and a decrease in right atrial pressure. Atropine: Duration of action is 1-1 1/2 hours. Parasympatholytic agent, increases heart rate decreases salivary secretions, increases gastric pH. Glycopyrrolate: same mechanism as atropine, less elevation of heart rate compared to atropine effects last longer - up to 2 - 3 hours with a peak effect in 30-45 minutes given subq or IM.

Injectable analgesic and anesthetic agents: Alpha 2 agonists

Used in veterinary medicine to produce: sedation, analgesia and anxiolysis. The addition of alpha 2 agonists may reduce requirements of inhalant anesthetics when used as a premedicant. Dexmedetomidine - Alpha 2 agonist- Produces profound sedation and analgesia but has significant cardiovascular effects. These include: vasoconstriction, bradycardia, decreased cardiac output. Reversal: Antisedan

Thiopental - Barbiturate

Reduction in blood pressure - peripheral vasodilation is the main action. Compensatory rise in heart rate - baroreceptor response. Commonly associated with ventricular arrhythmias - bigeminy rhythm not uncommon. Contraindications: Patient with known cardiac disease - particularly those with existing arrhythmias. Trauma patients - think of traumatic myocarditis!

Benzodiazepines

Midazolam and diazepam: Cause little or no direct myocardial depressant effects. May see increase in heart rate due to excitation with inadequate use of adjunctive agent, i.e. mu opioid.

Midazolam: Water soluble and is more useful for IM injections. When combined with an opioid, it will provide neuroleptanalgesia. NOT reliable as tranquilizers for dogs and cats when used as a sole agent!!
Patients may lose inhibitions and become excitable. When combined with an opioid as a CRI can be utilized to decrease MAC of inhalant agent. No analgesia provided. Effects may be reversed with Flumazenil.

Hypnotics- Etomidate: no direct myocardial depression. Safe to use with cardiac, critical and septic patients. Cardiovascular stability may be better due to maintained baroreceptor mediated responses. Will cause depression of adrenal function for 3-6 hours. Expense. Allows rapid induction/recovery. Non-cumulative. Propofol- Does cause direct myocardial depression as well as decrease in systemic vascular resistance. Decrease in contractility leads to increase in heart rate - will be transient - lasting several minutes. Profound bradycardia has been noted. Use cautiously in patients with heart disease and hypovolemia.

Mu opioids- Fentanyl is a pure mu agonist causes dose dependant bradycardia (increase in vagal tone). Bradycardia is responsive to anticholinergics - atropine/glycopyrrolate. Single dose IV is very short acting - up to 20 minute duration. Hydromorphone: morphine-like agonist, primary activity at the mu receptors. Cardiovascular effects: bradycardia due to central vagal stimulation, alpha-adrenergic depression causing peripheral vasodilatation, decreased peripheral resistance and baroreceptor inhibition. Oxymorphone- Similar effects to hydromorphone. Case management of side effects the same. Morphine- No direct myocardial effect. Dose dependent bradycardia - responsive to anticholinergics

Mixed agonist/antagonist agents= Buprenorphine: a partial mu agonist/antagonist. Slow onset of action, duration of 6-8 hours. Cardiovascular depression and respiratory depression not as profound as pure mu agonists. Butorphanol: partial agonist/antagonist. Similar to buprenorphine in cardiovascular/respiratory effects. Faster onset of action, shorter duration that buprenorphine. Recommended in multiple texts for premedication for cardiac patients due to sparing effects.

Dissociative Agents - Will indirectly stimulate the cardiovascular system by increasing sympathetic tone which may cause an increase in heart rate, cardiac output, mean arterial pressure, pulmonary arterial pressure and central venous pressure. Increase in rate causes an increase in myocardial work and oxygen demand/consumption. Ketamine- Does not produce a true anesthetic state - dissociation from the environment with analgesia and sensory loss. Heart rate and arterial pressure increase due to an increase in sympathetic tone (CNS derived). Peripheral vascular resistance is unchanged. Prior administration of benzodiazepine, acepromazine and/or inhalant agents may decrease or prevent cardiovascular effects. Telazol = Tiletamine and Zolazepam (benzodiazepine) - Clinical effects similar to ketamine. Prolonged or rough recovery when used as a sole agent.

Inhalant anesthetics. High cardiac output can delay anesthetic induction - blood flow through the lungs maintains the diffusion gradient between the alveoli and blood, i.e. - slower induction in excited patients vs. more rapid induction in decreased output patients - shock, hypovolemia, etc.

ALL inhalants cause dose dependant depression on the cardiovascular system. Some agents may sensitize the myocardium to catecholamine-induced arrhythmias.

MAC (minimal alveolar concentration) produces immobility in 50% of patients receiving noxious stimuli. Varies with agents and species. The lower the MAC, the higher potency of the anesthetic agent. Halothane- Acts to reduce cardiac output through direct depression of the myocardium. Heart rate changes may be minimal. Arterial blood pressure is decreased due to decreased output. Sensitizes the myocardium to catecholamine increasing the chances for arrhythmias. Isoflurane- Lesser degree of
myocardial depression than halothane. Heart rate is seen to increase slightly. Decrease in arterial blood pressure - main cause is decreased vascular resistance vs. decreased cardiac output. Sevoflurane- Causes mild myocardial depression (decreased contractility). Mild systemic vascular resistance and arterial blood pressure depression. Less likely to see an increase in heart rate, vasodilation when compared to isoflurane.

Local anesthetics
Lidocaine w/o epinephrine - Acts directly on the heart to reduce conduction velocity and myocardial contractility. Low plasma concentrations - beneficial. Higher dosages decreased cardiac output, vasodilation and hypotension. CRI at 25-30 mcg/kg/min for analgesic adjunct CRI at 50-100 mcg/kg/min for ventricular arrhythmia control.

Bupivicaine
More cardio toxic than lidocaine. Not used in CRIs for analgesia or arrhythmia control. Useful for local or regional blocks.

References:
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**Tech Tip**

**DON’T GET HYPERTENSIVE OVER HYPOTENSION**

Blood pressure is the driving force for blood flow (perfusion) through capillaries that supply oxygen to organs and tissue beds of the body. Blood pressure is needed to propel blood through high resistance vascular beds, including those of the brain, heart, lungs and kidneys. Blood pressure values are expressed in millimeters of mercury (mm Hg) and as three measurements: systolic, mean and diastolic. The systolic pressure is the pressure generated when the left ventricle is fully contracted. Diastolic pressure is the pressure measured when the left ventricle relaxes. Mean arterial pressure (MAP) is calculated as one third the systolic pressure plus two thirds the diastolic pressure. Mean blood pressure determines the average rate at which blood flows through the systemic vessels. It is closer to diastolic then systolic because, during each pressure cycle, the pressure usually remains at systolic levels for a shorter time than at diastolic levels. Most times, under anesthesia, a patient’s mean pressure is what the anesthetist focuses on. A mean arterial pressure of at least 60 mm Hg is needed to properly perfuse the heart, brain and kidneys.

Mean arterial blood pressures consistently below 60 mm Hg can lead to renal failure, decreased hepatic metabolism of drugs, worsening of hypoxemia, delayed recovery from anesthesia, neuromuscular complications and central nervous system abnormalities, including blindness after anesthesia. Prolonged hypotension (> than 15-30 minutes) can lead to nephron damage. Although the effects may not be immediately apparent since 65-75% of nephrons need to be damaged before renal disease becomes clinically observable, the effects may play a role in the onset of renal disease later in a pet's life. Severe untreated hypotension can lead to cardiac and respiratory arrest. Hypertension, or excessively high blood pressure, can lead to problems as well. Ideally, any animal under anesthesia should have regular blood pressure monitoring because most anesthetic drugs affect blood pressure in some way. Mean arterial blood pressure = cardiac output (CO) x systemic vascular resistance (SVR). Cardiac output is defined as the amount of blood pumped by the heart in a unit period of time. CO = Heart rate (HR) x stroke volume (SV = contractility). Systemic vascular resistance is the amount of resistance to flow through the vessels. Some vessels may be dilated, and therefore allow more flow at less resistance. Constriction of vessels may limit blood flow and require more pressure to get blood through. It's important to know that many of the drugs we use for anesthesia affect one or more of these systems in some way.

Pulse palpation: If no monitor is available, the manual palpation of an arterial pulse can give some indication of the state of the blood pressure. A palpable pulse pressure is the difference between the systolic and diastolic pressures. A difference of at least 30 mm Hg is necessary to palpate a strong pulse. Peripheral pulse palpation sites include the lingual, dorsal metatarsal, carpal, auricular and coccygeal. It is best to monitor the peripheral arteries because these pulses are lost at a much higher mean than the central (femoral) arteries. Potential cardiovascular abnormalities may be detected by regular palpation. Pulses should be assessed for strength, rate, and regularity and palpation should begin prior to induction so that differences in these can be tracked (monitor trends) from the very onset of anesthesia through recovery.

Blanching the mucous membranes with direct pressure should result in a refill time of less than 2 seconds. Delays in refill time can indicate intense vasoconstriction or hypotension.
Oscillometric devices work by picking up pulsation under an occlusion cuff placed over an artery. The cuff is connected to a monitor that can be programmed to measure blood pressure at specific intervals of time. These devices deliver systolic, mean and diastolic readings as well as the heart rate. Most have alarms that can be set to alert when readings are out of the accepted range. The cuff size should be approximately 40% of the circumference of the limb (or tail) around which it will be placed. Cuffs that are too large will lead to artificially low readings and too small a cuff will give false high readings. Ideally, cuffs should be placed on a limb that is close to heart level (the level of the right atrium is the zero mark for blood pressure). Limbs well above the heart may give artificially low readings. Legs hanging well below the heart will give false highs. The cuffs are usually marked with the proper placement over the artery. They must not be applied too tightly as this may occlude flow and cause inaccurate readings as well as swelling distal to the cuff. Poor pulse signals from poor flow (the rear limbs during a severe GDV or large abdominal mass), or any movement of the limb during a reading will interfere with the device and may cause it to fail or deliver an inaccurate reading. These devices do not usually work consistently or at all on very small patients, although there are some newer, veterinary specific monitors out that claim to work accurately on small animals.

Normal systolic blood pressures in the conscious patient are 100-160 mm Hg, normal diastolic pressures are 60-100 mm Hg and normal mean arterial blood pressure ranges are 80-120 mm Hg. Hypotension is classified as MAP of less than 60 mm Hg. It is important to be able to identify the cause of a blood pressure abnormality to know how to begin treatment for it. There are generally three things to consider when looking for causes of hypotension. Look for drugs or physiological/pathological factors that may reduce systemic vascular resistance (SVR), look at heart rate, and look for things that affect stroke volume (preload/contractility). As mentioned earlier, many of the drugs used in anesthesia cause some degree of hypotension, and less often, hypertension. Knowing the side effects of these drugs and how they work will help in determining treatment. Drugs that decrease SVR (and cause vasodilation) in a dose dependent manner include acepromazine, thiobarbiturates, propofol, isoflurane and sevoflurane. Other physiologic factors that may cause a decrease in blood volume or vascular tone include hemorrhage, inadequate volume administration or replacement, dehydration, shock, sepsis, anaphylaxis or severe hypercapnia (high CO2). Patients with acid/base abnormalities should be stabilized prior to anesthesia if possible to help reduce the possibility of hypotension. Drugs that can decrease heart rate include opioids, alpha 2 agonists, and the inhalant drugs isoflurane and sevoflurane. Patients with intracranial disease, hypothermic patients, and extremely fit pets may have low heart rates (bradycardia). Anesthetic drugs affecting the contractility of the heart include the inhalants, thiobarbiturates, propofol, and alpha 2 agonists. The inhalant drugs are potent vasodilators, with up to a 50% reduction in cardiac contractility at surgical planes of anesthesia as well. The other drugs' affect on contractility is more transient and less profound. Alpha 2 agonists and phenylephrine cause vasoconstriction of blood vessels which results in hypertension. The effects of hypertension from the alpha 2 agonists is transient, lasting only a few minutes before the vessels relax and hypotension can result. The dissociative drugs, Ketamine and Telazol have indirect positive effects on the cardiovascular system and thus increase heart rate, but this can cause a reduction in stroke volume. Patient positioning can affect blood pressure. Obese, bloated, or patients with large abdominal masses placed in dorsal recumbency may be hypotensive due to excessive pressure on the caudal vena cava. This pressure may compromise venous return and result in hypotension. The same can happen when positive pressure ventilation is used.

Certain disease states can cause hypertension including pheochromocytomas, pulmonic stenosis, heartworm disease, and hyperthyroidism. Ideally these patients will have their hypertension well controlled before surgery. The exception may be the pheochromocytoma patient.
whose hypertension may spike up during surgery when the tumor is manipulated. A nitroprusside CRI may be indicated for these patients. If a patient develops hypertension under anesthesia that is not related to a disease state, the cause is most likely related to inadequate anesthetic depth and/or inadequate analgesic administration. Adjusting anesthetic depth and providing additional pain medications should result in normotension.

Step one in developing a plan for treatment of hypotension is determining the cause. If the patient is otherwise normal and healthy, the anesthetic drugs are most likely the cause of hypotension. The effects of these drugs are dose related and therefore the best first treatment always involves reducing the dose of the drug, or reducing anesthetic depth. Anesthetic protocols that include appropriate analgesics, pre-operatively and peri-operatively will allow lower doses of all anesthetic drugs to be used, lowering the side effects of each drug as well. Any patient anesthetized with inhalant drugs and/or premedicated with acepromazine will have some degree of vasodilation. Intravenous fluid administration of crystalloids at a rate of 10 ml/kg/hr is recommended in any patient under anesthesia to help "fill the space" caused by vasodilation and to replace normal ongoing losses that occur for patients (with normal cardiovascular and renal function, patients with certain cardiac diseases may not be able to "handle" excessive fluid overload) under anesthesia. Fluid therapy is best begun before hypotension exists. For suspected hypovolemia a fluid bolus of "one hour's worth" the patient's maintenance rate may be given (i.e. 35 kg pet = 350 mls bolus, along with maintenance fluids). Reassess following the bolus. If the patient is instrumented with a Doppler monitor you may be able to hear the improvement and "stronger" flow. Blood loss should be replaced with 2-3 times the suspected amount of loss. One ml of blood loss should be replaced with 2-3 mls of crystalloid. Excessive hemorrhage may require replacement with colloids including Hetastarch and blood products.

If blood pressure fails to respond to these therapies, and surgical stimulation does not fix the problem, then pharmacologic intervention may be necessary. Pharmacologic agents stimulate the cardiovascular system through two primary mechanisms. Vasopressor effects increase MAP through changes in heart rate, myocardial contractility or affecting the tone of the vasculature. Inotropic effects increase contractility and cardiac output. The two most common drugs used for this purpose in dogs and cats are dopamine and dobutamine. Less commonly, ephedrine and phenylephrine can be used. In extreme circumstances, epinephrine and norepinephrine may be indicated. Before beginning dopamine or dobutamine therapy it is important to ensure propervascular volume. Side effects of these drugs include tachycardia and possible arrythmias. Tachycardia is more prevalent in hypovolemic patients or with overdose. ECGs should be monitored when beginning therapy. Therapy should be reduced or discontinued at any sign of side effects. The half life of both drugs is relatively short and side effects should diminish with the discontinuation of therapy. These drugs are given as a constant rate infusion with the dose varying from 0.5-20 mcg/kg/min. Infusions should be started slowly and increased to the desired effect while the heart rate and rhythm are monitored closely.

Blood pressure should be routinely measured on any patient undergoing general anesthesia. The best way to prevent hypotension is to detect changes in blood pressure as soon as they begin.

References:
Tech Tip

Editors Note: Canine Hip Dysplasia (CHD) is a common disease. As a Veterinary Technician or Veterinary Receptionist you should know this chronic painful disease can be avoided when diagnosed at a young age. The discussion below, although written to educate DVMs, should help you understand the need for early diagnosis. This in turn can be used to inform your clients with young puppies prone to the disease. (The bullet points are in bold type for a quick non medical read.)

Surgery STAT: Diagnosis and treatment of juvenile canine hip dysplasia

Oct/Nov 2009

By: William B. Henry Jr., DVM, Dipl. ACVS

DVM360 MAGAZINE

SurgerySTAT is a collaborative column between the American College of Veterinary Surgeons (ACVS) and DVM Newsmagazine. This month begins a two-part column by William B. Henry Jr., DVM, Dipl. ACVS. In Part 1 Dr. Henry writes about diagnosing canine hip dysplasia in young dogs. Part 2 in November will further discuss treatment of juvenile canine hip dysplasia, particularly the JPS procedure. Canine hip dysplasia (CHD) is a heritablepolygenic condition compounded with environmental factors that result in laxity of the femoral head ligament. Laxity of the ligament allows hip subluxation. This laxity, along with incongruity of the coxofemoral joint, damages the acetabular labrum and femoral head cartilage, resulting in osteoarthritis (OA) and clinical pain.

At 10 to 18 weeks of age, hip laxity seldom causes clinical signs unless it is severe in very large or overweight, active puppies. Diagnosis in the very young puppy can therefore be difficult.

Because hip laxity is the No. 1 risk factor for developing OA in the hip joint, it is ideal for all puppies to be evaluated during routine examination. Two methods used to assist in the diagnosis of CHD in puppies are the Ortolani test and PennHip evaluation.

The Ortolani maneuver described in children can be easily learned and used in sedated puppies 10 to 18 weeks old to determine the presence of pathologic hip laxity (Photo 1: a-c). It is done with the puppy in lateral or dorsal recumbency. The femur is slowly abducted while applying a steady dorsal force to the femur and feeling for subluxation and reduction of the femoral head in the acetabulum. A positive Ortolani confirms hip laxity. A negative test does not rule out hip laxity; it may be a result of insufficient patient relaxation, osteoarthritis or severe abnormality of the coxofemoral anatomy (Photo 2).

Photo 1: Palpation for Ortolani; The puppy is sedated and held in dorsal recumbency. Place your hand on the flexed knee and push the femur straight downward (dorsal) toward the acetabulum. A hip with pathologic laxity will subluxate out of the acetabulum (a). Continue to apply downward (dorsal) pressure on the femur and abduct it towards the table (b). At some point, usually between 20 and 45 degrees of abduction, the hip will relocate into the acetabulum. When this occurs there is a palpable drop as the femoral head seats in the acetabulum, often creating an audible sound (c). The palpable relocation of the femoral head back into the acetabulum is the Ortolani Sign, confirming pathologic laxity.
Photo 2: VD pelvic radiograph of an 11-week-old Pit Bull cross showing severe dysplasia. This dog did not have an Ortolani because he has "no hip joint." This is shown to emphasize the importance of radiographs as well as palpation when evaluating for hip dysplasia.

Photo 3: VD and PennHIP distraction views of an 18-week-old Golden Retriever puppy. The hip extended VD view looks fairly normal (a); however, the PennHip distraction view confirms laxity (b).

PennHIP radiographs are a series of three radiographic views (hip extended, distraction and compression views) that allow for assessment of degenerative changes and an objective measurement of hip laxity, reported as the distraction index (Photo 3 a,b). An ideal PennHIP Distraction Index (DI) is 0.30 or less. 0.40 and above are indicative of laxity that would result in CHD and secondary arthritis, especially in the 0.50 to 1.0 (at 1.0 there is no functional hip joint as in Photo 2, above).

The technique uses the dog’s neutral hip angle and a distraction device to yield the distraction index (DI). The DI is a number from 0 to 1 that quantifies the maximum amount the hip luxates out of the acetabulum under passive conditions. PennHIP distraction indices are highly predictive for the risk of development of osteoarthritis in puppies 4 months of age or older. The higher the DI, the greater chance of developing OA in life. This method cannot predict which dogs will have clinical signs of CHD, only the risk of developing OA. Puppies less than 18 weeks old that have a positive Ortolani and/or a PennHIP distraction index consistent with hip dysplasia are potential candidates for juvenile pubic symphysiodesis surgery, which will be discussed in Part 2 of this column.
PART 2:

Juvenile pubic symphysiodesis (JPS) surgery is a prophylactic procedure performed in puppies 10 to 18 weeks of age that have been diagnosed with hip dysplasia as discussed in last month’s column. This technique was developed as our ability to diagnose hip dysplasia (coxofemoral joint laxity) in the immature dog improved, along with the recognition of pubic symphysis abnormalities in children born with hip dysplasia. JPS is a relatively simple procedure associated with little postoperative morbidity.

JPS surgery causes premature closure of the cranial pubic symphysis. The pubic symphysis is responsible for much of the longitudinal growth of the pubis. Premature closure of the cranial pubic symphysis results in shortened acetabular branches of the pubic bones. This, combined with normal growth elsewhere in the pelvis, results in outward rotation of the acetabuli, thereby improving coverage of the femoral heads. This is similar to the effect gained by triple pelvic osteotomy (TPO), but it occurs gradually during the rapid growth phase.

Closure of the cranial pubic symphysis is accomplished either with an electrocautery needle applied through the physeal cartilage following a specific protocol for time and wattage, or by removal of the physeal cartilage with No. 12 and No. 15 scalpel blades, small bone rongeurs and curettes, followed by cauterization of the bone edges. With either technique, the insertion of the prepubic tendon must be removed to allow placement of a protective instrument to avoid urethral damage.

Following surgery, the puppies are walked on a leash, avoiding running and jumping for eight to sixteen weeks. They are reevaluated four to eight months after surgery by Ortolani palpation and standard ventrodorsal radiographs of the pelvis to assess acetabular coverage. The follow up evaluation time frame is based on the DI score and the remaining growth of the puppy i.e. a Springer Spaniel vs. a St. Bernard. Because hip dysplasia is a known heritable condition, neutering the pet is mandatory.

JPS is a minimally invasive, relatively inexpensive procedure associated with minimal morbidity and will eliminate or greatly minimize coxofemoral laxity and therefore the progression of OA. It is an day patient surgery, no overnight hospitalization is required.

Photo 1: PennHip distraction view of a Labrador puppy at 14 weeks. The DI is 0.55.

JPS surgery is more successful at an early age when a significant potential for growth remains, especially in puppies with high distraction indices. A successful outcome is one in which good femoral head coverage is achieved and hip joint laxity resolves, precluding the necessity for more invasive surgical intervention in the future (such as TPO, femoral head ostectomy or total hip arthroplasty) (Photos 1-3).
Hip dysplasia is a very prevalent, complex disease and, as such, veterinarians should strive to become competent in early detection of hip laxity and knowledgeable regarding the principles of the surgical options available. This will enable them to better counsel their technicians and clients regarding breeding strategies, exercise programs and dietary management, as well as potentially beneficial surgical options for affected puppies.

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Performing anesthesia is a task that most veterinary technicians undertake on a daily basis. Intra-operative monitoring is imperative for optimizing all anesthetic procedures. In addition to allowing informed, flexible and well-timed responses to changes in the patient’s status, it can also serve as a database for comparison prior to subsequent anesthetic episodes. A variety of equipment is available to monitor the patient’s physiologic parameters, including but not limited to stethoscopes, blood pressure monitors, electrocardiograph (ECG) tracings, pulse oximeters, end-tidal carbon dioxide monitors, and temperature probes. Is one monitor better than the others? One must first consider the overall effects of general anesthesia before evaluating monitors that would be ideal for assessing patients under general anesthesia. It is well known that inhalant anesthetics are potent respiratory depressants. They are potent vasodilators, readily causing hypotension at increased levels of anesthesia. Decreased cardiac output, central nervous depression, and muscle relaxation are also direct effects of inhalant anesthetics. With this information in mind, let’s examine the various monitoring modalities and exactly what they tell us about the anesthetized patient.

**Electrocardiography (ECG)**
ECG monitoring is commonplace during general anesthesia. It is important to ensure good contact of leads to skin by either using ECG paste or alcohol when placing ECG leads. Avoid wetting large areas of the skin and direct contact with the table. Exact lead locations are not as important as ensuring that all waves are present (even if they are inverted). The P-wave represents atrial depolarization. The QRS-wave represents ventricular depolarization. The T-wave indicates ventricular re-polarization. It is important to realize that an ECG tracing does not provide information about chamber size, or how efficiently the heart is ejecting blood. Therefore, the ECG should be used strictly for the detection of dysrhythmias during the peri-anesthetic period.

Induction agents and disease processes may predispose patients to cardiac arrhythmias. Other potential causes of cardiac arrhythmias may include an inadequate or excessive anesthetic depth, pain, hypoxia, hypercapnia, heart or lung disease, and traumatic myocarditis. Electrolyte imbalances and acidosis may also be a source of cardiac arrhythmias. It is not always necessary to treat arrhythmias unless they are causing adverse affects to the patient. Bradycardia is commonplace in patients undergoing general anesthesia and can be defined as a heart rate of <80 beats per minute in a small dog, or <60 beats per minute in large dogs. In cats, bradycardia is defined as <100 beats per minute. There are numerous causes for bradycardia, which may include drug side-effects, excessive vagal tone, hypertension, hyperkalemia, uremia, hypothermia, increased intracranial pressure, profound hypoxemia, and deep-level inhalants, among others. Tachycardia is defined as >180 beats per minute in a dog, and >200 beats per minute in cats. Tachycardic states can lead to hypotension. There are many causes of tachycardia, which may include but are not limited to, drugs, an inadequate plane of anesthesia, hyperthermia, anaphylactic reactions, hypovolemia, early-stage hypercarbia, and numerous disease states.

Pitfalls associated with interpretation of ECG tracings can include lead mal-positioning due to broken clips or loose connections to the monitor. Electrical interference caused by cautery or other operating room equipment can also be problematic. Rate inaccuracies can occur based on the size of the waveform, resulting in either double-counting or non-counting issues. Patient motion secondary to shivering or increased respiratory rates can cause blurred or erratic tracings. As a final caveat, electrical activity is often the last aspect to completely disappear prior to the pronouncement of death.

**Blood Pressure**
It is important to realize that all patients experience some degree of hypotension during general anesthesia, and if the patient has pre-existing conditions that decrease blood pressure, hypotension will be exacerbated during anesthesia. Normal arterial blood pressure values for canines are systolic 110-119mmHg & diastolic 55-110mmHg, and for felines, systolic 120-170mmHg & diastolic 70-120mmHg.
Although direct blood pressure monitoring is considered the "gold standard", it is highly impractical when it comes to routine blood pressure monitoring in most privately-owned veterinary facilities due to the advanced skill level required to place arterial catheters and the need for 24-hour care. Therefore, only indirect methods of blood pressure monitoring will be discussed. There are 2 methods to measure blood pressure indirectly - either by using a Doppler or an oscillometric device (e.g., Dinamap, Cardell, petMAP). Regardless of the method used, selection of the correct sized blood pressure cuff is imperative for providing the most accurate results. The width of the cuff should extend 40% around the circumference of the limb. When the cuff is determined to be too small, the next wider size should be selected. In cats, it is acceptable to use a cuff that is only 30% of the circumference of the limb. The cuff should be snug, but not too tight. It is acceptable to use a piece of tape to keep it from becoming dislodged during cuff inflation. Selection of an inappropriate cuff size is the most common source of errors. If the cuff is too narrow or too loose, the reading will be falsely high. If the cuff is too wide or too tight, the reading will be falsely low. Acceptable cuff locations include the forelimb, tail and hindlimbs, where the areas proximal to the carpus and tarsus work best. The ventral tail is a good choice in cats and short-legged breeds such as the Bassett hound and Dachshund.

Oscillometric methods detect intracuff changes caused by the pulse wave. They calculate the systolic, diastolic and mean arterial pressure (MAP) as well as the heart rate. They frequently can be programmed to obtain readings at various time intervals (e.g., once per minute, per hour.)

Doppler methods use a 'return-to-flow' principle to detect the systolic blood pressure. Doppler measurements are most accurate when the systolic blood pressure is within normal limits and when the patient has good peripheral perfusion. In cats it is hypothesized that the resultant reading probably represents the MAP, therefore a correction factor of 14mmHg is added to the reading to more accurately reflect actual feline systolic pressures. Because the 'white coat' phenomenon has been well documented in humans, the patient should be calm and as well-acclimated as possible to avoid an inadvertent false diagnosis of hypertension or hypotension. Be warned that a Doppler can mistake heavy respirations for blood flow. Profound arrhythmias, hypothermia, patient motion, low batteries, and electrical interference can also impede obtaining good readings.

There are drawbacks associated with indirect methods of blood pressure monitoring. In general they all tend to underestimate the actual blood pressure, and all work best when the MAP is between 60-100mmHg. Patient movement, smaller patient size (<5.0kg), cold or vasoconstricted patients, or patients with short-legs or excessive skin will all adversely affect results. Additionally, measurements may be difficult to obtain in patients with limb edema.

**Pulse Oximetry**

Pulse oximeters provide continuous and non-invasive monitoring of pulse and an estimate of arterial hemoglobin saturation (SpO2), but do not provide data on the amount (partial pressure) of oxygen in arterial blood, as dissolved in plasma (PaO2). Pulse oximeters can be used on the lip, tongue, ear pinna, prepuce, vulva, toe web or digits, metacarpus, tail, rectal mucosa or flank skin folds. If a skin-fold site is selected it should ideally be hairless, non-pigmented, and fairly thin-skinned (but not overly so). In large animals consider using the nostril/nasal septum as well.

There are 5 main types of hemoglobin: oxyhemoglobin, reduced hemoglobin (deoxyhemoglobin), methemoglobin, carboxyhemoglobin, and fetal hemoglobin. Since 95% of oxygen delivery to tissues is by oxyhemoglobin, saturation is of high clinical significance. Not all types of hemoglobin are capable of transporting oxygen, and as such are termed "dysfunctional hemoglobins." The presence of other light-absorbing types of hemoglobin such as methemoglobin and carboxyhemoglobin will cause the pulse oximeter to overestimate arterial oxygen saturation. Conversely, extraneous blood-borne dyes (such as methylene blue) are known to potentially lower SpO2 readings to 85%, regardless of the true saturation value. Pigmented substances such as bilirubin lipids (hyperbilirubinemia) may also affect arterial blood light absorption and alter SpO2 values. Other causes for erroneous SpO2 values include severe anemia or hemodilution. Moreover, the pulse oximeter may display an SpO2 reading of 100%, in spite of the considerable decrease in arterial blood oxygen content secondary to low hemoglobin values.
Further pitfalls of pulse oximetry use include erroneous and unreliable results or potential complete loss of function when peripheral pulsations are reduced or absent, as in the case of hypotension, hypothermia or hypovolemia. Other conditions that can contribute to unreliable pulse oximeter readings include arrhythmias and tachycardia, increased venous pulsations (e.g., right heart failure, tricuspid regurgitation, etc.), and movement artifacts (e.g., shivering.) Erroneous pulse oximeter readings may also occur when using certain Xenon arc surgery lights (resulting in an SpO₂ reading of 100% and a pulse rate of 180-225), without the probe being attached to a patient!

Finally, beware the pulse oximeter is surrounded by controversy in regards to its use as a monitoring device-it is either prized or despised. This is due, in part, to the oxyhemoglobin dissociation curve, which describes the non-linear relationship between PaO₂ and SpO₂. For example; patients breathing 100% oxygen may have a PaO₂ that is 5 times greater than the SpO₂ (e.g., PaO₂ = 500 mmHg: SpO₂ = 100%). Since the oxyhemoglobin dissociation curve is sigmoid shaped, the hemoglobin saturation would demonstrate only a very slight increase-going from 98% to 100%. Pulse oximeters are most beneficial when evaluating desaturation, such as when the reading drops to below 90%, which corresponds with a PaO₂ that is less than 60mmHg. Pulse oximeters are most accurate within 2% to 6%, and within the 80% to 100 percentile.

Carbon Dioxide
End-tidal carbon dioxide (ETCO₂) is the result of expired gases from the alveoli. End-tidal carbon dioxide analysis can be used to help assess acid/base status as well as the adequacy of patient ventilation in a variety of clinical situations. An abrupt decrease in ETCO₂ can be an early and reliable indication of an impending cardiovascular collapse or cardiac arrest. Consequently, ETCO₂ production can be used to assess the effectiveness of cardiopulmonary cerebral-resuscitation (CPCR) techniques since delivery of carbon dioxide from the lungs requires blood flow, cellular metabolism, and alveolar ventilation.

Capnometers and capnographs monitor ETCO₂ by evaluating samples of the patient’s exhaled gases taken from the anesthetic circuit via an adapter placed on the end of the patient’s endotracheal tube. This adapter must be placed precisely at the end of the patient’s nose to eliminate excessive dead space and prevent rebreathing of carbon dioxide. Capnometers provide only minimum and maximum ETCO₂ values, while capnographs provide a graphic display of exhaled carbon dioxide as each breath is taken. Diagnosing abnormalities in ventilation or anesthetic circuit function are easier using the graphical data provided by a capnograph.

Normal ETCO₂ values are 35-45mmHg. Under normal circumstances, ETCO₂ typically underestimates the arterial carbon dioxide partial pressure (PaCO₂) by a clinically insignificant 2-5mmHg. End tidal carbon dioxide values above 45mmHg indicate inadequate ventilation, necessitating ventilatory assistance via manual or mechanical means. Conversely, by allowing modest increases in ETCO₂ (up to 50mmHg) the anesthetist can bolster arterial blood pressure via endogenous catecholamine release. Nonetheless, the highest ETCO₂ permissible should be 60mmHg.

There are caveats to ETCO₂ monitoring: Esophageal intubation, occlusion of the endotracheal tube, inadequate seal on the endotracheal tube, anesthetic circuit dysfunction/disconnects, moisture within the sampling line, hyperventilation, or respiratory and/or cardiac arrest are all potential causes of failure to detect carbon dioxide. Elevated ETCO₂ levels may occur as a result of hypoventilation due to airway obstruction, pneumothorax, body positioning, or lung disease, or during periods of acutely increased metabolism (e.g., thyroid storm, or catecholamine release). Significant disparities between PaCO₂ and ETCO₂ indicate an inefficiency of gas exchange (e.g., dead space ventilation), which may be secondary to pulmonary embolism, thromboembolism, decreased cardiac output, or perhaps as a result of mechanical ventilation (intermittent positive pressure ventilation.) Explanations for elevated ETCO₂ and inspiratory carbon dioxide may include anesthetic machine malfunction (e.g., malfunctioning valves within the breathing circuit), unsuitable fresh gas flow rates (e.g., non-rebreathing circuits), or exhausted carbon dioxide granules. Therefore, end-tidal carbon dioxide is best analyzed in conjunction with an arterial blood gas sample to yield the most complete status of respiratory function.
Temperature

Hypothermia is not only one of the most common anesthetic complications, but also the easiest to document without special equipment. The hypothalamus closely regulates core body temperature. However, this regulation can be impaired in pediatric and geriatric patients, lean breeds, and those with organ failure, large wounds or infections. Almost all anesthetized or sedated patients will lose body heat under general anesthesia, with the exception of adult Nordic breeds (i.e., Samoyed, Siberian husky, Alaskan malamute), which can actually become hyperthermic. Small patients are at the greatest risk, due in large part due to their small body-surface-to-mass ratio. Hypothermia is exacerbated in prolonged surgical procedures, especially those which expose open body cavities or use cold irrigation solutions. Hypothermia-induced bradycardia is typically non-responsive to anticholinergics. Hypothermia contributes to delayed drug metabolism and decreased hepatic metabolism, resulting in prolonged recovery and potential drug toxicity. Clotting times can be prolonged due to impaired platelet function and hemoconcentration with sludging. Hypothermia also suppresses immune function and may lead to increased infection rates.

Obviously, prevention is key when addressing hypothermia. Re-warming should be considered when the patient temperature drops to < 97.6°F. There are a variety of ways to maintain an envelope of warm air around perioperative patients. Convection-type warm air devices (e.g., BAIR Huggers®) are the most effective, followed by circulating warm water blankets. At least 60% of the body surface area must be in contact with the external heat source for re-warming efforts to be most effective. If latex gloves or bottles of warm water are to be used for smaller patients, it is essential that they are initially warmed to a temperature of <107°F and removed once they cool to the temperature of the patient. Commercially available wire electric heating-pads and heat lamps have been associated with uneven heating, thermal injury and/or electrocution and should be avoided.

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** Presented at 2009 ACVS Symposium: Heidi Reuss-Lamky, LVT, VTS (Anesthesia) Oakland Veterinary Referral Services, Bloomfield Hills, Michigan
Tech Tip

OPTIMAL CONVENTIONAL AND DIGITAL RADIOGRAPHY

While conventional radiography is declining in veterinary medicine, it remains alive in many small animal practices. Digital radiography, on the other hand, is rapidly growing in popularity. Special techniques, such as those utilizing iodinated contrast media, are also changing due to the increasing availability of alternate imaging tools that include ultrasonography, CT and MR imaging. Ten key features for optimal radiography in small animal practice.

1. Patient positioning when you're by yourself. Flexible and rigid sandbags are useful for positioning and restraining dogs, particularly when sedation is not possible. With heavy sandbags encircling limbs or placed over the neck or back, and particularly with the use of a pet-positioner, dogs can be positioned in many different ways. Technicians can work more efficiently and help reduce patient motion during exposure.

2. Doing basic projections the right way. Inadequate patient positioning can lead to false interpretation. This is particularly crucial for head, spine and joints. The identification and assessment of a few anatomical landmarks can validate the quality of your images.

3. Special projections to consider. Stress views are useful when joint instability needs to be confirmed. Skyline projections can help detecting small bone fragments or help localizing soft tissue calcification. Other special views can help better highlighting tympanic bullae or the dens.

4. Fixing exposure issues in conventional radiography step by step. Film darkening relies on adequate mAs and kVp settings and proper development. In most instances, a chart is required and must be used adequately (patient measurements, etc.). Patient conformation and disease processes also influence film darkening. Fixing issues requires a systematic approach.

5. Adjusting DR images before saving and backuping. Image brightness and contrast must be evaluated on a proper monitor in low ambient lighting. Additional filtering can help highlight fine details such as bone margins. Unnecessary areas - particularly white areas - must be cropped. Patient ID and positioning (right-left) must be double-checked.

6. Treating grainy DR images. Grainy, or noisy, images result from insufficient signal, which can be due to insufficient x-rays reaching the digital detector (CR, DR plate or CCD), poor detector sensitivity, or inefficient transformation of x-rays into photons producing electric pulses. Signal-to-noise ratio (SNR) varies among systems, and can be optimized in some cases.

7. Referring patient or sending images for review. DICOM images represent raw information, and associated with larger image files (20-50MB). Ideally, this format is used for burning a CD/DVD or sending images by the web to consulting
radiologists. Images can then be reviewed with native format (megapixel resolution) and greyscale. When burning a CD/DVD, make sure to include proper DICOM reading software in case the consultant does not have one. JPEG compression reduces image file but results in image degradation - giving a pixelated look - which can significantly hamper interpretation. JPEG2000 and lossless JPEG formats are offered by some vendors and can be used for interpretation.

8. **Talking care of DR systems.** A few simple steps can increase the longevity of a digital system. Among those, turning off the computer and monitor at night, and keeping it free from dust and hair, can be crucial. Making sure patient data - including body markers - is accurate can save a lot of time when reviewing images, and can prevent redos. Limiting unnecessary exposures to the digital plate, and particularly with CR, can increase longevity.

9. **Using contrast media.** Now that ultrasound (US), CT and MRI have become widely available, the need for radiographic contrast procedures in veterinary medicine has declined. Yet, several can be useful in practice. Esophagography remain the only way to provide both functional and structural assessment of the thoracic esophagus. Urethrocystography is particularly useful for detecting tears and to assess the intrapelvic ureter. Barium studies can complement US in the detection of foreign bodies, masses, and strictures, particularly when the GI system is gas distended. With US guidance, renal pyelography can help confirm ureteral obstruction or rupture on radiographs. Myelography remains useful in some instances and particularly when MRI is not available.

10. **Proper utilization and maintenance of contrast media.** Light, temperature, and air can alter contrast media in different ways and limit their longevity. If seldom used contrast material is used check expiration dates. Limiting contamination through proper contrast medium manipulations is also crucial. Spillage or leakage of contrast media on or around the patient will make interpretation more difficult.
TECH TIP

POST ARREST CARE: A PERFECT SCENARIO TO APPLY "THE RULE OF TWENTY"
Louisa Rahilly, DVM DACVECC

At the conclusion of cardiopulmonary resuscitation (CPR), there is hopefully a thrilling moment when we have achieved return of spontaneous circulation (ROSC). This joyous moment quickly becomes tempered with the panicked thought...."What Now?". While the survival to discharge rate in human medicine for people who survived the initial arrest episode is only 30-40%, this figure is markedly lower in veterinary medicine with survival rates at 2-10%. While the cause of the arrest and the presence of end stage underlying disease need to be considered in the ultimate outcome, these figures suggest that there is much room for improvement in veterinary post arrest management. "The rule of twenty" is a concept introduced by Dr. Rebecca Kirby in the context of sepsis and the Systemic Inflammatory Response Syndrome (SIRS). It is a list of parameters which have been recognized as essential for critically ill patients in general. It is however, a great rule of thumb for any sick patient, but is an excellent outline in the approach to a post arrest patient.

The first step in managing a post arrest case is to determine and address the underlying cause of the arrest. The following guidelines should be applied to the patient and considered in the context of the underlying disease process. One must always remember, however, that these patients are suffering from both their underlying disease and the effects of having died and undergone CPR. This means they had a period of major organ (including brain, heart, gastrointestinal tract and kidney) ischemia and potentially some trauma sustained from the CPR itself (ie. chest compressions causing pulmonary contusions or rib fractures - good chest compressions can do this!). They will also systemically suffer from reperfusion injury resulting in intracellular organelle and membrane damage which will ultimately result in organ dysfunction and potentially failure. The immediate post arrest period is a critical period in which all of these factors must be considered and addressed concurrently.

Below is Dr. Rahilly borrowing a page from Dr. Kirby's book: an application of the rule of twenty to post arrest cases.

1. **FLUID BALANCE**: Careful administration of fluids including isotonic crystalloids, hypertonic saline, synthetic or natural colloids and blood products is essential to post arrest care. Reperfusion injury may result in endothelial damage and vascular leak into the interstitium. Serial patient assessment to monitor interstitial hydration status as well as close attention to fluid losses (urine quantification in a collection system or weighing of bedding, vomiting/ diarrhea, and panting resulting in increased insensible losses) is necessary. The RECOVER initiative concluded that there is no clear consensus based on the evidence on the type or amount of fluids that are need post arrest, but it is clear that most cases need some fluids. The only exception would include cases which are fluid overloaded or in congestive heart failure as part of the reason for the arrest event. The type of fluids administered should be tailored to each case.

2. **ANALGESIA**: Many patients are comatose or severely neurologically depressed following an arrest episode due to a period of cerebral ischemia. One should consider the degree of reaction/awareness to painful stimuli as well as the presence of injury (existing pre arrest or potentially incurred during CPR) and treat accordingly. The RECOVER initiative did not specifically look at analgesic medications used in the post arrest period, but analgesia at this point is similar to that of many critically ill
patients. Reversible agents such as pure mu agonists (Fentanyl, Remifentanil, Hydromorphone, Oxymorphone, Methadone) should be considered so that they can be completely reversed if arrest recurs or the degree of sedation is deemed inappropriate (ie. hypoventilation). One should keep the respiratory depression of opioids in mind and titrate medications to effect so as not to contribute to hypoventilation and hypercapnea. Non-steroidal anti-inflammatory agents which may contribute to gastrointestinal or renal injury should be avoided. Other agents to consider are Ketamine or Lidocaine infusions. Ketamine increases metabolic demand and therefore is not an ideal choice when one considers the need to maximize adequate cellular oxygenation. There is also controversy surrounding Ketamine use in cases with brain injury (and post arrest cases suffered some brain injury in the form of ischemial). Lidocaine (initially as a bolus of 1-2mg/kg followed by a CRI of 50mcg/kg/min) is a good choice in dogs as it provides analgesia with minimal respiratory or cardiac compromise and can serve as an anti-oxidant.

3. **ONCOTIC PRESSURE:** Adequate Colloid Oncotic Pressure (COP ~17mmHg) is necessary to help maintain intravascular volume and minimize fluid leakage into the interstitium, which can decrease organ function. Oncotic pressure can be augmented through the administration of synthetic colloids, plasma and albumin infusions (canine or human). Careful consideration of protein losses and patient COP relative to colloid administration is necessary as endogenous albumin production is triggered by low COP; over-zealous augmentation of oncotic pressure with synthetic colloids can therefore stifle the production of albumin. Patients with high protein losses (severe peritonitis/ diarrhea) may need 1-2mL/kg/hr of a synthetic colloid while those with minimal to no on-going protein losses but a low total protein due to underlying disease or historic protein losses often only require 0.5-1mL/kg/hr.

4. **ALBUMIN CONCENTRATION:** Albumin contributes the bulk of colloid oncotic pressure (COP), but also has important functions in wound healing, systemic buffering and drug transportation. Hypoalbuminemia has been shown to be a risk factor for mortality in multiple disease states. Endogenous albumin production can be maximized clinically through careful titration of colloids (see above) and providing nutrition. Anorexia causes albumin production to stop within 24 hours and no further production will occur until nutrition is instituted.

5. **BLOOD PRESSURE (CARDIOVASCULAR SYSTEM):** Ensuring adequate tissue perfusion is absolutely necessary for the recovering brain and other major organ systems in the post arrest patient. Analysis of the evidence in the RECOVER initiative found that normal, or perhaps even mild to moderate hypertension (MAP >150mmHg) results in better neurologically intact survival. Vasopressor and/or cardioactive drugs may be required to achieve this outcome. Which drugs and the optimal goal blood pressure are still unknown. It is clear, however that hypotension is unacceptable. Monitoring tissue perfusion through such parameters such as lactate, base excess and central venous oxygenation improve the sensitivity of detecting cellular hypoxia and on-going occult shock.

6. **BODY TEMPERATURE:** The RECOVER initiative found that there is evidence to suggest that post arrest hypothermia initiated as soon as possible in comatose post arrest patients and maintained for >12 hours is beneficial for survival. The recommendation is to cool to approximately 32-34 degree C. 89-93 degree F. Veterinarians should note, however, that these numbers are in human patients and experimental cases and not in clinical small animal patients who are warmer than humans in health. Details of how to achieve the hypothermia and the duration of which are not known. Practically for small animals in a clinical setting, achieving hypothermia is often not a challenge as many cases post arrest are cold. My approach is to not actively re-warm them unless they become <92 degree F. If re-warming is necessary, it should be done slowly (<1 degree C per hour).
7. VENTILATION, OXYGENATION: Evidence as presented in the RECOVER initiative demonstrates that profound hyperventilation (to a low CO2) and hypoventilation (with a high CO2) result in decreased neurologic recovery. The current recommendations are to aim to achieve mild to moderate hyperventilation (mildly low CO2) if an animal is mechanically ventilated or normocapnea. Similarly, the precise goal for oxygenation is unclear. What studies have demonstrated, however, is that hyperoxia and hypoxia are detrimental. Hyperoxia may result in exacerbation of reperfusion injury with the generation of more reactive oxygen species. Hypoxia may result in decreased oxygen delivery to the tissues. Careful pulse oximetry and/or arterial blood gas monitoring to evaluate oxygenation is imperative in post arrest cases.

8. ELECTROLYTES, ACID-BASE BALANCE: There are currently no guidelines for goal electrolyte levels or acid-base parameters in post arrest patients. As critically ill patients, careful attention to sodium levels as a marker of free water status is a necessity. Potassium, calcium, phosphorus and magnesium should also be monitored as these electrolytes all function in important physiologic activities including smooth muscle contraction and vascular tone, cellular energy production, and skeletal muscle strength necessary for adequate ventilation. Acidosis may occur due to hypoventilation or decreased perfusion in these patients and should be treated accordingly as it can result in cardiovascular depression.

9. CARDIAC RATE, RHYTHM, FUNCTION: Myocardial ischemia during the arrest may result in arrhythmias and/or decreased systolic function following ROSC. Continuous ECG monitoring for arrhythmias and treatment as indicated is necessary. Inotropic medications such as Dobutamine infusions may also be necessary if there is depressed cardiac contractility post ischemia.

10. COAGULATION: Endothelial and cellular damage through ischemia and reperfusion injury may result in coagulation disorders and disseminated intravascular coagulopathy (DIC) in post arrest patients. Monitoring of platelet levels and coagulation parameters is important to attempt to "catch" DIC in its earlier phases and treat accordingly. Plasma administration in cases which show clotting factor consumption through elevation of clotting times should be considered. Theoretically, clinicians should also consider anticoagulant therapy as the inflammation associated with reperfusion injury may trigger a hypercoagulable state.

11. RENAL FUNCTION: Renal function can be monitored directly through quantification of urine output and regular assessment of BUN and creatinine. It is important to monitor these values daily as urine function may decrease in the days following a renal ischemic event, such as cardiopulmonary arrest. Renal function is also indirectly evaluated in the assessment of electrolytes and acid-base status as tubular injury may result in diuresis or metabolic acidosis in the absence of a rising BUN or creatinine.

12. GASTROINTESTINAL INTEGRITY: The gastrointestinal tract of the dog is very susceptible to ischemia and is considered to be the source of systemic toxins/inflammatory cytokines and activated white blood cells following an ischemic incident and subsequent reperfusion. Antibiotic coverage to "protect" from bacterial translocation from the gastrointestinal is somewhat of a controversial topic in critical care as the development of resistance is a concern, and the question of prophylactic antibiotic use was not specifically addressed in the RECOVER initiative. Measures to improve intestinal integrity, such as enteric nutrition and ensuring adequate gastrointestinal perfusion through cardiovascular optimization, however, make sense as supportive measures which are unlikely to cause harm.
13. **NUTRITION:** Although nutritional status of the post arrest patient was not specifically addressed in the RECOVER initiative, adequate nutrition in critically ill patients is known to maximize immune function and is necessary for endogenous albumin production. Enteric feeding is the optimal route of nutrient administration as it helps to maintain intestinal motility, function and integrity. Calculation of the patients' resting energy requirements (RER) with the goal of feeding 50-100% of RER is recommended, as over-feeding can result in increased CO2 production in an animal with potentially compromised ventilatory reserves to maintain normocapnea. Over-feeding in a neurologically or respiratory compromised animal may result in a respiratory acidosis.

14. **GLUCOSE:** Blood glucose levels should be monitored frequently to ensure normoglycemia as hypoglycemia can be detrimental to neurologic function and recovery and hyperglycemia has been shown to be detrimental to patient outcomes. Dextrose supplementation and conversely short-acting insulin should be utilized as needed to maintain normal blood glucose.

15. **ANTIBIOTICS/WBC COUNT:** Complete blood counts should be performed every 2-3 days during the critical period (more often if indicated) and peripheral blood smears should be evaluated daily for a manual white blood cell count and evidence of toxic change and/or left shifting. Judicious antibiotic use as indicated for the underlying disease state or developing nosocomial infections is prudent.

16. **RED BLOOD CELLS:** A packed cell volume should be monitored at least twice a day to watch for anemia and as an indicator (along with total solids) of hydration status. Clinicians should have a quick trigger for red blood cell transfusions in post arrest cases as ensuring adequate oxygen delivery is a top priority.

17. **DRUG DOSAGE, METABOLISM, INTERACTIONS:** As with all critically ill patients, one must consider the entire physiologic picture of post arrest cases. Drug dosages or frequency may need to be adjusted based on liver or renal dysfunction and associated increased half-life of hepatically metabolized and/or renally excreted medications. The albumin level should also be considered for drugs which are highly protein bound.

18. **MENTATION:** Many post arrest cases will have decreased mentation and potentially even be in a coma. The clinician must monitor these cases closely to ensure adequate ventilation and gag reflex and consider mechanical ventilation or intubation with close monitoring if either of these parameters are sub-optimal. The RECOVER initiative evaluated the prophylactic use of anti-seizure medications post arrest as seizures are known to be relatively common in humans following ROSC. There is currently no evidence indicating a benefit to seizure prophylaxis post arrest. However, there are some bundled therapy studies that evaluated seizure prophylactic medications (Thiopental and Phenytoin) which used these medications among other interventions and found potential benefit. It is not clear, however, if it was the seizure prophylaxis specifically, another component of the bundle or the entire package which allowed for the benefit. Cerebral function and signs of elevated intracranial pressure should be monitored closely. Signs of elevated intracranial pressure include pupillary changes (miosis followed by anisocoria and mydriasis with progressive intracranial pressure elevations), limb and/or jaw rigidity, decreasing mentation and hypertension with concurrent bradycardia. Suspected elevations in intracranial pressure should be treated with hypertonic saline (3-4mL/kg) if the patient is hemodynamically unstable or mannitol (1g/kg) and/or hypertonic saline if the patient is stable. Brain protective measures such as keeping the head elevated 15-30° and making sure there are no bends of
the neck should be taken to ensure adequate cerebral venous drainage in order to minimize intracranial pressure.

19. **NURSING ORDERS:** Detailed nursing orders ensuring constant attention to mental and hemodynamic status are essential. For comatose patients, measures including applying eye lubrication and anti-bacterial oral rinses to avoid ulcers and bacterial colonization should be taken. Regular turning of the patient allows for improved pulmonary function and passive range of motion exercises keeps interstitial fluid moving and lymphatics flowing.

20. **TENDER LOVING CARE:** The most important, but hard to directly institute on our treatment sheets, aspect of critical care is tender loving care. Clean bedding and ensuring that the patient is comfortable, free from anxiety, and clean and dry at all times decreases the risk of nosocomial infection and will ultimately contribute to patient well-being and hopefully survival.

As is evidence by the extensive list of parameters to concurrently monitor and consider in post arrest patients, these cases are intensive. There are no studies in veterinary medicine evaluating the survival effect of these patients being treated by a criticalist specifically, but at this point the recommendation is to hospitalize these patients at a 24 hour facility with the ability to closely monitor and treat critically ill patients on a minute-by-minute basis.

**REFERENCES:**
TECH TIP

Editor's Note: We have used cold compress therapy during the first 24 hours post-op for the past several years on all our orthopedic surgery cases. Our surgery technicians use hand compression rather than pneumatic compression as was used in this study. We feel it helps minimize post-op pain. This paper was given at the ACVS Symposium in 2012 substantiated our subjective assumption.

Prospective Evaluation of Cold Compression Therapy On Postoperative Pain, Swelling, Range of Motion and Lameness Following Tibial Plateau Leveling Osteotomy

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Introduction: Cold compression therapy (CCT), the use of cold therapy combined with intermittent pneumatic compression, is currently used in human medicine to treat postoperative pain, decrease swelling and improve limb function following knee surgery. Our objective was to determine the effect of CCT on postoperative pain, swelling, range of motion and lameness in dogs undergoing tibial plateau leveling osteotomy (TPLO).

Materials and Methods: Thirty-four dogs undergoing TPLO were included in the study and randomly assigned to one of two groups. Group 1 received CCT during the 24 hour postoperative period and group 2 received no CCT. Pain, degree of lameness, stifle range of motion and swelling were evaluated preoperatively, 24 hours, 14 days and 28 days postoperatively. Logistic regression and linear regression analysis were used to compare the measured variables. P < 0.05 was considered significant.

Results: Treatment resulted in significantly lower pain scores (p=0.004), decreased lameness (p=0.001), increased range of motion (p=0.003) and decreased stifle swelling (p=0.008) 24 hours postoperatively. No difference in the outcome measures were observed at 14 and 28 days postoperatively.

Discussion/Conclusion: Our study supports the use of CCT as part of a multimodal approach to decrease pain and swelling and improve limb function in the immediate 24 hours following TPLO. The benefits of CCT reported here are likely related to the decrease in pain and inflammation and improved regional tissue perfusion achieved during the treatment period.

Acknowledgments: Cold compression units were provided by Game ReadyEquine, Coolsystems Inc.
Tech Tip

ISOFLURANE OR SEVOFLURANE - DOES IT REALLY MATTER?

With the removal of halothane from the US veterinary market, veterinarians who hadn't already transitioned to use of isoflurane or sevoflurane are faced with selecting between them. The question of whether a practice should switch from isoflurane to sevoflurane or have both is also frequently raised. We currently use both depending on the patients age, general health, length of the surgical procedure, and surgeon's preference. The following discussion may help you understand the differences between the two gas anesthetics we have available and commonly use.

Blood Gas Solubility
Speed of anesthetic onset and recovery and the ability to change anesthetic depth precisely and rapidly are directly related to the agent's blood gas solubility. When all other factors are equal, changes occur faster when blood gas solubility decreases and so is most rapid for sevoflurane and slowest for halothane. Changes occur more rapidly with sevoflurane than with isoflurane. Clinically this impact is most notable in patients being anesthetized with an inhalant agent in the absence of other drugs (less common approach in today's practice except perhaps with small mammals and birds). While efficiency may be improved with a more rapid onset, offset and change in anesthetic depth, patient monitoring is critical to avoid an anesthetic overdose.

Vapor Pressure
The vapor pressure at a given temperature determines the maximum concentration of the inhalant. So for example, at sea level concentrations approximating 32% are possible for halothane and isoflurane, 21% for sevoflurane. Because these concentrations are well above the necessary anesthetic dose in human beings and animals, these drugs are administered using vaporizers calibrated to specifically administer clinically relevant concentrations. Agents with similar vapor pressures (e.g., halothane and isoflurane OR sevoflurane) have used interchangeably in the same vaporizer after thorough cleaning and recalibration. This has been historically used as a cost saving mechanism, but in today's environment manufacturers will frequently provide a vaporizer if the practice purchases sufficient inhalant agent.

While Mean Alveolar Concentration (MAC ie. the percentage of gas administered) vary slightly between species, for a specific agent they tend to be within a fairly tight numerical range. MAC is a good standard of comparison when evaluating physiologic effects (e.g., blood pressure, PaCO2) of a fixed concentration of the inhaled agents between species or effects at different concentrations within a species. While MAC may be used to guide anesthetic delivery in clinical patients, it is important to keep in mind that MAC is determined in healthy patients in the absence of modifying drugs. However, it does provide and indicator of drug potency; higher MAC values reflect a less potent drug or the need to get to a higher concentration to have a similar effect. Halothane is more potent than isoflurane which in turn is more potent than sevoflurane. This partially offsets the clinical effects of the differences in blood gas solubility observed for these agents. For example, the blood gas solubility for sevoflurane is approximately half that of isoflurane, but the MAC is approximately double. So while it takes less time for anesthetic uptake with sevoflurane, the concentration necessary to anesthetize a patient to a given depth is higher.
Cardiovascular and Respiratory Effects
Cardiovascular and respiratory depression occurs in a dose related manner with all the aforementioned inhaled agents. The magnitude of these changes at a given dose is both agent and species specific. Of the agents, halothane causes more cardiovascular depression, but less respiratory depression than isoflurane and sevoflurane which have roughly equivalent effects in the clinical dose range.

Other Considerations
Two other factors that should be considered when selecting between isoflurane and sevoflurane today are the reactivity of the compounds and their cost. Sevoflurane does react with carbon dioxide absorbents to form Compound A which has the potential, albeit limited or in extreme clinical circumstances, to be nephrotoxic. To minimize the level of Compound A present in the circuit, low flow or closed circuit anesthesia with sevoflurane is not recommended. This has an additional impact on the cost of using this agent which is while recently reduced in prices is still considerably more expensive than isoflurane. Example: Isoflurane @1 liter of O2 per minute 1.4 %, costs 63 cent per hour; Sevoflurane @1 liter of O2 per minute 2.3%, cost $3.68 per hour.

For many years, isoflurane has been the predominant inhalation agent in small animal practice in the US (vs. halothane). It offers greater cardiovascular stability, an ability to change depth more rapidly and is metabolized to a lesser degree than halothane. Veterinary patients unlike human patients do not seem to react adversely to isoflurane when administered by a mask. With the availability of the newer agent sevoflurane which like isoflurane is licensed for use in dogs, veterinarians have been faced with an additional choice of inhaled agent. The advocacy to switch to this agent has been largely driven by marketing of its rapid onset and offset. The literature is mixed in this regard and clinical impression suggests that in the presence of modifiers (premedications, injectable induction agents, analgesics, etc.) commonly used in peri-anesthetic patient management today, these potential advantages are less. The ability to quickly change anesthetic depth with sevoflurane can be an advantage as long as someone knowledgeable in its use is present during the anesthetic to monitor the patient. Cardiovascular and respiratory effects are very similar for the two agents. While likely not to be clinically important in the majority of patients the question of sevoflurane use in patients with renal compromise remains. The cost difference is not as substantial now as when sevoflurane was first introduced, but should be considered when choosing between the two agents.