Special Anesthetic Considerations: Managing Higher-Risk Patients (Parts 1 & 2)
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The key to avoiding peri-anesthetic complications is to anticipate possible concerns. This process begins with the anesthesia work-up. Patient signalment, history, including co-morbidities, current medications and previous anesthetic procedures, along with a complete physical examination and laboratory values allow the anesthetist to assign a physical status to the patient. It is also important to identify anticipated complications based on any of the above information and/or the primary disease/complaint, to monitor the patient closely and have an action plan should any of these complications arise.

Anesthetic drugs alter the patient’s normal physiology and ability to maintain homeostasis. Therefore, proper pre-operative patient preparation including rehydration, correction of acid/base and electrolyte abnormalities, anemia and hypoproteinemia will help the patient better compensate during anesthesia and surgery.

Patients with liver disease
Anesthetic concerns for patients with liver disease include:

Glucose homeostasis => Hypoglycemia
- ↓ Drug Metabolism => Prolonged recovery
- ↓ Protein Synthesis:
  - Drug binding- drugs protein bound, ↑ unbound drug, ↑ effect
  - Oncotic pressure - albumin 80% oncotic pressure, Hypotension
  - Coagulation Factors- ↑ hemorrhage, blood loss

Hypoglycemia
Pre-operative hypoglycemia (<60-70mg/dl) should be corrected before anesthesia and monitored intra-operatively every hour. 2.5% Dextrose can be added to the intra-operative IV fluids. The clinical signs of hypoglycemia (seizures, CNS depression, coma) are masked by anesthesia, therefore serial blood glucose measurement is necessary.

Drug metabolism
There are four basic pathways for drug metabolism: Phase I reactions include oxidation (CYP450)**, reduction and hydrolysis, Phase II reactions include conjugation with glucuronic acid***(most common), sulfate, glycine, glutamate, glutathione, acetylation, methylation. The CYP 450 oxidation metabolic pathways may be the most susceptible in liver dysfunction; glucuronide conjugation is the most preserved (except in cats which are deficient in glucuronyl transferase). Rather than memorize all of the metabolic pathways, there are several strategies anesthetists can use when choosing drug protocols for patients with liver disease:

1. Use drugs not metabolized by the liver
The newer inhalant anesthetics are minimally metabolized by the liver (isoflurane ~0.1- 0.2%, sevoflurane ~3 - 5% compared to halothane ~20- 25% and methoxyflurane ~50%). Nitrous oxide can also be used as an adjunct analgesic/anesthetic. It is eliminated via respiration, with no documented liver metabolism. The MAC of nitrous oxide in animals is reported to be 150-200% and therefore, it will only decrease the MAC of Isoflurane by ~25%. Propofol is metabolized by CYP 450 but it also has extra-hepatic sites of metabolism and its short duration of action make it a good choice as an induction agent in patients with liver dysfunction.

2. Use drugs that are reversible
The use of pure mu agonists such as hydromorphone, morphine, oxymorphone or fentanyl can be used for analgesia and to decrease the MAC of inhalant anesthetics. Inhalant anesthetics cause dose dependent cardiodepression and vasodilation. Patients with hypoalbuminemia will have decreased colloid oncotic pressure and will be prone to hypotension during general anesthesia. Therefore, using pure mu agonists to decrease the amount of inhalant needed will decrease the negative effect of the inhalant on blood pressure. Mu agonists can be fully or partially reversed with naloxone or butorphanol (.1ml (1mg) Butorphanol + .9ml NaCl, give in .2ml (.2mg) increments). The use of benzodiazepines such as diazepam or midazolam has the advantage of being reversible (flumazenil) and minimal cardiovascular effects. However, their use in patients exhibiting signs of hepatocerebralpathy is controversial.

3. Use lower doses of highly protein bound drugs, avoid non-reversible/long lasting drugs with significant cardiovascular effects
The decreased albumin levels in patients with liver disease will affect protein binding of many anesthetic drugs, resulting in higher plasma levels of unbound or active drug and greater clinical effect. Avoid drugs such as acepromazine which has a long duration of action (4-6hrs), is non-reversible and can cause vasodilation and hypotension.
Hypoproteinemia/hypoalbuminemia
Drug binding – see drug strategies above

Colloid oncotic pressure (COP) – Albumin accounts for 70-80% of plasma COP. It is the most oncotypically active plasma protein and is responsible for most of the intravascular fluid retention. General anesthesia and major abdominal surgery results in redistribution of albumin from the intravascular space to extracellular sites, causing a decrease in plasma COP, increased interstitial fluid and a reduction of plasma volume. The decrease in plasma volume can contribute to decreased stroke volume and cardiac output, contributing to hypotension intra-operatively.

Treatment should begin pre-operatively if possible. Fresh frozen plasma is used to treat hypoalbuminemia. However, to increase the albumin by 1gm/dl, a dose of 45ml/kg is required and can be expensive in larger patients. Therefore, the best approach is to use synthetic colloids in addition to plasma. Hetastarch at 2ml/kg/hr can be administered pre-operatively and intra-operatively, along with monitoring of COP.

Hemorrhage/clotting abnormalities
Most of the factors involved in coagulation are primarily or exclusively produced in the liver except for factor VIII. Coagulation inhibitory factors including anti-thrombin III, protein C, protein S and fibrinogen are also formed in the liver. Clotting abnormalities may result from decreased synthesis of coagulation factors (II, V, VII, IX, X, XIII, fibrinogen), shortened circulating half-lives of coagulation factors due to loss from bleeding and/or disseminated intravascular coagulation and platelet abnormalities including decreased platelet count and impaired platelet function. Prothrombin time (PT) and Partial thromboplastin time (PTT) are recommended pre-operatively to assess coagulation. Platelet count and buccal mucosal bleeding time may give additional information regarding primary hemostasis and platelet function. Pre-operative or intra-operative administration of Fresh Frozen Plasma may be indicated to provide clotting factors and albumin.

Managing Acute intra-operative hemorrhage
Delivery of oxygen (DO2) to tissues is essential to patient survival. DO2 is dependent on cardiac output (CO) and the content of oxygen (CaO2) in the blood (DO2 = CO x CaO2). The oxygen content in blood is made up of the O2 bound to hemoglobin (HbO2) and the oxygen dissolved in blood (PaO2). Normal healthy animals with a PCV of 20 can compensate for a decreased O2 content (RBC) by increasing CO via increasing heart rate and vasoconstriction. Anesthesia interferes with the ability to compensate by causing vasodilation and many anesthetic drugs also decrease HR.

Therefore, the following pre-operative transfusion triggers are recommended
- PCV < 20  => transfusion recommended
- PCV 20-30  => maybe, depends on organ reserve, expected losses, chronicity/regeneration, expected blood loss for procedure
- PCV > 30  => no transfusion

Even if a patient has a normal PCV pre-operatively, the anesthetist should be prepared to quantitate and treat intra-operative hemorrhage if it occurs. Induction of general anesthesia decreases PCV, TP and COP even if no crystalloid IV fluids are administered, therefore, a PCV, TP and/or COP should be measured after induction to obtain an accurate starting point for these values. The total blood volume should be calculated for the patient and then increments of loss (10%, 20% and 30%) as this corresponds to modalities of treatment.

- Calculate total blood volume
  - Dog 90ml/kg, cat 70ml/kg
    - Examples:
      - 20kg dog x 90ml/kg = 1800ml
      - 2.5kg cat x 70ml/kg = 175mls
  - Then calculate allowable blood loss at 10, 20 & 30%
    - Examples:
      - 20kg dog, total blood volume = 1800ml => 10% = 180ml, 20% = 360ml, 30% = 540ml
      - 2.5kg cat, total blood volume = 175ml => 10% = 17.5ml, 20% = 35ml, 30% = 52.5mls

Depending on an individual’s ability to compensate while under general anesthesia, clinical signs (hypotension, +/- tachycardia) may be seen when ~ 10% blood loss has occurred, therefore, blood loss should be replaced as it occurs.

Quantitating intra-operative blood loss
- Q-tip .1ml
- 4x4 sponge 5-15ml
- Lap sponge 50ml
- Weight 1gm = 1ml
- Suction cannister: mls blood loss = PCV of fluid x Volume of fluid in suction PCV of patient

Treatment of blood loss starting with normal PCV
- 10-15%  => replace with crystalloid
• 15-25% => replace with colloid
• >25-30% => replace with blood

**Blood products**
• Whole Blood - RBC, protein, platelets, clotting factors; for severe blood loss (> 30-50%)
• Packed RBC- RBC, PCV 80-90%; for less severe blood loss or with plasma for severe blood loss
• mls blood required = blood volume x (desired PCV–recipient PCV)
  Donor PCV
• Fresh Frozen Plasma - Albumin, plasma proteins, clotting factors; for hypoalbuminemia (along with colloids), prolonged clotting times
  o Need high doses; 45ml/kg to raise albumin 1g/dl
• Canine/Human Albumin
  o Human – ½ of dogs have severe, sometimes fatal transfusion reactions
  o Canine – new, no independent, controlled studies

**Hypotension**
• Hemorrhage – see above
• Colloid oncotic pressure – see above

Treatment of hypotension consists of assessing/reducing anesthetic depth, decreasing the amount of inhalant anesthetics, increasing vascular volume and the use of positive inotropes or vasopressors. Inhalant anesthetics cause dose dependent cardiovascular depression, therefore, limiting inhalant % by supplementing with opioids, nitrous oxide or other adjunct analgesics will decrease the MAC of inhalant and cause less CV depression. Treatment of vagal induced bradycardia, especially when using moderate to high doses of pure mu opioid agonists, with anti-cholinergic drugs is also indicated if the patient is hypotensive; recommended target HR are >60bpm for medium to large dogs > 80bpm for small dogs, >90bpm for cats. Anti-cholinergic drugs will be less effective or not effective in the face of moderate to severe hypothermia. Stroke volume can be improved with IV fluids; crystalloid bolus of 2 - 5mls/kg, hetastarch at 2 – 5ml/kg (up to 20ml/kg), hypertonic saline at 2-4ml/kg or blood products if indicated (blood loss > 20-30% of blood volume). Use of crystalloid IV fluids should be limited in patients that are hypoalbuminemic; instead, colloids (plasma or hetastarch) are indicated.

Positive inotropes may be used to increase contractility and therefore SV and vasopressors are used to counteract drug induced vasodilation. Ephedrine has both direct and indirect sympathomimetic properties, has greater β1>β2 activity leading to positive inotropy, increase contractility and also stimulates α mediated vasoconstriction (dose: .03-.1mg/kg IV). Dopamine at a dose of <2.5 μg/kg/min stimulates DA1 & DA2 dopamine receptors and causes vasodilation especially in the kidney, at a dose of 2.5-5 μg/kg/min, it’s primary effect is β1 agonist and + inotropy, doses >5-10 μg/kg/min stimulate a1 & a2 receptors leading to vasoconstriction and increased afterload so, although blood pressure increases so does myocardial work. Dobutamine is a β1 agonist that ↑contractility and does not have an effect on SVR, it has some β2 and α effects (Dose: 1-10 μg/kg/min)

**Patients with neurologic disease**

Understanding of normal cerebral physiology and pathobiology, the effects of drugs and anesthesia on physiologic responses, and necessary therapeutic interventions on cerebral blood flow are all essential in meeting the demands of the patient with intracranial disease.

Within the cranial vault, the components of cerebrospinal fluid, blood, and parenchymal tissue comprise approximately 10%, 10%, and 80%, respectively, of the total volume. The Monroe-Kellie Doctrine states that an increase in volume of one of the cranial constituents must be compensated by a decrease in volume of another, usually a decrease in CSF production and an increase CSF absorption. Brain tumor, trauma with brain edema, infectious disease/abscess, seizures leading to brain edema, hydrocephalus all increase the volume of the brain tissue within the cranial vault. Changes that occur in any single component result in compensatory changes in the other components to maintain constant ICP, within limits. Changes that exceed the limitations of the system result in elevations of ICP that may cause brainstem herniation and death. The importance of auto regulation of CBF lies in its relationship to intracranial pressure (ICP). If CBF increases, the cerebral blood volume increases and may increase ICP in patients that are maximally compensated for the increase in intra-cranial volume. Conversely, a reduction in CBF may produce a therapeutic decrease in the cerebral blood volume and ICP.
Cerebral blood flow (CBF) is auto regulated and held constant over a mean arterial pressure range of 50 to 150 mm Hg. A reduction in arterial oxygen tension to below 60 mm Hg also dramatically increases cerebral blood flow. But the primary driver of cerebral blood flow is under chemical regulation and varies directly with arterial carbon dioxide tension over the range of 25 to 70 mm Hg. Changes in arterial oxygen and carbon dioxide are common in anesthetized animals. Therefore, it is important to avoid hypoxemia and hypercarbia in patients with intracranial disease. Patients should be pre-oxygenated prior to anesthetic induction with 50-100mls/kg/min for 3-5 minutes. Positive pressure ventilation is necessary to maintain EtCO2 (and therefore PaCO2) within the normal range (EtCO2 35 – 37 mmHg, corresponding to a PaCO2 of 35 – 40mmHg). Maintain MAP > 60mmHg.

Anesthetic drugs can alter cerebral blood flow. Injectable anesthetics, with the exception if ketamine, tend to preserve or decrease cerebral blood flow and cerebral oxygen consumption, thereby providing some measure of neuroprotection. Inhalant anesthetics, at greater than 1.0-1.5 MAC may cause cerebral vasodilation, increased cerebral blood flow, and raised intracranial pressure. At low concentrations, inhalant anesthetics combined with controlled ventilation can be effective in preventing exacerbation of raised intracranial pressure. In patients where raised intra-cranial pressure is suspected, propofol (+/- fentanyl) constant rate infusion, along with IPPV may be necessary to avoid increasing intra-cranial pressure.

Anesthetic considerations
- Avoid seizure promoting drugs (ketamine, methohexitol)
- Treat seizure activity with GABA agonist (benzodiazepine, propofol)
- Provide analgesia and inhalant sparing effects with opioids
- Prevent coughing at intubation
- Maintain < 1.0 MAC inhalant
- Monitor EtCO2 or PaCO2 – maintain in normal range with IPPV
- Monitor blood pressure – treat hypotension
- Avoid positioning patient where venous congestion is created (head below heart position, occlusion of jugular veins)

Monitor and be aware
- SpO2, EtCO2, MAP, HR => Sudden, severe decrease in heart rate may indicate occurrence of the Cushing’s Reflex (CPP = MAP – ICP), if ICP increases to the point of decreasing cerebral perfusion pressure, the body will respond by a severe increase in MAP causing a reflex bradycardia – this reflex is driven by cerebral ischemia due to increased ICP and risk of brain herniation and imminent death. Treatment: hyperventilation, hypertonic saline 2-4ml/kg and mannitol - *Induction is a critical time

Patients with cardiac disease

General strategies
- Maintain cardiac output, maintain good oxygenation/ventilation, avoid fluid overload, avoid hypo- or hypertension, avoid bradycardia or tachycardia, avoid increased myocardial work & O2 consumption, and avoid drugs that cause arrhythmias & myocardial depression
- Use drugs with mild CV effects
  - Opioids – pure u-agonists: hydromorphone, oxymorphone, fentanyl, morphine and benzodiazepines – midazolam, diazepam, etomidate, alphaxalone, nitrous oxide, balanced Anesthesia’ & multi-modal approach
    - Low dose Acepromazine to decrease stress and promote forward flow
    - Judicious use of anti-cholinergics, pre-oxygenate, decrease IV fluid rate

Dogs
- A-V valve insufficiency – Mitral Regurgitation - most common degenerative heart disease in dogs, progressive disease/need to establish extent of cardiac dysfunction prior to anesthesia.
Cats
Hypertrophic cardiomyopathy - associated with hyperthyroidism, tachycardia, murmur, ‘gallop’ rhythm, hypertension, renal failure
Management
- Stabilize pre-op with anti-thyroid, cardiac medications
- Avoid stress, tachycardia
- Opioid, +/- benzodiazepine, +/- low dose Acepromazine, +/- low dose Dexmedetomidine

Patients with respiratory disease
- Lower airway disease: pneumonia, asthma, contusions
- Extra-pulmonary disease: pneumothorax, pleural effusion, Diaphragmatic hernia- !! Evacuate air, fluid !!
- Sedation/pre-med depends on patient status, preoxygenate, rapid IV induction/intubation
- 100% O2, +/- IPPV, +/-PEEP

Brachycephalic airway obstructive syndrome
- Stenotic nares, elongated soft palate, excessive pharyngeal tissue, everted laryngeal sacculles, hypoplastic trachea => Upper Airway Obstruction (UAO)
- Peri-operative mortality ~3% (~25x risk of Class I or II)

Premedication
Prevent peri-operative vomiting => Maropitant 1hr prior to opioid administration, NK-1 antagonist – FDA approved anti-emetic
Assess degree of upper airway obstruction - +/- LOW dose Acepromazine to relieve stress from UAO/hypoxemia, +/- Anticholinergics - high vagal tone from UAO, continuous observation after pre-medication. Have a variety of ET tube sizes 6.0-10mm available and leak checked.
Induction: pre-oxygenate, rapid IV induction/intubation, propofol - drug of choice – quick recovery without residual effects

Recovery
Continue O2, monitor SpO2, leave in IV catheter, sternal position, head elevated, quiet/dim light surroundings, leave in ET tube as long as possible, be prepared to re-intubate (have laryngoscope, ET tube, induction agent, O2 source/IPPV - anesthetic machine available) => post-operative nasotracheal O2, monitor SpO2 after extubation, sternal, prop open mouth, extend tongue.

Patients with renal disease
Acute renal disease
Patients in acute renal failure may present in a wide spectrum of clinical states from nearly normal to extremely depressed, obtunded and in shock. Stabilization with regard to intravascular volume, acid/base and electrolyte abnormalities is necessary to avoid life threatening complications during general anesthesia. The most common causes in small animals include urethral obstruction, traumatic urinary bladder rupture.
Metabolic abnormalities include: dehydration, metabolic acidosis, uremia (increased BUN, creatinine), hyperkalemia, hyponatremia, hypochloremia and respiratory compromise in the case of uroabdomen. Acid base and electrolyte levels should be analyzed pre-operatively and corrected prior to anesthesia. An electrocardiogram should be performed on all patients to check for cardiac arrhythmias due to hyperkalemia. Serum potassium levels greater than 5.5 to 6.0 mEq/L need be treated before general anesthesia and corrected as much as possible.
These cases should be treated as a medical NOT surgical emergency => **Pre-op stabilization is key to avoiding anesthetic complications** and should consist of:
Correct dehydration, hyperkalemia, acidosis, Na, Cl, +/- drain fluid from abdomen slowly (dogs), passage of urinary catheter in cats with urethral obstruction (this can be accomplished with sedation and a caudal epidural block).
Decrease serum potassium levels => using potassium free fluids such as 0.9%NaCl, +/- glucose (2.5 – 5.0% dextrose will shift potassium into cells (effects in 15-30 minutes), +/- insulin (0.5-1 U/kg insulin), sodium bicarbonate. If cardiac arrhythmias are present, stabilize myocardium using 10% calcium gluconate 0.5mg/kg IV slowly.
Avoid drugs that are excreted unchanged by the kidney (ketamine in cats)
Intermittent positive pressure ventilation and monitoring of EtCO2 – hypercarbia will cause a respiratory acidosis (in addition to the metabolic acidosis from the urinary obstruction) and can cause serum potassium levels to increase (K+ comes out of cells in exchange for H+ into cells to maintain electroneutrality).
*Caudal epidural technique
A caudal epidural technique at the sacrococcygeal or between the first two coccygeal vertebra has been described to help relieve urinary obstruction in cats. It may also be used to provide anesthesia/analgesia to the perineum, penis, urethra, colon, and anus by blocking the pudendal, pelvic, and caudal nerves without loss of motor function to the hind limbs. Preservative free lidocaine or bupivacaine (0.1–0.2 mL/kg) may be used depending on the duration of anesthesia/analgesia needed.
Chronic renal disease
The kidneys receive ~25% of the body’s cardiac output and thus are highly dependent on blood flow for proper function. Anesthetic agents decrease glomerular filtration rate (GFR) and renal blood flow, so general anesthesia should not be considered an innocuous process for patients with preexisting renal disease. When general anesthesia is necessary, patient preparation is crucial and steps should be taken to minimize the detrimental impact to remaining nephron function and the duration of general anesthesia should be minimized as much as possible.

Patients with chronic renal failure should be admitted the evening before general anesthesia and placed on IV fluids to ensure optimal hydration, correction of azotemia and electrolyte abnormalities before general anesthesia. Inhalant anesthetics produce profound vasodilation and a reduction in cardiac output that can be very detrimental in the face of hypovolemia. Anemia should be identified and blood products administered if needed (PCV < 20% in dogs, <18% in cats) since anemia will result in a failure to deliver adequate oxygen to tissues.

Maintain renal blood flow
Use drugs which have minimal cardiovascular effects (opioids, benzodiazepines, nitrous oxide) to reduce the amount of inhalant.

Monitor blood pressure
The method of BP monitoring is dependent on patient status and the duration of anesthesia. Critically ill patients having all but the shortest of procedures (<30 minutes) should be monitored via invasive/direct blood pressure monitoring. Less compromised patients or very short duration anesthesia may be managed with indirect monitors. Indirect, cuff-based monitors are easy to use in patients but are more prone to inaccuracy. Direct arterial pressure monitoring requires more skill and expense but gives continuous, accurate readings and permits easy access for blood gas analysis.

Avoid hypotension
Mean arterial blood pressure is considered an estimate of tissue perfusion pressure and should be maintained > 60 mm Hg in small animal patients to provide sufficient blood flow to vital organs; Mean arterial pressures > above 70 mm Hg are desirable if renal disease is present.

Fluid administration
Higher than normal rates to promote diuresis (10-20ml/kg/hr) – caution with colloids
- Ensure absence of severe cardiac disease, congestive heart failure, pulmonary edema, anuria
- Avoid drugs excreted unchanged by the kidney or that have active metabolites – ketamine in cats
- Avoid hypothermia and ensure adequate analgesia
- Caution with NSAIDS

Patients with endocrine disease
Diabetes mellitus
- Blood glucose regulation prior to anesthesia
- Stress/fasting disrupt BG regulation
- Schedule for early morning
- Check BG in a.m., give ½ insulin dose
- Monitor BG intra-op
- Quick recovery to return to normal eating patterns

Canine hypothyroidism
- ↓ metabolic rate
- ↓ drug metabolism
- Hypothermia
- Delayed recovery
- Obese
- Hypoventilation => IPPV
- Peripheral Neuropathy => laryngeal paralysis
- See BAOS pre-op & induction management
- Replacement and re-check thyroid levels pre-op

Hyperadrenocortisim – Cushing’s
- Weak muscles => IPPV
- Hypercoagulable
- ↓ wound healing
Patient’s with ocular disease

- Glaucoma, penetrating eye wounds, intra-ocular surgery (cataracts)
- Avoid increasing IOP - Ketamine, tiletamine, etomidate
- Avoid vomiting – Maropitant (Cerenia) 1 hour before opioid
- Avoid coughing/gagging at intubation
- Deep anesthesia, Lidocaine adjunct 1-2mg/kg IV dogs
- Avoid head down position/venous congestion
- Avoid hypercarbia & hypoxemia
- Requirements for Ocular Surgery:
  - Central eye position & Akinetic/still eye; no nystagmus or blinking
  - Neuromuscular Blockers, IPPV
  - Concurrent disease – Diabetes Mellitus – pre-operative Insulin dose, peri-op glucose monitoring
  - Oculocardiac Reflex: Traction or pressure on eye - bradycardia, AV block, asystole
  - Anti-cholinergics: Atropine, Glycopyrrolate
- Quiet, smooth recovery - adequate pre-medication, analgesia and sedation for recovery, eye protection

Geriatric patients

No single definition in small animals - >75-80% of lifespan, Dogs > 8, Cats > 12 years of age or by species, breed/size: small dogs (<9kg) – 9-13 years, medium dogs (9–23 kg) – 9-11.5 years, large dogs (23-41kg) – 7.5-10 years, giant dogs (>41kg) – 6-9 years and cats at any weight 8-10 years.

Cardiovascular changes

Decreased functional reserve, reduced cardiac output, decreased blood pressure and baroreceptor activity, increased circulation time and vagal tone and reduced ability to autoregulate blood flow. These changes significantly decrease cardiac reserve capacity. Geriatric patients also have an increased rate of heart disease, most commonly are degenerative valvular disease and conduction abnormalities leading to cardiac arrhythmias.

Respiratory

Geriatric patients lose thoracic compliance, alveolar elasticity and have atrophy of the intercostal muscles, leading to decreased arterial oxygen concentration. This, together with a decreased response to increased carbon dioxide and decreased oxygen puts them at increased risk for hypercapnia and hypoxemia.

Renal

Geriatric patients have a decrease in functional nephrons, decreased renal blood flow and GFR and difficulty retaining sodium and water. The renin–angiotensin system becomes less responsive, making geriatric patients less able to tolerate hypovolemia and electrolyte and acid–base disturbances.

Liver

Decreased functional liver capacity and decreased cardiac output leading to decrease liver blood flow may both affect the capacity to metabolize drugs. See anesthesia for liver disease above.

CNS

Loss of functional neurons, decreases MAC of inhalants, prone to dysphoria, emergence delirium – drugs used for treatment of cognitive dysfunction in dogs can have serious drug interactions with anesthetics and analgesics; it is important to obtain a complete and accurate medication history.

Orthopedic disease

Many geriatric patients have osteoarthritis, attention to patient positioning and proper padding is essential.

Proper patient preparation and monitoring are the best defense against anesthetic problems in the geriatric patients.

Preparation

- History (including current medications)
- Physical examination with cardiopulmonary auscultation
- Complete blood count
- Serum biochemical profile
- Electrocardiogram
- Urinalysis

Monitoring

Blood pressure, electrocardiogram, end-tidal capnography, pulse oximetry, temperature, IV access, goal directed IV fluid therapy

Judicious use of anti-cholinergics – potential danger of indiscriminate use of anti-cholinergics is that sinus tachycardia may result which will increase myocardial oxygen while at the same time decreasing myocardial blood and oxygen delivery and when coupled with decreased cardiac reserve may cause myocardial hypoxia, ischemia and arrhythmias. When needed to counter-act hypotension
associated with bradycardia from vagotonic drugs such as opioids, the dose should be titrated to effect at ¼ - ½ the normal mg/kg calculated dose.

Anesthesia for pediatric/neonatal patients

- Neonate: up to 4 weeks (dog/cat), Pediatric 4 weeks – 4 months
- Anesthetic concerns:
  - Liver: decreased metabolism, hypoglycemia, decreased albumin
  - CNS: increased BBB permeability, sympathetic nervous system immaturity
  - Cardiovascular: heart rate dependent for cardiac output
  - Resp: High metabolic rate/RR, easily fatigued
  - Poor thermoregulation

General strategies

- Drugs: lower doses - decreased protein binding, increased BBB permeability lead to exaggerated drug effect, drugs not metabolized by liver or reversible: Inhalants, opioids, benzodiazepines, propofol
- +/- Anticholinergics – maintain HR
- Monitor blood pressure and treat hypotension
- Monitor/supplement glucose in IV fluids
- Monitor temp/active patient warming