**Anesthesia and Analgesia for Patients with Comorbidities**

**NOTE: In this session we will cover SPECIFIC Comorbidities**

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Our veterinary patient population has changed as our medical skills have progressed and we have become capable of supporting patients with advanced disease and/or advancing age. All tranquilizers, induction drugs and inhalant drugs cause CNS depression and most cause some degree of dose-dependent physiologic dysfunction. In healthy patients, many of the physiologic effects of anesthetic drugs are tolerated or can be counteracted by routine measures such as administration of oxygen or

intravenous (IV) fluids. In compromised patients, these effects can be quite dangerous as they may magnify pre-existing disease-related physiologic dysfunction. Patient needs should be addressed in each of the 4 distinct and equally important periods: 1) preparation /premedication; 2) induction; 3) maintenance and 4) recovery. In healthy patients, many of the physiologic effects of anesthetic drugs are tolerated or can be counteracted by routine measures such as administration of oxygen or intravenous (IV) fluids. In compromised patients, these effects can be exacerbated, further contributing to the demise of the patient. Successful anesthesia in compromised patients is highly dependent on adequate patient stabilization, diligent patient support and monitoring, and the use of appropriate anesthetic drugs at appropriate **dosages.**

**Preparation for Anesthesia & Premedications**

*Stabilization of critical or challenging patients prior to sedation and/or anesthesia is imperative.* Both increasing American Society of Anesthesiologists (ASA) scores and increasing urgency of the procedure increase risk of anesthetic death (Brodbelt 2009). Most critical patients have disease or conditions that cause at least some degree of cardiovascular and/or respiratory compromise, potentially resulting in decreased tissue oxygen delivery. Most anesthetics also cause at least some degree of cardiovascular and respiratory compromise, also potentially resulting in decreased tissue oxygen delivery. One of the critical roles of the anesthetist is to support oxygen delivery by promoting normal function in both the cardiovascular and respiratory systems.

*Analgesia should be part of stabilization.* Pain creates a tremendous sympatho-adrenal stress response and can contribute to **morbidity** and perhaps even **mortality**. Relief of pain can provide hemodynamic and respiratory stabilization, along with many other positive benefits. If relieving pain does **not** provide stabilization, the veterinarian will know to rapidly continue diagnostics as something other than pain is the main cause of the patient’s condition. Safe, reversible drugs like the opioids are excellent choices for most challenging patients. When possible, decreased fear/anxiety/stress (FAS) should also be part of stabilization as FAS can often exacerbate the negative components of underlying disease through the FAS-induced stress response and can increase pain intensity. Drugs like gabapentin, trazodone and alfaxalone are generally safe and often administered to hospitalized patients if not administered before the patient left home.

*Sedatives / tranquilizers:*Although not intuitive that critical patients need premedicants, the use of premedicants will decrease the dose of induction and maintenance anesthetic drugs. Since adverse effects are dose dependent, decreasing the dosages will improve anesthetic safety.

* *Opioids - morphine, fentanyl, methadone, butorphanol, buprenorphine:* Advantages: Provide moderate to profound analgesia, minimal to no cardiovascular effects, minimal respiratory effects, allow a decrease in dosage of maintenance drugs, reversible, many are inexpensive, provide sedation, versatile (can be administered PO, IM, IV, SQ, in the epidural space, in the intra-articular space, etc...). Disadvantages*:* cause vomiting (administer maropitant), relatively short duration of action when compared to the duration of most pain (administer as an infusion).
* *Benzodiazepines – Diazepam, midazolam:* Advantages: Wide safety margin, minimal to no cardiovascular or respiratory effects, reversible. Excellent choice for critical patients – either as a premed or a part of induction. Disadvantages: Minimal to no sedation when used alone in healthy patients and can cause paradoxical excitement, especially in stressed or fractious patients, no analgesia.
* *Alfaxalone* – Advantages: Can provide dose-dependent light to moderate sedation and can be administered IM or IV. Disadvantages: Some dose-dependent cardiovascular & respiratory effects – VERY minor at the sedative dose, volume limited to small patients, can cause ‘rough’ recoveries – unlikely at the sedative dose, no analgesia.
* *Acepromazine –* not commonly used in compromised/challenging patients (the exception is patients with upper airway compromise that need long-term sedation and some patients with cardiovascular disease that would benefit from a reduction in afterload). Disadvantages: not reversible, causes vasodilation which could contribute to hypotension in compromised patients.
* *Alpha-2 agonists –* not commonly used in compromised patients because they rarely need profound sedation but appropriate in stable emergency patients that need sedation/analgesia. Advantages: Provide both sedation and analgesia, effects are reversible. Disadvantages: Causes increased cardiac work.

*Other drugs:**Maropitant* is recommended, both for its anti-emetic effects and its potential for contributing to analgesia. Vomiting itself, with the intense contraction of abdominal muscles, is painful. This can greatly exacerbate the pain level in patients with pre-existing abdominal pain. Disease-specific drugs might also be necessary, as an example, a lidocaine infusion could be necessary in patients with ventricular tachyarrhythmias.

**Induction**

REMINDER:All tranquilizers, induction drugs and inhalant drugs cause CNS depression and most cause some degree of **dose-dependent** respiratory and cardiovascular dysfunction. **All drugs should be dosed ‘to effect’**.

* *Propofol:* Advantages: rapid induction and recovery, easy to titrate ‘to effect’, multiple routes of clearance from the body, good muscle relaxation. Disadvantages: Causes mild to moderate dose-dependent respiratory and cardiovascular depression
* *Alfaxalone:* Advantages: rapid induction and recovery, easy to titrate ‘to effect’. Disadvantages: Causes mild to moderate dose-dependent respiratory and cardiovascular depression. Can cause rough recoveries, uncommon in appropriately sedated patients.
* *Ketamine:* Advantages: inexpensive, can be administered IM, mild respiratory depression, no cardiovascular depression in heart-healthy patients. Disadvantages: can cause cardiovascular depression in patients with cardiovascular compromise, can cause muscle rigidity.
* *Etomidate*: Advantages: no cardiovascular effects. Disadvantages*:* expensive, poor muscle relaxation, vocalization, maybe not appropriate in septic patients due to adrenocortical suppression.
* ***Inhalant induction is NOT appropriate*** *for almost all dogs and cats****.*** The dose of the inhalant is entirely too high when used alone (side effects of inhalants are dose-dependent) and the induction will be stressful and will be prolonged. Furthermore, use of inhalants alone for induction and maintenance increases the risk of anesthesia-related death (Brodbelt 2009).

**Maintenance**

Inhalant anesthesia is generally the safest and most effective way to maintain anesthesia that will last 30 minutes or more. However, inhalant anesthetic drugs should never be used as the sole anesthetic drug since inhalants can cause significant hypotension and hypoventilation. Our goal should always be to keep the vaporizer as low as possible. Often, *analgesia* must be provided in order to minimize *anesthesia* drug doses. *Advantages*: easy to administer, relatively inexpensive, are eliminated with minimal metabolism. *Disadvantages*: DOSE DEPENDENT contribution to hypoventilation, hypotension and hypothermia. MONITOR, MONITOR, MONITOR. **NOTE:** The advantages and disadvantages of the inhalant drugs are class effects and apply to all inhalants. However, sevoflurane has an advantage in critical patients since it is more easily dosed ‘to effect’ because of its lower solubility coefficient.

*Analgesic Drugs & Techniques*

Maintenance of anesthesia is much easier and safer if analgesia is provided prior to the painful stimulus. Most anesthetic drugs, including the anesthetic gases, block the brain's perception that pain has occurred but don't actually block pain. If pain is severe enough, the brain can still respond and make the patient appear to be inadequately anesthetized. This usually leads to an increased inhalant dose and the brain ceases to respond, but the patient is now too deeply anesthetized and can be at a very dangerous physiologic plane. A more appropriate response would be to block the pain and maintain anesthesia at a light, safe depth. The advantage to all of the drugs and techniques listed below is that they are anesthetic-sparing, meaning that they allow a decrease in the anesthetic dose necessary to maintain unconsciousness.

* *Opioids:* Advantages: provide moderate to profound analgesia, cause minimal cardiovascular or respiratory effects, are reversible. Disadvantages: Previously discussed opioid-meditated adverse effects.
* *Local anesthetic drugs & locoregional techniques:* Advantages: Inexpensive, easy to administer, very effective. Drugs block the pain impulse from getting to the dorsal horn of the spinal cord and thus decrease the incidence of central sensitization. This results in pain that is much lower, not only during the block, but even beyond the expected duration of the drug itself. Local blockade also decreases the likelihood that chronic pain will develop secondary to acute pain. Disadvantages: Relatively short duration of action when compared to the duration of pain, except for NOCITA®.

**NOTE:** Local anesthetics are underutilized, yet they are easy to use, inexpensive and highly effective.

* *Constant rate infusions (CRIs):* Advantages: EASY, inexpensive, effective, many drug choices (opioids, lidocaine, ketamine, alpha-2 agonists and combinations). Disadvantages: Almost none because of the low dose delivered but side effects from any drug could always occur. There is a very useful open-access CRI calculator at IVAPM.org under the ‘professionals’ tab.

*Monitoring & Support*

*Monitoring:* Anesthesia causes changes in all organ systems but the changes in the CNS, cardiovascular and respiratory systems are the most immediately life-threatening so monitoring and support is focused on these systems. Also, support of these systems will provide support for other systems by providing adequate oxygen delivery to the organs/tissues of that system. Don’t forget the basics: mucous color, capillary refill time, jaw tone, eye position, etc… Utilize SpO2 (pulse oximeter) and end-tidal CO2 to assess respiratory function. Utilize ECG and **blood pressure** to assess cardiovascular function. Measurement of blood pressure is IMPERATIVE in critical patients.

*Cardiovascular support* includes use of IV fluids & positive inotropic and antiarrhythmic drugs.

* + *IV fluids* should be used, as needed, to rehydrate the patient and replace ongoing losses. Do not overhydrate – excessive administration of fluids can cause edema.
  + Many critical patients would benefit from the use of *colloids* in addition to crystalloids. Voluven (Vetstarch) is commonly used and the total dose in the dog is <50 ml/kg in a 24-hour period. Cats should generally receive <30-40 ml/kg in a 24-hour period.
  + If patients have hemorrhaged, if severe hemorrhage is expected intraoperatively or if the patient is anemic (PCV < 18-20%), collect blood for a *blood transfusion* prior to anesthesia.
  + Oxygen bound to hemoglobin is the main source of oxygen delivered to the tissues. If the patient is hypoproteinemic (albumin <2 g/dl), administer *plasma* prior to anesthesia.
  + If the patient is hypotensive:
    - * 1. If anesthetized, TURN DOWN THE VAPORIZER.
        2. Give boluses of fluids (5-10 ml/kg rapidly) or colloids (5 ml/kg rapidly).
        3. Consider positive inotropes like dopamine or dobutamine. Dose of each is 1-10 microg/kg/min (up to 15 with dopamine). Patients with conditions that cause decreased cardiac contractility (eg, sepsis, etc…) are likely to need positive inotropes for effective blood pressure support.
        4. If these measures are not effective or if the patient is severely vasodilated, vasopressors (eg, norepinephrine, vasopressin) may be necessary.

*Respiratory support* includes oxygen delivery and maintenance of ventilation.

* Oxygen is inexpensive and profoundly beneficial in many critical patients. When in doubt, administer oxygen!
* If the patient is having any trouble ventilating (head trauma, thoracic trauma, profound CNS depression, impingement on thorax by GI contents, etc…) ADMINISTER OXYGEN.
* If the ventilatory depression is moderate, consider intubation. If severe, INTUBATE.
* Obviously most anesthetized patients would be intubated. Intubate rapidly and quickly inflate the endotracheal tube cuff to an appropriate pressure.
* Many compromised patients will require assisted ventilation because the respiratory drive in-response to hypoxemia and/or hypercarbia may be impaired and/or the patient may not physically be able to ventilate normally (muscle weakness, thoracic trauma, electrolyte imbalance, GI distension, etc…). Assisted ventilation: 2 breaths/min to 15-20 cmH2O on the manometer. Controlled ventilation: 6-10 breaths/min to 15-20 cm H2O on the manometer. If a ventilator is available, set tidal volume to 15-20 ml/kg. MONITOR End-tidal CO2 – normal is 35-55 mmHg in the anesthetized patient (35-45 mmHg in conscious patients); Do not over ventilate.

**Recovery**

Unfortunately, most anesthetic deaths occur in recovery and the majority of those occur within the first 3 hours of recovery (Brodbelt 2009). The cause is likely a decrease in anesthetist vigilance in recovery. Support and monitoring should be continued into the recovery phase, especially for challenging patients. Analgesia should also be re-addressed. If effective analgesia is utilized pre- and intra-operatively, the analgesic needs of the patient may be minimal. Opioid boluses and constant rate infusions are excellent choices during the recovery period. NSAIDs may be appropriate depending on the disease. The drugs diminish pain at its source (inflammation) making them very powerful. Administer NSAIDS if not contra-indicated.

**Specific Comorbidities**

***Patient with cardiovascular disease/dysfunction***

The cardiovascular system includes the heart, blood vessels and blood/plasma. Thus, cardiovascular disease or dysfunction encompasses `conditions ranging from decreased cardiac contractility to arrhythmias to anemia. Diseases that are not necessarily cardiovascular diseases but which affect the cardiovascular system (eg, hyperthyroidism, sepsis, etc…) should also be considered in this category when making anesthetic plans for patients with those diseases. In addition, anesthetic drugs (e.g. inhalants), perioperative manipulations (e.g. recumbency, positive pressure ventilation) and surgical complications (e.g. uncontrolled pain, hemorrhage) can exacerbate cardiovascular dysfunction – and can even cause cardiovascular changes that mimic cardiovascular disease. Thus, a fair number of anesthetized patients may need cardiovascular support, even in the absence of cardiovascular disease. Because of the vast number of diseases/conditions that affect the cardiovascular system, one anesthetic protocol may not be appropriate for all patients in this category, but an understanding of cardiovascular physiology and the cardiovascular effects of the anesthetic drugs will promote appropriate anesthetic/analgesic protocol selection. In addition to appropriate dose/drug selection, diligent patient monitoring and support are crucial.

*Physiology of the Cardiovascular System & Anesthesia Goals*

The ultimate goal of the cardiovascular system is to work in concert with the respiratory system to provide adequate oxygen delivery (DO2) to the working cells. The cardiovascular system’s role in this goal is achieved through support of cardiac output, which is a product of heart rate (HR) and stroke volume (SV). Stroke volume is determined by preload, afterload and myocardial contractility (inotropy). In all patients, the focus should be on support of normal physiologic processes in order to optimize tissue oxygen delivery.

*Preanesthetic preparation*

STABILIZE the patient! Long term if possible (eg, send home on drugs that improve cardiac function) or short term in not possible (eg, administer fast-acting anti-arrhythmic drugs to treat arrhythmias). However, drugs that may decrease blood pressure (eg, beta-blockers, calcium channel blockers) should be withheld on the morning of anesthesia. Once ready for anesthesia, preoxygenate to decrease the likelihood of decreased oxygen delivery. Start on JUDICIOUS rate of IV fluids if the patient is hypovolemic at a suggested rate of 2-5 ml/kg/hr depending on the disease and patient. Monitors should be connected to the patient prior to induction. Numerous physiologic changes can happen at induction. Warming should start now since body temperature starts to drop at induction. Hypothermia causes adverse cardiovascular effects like decreased myocardial contractility, arrhythmias and bradycardia.

Excitement, struggling and fear cause tachycardia and increased peripheral resistance, arterial blood pressure, cardiac work and cardiac oxygen consumption. These changes are generally well-tolerated in patients with a healthy cardiovascular system but are extremely dangerous in a patient with cardiovascular disease, possibly resulting in decompensation and cardiac failure. Pain causes the same sympathetic response as excitement, struggling and fear. Therefore, all of these stressors must be avoided in patients with cardiovascular disease and calm handling, along with the administration of a low-dose of a tranquilizer and preemptive analgesic drug, are crucial. **Opioids** have minimal adverse impact on the cardiovascular system and are the drug class of choice for patients with cardiovascular disease.

*Disease-specific comments*  
Hypertrophic cardiomyopathy (mostly cats) and dilatative cardiomyopathy/regurgitant valvular disease (mostly dogs) have different physiologic impacts and, thus, different anesthetic concerns.

* Hypertrophic cardiomyopathy: In this disease the cardiac muscle is overworked so avoid drugs that further increase cardiac work (through increased rate or contractility). Increased work will increase myocardial oxygen need - but the hypertrophic cardiac muscle is generally not matched by increased vasculature, thus oxygen delivery can be decreased. This could include ketamine (no concern at CRI dose), anticholinergics, etc… Inotropes are somewhat controversial as some clinicians feel that they are contraindicated but research shows they are safe and effective when used at low dosages. Both dopamine and phenylephrine improved blood pressure but only dopamine improved cardiac output. (Wiese et al 2012). Slower heart rates (allow time for ventricles to fill) and vasoconstriction (increased afterload which can decrease left ventricular outflow obstruction) is often beneficial in these patients, thus alpha-2 agonists may be considered (Lamont et al. 2002) if needed to adequately control the cat (Lamont et al. 2002).
* Dilatative cardiomyopathy/regurgitant valvular disease: In this disease the cardiac muscle is inefficient for appropriate ejection of adequate blood volume or the dysfunctional valve promotes inadequate ejection blood volume. In these patients, the goal is myocardial contractility support and decreased ejection resistance (ie, vasodilation). Low-dose acepromazine may be beneficial. Ketamine can be very useful as it increases both rate and contractility through SNS stimulation. Increased heart rate may be necessary so anticholinergics may be indicated. Inotropes are beneficial.

*Preanesthetic drugs: Specific cardiovascular (CV) effects/concerns*

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| --- | --- |
| Opioids | Minimal CV effects – no change in contractility, some vagally-mediated bradycardia; ‘cardio sparing’ |
| Alpha-2 Agonists | Increased cardiac work from vasoconstriction. Generally contraindicated, but may be beneficial in some diseases2 |
| Acepromazine | Low dose = decreased afterload but high dose = hypotension |
| Benzodiazepines | No CV effects; Cardio sparing; Not very sedating – combine with an opioid |

*Preoxygenate:* Decreases the likelihood of decreased oxygen delivery. Preoxygenation for only 3 minutes increases the time do desaturation (SpO2<90%) approximately 1 minute to 6 minutes.

*Induction drugs: Specific cardiovascular effects/concerns*

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| --- | --- |
| Propofol | Mild to moderate dose-dependent cardiovascular (CV) depression.3 Administer premeds to decrease the dose of induction drug required to produce anesthesia. |
| Alfaxalone | Mild to moderate dose-dependent CV depression.3 Administer premeds to decrease the dose of induction drug required to produce anesthesia. |
| Ketamine | Increased HR & contractility through SNS in healthy hearts but direct myocardial depression in uncompensated heart failure; MAY exacerbate tachyarrhythmias? CRI dose is not concerning. |
| Telazol | Probably same effects as ketamine. |
| Etomidate | Minimal to no impact on CV system. Drug of choice for profound disease. |
| Inhalants | Moderate to profound dose-dependent CV depression. Don’t induce with inhalants! |

**TIP:** Administer a low-dose benzodiazepine or fentanyl bolus just before induction to decrease the dose of induction drug.

*Maintenance*Inhalants can cause hypotension since they cause both dose-dependent decreased cardiac contractility and vasodilation. KEEP THE DOSE LOW. Add **analgesia**! Opioid boluses, local blocks and infusions of opioids, lidocaine and/or ketamine are all good options. Ketamine at the infusion dose used for infusions is unlikely to cause adverse effects and is commonly used for patients with cardiac disease.

*Monitoring:* Blood pressure, ECG, SpO2 and ETCO2. Need to monitor both the respiratory and cardiovascular systems to insure oxygen delivery.

*Support:* Maintain MAP >8 kPa (>60 mmHg). The steps to promote normotension are:

* DECREASE the INHALANT DOSE
* **Support cardiovascular function with inotropes** 
  + Dopamine, dobutamine
  + In a patient with myocardial disease/dysfunction, decreased contractility is the most likely cause of hypotension so start inotropes early.
* Check the heart rate – fix if necessary
* JUDICIOUS use of IV fluids if hypovolemia is present
  + 2-10 ml/kg/hr intra-op
  + Balanced electrolyte solution
  + Blood or plasma if necessary
* USE COLLOIDS – eg, hetastarch
* Treat arrhythmias appropriately
  + Arrhythmias can affect cardiac output

*Recovery*

DON’T STOP MONITORING and SUPPORT

The more compromised the patient, the longer monitoring and support should continue.

**Keep warm**. Shivering can increase oxygen consumption by up to 200%, which may not be met by oxygen delivery in patients with cardiovascular disease.

Readdress analgesia; USE TRANQUILIZERS if necessary – DON’T allow a rough recovery! The stress physiologic stress can exacerbate cardiac dysfunction. NSAIDs if appropriate: No negative CV effects, Anti-inflammatory

*Sample Protocol**Preanesthesia*: Physical exam, complete blood work, thoracic radiographs, ECG. Consider maropitant.  
*Premedication:* Opioid IM (unless catheter already placed); preoxygenate if possible  
 If the patient is really sick, skip the IM premed and use fentanyl IV at induction.  
*Induction:* 0.2 mg/kg midazolam or diazepam IV followed by propofol, alfaxalone or etomidate  
 SLOWLY to effect. Fentanyl (2-5 microg/kg) can be substituted for or added to the  
 benzodiazepine.  
*Maintenance:* LOW DOSE inhalant; use CRIs (especially fentanyl or other opioid) & local blocks  
 Monitor ECG and blood pressure; use dopamine CRI for hypotension; use active warming.  
*Recovery:* Keep monitoring until patient is fully awake; Provide analgesia with opioids  
 Administer NSAIDs if there are no contraindications.

***Patient with respiratory disease/dysfunction***

The respiratory system includes all of the structures from the nares to the alveoli, and anything that impacts gas exchange. This includes brachycephalic airway syndrome, laryngeal paralysis, collapsing trachea, pneumonia, pneumothorax (and any other foreign material in the thorax), diaphragmatic hernia, etc… Although not truly airway disease, anything obstructing normal movement of the thorax also decreases gas exchange – like fractured ribs, or GI distension affecting diaphragmatic movement. Faulty equipment can also affect gas exchange – when a patient is connected to an anesthesia machine, that machine and the breathing system (nonrebreathing vs rebreathing) become part of the respiratory system!

*Physiology of the Respiratory System*

The ultimate goal of the respiratory system is to provide adequate gas exchange, which means support of adequate oxygen delivery (DO2) to the tissues by maintenance of adequate arterial oxygen content (CaO2) and support of normal acid-base balance by the elimination of CO2. Respiratory diseases generally impair gas exchange by causing hypoventilation via decreased tidal volume or respiratory rate, decreased alveolar surface area for diffusion of gases (eg, lung consolidation or tumors), and/or impaired diffusion of gases at alveoli. Many anesthetic drugs also impair gas exchange by causing hypoventilation via decreased tidal volume or respiratory rate, decreased alveolar surface area for diffusion of gases (eg, atelectasis), decreased FiO2 (human error or equipment malfunction!) and decreased elimination of CO2 (human error or equipment malfunction!).

*Anesthesia Goals for Patients with Respiratory Disease*

This is more of a management issue than a drug issue, thus most anesthetic drugs are acceptable.

*Preanesthetic preparation*

* Have airway equipment ready (laryngoscope, varied tube sizes, etc…)
* Have oxygen ready, maybe have ventilator (machine or person) ready (lower airway)
* KEEP THE PATIENT CALM!
  + Administration of sedatives and analgesic drugs is critical! Stress, fear and pain can cause increased respiratory drive, which can cause increased negative pressure in the airway, which can exacerbate airway collapse.
  + Remember that any narrowing of the area causes a major impact on air flow. Airway resistance (***R*aw**) is governed by Poiseuille’s law: ***R*aw = 8 h L/r4**. This means that *R*aw is directly related to the air viscosity (h) & to the length (L) of the tube, and inversely related to its radius by the 4th power. Thus, halving the radius of the airway increases the resistance sixteen-fold. This is important during changes in radius due to pathology – and during selection of endotracheal (ET) tubes – choose the biggest tube you can.

*Preanesthetic drugs: Specific respiratory effects/concerns*

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| Opioids | Some, usually insignificant, respiratory depression |
| Alpha-2 Agonists | Profound sedation, MONITOR! |
| Acepromazine | Minimal to no respiratory changes; |
| Benzodiazepines | Minimal to no respiratory changes |

*Preoxygenate.* Decreases the likelihood of decreased oxygen delivery. Preoxygenation for only 3 minutes increases the time do desaturation (SpO2<90%) approximately 1 minute to 6 minutes.

*Induction drugs: Specific respiratory effects/concerns*

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| Propofol | Dose-dependent respiratory depression |
| Alfaxalone | Dose-dependent respiratory depression |
| Ketamine | Minimal to no change in respiratory function. CRI dose is not concerning. |
| Telazol | Minimal to no change in respiratory function |
| Etomidate | Minimal to no change in respiratory function |

*Intubation: CRITICAL PART OF SUCCESS*

* Use a laryngoscope – especially if the problem is in the upper airway
* Use lidocaine on arytenoids (cats)
* Use a stylet if you need one
* Be ready for a tracheostomy if the problem is upper airway (not likely to need one but good to be prepared!)

*Maintenance*

Inhalants cause hypoventilation. KEEP DOSE LOW. Add **analgesia**. The impact of inhalants may be more obvious in recovery – if the patient has been too deep for too long, complications in recovery (when the patient is no longer on 100% oxygen and maybe no longer intubated) may be more profound. To prevent this complication, keep the inhalant dose low and discontinue inhalants as early as possible. Analgesia should be based on the expected pain level of the procedure and the patient’s health. Consider local anesthetics and NSAIDs since they are non-sedating. Excess sedation in recovery could impair ventilation. HOWEVER, opioids should be used if pain is moderate-to-severe.

*Monitors:* Need to monitor both cardiovascular and respiratory function in order to support DO2. The pulse oximeter is a monitor of oxygenation, not ventilation, and true respiratory function should be monitored using end-tidal CO2 (ETCO2) – or blood gases.

*Support:* Keep SpO2>95% and ETCO2 between 30-50mmHg

* Provide ventilation if necessary – can be machine or person
  + 6-15 breaths/min
  + 10-15 (up to 20) cmH20 PIP
  + 10-15 ml/kg tidal volume
* Maintain cardiac output/blood pressure to optimize oxygen delivery
  + However, positive pressure ventilation (IPPV) can decrease cardiac output secondary to compression of blood vessels in the thorax.

*Recovery*

Can be the most critical part Especially for patients with upper airway disease

DON’T STOP MONITORING and SUPPORT

The more compromised the patient, the longer monitoring and support should continue

Readdress analgesia; USE TRANQUILIZERS if necessary – DON’T ALLOW EXCITEMENT

*Upper Airway (Brachycephalics, patients with laryngeal paralysis, etc):*

* **Most critical part of the entire anesthetic period.**
* USE OXYGEN
* Make sure the patient is fully awake but calm and that pain is alleviated prior to extubation.
* Leave ET tube in as long as possible.
* Stretch out neck, pull out tongue to help open up airway
* Be prepared to re-anesthetize and reintubate if the patient can’t breathe.
  + Have propofol and laryngoscope ready.
* Consider a tracheotomy if the upper airway dysfunction is severe.
* Administer steroids if upper airway inflammation is moderate to severe.

*Lower Airway (Patients with pneumonia, lung contusions, etc):*

* Keep patient on oxygen as long as needed
  + Can monitor with pulse oximeter
* Make sure the patient is fully awake but calm and that pain is alleviated prior to extubation.
* Leave ET tube in as long as possible.

Sample Protocols  
*Upper Airway*

*Preanesthesia*: Physical exam, complete blood work, ECG  
 opioid + 0.02 mg/kg acepromazine; PREOXYGENATE  
 maropitant to decrease the incidence of vomiting with potential for aspiration pneumonia  
*Induction:* 0.2 mg/kg midazolam or diazepam IV followed by propofol or alfaxalone to effect  
 Ketamine + benzodiazepine is also an appropriate choice  
 INTUBATE!!! Do upper airway exam as intubating  
*Maintenance:* LOW DOSE inhalant; use CRIs and local blocks – aggressive analgesia in the maintenance phase means a more comfortable patient in the recovery phase.  
 Monitor ECG and blood pressure; support as needed  
*Recovery:* KEEP PATIENT INTUBATED AS LONG AS POSSIBLE;   
 Keep monitoring until patient is fully awake; Provide analgesia with opioids if needed.  
 Use NSAIDs if there is no contraindication

*Lower Airway*

*Preanesthesia*: Physical exam, complete blood work, thoracic radiographs, ECG  
 opioid IM +/- acepromazine or dexmedetomidine; PREOXYGENATE  
*Induction:* 0.2 mg/kg midazolam or diazepam IV followed by propofol or alfaxalone to effect  
 Ketamine + benzodiazepine also good choice  
 INTUBATE and start assisting (or controlling) ventilation right away  
*Maintenance:* LOW DOSE inhalant; use CRIs and local blocks; Monitor ECG and blood pressure; assist  
 ventilation; support BP with fluids, colloids and dopamine if needed  
*Recovery:* KEEP PATIENT ON OXYGEN AS LONG AS POSSIBLE. Use the pulse oximeter to determine when the supplemental oxygen can be discontinued.  
 Keep monitoring until patient is fully awake; Provide analgesia with opioids  
 Use NSAIDs if there is no contraindication

***Patient with Chronic Kidney Disease***

1. *Main preanesthetic concerns* 
   1. Biochemistry/CBC: Increased BUN, creatinine (Cr), hypokalemia or hyperkalemia, hypoproteinemia, hyperphosphatemia, anemia, acid-base imbalance (usually metabolic acidosis)
   2. Physiologic/physical exam: Dehydration/hypovolemia, poor body condition, hypertension
   3. Recommended additional diagnostics: Urine protein:creatinine ratio, systolic blood pressure
2. *Stabilization: Prior to scheduling anesthesia*
   1. **Stabilization based on IRIS score**. See IRIS staging guidelines: <http://www.iris-kidney.com/guidelines/staging.html> and <http://www.iris-kidney.com/pdf/IRIS_Pocket_Guide_to_CKD.pdf> . Stabilization may include anti-hypertensive medications, renal diet, anti-emetics, appetite support, etc...
   2. Repeat biochemistry/CBC at 1-4 weeks, depending on the severity of the disease/urgency of anesthesia.
   3. Unless the procedure requiring anesthesia is urgent, do not anesthetize the patient until the disease is controlled as defined by BUN/Cr improved or stable, proteinuria resolving, systolic arterial pressure <140 mmHg.
3. *Stabilization: Day of anesthesia*
   1. Repeat all biochemical and physiologic tests and correct abnormalities that are correctable. Main concerns and corrective actions:
      1. Dehydration/Hypovolemia/Decreased renal perfusion: Start on IV fluids prior to anesthesia as determined by IRIS stage. Minimum 2 hours, maximum 24 hours.
      2. Hypokalemia: Add potassium to the IV fluids if K+<3.5 mEq/L. Administer at a rate not to exceed 0.5 mEq/kg/hr until K+ is 4-5 mEq/L.
      3. Hyperkalemia: Evaluate ECG to determine the impact of hyperkalemia. Dilute K+ using IV fluids, consider treatment with calcium and/or with insulin/dextrose. Do not anesthetize if K+ >6 mEq/L.
      4. Metabolic acidosis: normally resolved with appropriate IV fluid administration.
      5. Anemia and/or hypoproteinemia: Consider a pre-anesthesia blood transfusion if PCV is <20% and administration of plasma if albumin is <2 g/dl or total protein <3.5 g/dl.
   2. Repeat specific tests prior to induction to ensure abnormalities are resolved/resolving.
   3. Anesthetize when all correctable abnormalities are near to or within normal limits.
4. *Preanesthetic drugs: Specific renal effects/concerns*

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| Opioids | Nothing specific – respiratory depression could lead to decreased oxygen delivery but unlikely. |
| Alpha-2 Agonists | Somewhat ‘controversial’ – concern that vasoconstriction could decrease renal blood flow but flow to internal organs is generally preserved by vasoconstriction in peripheral vessels. Reversible. |
| Acepromazine | Nothing specific – vasodilation could decrease blood pressure and thus renal oxygen delivery. |
| Benzodiazepines | No effects/concerns. |

1. *Preoxygenate.* Decreases the likelihood of decreased oxygen delivery. Preoxygenation for only 3 minutes increases the time do desaturation (SpO2<90%) approximately 1 minute to 6 minutes.
2. *Induction drugs: Specific renal effects/concerns*

|  |  |
| --- | --- |
| Propofol | Nothing specific. Could cause decreased oxygen delivery via cardiovascular and respiratory depression. Dose to effect. |
| Alfaxalone | Same as propofol. |
| Ketamine | Supports normal cardiovascular and respiratory function. Cleared in-part unchanged in the kidney in cats so could continue to circulate as an active drug and delay recovery. Unlikely to be a major clinical impact. CRI dose is not concerning. |
| Telazol | Probably same as ketamine. |
| Etomidate | No specific concerns. |

1. *Maintenance phase of anesthesia*
   1. *Physiologic Monitoring:* Blood pressure, ECG, SpO2 and ETCO2. Need to monitor both the respiratory and cardiovascular systems to **insure oxygen delivery**.
   2. *Physiologic Support:* Maintain MAP >60-70 mmHg.
   3. *Intra-anesthesia* biochemistry/CBC monitoring: No specific tests unless pre-existing abnormalities were not completely stabilized or are likely to change, examples for renal disease would be electrolytes and acid-base imbalance.
2. *Recovery/discharge:* 
   1. Physiologic monitoring/support: Continue monitoring and supporting cardiovascular and respiratory function along with body temperature as indicated by the patient’s physiologic status and concerns that arose intraoperatively.
   2. Biochemistry/CBC monitoring: Recheck any correctable abnormalities that were present pre-anesthesia or that developed during anesthesia, like changes in electrolytes or acid-base. BUN and CRE can be measured but may be temporarily increased after anesthesia.
3. *Post-Procedure* Recheck renal values, urinalysis, electrolytes and acid-base status in 3-14 days depending on severity of disease and patient health status.

***Hepatic Disease/Insufficiency***

1. *Main preanesthetic concerns*
   1. Biochemistry concerns: Elevated AST, ALT; hypoalbuminemia, hypokalemia, hypoglycemia, coagulopathy
   2. Potential additional diagnostics: Bile acids, clotting times, abdominal ultrasound
   3. Physiologic/physical exam concerns: hypovolemia (from vomiting/diarrhea), hypotension, ascites
2. *Stabilization: Prior to scheduling anesthesia*
   1. Stabilization according to internal medicine guidelines (reference). Stabilization may include liver diet, appetite support, anti-emetics, liver support etc...
   2. Repeat biochemistry/CBC at 1-4 weeks, depending on the severity of the disease/urgency of anesthesia.
   3. Unless the procedure requiring anesthesia is urgent, do not anesthetize the patient until the disease is controlled as defined by ALT/ALP that is improved or stable (they may not decrease) in a patient has no/minimal clinical signs of hepatic disease (eg, no vomiting, jaundice, etc..).
3. *Stabilization: Day of anesthesia*
   1. Repeat all biochemical and physiologic assessments and correct abnormalities that are correctable. Main concerns and corrective actions:
      1. Dehydration/Hypovolemia/Decreased hepatic perfusion: Start on IV fluids prior to anesthesia for approximately 2-4 hours. Choose a fluid with acetate as the buffer as lactate requires hepatic metabolism to become a buffer.
      2. Hypokalemia: Add potassium to the IV fluids if K+<3.5 mEq/L. Administer at a rate not to exceed 0.5 mEq/kg/hr until K+ is >3.5 mEq/L.
      3. Hypoalbuminemia/hypoproteinemia: Administer plasma if albumin is <2 g/dl or total protein <3.5 g/dl.
      4. Hypoglycemia: Administer IV fluids with dextrose if glucose <100-120 mg/dL.
4. Coagulopathy: Consider fresh-frozen plasma or more advanced therapy like human albumin.
5. Repeat specific tests prior to induction to ensure abnormalities are resolved/resolving.
6. Anesthetize when all correctable abnormalities are near to or within normal limits.

*Intra-anesthesia* biochemistry/CBC monitoring: No specific tests unless pre-existing abnormalities were not completely stabilized or are likely to change, analytes of interest for hepatic disease are albumin and glucose.

1. *Preanesthetic drugs: Specific hepatic effects/concerns*

|  |  |
| --- | --- |
| Opioids | Nothing specific – respiratory depression could lead to decreased oxygen delivery but unlikely. Reversal so does not require hepatic metabolism. |
| Alpha-2 Agonists | Somewhat ‘controversial’ – concern that vasoconstriction could decrease renal blood flow but flow to internal organs is generally preserved by vasoconstriction in peripheral vessels. Reversible so does not require hepatic metabolism |
| Acepromazine | Requires hepatic metabolism and is not reversible so can lead to prolonged recoveries. Generally avoided unless disease is mild. |
| Benzodiazepines | Reversible so does not require hepatic metabolism. Hepatic failure has only been reported with REPEATED ORAL doses of diazepam in cats. Benzodiazepines are unlikely to cause hepatic encephalopathy but might worsen pre-existing hepatic encephalopathy. |

1. *Preoxygenate.* Decreases the likelihood of decreased oxygen delivery. Preoxygenation for only 3 minutes increases the time do desaturation (SpO2<90%) approximately 1 minute to 6 minutes.
2. *Induction drugs: Specific hepatic effects/concerns*

|  |  |
| --- | --- |
| Propofol | Nothing specific. Could cause decreased oxygen delivery via cardiovascular and respiratory depression. Dose to effect. |
| Alfaxalone | Same as propofol. |
| Ketamine | Supports normal cardiovascular and respiratory function. Does require hepatic metabolism, especially in dogs. Unlikely to be a major clinical impact. CRI dose is not concerning. |
| Telazol | Probably same as ketamine. |
| Etomidate | No specific concerns. |

1. *Maintenance phase of anesthesia*
   1. *Physiologic Monitoring:* Blood pressure, ECG, SpO2 and ETCO2. Need to monitor both the respiratory and cardiovascular systems to **insure oxygen delivery**.
   2. *Physiologic Support:* Maintain MAP >60-70 mmHg.
   3. *Intra-anesthesia* biochemistry/CBC monitoring: No specific tests unless pre-existing abnormalities were not completely stabilized or are likely to change, examples for hepatic disease would be albumin and glucose.
2. *Recovery/discharge physiologic monitoring:* 
   1. Physiologic monitoring/support*:* Continue monitoring and supporting cardiovascular and respiratory function along with body temperature as indicated by the patient’s physiologic status and concerns that arose intraoperatively.
   2. Biochemistry/CBC monitoring: Recheck any correctable abnormalities that were present pre-anesthesia or that developed during anesthesia, like changes in electrolytes or acid-base. BUN and CRE can be measured but may be temporarily increased after anesthesia.
3. *Post-procedure:* Biochemistry/CBC monitoring: Recheck any correctable abnormalities that were present pre-anesthesia or that developed during anesthesia. The ALT/ALP can be measured but may be temporarily increased after anesthesia

**Resources**

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