Overview
Emergent and critically ill patients often times have a wide variety of disease conditions that can make anesthesia and analgesia a challenge. Some disease conditions that warrant special anesthetic consideration include cardiac disease, respiratory disease, trauma, GI disease, renal disease, hepatic disease, and reproductive disease. Over the last several years, the concepts of pain and pain management have become more prevalent and are being given more research. Pain, if left unidentified and untreated, contributes to greater morbidity, mortality, and animal suffering as well as a poor quality of life.

Anesthetic Risk
Prior to providing anesthesia to any patient, their anesthetic risk needs to be determined. The American Society of Anesthesiologists (ASA) created a classification system based on a patient’s physical and physiological status. This system helps to prepare and assess a patient’s anesthetic risk, taking into consideration age, level of systemic disease, type of procedure, and urgency of surgical condition. In the emergency setting, most patients requiring anesthesia are classified between ASA III-V. Understanding the critically ill patient’s underlying disease pathophysiology allows for better anesthetic preparation and monitoring.

Anesthetic Patient Preparation
The pre-anesthetic period can range from 30-45 minutes to several hours, depending on the emergent status of the patient. Some key tasks that should be performed as part of initial stabilization and preparation include venous access, minimum database diagnostics, discussion of advanced directive orders, setup of the operating room suite, and pre-anesthetic drug administration.

Cardiac Anesthesia
Cardiac disease is perhaps the most challenging anesthesia, as nearly all anesthesia-related agents have an effect on the cardiovascular system. These include potentially negative effects on heart rate (HR), stroke volume (SV), contractility, and systemic vascular resistance (SVR). Anesthesia also becomes more difficult in patients with pre-existing cardiac disease, as they have anatomical or physiological alterations to their heart function.

Cardiac patients have a greater chance of decompensation and therefore require more pre-anesthetic care. During pre-anesthetic exam, the patient should be auscultated for heart murmurs, gallop rhythms, arrhythmias, heart rate, and lung sounds. Additional cardiovascular parameters to assess include pulse rate, pulse quality, mucous membrane (mm) color, capillary refill time (CRT), and core versus peripheral temperature. Screening thoracic radiographs and/or echocardiogram may be beneficial to determine the severity of heart disease (i.e. size of heart, blood flow, valve function). A blood pressure should be taken frequently to help assess peripheral perfusion and cardiac output. If the patient is on specific heart medications, pre-anesthetic and induction agents should be reviewed for potential adverse effects. Pre-anesthetic agents that should be used with caution in cardiac disease patients include acepromazine, alpha-2 agonists, anticholinergics (unless there is a responsive arrhythmia), ketamine, and propofol. While there is no ideal anesthetic protocol, agents that have minimal cardiovascular effects include etomidate, alfaxalone, and neuroleptanalgesia (combination of opioid + benzodiazepine). Another consideration is to perform total intravenous anesthesia (TIVA), since all inhalant anesthetics cause cardiovascular depression.

Respiratory Anesthesia
Respiratory disease is the next challenging anesthesia, because generally speaking, most anesthetic agents are dose-dependent respiratory depressants. Patients with pre-existing respiratory disease also have anatomical or physiological alterations to their pulmonary function. Alterations in pulmonary function can lead to ventilation-perfusion mismatch, altered oxygen carrying capacity (hypoxemia), altered oxygen delivery to tissues (hypoxia), and decreased cardiac output.
Respiratory patients should be stabilized as much as possible prior to anesthetizing. Oxygen supplementation should be provided to any patient experiencing respiratory distress, and can be provided via flow-by, cage system, or nasal cannulas. During pre-anesthetic exam, the patient should be assessed for stertor or stridor as well as auscultated in all lung fields for wheezes or crackles. Screening thoracic radiographs and/or thoracic ultrasound may be beneficial to determine the severity of lung disease (i.e. pleural effusion, pneumothorax, pulmonary infiltrates/contusions). A pulse oximetry (SpO2) or venous/arterial blood gas analysis should be taken to help assess oxygenation status. Once intubated, end-tidal CO2 (ETCO2) should be used to help assess ventilation status (this can also be assessed pre-operatively on a venous blood gas). Sedative agents such as acepromazine, butorphanol, or benzodiazepines can be used to calm the patient and ease their work of breathing in the pre-anesthetic period. Other pre-anesthetic agents that can be used include opioids (not at high doses) and alpha-2 agonists (minimal respiratory effects). All respiratory patients should be pre-oxygenated for a minimum of five minutes prior to induction. Induction agents such as ketamine, propofol, alfaxalone, and etomidate all produce an apneustic breathing pattern, so they should be administered slowly and dosed to effect only. Intubation should occur in a rapid sequence to preserve a patent airway. The use of antiemetics and gastroprotectants are also recommended in respiratory disease to decrease the chance of regurgitation/vomiting (which could lead to aspiration) and acid secretions. During the anesthetic procedure, positive-end expiratory pressure (PEEP) can be utilized to improve pulmonary compliance. Post-anesthetic care includes continued supplemental oxygen and ensuring appropriate gag and swallow reflex prior to extubation.

Trauma Anesthesia

Traumatic injuries, both internal and external, result in a hypovolemic shock state. Hypovolemic shock occurs when there is a decrease in circulating blood volume, such as active hemorrhage. The loss of circulating blood volume results in decreased venous return to the heart (preload), which decreases cardiac output. The hypovolemia should be stabilized as much as possible prior to anesthesia. This includes oxygen support, shock IV fluid therapy, and blood component therapy. A minimum of two IV catheters should be placed to facilitate administration of fluids and medications in one and blood component therapy in the other. Any external traumatic injuries should be clipped/cleaned and pressure wrapped to contain hemorrhage. A packed cell volume (PCV) should be used to help assess red blood cell level. Full body radiographs and scanning ultrasound can be used to assess potential internal injuries and hemorrhage. Anesthetic protocols will vary more based on the cause of traumatic injury.

GI Anesthesia

Gastrointestinal disease can present in a wide range of patient physical status based on the type and severity of disease. Common disease conditions that affect the GI tract include foreign body ingestion, gastric dilatation volvulus (GDV), and sepsis (i.e. septic peritonitis from GI perforation). Often times, GI disease results in either a hypovolemic, distributive, or mixed hypovolemic-distributive shock state.

While GI disease can be more stable compared to cardiac or respiratory, there is the potential for the patient to decompensate. In addition to pre-anesthetic minimum database, close monitoring of hemodynamic parameters should be routinely done. If arrhythmias develop, ECG monitoring and anti-arrhythmic therapy should be instituted. If the patient remains hypotensive despite fluid resuscitation, vasopressor therapy may be required. GDV patients may require emergency trocharization to decompress the stomach. Antibiotic therapy may be needed in cases of sepsis for foreign body ingestion to get ahead of bacterial translocation. Opioids and antiemetics should be included as part of the pre-anesthetic drug administration, as most GI disease is painful. Induction agents should be based off the patient’s level of cardiovascular stability. Depending on the severity of disease, patients may been to be anesthetically ventilated. Post-anesthetic care includes ensuring appropriate gag and swallow reflex prior to extubation to reduce the chance of regurgitation/vomiting which can lead to aspiration.

Renal Anesthesia

Renal disease can present as either an acute condition or chronic kidney disease. Renal azotemia can be pre-renal, intrinsic renal, or post-renal in nature. Pre-renal refers to “before” the kidney, meaning injury is caused by other physiological or hemodynamic factors (i.e. hypovolemia, dehydration, cardiac compromise, or vasodilatory diseases). Intrinsic renal refers to direct damage to the renal parenchyma (i.e. renal ischemia, exposure to toxic agents, infectious insult, or neoplasia). Post-renal refers to “after” the kidney, meaning there is an obstruction or impediment in the outflow of urine that
prevents urine from being eliminated from the body (i.e. urethral obstruction, prostatic disease, urolithiasis, trauma, or neoplasia).

Anesthetic goals for the renal patient include maintaining fluid balance, addressing acid-base disturbances, correcting electrolyte abnormalities, and promoting excretion. Another anesthetic concern is to remember that the kidneys are the major organ system involved in drug excretion. In the pre-anesthetic period, the patient should be stabilized as much as possible with IV fluid therapy. A blood pressure should be taken, as hypertension is a common finding. If the patient is taking medications for their renal disease (i.e. enalapril, amlodipine) or has concurrent heart disease, these factors needs to be taken into consideration in the anesthetic drug selection. Opioids are generally a safe class of drugs, but acepromazine and alpha-2 agonists should be used with caution, as they may contribute to alterations in cardiac output and systemic vascular resistance.

Hepatic Anesthesia
Hepatic disease is primarily a concern due to the liver being the major organ system involved in drug metabolism. Common disease conditions that affect the liver include pancreatitis, cholangiohepatitis, cholecystitis, gall bladder mucocele, hepatic lipidosis, and hepatic failure. It’s important to remember that two functions of the liver are gluconeogenesis (production for glucose) and production of clotting factors, both of which may be altered with hepatic disease. Another anesthetic concern is to remember that the liver the major organ system involved in drug metabolism.

Anesthetic goals for the hepatic patient include stabilizing hemodynamic and liver parameters (i.e. perfusion, coagulopathy, encephalopathy, glucose regulation), taking into consideration anesthetics that undergo enterohepatic recirculation, and adjusting anesthetic drug dosing based on the severity of liver disease. Pre-anesthetics such as acepromazine, benzodiazepines, alpha-2 agonists, and propofol should be avoided.

Reproductive Anesthesia
The most common reproductive emergency is dystocia, which is challenging in that there rare both maternal and fetal patient considerations. During pregnancy, material physiologic changes include increased blood volume (causing a relative anemia), increased cardiac output from estrogen release, increased minute ventilation due to progesterone release (increased oxygen consumption), and increased abdominal volume causing displacement of the diaphragm (higher risk of regurgitation). Fetal physiologic changes include an immature hepatic system until 3-5 weeks old (lower tolerance of drug metabolism) and greater oxygen demands.

The major Caesarean (C-section) anesthetic goal is to avoid decreasing uterine blood flow, as decreased blood flow may result in fetal hypoxia, distress, and death. It is important to maintain maternal blood pressure, which can be done by maintaining perfusion and avoiding anesthetic drugs that cause cardiac depression. The placenta is highly permeable to anesthetic drugs and therefore should be minimized. One consideration is to utilize local or regional anesthetic blocks (i.e. epidural). Pre-anesthetic agents for the mother should be given to reduce maternal stress preoperatively; these include opioids and antiemetics for gastroprophylaxis. Drugs that are contraindicated in C-sections are acepromazine, benzodiazepines, and alpha-2 agonists, as these all cause fetal depression and contribute to greater fetal mortality. In addition, careful positioning of the mother for the surgical procedure. The mother should also be pre-oxygenated prior to induction for at least five minutes. Since all induction agents have the potential to cross the placental barrier, the target time from induction to fetal delivery is 10-15 minutes. When positioning the mother on the surgical table, consideration should be given to the weight of the uterus on the caudal vena cava; if possible, the mother’s trunk should be inclined so that the uterus is angled downwards toward the vulva rather than the spine.

Physiology of Pain
The International Association for the Study of Pain defines pain as, “an unpleasant sensory and emotional experience associated with actual or potential tissue damage”. Nociceptors (pain receptors) are present in the nervous system and become stimulated by a noxious stimulus (stimulation of a nerve ending). Nociception is the sensory process that involves a series of electrical events that start at the site of tissue injury, convey signals to the central nervous system, and result in the perception of pain. Nociception is essentially the pain pathway, and can be broken down into four processes: transduction, transmission, modulation and perception.
A noxious stimulus occurs at the site of tissue damage, which initiates the transduction process the pain pathway. Transduction is the conversion of physical energy into electrical energy by the nociceptor. The electrical energy then becomes a nerve impulse that can travel in the pain pathway along the nervous system. Transmission is propagation of the electrical nerve impulses in the nervous system. This moves the nerve impulse from the site of tissue damage to the spinal cord. Modulation is amplification, inhibition or suppression of nerve pain signals within the spinal cord. Perception is the integration, processing and recognition of nerve signals in the brain. Perception is how the animal feels pain and is a subjective experience.

### Recognition of Pain

Signs of pain can be classified as either behavioral or physiological. Behavioral signs are usually recognized first as they occur outwardly and are more readily observed. Physiological signs are systemic in nature, and therefore require a more hands on approach for assessment.

Behavioral signs of pain include vocalizations (i.e. whining, whimpering, groaning, growling, hissing, howling, purring), inability to rest, reluctance to lie down, agitation, change in temperament (i.e. aggression, timidity, withdrawal), drop in activity level, attention seeking actions, lethargy/depression, abnormal postures or movements (i.e. guarding, hunched, prayer position), immobility, trembling, muscle tension, change in facial expressions (i.e. staring, dull eye, drooping ears), insomnia, inappetence, lack of grooming (in cats), hiding, or self-mutilation (i.e. licking, kicking, biting or scratching a painful area).

Sometimes, an animal’s behavior changes depending on changes in their environment. For instance, an animal in a hospital setting may exhibit different behaviors compared to their more comfortable and familiar home environment. It’s important to obtain a thorough history from the owner about what’s normal and abnormal in their pet’s behavior. Behavior can also be modified in times of fear, anxiety and stress, which a hospital setting can also contribute to. When observing behaviors, it’s important to evaluate only the behaviors that are pain responsive/specific during your pain assessment.

Physiological signs of pain occur from the body’s increase in sympathetic tone, which is also known as the nervous system’s fight or flight response. These signs include increased heart rate (leads to increased myocardial oxygen demand, arrhythmias), variable cardiac output, increased respiratory rate, atelectasis (secondary to decreased tidal volume), retention of viscous respiratory secretions (leads to higher incidence of pneumonia), increased blood pressure (from peripheral vasoconstriction), decreased tissue oxygen delivery (which can lead to shock), increased temperature, increased metabolic rate, stress hormone release, decreased GI blood flow (leads to increased potential for ulceration), increased blood viscosity (leads to prolonged clotting times, platelet aggregation, and fibrinolysis), and immunosuppression (from compromised cardiovascular and respiratory systems, poor nutritional intake and recumbency).

The use of pain scoring systems can be a useful tool in helping assess an animal’s pain level. Pain scales are a means of documenting an objective measurement and assessment of pain. While no pain scale has been universally adopted, it would be ideal to sample different pain scales to find one that’s suitable to your practice and patients. Standardization of what pain scale is used ensures everyone is evaluating a patient’s pain with the same tool, and lowers the subjective aspect of pain assessment. Some examples of pain scoring systems include the visual analog scale (VAS), numerical rating scale (NRS) and the Glasgow Composite Measures Pain Scale (GCMPS). The VAS takes the form of a straight line, with one end representing no pain and the opposite end representing the worst possible pain. The VRS involves the patient pointing to where along the line their pain is, which unfortunately is not possible for our patients. The NRS uses multiple categories to evaluate patient behavior, such as vocalization or movement, which are given a numerical value within each category. Additionally, changes in physiologic parameters, such as HR, RR, or BP, can be included in the NRS score. The GCMPS is a multidimensional pain scale primarily used for acute postoperative pain; it includes several categories such as posture, comfort, vocalization, demeanor, and mobility. Again, the major limitation of GCMPS is it takes the form as a questionnaire, which is obsolete for use in our patients. In the author’s experience, the use of Colorado State University’s canine and feline acute pain scales has shown to be most objective in veterinary pain assessments. The scoring system is a combination of a VRS and NRS system that has been adapted for animal patients.
Pain Management

Analgesia is defined as absence of pain, or loss of sensitivity to pain, in response to a stimulus that is normally painful. An analgesic is a drug or agent that causes or allows pain relief. The analgesia options available in veterinary medicine are ever evolving, as more awareness and research is invested into the understanding of pain.

Opioids are the most effective class and often the first line drug used to provide analgesia. Opioids are centrally acting analgesics that limit nociception. Opioids exert their effects based on different receptors within the brain, spinal cord and peripheral nerves. There are four classes of opioids: pure agonists, partial agonists, agonists-antagonists, and antagonists. Pure agonists provide the most profound analgesic effects; examples include morphine, hydromorphone, oxymorphone, fentanyl, remifentanil, and methadone. Partial agonists provide a less pronounced analgesic effect; an example is buprenorphine. Agonist-antagonists provide an agonist effect on one receptor and an antagonist effect on another receptor; an example is butorphanol. Antagonists compete for the same receptors agonists use, thus acting as a reversal agent and providing no analgesic effect; an example is naloxone. Adverse effects of opioids include sedation, anxiolytic, bradycardia (at high doses), respiratory depression (at high doses), cough suppressant activity, urine retention (associated with increased urethral sphincter smooth muscle tone), nausea/vomiting (due to activation of the CRTZ), ileus, liver metabolism, and changes in temperature (from alteration of the central thermoregulatory set point).

Alpha-2 agonists provide centrally mediated (brain and spinal cord) analgesia and profound sedation. Their analgesic effects last anywhere from 30-90 minutes while their sedative effects can last much longer (hours). The most common examples of alpha-2 agonists include dexmedetomidine, medetomidine and xylazine. Just like with opioids, alpha-2 agonists have a reversal agent, atipamezole. Adverse effects of alpha-2 agonists include profound sedation, bradycardia, and hypotension.

Non-steroidal antiinflammatory drugs (NSAIDs) decrease the production of inflammatory mediators at the site of tissue injury. Inflammatory mediators contribute to increased transmission of pain signals by stimulating nociceptors. NSAIDs exert their effect by inhibiting prostaglandin production within the cyclooxygenase (COX) pathway. COX-1 prostaglandins are responsible for normal homeostasis of the GI tract and kidneys as well as maintaining hemostasis. COX-2 prostaglandins are responsible for gastric mucosal healing and homeostasis of the brain, kidneys and reproductive systems. NSAIDs are not specific to either COX-1 or COX-2 inhibition, rather they have varying degrees of inhibition for both. Examples of veterinary-approved NSAIDs include carprofen, meloxicam, deracoxib, and robenacoxib. Since inflammation can play a significant role in the pain pathway, the use of NSAIDs may prove beneficial. Adverse effects of NSAIDs are primarily GI (risk of ulceration with prolonged use) and renal (compromised renal blood flow and glomerular filtration rate if given to hypovolemic, dehydrated or hypoperfused patients) effects. It should also be noted that the use of steroids, other NSAIDs or nephrotoxic drugs are contraindicated, as they potentiate the risk of GI or renal injury.

There are also agents and therapies available that can have synergistic activity with, or potentiate the effects of, primary analgesics. These adjunctive agents include sedatives (i.e. benzodiazepines), anxiolytics (i.e. trazadone), gabapentin, ketamine, local anesthetics, and low-level laser therapy.

Since there are several mechanisms in which pain can be produced and perceived, it’s often advantageous to use more than one type of analgesic. Balanced analgesia and combination therapy is known as multimodal analgesia, which is the practice of using multiple analgesic agents to provide better analgesia than with a single agent alone. Examples of combinations include an opioid + benzodiazepine, an opioid + NSAID, an opioid + ketamine, an opioid + local anesthetic, etc.

References