

## Tech Tip

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

### CARDIAC EFFECTS OF ANESTHETIC AGENTS

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

Sympathetic activity is controlled by mediation or release and uptake of the hormones epinephrine, norepinephrine and related adrenergic hormones. (Fight or flight) The sympathetic nervous system often called the adrenergic nervous system. Direct-acting adrenergic receptor agonists (sympathomimetic agents) interact with and activate adrenergic receptors:

Alpha adrenergic receptors-  $\alpha_1$  stimulation causes the blood vessels to constrict

Beta ( $\beta$ ) adrenergic receptors-  $\beta_2$  stimulation causes the blood vessels to dilate Dopamine receptors

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

Injectable analgesic and anesthetic agents: Alpha 2 agonists

Used in veterinary medicine to produce: sedation, analgesia and anxiolysis.

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

Thiopental - Barbiturate

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

Benzodiazepines

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

Midazolam: Water soluble and is more useful for IM injections. When combined with an opioid, it will provide neuroleptanalgesia. NOT reliable as tranquilizers for dogs and cats when used as a sole agent!!

Patients may lose inhibitions and become excitable. When combined with an opioid as a CRI can be utilized to decrease MAC of inhalant agent. No analgesia provided. Effects may be reversed with Flumazenil.

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

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

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

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

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

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

MAC (minimal alveolar concentration) produces immobility in 50% of patients receiving noxious stimuli. Varies with agents and species. The lower the MAC, the higher potency of the anesthetic agent. Halothane- Acts to reduce cardiac output through direct depression of the myocardium. Heart rate changes may be minimal. Arterial blood pressure is decreased due to decreased output. Sensitizes the myocardium to catecholamine increasing the chances for arrhythmias. Isoflurane- Lesser degree of

myocardial depression than halothane. Heart rate is seen to increase slightly. Decrease in arterial blood pressure - main cause is decreased vascular resistance vs. decreased cardiac output. Sevoflurane - Causes mild myocardial depression (decreased contractility). Mild systemic vascular resistance and arterial blood pressure depression. Less likely to see an increase in heart rate, vasodilation when compared to isoflurane.

#### Local anesthetics

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

#### Bupivacaine

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

#### References:

1. Lumb and Jones' Veterinary Anesthesia, 3rd edition, Lippincott, Williams & Wilkins, 1996
2. Small Animal Anesthesia and Analgesia, G.L. Carroll, editor, Blackwell Publishing 2008
3. Textbook of Medical Physiology, A. C. Guyton editor, 8th edition, W. B. Saunders, 1991
4. Manual of Canine and Feline Cardiology, Tilley, Smith, Jr., Oyama, Sleeper, editors, 4th edition, W. B. Saunders, 2001
5. Small Animal Clinical Pharmacology, Madison, Page, Church, editors, 2nd edition, W. B. Saunders, 2008
6. Anesthesia workshop, Hanson, Waddell, proceedings, IVECCS, 2007