PERIANESTHETIC ARRHYTHMIAS—WHAT IS IT AND WHAT DO I DO NOW? **ANESTHESIA**

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Cardiac arrhythmia may be defined as an irregularity in the cardiac rhythm. They are typically classified according to origin, rate, or regularity. Arrhythmias may occur from cardiac and/or extra cardiac reasons.

Bradycardia is commonly seen with the anesthetized patient. Commonly the cause is dose related to the anesthetic and/or analgesic agents used. If the heart rate is low with a normal sinus rhythm and supporting blood pressure, be aware, be prepared to treat but be tolerant of a slow rate. If hypotension is noted in addition to a slow rate, an anticholinergic may be indicated to increase output by raising rat; this is particularly true with pediatric and juvenile patients.

Normal sinus rhythm is seen on the ECG as a P wave associated with every QRS complex with a regular rhythm. In awake patients, the rate for canine patients will vary between 60 bpm to 180 bpm dependent on age, size, and degree of body conditioning. The feline patient will have a rate that can very between 140 bpm and 240 bpm depending on the anxiety level of the cat in the hospital setting.

The canine patient can have a normal pattern of irregularity, sinus arrhythmia that occurs as the heart rate increases during inspiration and decreases during exhalation in a regular cyclical pattern.

Sinus bradycardia, a normal sinus rhythm with an abnormally slow rate, may be due to normal physiology: i.e., natural sleep the heart rate may drop to the 30 bpm range; athletic dogs at rest may have a naturally low heart rate. An increase in vagal tone frequently results in a sinus bradycardia. In the anesthetized patient, bradycardia is defined as a heart rate less than 60 bpm for large/giant breeds and 60–70 bpm for the majority of adult canine patients. It is important to remember that neonate and pediatric patients have a need for a higher heart rate as their blood pressure is rate dependent and will require intervention to maintain heart rate at a greater number of beats per minute to maintain blood pressures. The feline patient may be considered bradycardic with a heart rate of 100–120 bpm.

There are many different causes for bradycardia. High vagal tone—dose dependent, the use of mu opioids such as oxymorphone, fentanyl, and morphine and alpha 2 agonists will cause an increase in vagal tone thus reducing the heart rate.

- Brachycephalic breeds—due to their anatomy
- Intubation—stimulation of the vagas nerve during sternal intubation
- GI disease/surgical manipulation—traction on the viscera
- Neurologic/spinal cord lesions
- Ophthalmological procedures—pressure or traction on the orbit
- Manipulation of vagas nerve during cervical/thoracic procedures
- Electrolyte imbalances: i.e., hyperkalemia regardless of metabolic dysfunction
- Hypothermia
- Excessive depth of anesthesia
- Profound hypoxemia

First degree atrioventricular block occurs when there is a delay of conduction through the AV node that produces a prolongation of the PR interval of greater than 0.13 seconds in the canine and greater than 0.09 seconds in the feline. First degree AV block as an isolated occurrence may not impact the hemodynamic status of the patient but can be an indicator of early nodal disease.

Second degree atrioventricular (AV) block can be attributed to drug administration as well as inherent cardiac nodal disease. The atrial rate is faster than the ventricular rate. Second degree AV block is further divided into two types. Mobitz type 1 (Wenckeback) is demonstrated by a progressive prolongation of the interval between the P wave and the QRS complex. Mobitz type II is seen as a regular P wave rate with the QRS complex periodically dropping out. The block may respond to anticholinergic agents but a worsening of the block may be seen initially as the atria respond initially to the action of the anticholinergic.

Advanced second degree block is diagnosed when the heart rate is greater than 40 beats per minute. No relationship exists between the P wave and the QRS complex.

In third degree AV block, there will be no association between the P waves and the QRS complexes. Frequently, this ventricular rate is based solely on escape or fusion beats and is lower than 40 beats per minute. Oftentimes this patient presents with episodes of syncope and is non-responsive to an atropine challenge. This patient should be referred to a cardiology service for pacemaker implantation as they are at a high risk for sudden death. Antiarrhythmic ventricular escape beats should never be treated with antiarrhythmic agents as this is the only driving ventricular beat.

In an emergency basis, heart rate may be driven chemically with the CRI administration of isoproterenol, a chronotrophic agent. Be cognizant of the fact that typically, administration of isoproterenol will detrimental to systemic blood pressure.

Tachyarrhythmia, bradyarrhythmia, also known as sick sinus syndrome, is more commonly seen in cocker spaniels, miniature schnauzers, and dachshunds. The arrhythmias seen will include sinus bradycardia, sinus arrest, alternating brady-tachycardia, and escape beats. This patient should be referred for a temporary or permanent pacemaker should anesthesia be indicated for diagnostics or a surgical procedure.

Treatments/interventions to manage perianesthetic bradyarrhythmias: Determine the origin bearing in mind that there may be more than one factor affecting the rate.

Opioid administration—judicious use of anticholinergic agents should be designed to maintain the heart rate within normal parameters for the breed and age of the patient. Should the heart rate be elevated due to pain or anxiety, premedicate the patient with an opioid, wait 10–15 minutes to judge the effect of the opioid on the heart rate, and determine whether or not an anticholinergic agent should be given. It is desirable that a plasma level of the anticholinergic agent be in place prior to an anticipated intra-operative CRI of a mu opioid. Opioids may be reversed if the bradycardia does not respond to administration of an anticholinergic or if the patient's condition is deteriorating. In the recovery phase, reversal with a mixed agonist/antagonist such as butorphanol or buprenorphine will help correct the bradycardia while leaving some analgesia in effect provided that the pain level is deemed mild to moderate.

Brachycephalic breeds—a dose of an anticholinergic agent should be calculated and available to be administered during the post premedication and induction period should the heart rate drop to an unacceptable rate and is not supporting systemic blood pressure.

Patients with existing GI disease that are undergoing anesthesia for diagnostic or surgical procedures should also have an anticholinergic dose calculated and at hand to be administered should the need arise. Frequently, those patients undergoing diagnostic procedures such as endoscopy are premedicated with a mixed agonist/antagonist agent that does not produce a significant decrease in heart rate.

Neurologic patients are often found to be bradycardic. As long as the heart rate supports the systemic blood pressure, it is often not necessary to treat the heart rate with an anticholinergic agent. It can be quite unnerving to monitor a canine patient with a heart rate in the 50 bpm range that has adequate blood pressure and is reassuring to have a rescue dose of an anticholinergic agent pre-drawn and in hand.

Ophthalmology patients frequently are given additional agents intra-operatively: i.e., neuromuscular depolarizing agents that can decrease heart rate. In addition to vagal stimulation provided by surgical manipulation this can be an unfortunate combination if not anticipated by the administration of an anticholinergic to maintain the heart rate within a normal parameter.

Manipulation of the vagas nerve during cervical or thoracic procedures will induce bradycardia that can be gauged by asking the surgeon to cease momentarily to judge the heart rate response; if it is inadequate, the administration of an anticholinergic would be advisable.

Electrolyte imbalances can be managed preoperatively by having lab results available prior to induction of general

anesthesia. Hyperkalemia due to urinary blockage/rupture of urinary bladder. Decreased elimination due to oliguric or anuric renal failure. Addison's disease that may not be stabilized. Potassium-sparing diuretics and ACE inhibitors. Excessive administration of supplements either oral or by IV fluid additive.

Hypothermia—mild hypothermia can result in elongation of PR intervals, QRS duration, and QT interval. Additionally, atrial premature complexes may be seen as well as inverted T waves. Moderate hypothermia can produce atropine resistant bradycardia and ventricular arrhythmias, while severe hypothermia may lead to ventricular fibrillation and possible cardiac arrest.

Excessive depth of anesthesia—assess other physical parameters: i.e., eye position, jaw tone, anal tone, and decrease inhalant +/- CRI agents to "lighten" patient to correct plane of anesthesia. A rescue dose of an anticholinergic may be necessary to maintain heart rate during the interval and existing heart rate.

Profound hypoxemia—this can be seen as a terminal event as typically ventricular tachycardia precedes bradycardia as hypoxemia progresses. Availability of arterial blood gases to assess ventilation can determine the patient's status and in the interim if intubated, rule out mechanical obstruction of the endotracheal tube, increase oxygen flow rate, increase respiratory rate and/or tidal volume. If not intubated, be prepared to intubate and provide intermittent positive pressure ventilation.

Tachyarrhythmias may present as sinus tachycardia, atrial tachycardia, atrial fibrillation, ventricular tachycardia, or supraventricular tachycardia.

Sinus tachycardia increases myocardial work, oxygen demand, as well as decreasing diastolic filling time. Approximately 70 percent of the perfusion of the myocardium occurs during diastole, therefore it is valuable to control excessive heart rates.

Potential causes of sinus tachycardia:
Administration of anticholinergic agents, especially IV administration. Increase in sympathetic tone: ketamine given as a bolus will increase sympathetic tone thus increasing the heart rate. If a ketamine CRI is deemed necessary during the intra-operative/postoperative period be aware that effective analgesia levels are not normally reached without the administration of the initial bolus. Administration of excessive thyroid supplementation. Administration of methylxanthines, i.e., theophylline. Administration of catecholamines, i.e., epinephrine, dobutamine.

1. Patient awareness may be handled with a bolus of an appropriate opioid agent, which will also assist with pain management and should return the heart rate to within an expected rate.
2. Hypovolemia should be corrected as soon as possible by the administration of IV crystalloid therapy or a combination of colloid and crystalloid infusion. Acute hypovolemia may be treated with 1/4 of the calculated shock dose of fluids given as a bolus or should the patient be hypoproteinemic, consider using a synthetic colloid at a dose of 3–5 ml/kg over 15–20 minutes. This a compensatory mechanism to maintain cardiac output by increasing the heart rate in view of decreased cardiac output.
3. Hypotension that is not caused by hypovolemia but perhaps by anesthetic agents causing peripheral vasodilation may be treated with positive ionotropes as a CRI, whether dobutamine or dopamine is selected will be based on patient overall assessment. Administration of thiopental and propofol will cause transient decreases in systemic blood pressure due to myocardial depression and peripheral vasodilation. Acepromazine will also cause peripheral vasodilation that is longer lasting and must be support by fluid administration with or without the addition of a positive ionotrope.
4. Anemia may be managed by the administration of packed red blood cells or fresh whole blood. This will increase the oxygen carrying capability of the cardiovascular system. Synthetic hemoglobin carriers may be used in lieu of blood products.
5. Hypoxemia may be corrected by increasing the inspired oxygen whether the patient is intubated or receiving flow by oxygen support. Be aware that in the awake patient in this situation that intubation may need to occur in the near future.
6. Hyperthermia can be corrected by active cooling. Discontinuing any active rewarming support (i.e., forced warm air blankets) administration of cooled IV fluids will aid in decreasing body temperature.
7. Myocardial disease: Those patients with existing cardiac disease may already be on beta blockers for heart rate control. If timing of the anesthesia can be controlled, request that the patient receive a 1/2 dose of the prescribed

beta blocker the morning of the surgical procedure and premedicate ONLY as needed with an anticholinergic agent. Should blood pressure management be required intra-operatively, positive ionotrope support may not be as effective as desired and a pressor agent may be necessary as oftentimes cardiac patients are not able to receive fluid volume due to the possibility of pulmonary edema.

Ventricular arrhythmias are noted as premature contractions, tachycardia, and fibrillation. Again, these rhythms may cardiac or extracardiac in origin.

Multiform VPCs—Encountering arrhythmias in those patients with existing cardiac disease should not be unexpected depending on the nature of the disease. Those patients with conditions causing atrial enlargement are predisposed to the development of supraventricular arrhythmias, supraventricular tachyarrhythms (SVY), atrial premature contractions (APCs), atrial fibrillation, or atrial flutter while those with ventricular enlargement may be predisposed to ventricular premature contractions (VPCs), ventricular tachyarrhythmia, or ventricular fibrillation. Indeed the very act of providing anesthesia may predispose patients with normal myocardium function to show arrhythmias.

Supraventricular rhythms are rapid rhythms arising from the atria or the atrioventricular junction. Supraventricular tachyarrhythmias (SVT) may be attempted to be broken by the use of a vagal maneuver. Applying pressure on both eyes or carotid massage may increase vagal tone to the heart thereby resulting in the slowing of the sinoatrial node discharge and the atrioventricular nodal conduction time. Failing response to a vagal maneuver, procainamide at 20mg/kg may be administered over a 20-minute period or to effect. If the tachyarrhythmia persists, diltiazem at 0.125–0.35 mg/kg may be given slow IV. Diltiazem is a calcium channel blocker and should not be considered if the patient is hypotensive or has sick sinus syndrome. Adverse side effects may be seen as bradycardia, hypotension, or heart block. Esmolol, an ultra-short acting beta blocker, may also be used should rhythm conversion fail with previous treatments. It requires a loading dose of 200–500 mcg/kg IV to be given over a 60-second period and must be followed by a CRI of 25–200 mcg/kg/minute. If esmolol administration precedes diltiazem therapy, a 30-minute wait is recommended. Transient hypotension and bradycardia may be seen with esmolol administration.

Those patients exhibiting the occasional VPC, unifocal VPCs less than three in a row should not require intervention unless they are hemodynamically unstable. If the VPCs occur more often than approximately 10 per minute, occur as a “run” of more than three in a row, are multifocal or cause a decrease in systemic blood pressure, therapy should be instituted. A 2 mg/kg bolus of lidocaine given IV should be the first line of treatment. Lidocaine is a class IB antiarrhythmic agent and works by inhibiting sodium channels. If the arrhythmia persists, up to four boluses, for a total dose of 8 mg/kg, may be delivered over a 15-minute time frame. If the bolus is given too rapidly, a fall in blood pressure may be seen. The onset of action is two minutes and has a 10- to 20-minute duration. If the arrhythmia converts to a normal sinus rhythm, a CRI may be initiated. The usual starting rate for arrhythmia control is calculated at 50 mcg/kg/minute and may be up titrated to 100 mcg/kg/minute. Procainamide, a class IA antiarrhythmic agent, works by slowing conduction velocity and is used for the treatment of VPCs that are not controlled by lidocaine administration. Typically a dose calculated at 20 mg/kg is given IV over a 20-minute period or until the rhythm converts. If conversion is successful, a CRI may be delivered for further control. Long-term treatment is usually achieved by using a beta blocking agent if indicated once the patient has recovered from anesthesia.