CARDIOVASCULAR MONITORING AND YOU: PERIOPERATIVE ECG AND BLOOD PRESSURE MANAGEMENT
Kim Spelts, BS, CVT, VTS

Monitoring patients under anesthesia is crucial in order for a technician to be able to assess whether a patient is adequately anesthetized, whether his or her pain is adequately managed, and whether the autonomic nervous system is adequately subdued. Most importantly, appropriate anesthesia monitoring allows the observer to assess the current physiologic consequences of anesthesia in order to help improve the safety of the anesthetic procedure. Maintaining cardiac output is key. ECG and blood pressure are two monitoring tools the anesthetist has that help assess cardiovascular function.

**ECG Management**
Cardiac dysrhythmias are common under anesthesia, especially in critical patients. The decision to treat or not to treat these dysrhythmias is based on the hemodynamic consequences of the abnormal rhythm, as well as the likelihood for further deterioration.

**Bradydysrhythmias**
Under anesthesia, bradydysrhythmias are most frequently caused by a drug-induced increase in vagal tone, such as that caused by the use of opioids and alpha-2 agonists.

Sinus bradycardia is a common side effect of opioid administration. The decision to treat should be based on the patient’s overall status. Remember that CO = HR x SV, where CO = cardiac output, HR = heart rate, and SV = stroke volume (a function of preload, afterload, and contractility). If the heart rate is low enough that it causes a significant decrease in cardiac output (and potentially a decrease in blood pressure), then an anticholinergic should be administered to offset the high vagal tone (atropine 0.01 mg/kg IV or glycopyrrolate 0.005 mg/kg IV).

1st-degree atrio-ventricular (AV) block results in a prolonged but consistent P-R interval on the ECG. This rhythm may be seen in patients who are taking beta-blockers, calcium channel blockers, or digoxin. The effect on cardiac output of this dysrhythmia is usually not significant enough to warrant treatment.

2nd-degree AV block is a common dysrhythmia seen with very high vagal tone (such as that seen with alpha-2 agonists) and with administration of IV atropine. There are two sub-types:

- **Mobitz Type 1 (Wenckebach):** On the ECG, this type of AV block is seen as a gradually increasing P-R interval until the QRS complex is dropped altogether.
- **Mobitz Type 2:** On the ECG, this type of AV block shows a consistent and normal PR interval with occasional dropped QRS complexes. This dysrhythmia has the potential for deteriorating into 3rd-degree AV block and should always be treated.

Both types of 2nd-degree AV block can be treated with IV anticholinergics. If the dysrhythmia was originally caused by administration of IV atropine, give another 0.01 mg/kg dose IV.

3rd-degree AV block is a serious, potentially lethal dysrhythmia. This block is marked by a regular atrial rhythm and a regular ventricular rhythm, but the two are independent of each other due to a complete lack of communication between the SA and AV nodes. The ventricular beats are usually slow and ineffective, resulting in a marked decrease in cardiac output. 3rd degree heart block has a potential for progressing to asystole. It is unresponsive to anticholinergics. Temporarily, IV isoproterenol may be administered for its beta-1 and beta-2 receptor agonist effects (increase in heart rate, contractility, conduction velocity, as well as vasodilation). For long-term management, a pacemaker should be implanted.

**Tachydysrhythmias**
Sinus tachycardia is frequently seen in anesthetized patients and is characterized by fast, but normal, ECG complexes. Sinus tachycardia is caused by high sympathetic tone, often generated by pain, a high dose of anticholinergics, and/or a light plane of anesthesia. Hypovolemia can also generate sinus tachycardia as the body compensates for the drop in stroke volume by increasing the heart rate in an attempt to maintain cardiac output.
Supraventricular tachycardia (SVT) is a fast rhythm, the origin of which occurs somewhere above the ventricles. Sinus tachycardia is a type of SVT, but traditionally SVT is considered to be a tachycardia that originates from somewhere other than the sinus node but above the AV junction. Consequently, an ECG strip of SVT will not show P waves. This is rarely seen under anesthesia, but it should be addressed quickly. Prolonged SVT can lead to progressive myocardial failure as well as congestive heart failure. Treatment for SVT is usually with esmolol 0.25–0.5 mg/kg followed by a CRI of 50–200 mcg/kg/min if necessary.

**Ventricular Dysrhythmias**

Premature ventricular complexes (PVCs or VPCs) are common occurrences. They are generated by an irritable site (or sites) in the ventricles. They appear on the ECG as wide and bizarre QRS complexes that occur earlier than would be expected given the underlying cadence of the normal rhythm. They are followed by a compensatory pause in the rhythm, the sound of which is easily discernible with an ultrasonic doppler. PVCs that look alike are termed *uniform*, while those that appear different from each other are termed *multiform*. Multiform PVCs are more serious than uniform PVCs, because they indicate a greater degree of myocardial irritability.

Treatment for rare or occasional uniform PVCs is generally not required. Frequent and/or multiform PVCs may have a significant impact on cardiac output. IV lidocaine may be administered at 2–4 mg/kg in dogs, 1–2 mg/kg in cats. Some patients may require a constant infusion of lidocaine (20–80 mcg/kg/min) for management of PVCs. PVCs are common in patients with hemoabdomen, bloat, and gastric dilatation-volvulus (GDV).

**Atrial Dysrhythmias**

Atrial dysrhythmias are less common under anesthesia. Premature atrial complexes (PACs) generate an audible compensatory pause on a doppler, similar to PVCs. However, they rarely have a significant effect on the patient’s cardiovascular status.

Atrial fibrillation may be seen in some giant breed dogs (Great Danes, Irish wolfhounds) and in some horses. The only treatment for this dysrhythmia is to attempt an electric cardioversion. Cardiac auscultation has been described as “tennis shoes in a dryer” or “jungle drums.” An ECG strip will reveal an “irregularly irregular” series of complexes with no P waves.

**Other Dysrhythmias**

Pain or a light plane of anesthesia may cause sinus tachycardia within the perianesthetic period, or it may result if a patient is in shock and/or hypovolemic. If concurrent hypotension is observed, an IV bolus of crystalloid or colloid should be administered. Intraoperative opioids will also help to stabilize the plane of anesthesia and provide additional pain control.

Pulses alternans may also be observed in hypovolemic patients, especially those receiving intermittent positive pressure ventilation (IPPV). Pulses alternans appears on a monitor as a normal ECG with every other pulse wave (on the pulse oximeter and/or arterial blood pressure monitor) lower in amplitude or absent. Decreased ventricular filling results in every other cardiac contraction being less powerful, resulting in a smaller pulse waveform. An IV crystalloid or colloid bolus should resolve the pulses alternans.

**Blood Pressure Management**

Hypotension is probably the most common complication seen during the perianesthetic period. Most anesthetic drugs negatively affect blood pressure by decreasing cardiac output. In most cases, hypotension can be resolved easily if the anesthetist is able to correctly identify and treat the underlying cause.

There are several ways to monitor blood pressure during an anesthetic procedure, the most common being ultrasonic doppler or noninvasive oscillometric device. Each of these methods is inherently inaccurate, but they are simple to use. The gold standard of blood pressure measurement is placement of an arterial catheter. This is an advanced technical skill, but it also provides an accurate measurement of a patient’s blood pressure in real-time.

Regardless of the method used, the goal should be to maintain the following low limits in systolic (SAP), diastolic (DAP), and mean (MAP) arterial pressures:
MAP >70 mmHg: The autoregulation of blood flow to renal and cerebral tissues occurs between MAP of 60 and 160 mmHg.

SAP >90 mmHg: If no method of measuring MAP available (e.g., blood pressure monitored with ultrasonic doppler); helps ensure MAP >60 mmHg.

DAP >50 mmHg: Myocardial reperfusion occurs during diastole and requires DAP >40 mmHg.

The underlying cause of a patient’s hypotension varies depending on the combination of injectable and inhalant anesthetic agents utilized, along with the patient’s underlying physical condition. In order to choose a correct course of treatment for hypotension, it is important to first determine the correct underlying cause. Keep in mind the following physiological relationships:

\[
\text{MAP} = \text{CO} \times \text{SVR}
\]

\[
\text{CO} = \text{HR} \times \text{SV}
\]

Clinical hypotension (MAP <60 mmHg) results when the systolic and/or diastolic arterial pressures are low due to low cardiac output and/or systemic vascular resistance. One must be able to assess all pressure values and understand what is going on with the patient as a whole in order to determine the appropriate course of treatment. It is important to recognize that an increase in blood pressure does not necessarily correlate to an increase in cardiac output. For example, IV sedation with alpha-2 agonists may yield high blood pressures; however, this is due to high SVR caused by vasoconstriction. This increase in afterload actually decreases cardiac output and reduces peripheral perfusion.

Bradycardia is common under anesthesia, especially during systemic opioid administration. A hypotensive patient who is also bradycardic should be treated with an anticholinergic to increase the heart rate and therefore increase the cardiac output. This is a common source of hypotension in puppies and kittens, who are more reliant upon heart rate to maintain their cardiac output than adults.

Cardiac output may also drop as a result of decreased stroke volume, usually due to decreased contractility (a common side effect of inhalant anesthetics) and/or decreased preload. A decrease in contractility may be indicated by a low SAP, and administration of an inotropic agent (coupled with decreasing the inhalant, if possible) is appropriate. A decreased DAP may indicate a low systemic vascular resistance and/or decreased preload; in these cases, an IV crystalloid and/or colloid bolus should be considered.

Inotropic agents are effective at treating hypotension related to decreases in contractility and/or severe drops in systemic vascular resistance (such as that seen in severe sepsis or shock). They work by stimulating alpha- and/or beta- adrenergic receptors. Briefly:

- Stimulation of alpha-1 receptors results in vasoconstriction;
- Stimulation of alpha-2 receptors results in sedation and analgesia as well as peripheral and coronary vasoconstriction;
- Stimulation of beta-1 receptors results in increased contractility and heart rate;
- Stimulation of beta-2 receptors results in vasodilation and bronchodilation.
**Ephedrine** stimulates the release of endogenous stores of norepinephrine, although it also has some direct stimulation of beta-1 and -2 receptors.

**Dobutamine** is a beta-1 agonist that provides an increase in contractility without an increase in heart rate (except at very high rates). It is the most desirable drug for increasing blood pressure by increasing cardiac output. It should be pointed out, however, that dobutamine (and all beta-agonists) are ineffective in the presence of acidemia, since beta receptors become less responsive under those conditions.

**Dopamine** has different effects depending on the rate. At low rates, it stimulates dopaminergic receptors to dilate renal arterioles. Slightly higher rates result in stimulation of beta-1 receptors. The concurrent increase in contractility and heart rate often seen with dopamine may result in a greater myocardial oxygen demand, increasing the risk of dysrhythmias. This is potentially dangerous in patients with underlying heart disease. Higher rates of dobutamine result in primarily alpha-1 stimulation, which increases blood pressure at the expense of peripheral perfusion.

**Norepinephrine** stimulates alpha-1 receptors. Norepinephrine may be the drug of choice in septic/endotoxemic patients, since it corrects inappropriately dilated areas. Careful monitoring of blood pressure and mucous membrane color should occur to guard against over-constriction and subsequent decrease in peripheral perfusion.

If treatment with inotropes is not effective, check the patient’s acid-base and electrolyte status. Inotropes have a decreased effect on cardiac contractility in the presence of acidemia. Also, low ionized serum calcium can result in decreased contractility.

---

**Table 1. Drug Infusion Worksheet**

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>Desired dose (mcg/kg/min)</th>
<th>B</th>
<th>Body weight (kg)</th>
<th>C</th>
<th>Desired diluent volume (ml)</th>
<th>D</th>
<th>Drip set used (drops/ml)</th>
<th>E</th>
<th>Concentration of drug (mg/ml)</th>
<th>F</th>
<th>Desired drip rate (drops/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ml of drug to add to diluent =</td>
<td></td>
<td>A x B x C x D</td>
<td>E x F x 1000</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 2. Hypotension: Drugs, Dosages and Indications**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Drug Class</th>
<th>Dose Range</th>
<th>Route</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>Anticholinergic</td>
<td>0.01–0.04 mg/kg</td>
<td>SQ, IM, IV</td>
<td>Bradycardia</td>
</tr>
<tr>
<td>Glycopyrrolate</td>
<td>Anticholinergic</td>
<td>0.005–0.02 mg/kg</td>
<td>SQ, IM, IV</td>
<td>Bradycardia</td>
</tr>
<tr>
<td>Ephedrine</td>
<td>Beta agonist</td>
<td>0.1 mg/kg*</td>
<td>IV</td>
<td>Increase contractility</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>Beta agonist</td>
<td>0.5–10 mcg/kg/min</td>
<td>IV CRI</td>
<td>Increase contractility</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Dopaminergic agonist</td>
<td>1–5 mcg/kg/min</td>
<td>IV CRI</td>
<td>Dilate renal arterioles via DA1 receptors**</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Beta agonist</td>
<td>5–10 mcg/kg/min</td>
<td>IV CRI</td>
<td>Increase contractility</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Alpha agonist</td>
<td>&gt;10 mcg/kg/min</td>
<td>IV CRI</td>
<td>Increase SVR</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>Mixed alpha and beta agonist</td>
<td>0.05–0.2 mcg/kg/min</td>
<td>IV CRI</td>
<td>Increase SVR, increase contractility</td>
</tr>
</tbody>
</table>

*May lose effectiveness over time due to depletion of endogenous norepinephrine stores.

**Cats may not have DA1 receptors.**

**Suggested Reading**

