Perioperative Monitoring of Heart Rate and Rhythm

Mark A. Oyama, DVM

KEYWORDS
- Arrhythmia • Antiarrhythmics • Electrocardiography

KEY POINTS
- When assessing perioperative arrhythmias, it is important to obtain an accurate electrocardiographic diagnosis, assess patient hemodynamic status during the arrhythmia, and determine whether underlying primary cardiac disease is present.
- The decision on whether to treat a specific arrhythmia should be based on the presence or absence of hemodynamic signs and risk of sudden death.
- If antiarrhythmic therapy is deemed necessary, consider the likely mechanisms of arrhythmia, address transient imbalances that contribute to arrhythmia formation, and select antiarrhythmic agents based on mechanism of action and arrhythmia diagnosis.
- Whether or not treatment is initiated, continuous and careful monitoring of electrocardiogram rhythm and hemodynamics is advisable.

NATURE OF THE PROBLEM

Introduction

Disorders of heart rate and rhythms in the perioperative period are common and can occur in animals with or without underlying primary cardiac or conduction system disease. Primary cardiac diseases that are associated with high baseline risk for clinically important arrhythmias include myocardial diseases such as hypertrophic, dilated, and arrhythmogenic cardiomyopathy; pericardial disorders; conduction system diseases such as sick sinus syndrome or atrioventricular (AV) nodal block; and, to a lesser extent, myxomatous mitral valve disease. Observational studies reveal that perioperative arrhythmias are commonly encountered in noncardiac conditions (Box 1) such as gastric dilatation-volvulus (GDV), hemoabdomen, and splenic mass, wherein the incidence of ventricular arrhythmias is 50.6% to 77.4%,1,2 32%,3 and 28.4%,4 respectively. Some of these studies indicate that the presence of perioperative arrhythmias is associated with an increase in mortality,3,4 whereas others do not.1 Although these observational studies provide evidence that perioperative arrhythmias are a common

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Department of Clinical Studies, Matthew J. Ryan Veterinary Hospital, University of Pennsylvania, School of Veterinary Medicine, 3900 Delancey Street, Philadelphia, PA 19104, USA
E-mail address: maoyama@vet.upenn.edu

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clinical occurrence, they are unable to indicate whether intervention to suppress these arrhythmias improves outcome. In one retrospective study of dogs undergoing surgery for GDV, prophylactic perioperative lidocaine administration was not associated with reduced mortality.\(^5\) Important aspects of the problem include the following:

- Perioperative cardiac arrhythmias are common.
- Their effect on morbidity and mortality is largely unknown.
- The benefit in treating or not treating perioperative arrhythmias is likely influenced by many different factors including the following:
  - Presence or absence of underlying primary cardiac disease
  - Presence or absence of underlying extracardiac disease
  - Nature of the cardiac arrhythmia (eg, rhythm, rate, frequency)
  - Presence or absence of aggravating factors or transient physiologic imbalances (discussed later)
- The precise effect of these and other factors in overall survival is unknown, which makes decision making around when and what to treat extremely difficult.

**Mechanisms of Arrhythmia Formation**

Arrhythmias arise from disorders of impulse formation or conduction (Box 2). Injury to cardiac tissue and arrhythmogenesis can occur from a variety of insults, including ischemia, fibrosis, inflammation, necrosis, or oxidative stress. In many patients with perioperative arrhythmias, the exact nature of the cardiac injury is unknown. Careful inspection of the electrocardiogram (ECG) and cardiac blood tests such as cardiac

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**Box 1**  
Diseases commonly associated with perioperative arrhythmias

- Splenic or hepatic masses or neoplasia
- Gastric dilatation and volvulus
- Pericardial disease requiring pericardiectomy
- Pulmonary disease requiring lung lobectomy
- Pulmonary stenosis undergoing balloon valvuloplasty

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**Box 2**  
Mechanisms of arrhythmogenesis and examples of commonly associated arrhythmias

- Disorders of impulse formation
  - Enhanced or suppressed normal automaticity: examples include sinus bradycardia, sinus arrest, sinus tachycardia, accelerated junctional or ventricular escape rhythms.
  - Abnormal automaticity: examples include supraventricular or ventricular premature beats or tachycardia.
  - Triggered activity (early or late afterdepolarizations): examples include supraventricular or ventricular premature beats or tachycardia.

- Disorders of impulse conduction
  - Bundle branch blocks: examples include right or left bundle branch block, which do not require treatment in the absence of other arrhythmias or ECG abnormalities.
  - Reentry circuits: examples include supraventricular or ventricular premature beats or tachycardia.
troponin-I might help identify likely causes. For instance, ST segment depression and increased cardiac troponin-I level are suggestive of myocardial ischemia and necrosis.

**Role of Transient Imbalances**

The importance of transient imbalances in the genesis of perioperative arrhythmias is well accepted. Transient imbalances are temporary circumstances that provide a substrate for arrhythmia formation and lead to the development or worsening of arrhythmias (Box 3). Transient imbalances in the presence of cardiac injury might be sufficient to lead to life-threatening arrhythmias. Identification and treatment of such imbalances plays an important role in the management of perioperative arrhythmias such that removal of transient imbalances could be all that is needed to reduce the incidence or severity of arrhythmia formation.

**APPROACH TO MANAGEMENT**

**Diagnosis of Perioperative Arrhythmias**

A complete review of ECG evaluation is beyond the scope of this article and the reader is referred to a variety of excellent sources for additional information. Several of the most important ECG characteristics of perioperative arrhythmias are listed in Box 4.

**Decision to Treat**

The decision of when to treat perioperative arrhythmias (Fig. 1) is depends on the answer to the following 2 questions:

- Are there clinical signs caused by altered hemodynamics during the arrhythmia?
- Does the arrhythmia predispose to a high risk of sudden arrhythmic death (ie, ventricular fibrillation)?

<table>
<thead>
<tr>
<th><strong>Box 3</strong></th>
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</thead>
<tbody>
<tr>
<td><strong>Partial list of transient imbalances that promote arrhythmogenesis</strong></td>
</tr>
<tr>
<td><strong>Autonomic imbalance</strong></td>
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<tr>
<td>- Increased sympathetic tone caused by stress, pain, sympathomimetic drugs.</td>
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<tr>
<td>- Increased parasympathetic tone caused by gastrointestinal, respiratory, or central nervous system disease; opioids; parasympathomimetic drugs; laryngoscopy; or tracheal intubation.</td>
</tr>
<tr>
<td><strong>Electrolyte or metabolic disturbances</strong></td>
</tr>
<tr>
<td>- Hyperkalemia caused by urinary obstruction, hypoadrenocorticism, renal failure, drugs, acidosis.</td>
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<tr>
<td>- Hypokalemia caused by vomiting, diarrhea, polyuria, diuresis, drugs.</td>
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<tr>
<td>- Metabolic acidosis caused by poor tissue perfusion, toxins, diabetic ketoacidosis, diarrhea.</td>
</tr>
<tr>
<td>- Respiratory acidosis caused by inadequate ventilation, pulmonary disease, poor cardiac output.</td>
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<tr>
<td><strong>Myocardial ischemia caused by poor cardiac output, structural heart disease, thromboembolism, hypoxemia, anemia.</strong></td>
</tr>
<tr>
<td><strong>Indwelling catheters or devices such as pacemakers, tracheal tubes, central venous or pulmonary artery catheters, chest tubes.</strong></td>
</tr>
<tr>
<td><strong>Systemic derangements associated with inflammation, cytokine release, sepsis, myocarditis, paraneoplastic syndromes.</strong></td>
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</tbody>
</table>
Patients with findings related to poor hemodynamics, such as hypotension, weakness, syncope, poor tissue perfusion, congestion, and hypothermia, benefit from interventions to correct the arrhythmia. In the absence of clinical findings indicating poor hemodynamics, the decision to treat is based on whether the arrhythmia is thought likely to degenerate into a clinically significant or fatal arrhythmia. Unlike the former indication, the decision to treat based on future risk of signs or sudden death is often extremely difficult to make. The following criteria are often used as indicators of increased risk for sudden death and triggers to begin treatment:

- Heart rate: rates greater than 180 beats per minute (bpm) (dog) or greater than 220 bpm (cat) or less than 50 bpm (dog) or less than 90 bpm (cat) tend to be associated with increased risk for clinical signs.
- Frequency: how often does the arrhythmia occur? Frequent or sustained (>30 seconds in duration) arrhythmias tend to be more associated with clinical signs.
- Origin of beats: are the ectopic beats originating from or above the AV node (supraventricular origin) or from the ventricle (ventricular origin)? Ventricular origin beats tend to be more associated with a high risk for hemodynamic compromise or sudden death versus supraventricular origin beats.
- AV synchrony: is synchrony between atrial and ventricular contraction maintained? Loss of AV synchrony is associated with greater risk for significant reduction in cardiac output. Examples of ECG rhythms with loss of AV synchrony include ventricular tachycardia, complete AV nodal block, and atrial fibrillation.

Box 4
Important electrocardiographic characteristics of perioperative arrhythmias

- Heart rate: what is the heart rate of the arrhythmia? Arrhythmias with rates greater than 180 bpm (dog) or greater than 220 bpm (cat) or less than 50 bpm (dog) or less than 90 bpm (cat) tend to be associated with increased risk for clinical signs.
- Frequency: how often does the arrhythmia occur? Frequent or sustained (>30 seconds in duration) arrhythmias tend to be more associated with clinical signs.
- Origin of beats: are the ectopic beats originating from or above the AV node (supraventricular origin) or from the ventricle (ventricular origin)? Ventricular origin beats tend to be more associated with a high risk for hemodynamic compromise or sudden death versus supraventricular origin beats.
- AV synchrony: is synchrony between atrial and ventricular contraction maintained? Loss of AV synchrony is associated with greater risk for significant reduction in cardiac output. Examples of ECG rhythms with loss of AV synchrony include ventricular tachycardia, complete AV nodal block, and atrial fibrillation.

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- Sustained versus nonsustained arrhythmias: those persisting for more than 30 seconds at a time tend to be more associated with increased risk for clinical signs.
- R-on-T phenomenon: single or multiple premature beats with rapid rates wherein the R wave of the ectopic beat encroaches on the preceding beat’s T wave are considered to signal increased risk for malignant ventricular arrhythmias.
- Presence of underlying structural heart disease: in particular, those diseases such as dilated, hypertrophic, restrictive, or arrhythmogenic cardiomyopathy that tend to be associated with electrical instability and sudden death.
- Cardiac troponin-I: markedly increased serum or plasma concentrations tend to be associated with more severe myocardial injury and dysfunction.

Arrhythmias that commonly cause hemodynamic compromise include the following:

- Rapid and sustained supraventricular or ventricular tachycardia.
- High-grade second-degree AV nodal block.
- Third-degree or complete AV nodal block.
- Sinus arrest greater than 4 seconds.
- Atrial fibrillation with rapid ventricular heart rate (>160–180 bpm).

These arrhythmias might require treatment because of their effects on hemodynamics regardless of the risk for development of ventricular fibrillation. In general,
arrhythmias that commonly do not require specific antiarrhythmic treatment include the following:

- Infrequent supraventricular or ventricular premature beats
- Accelerated ventricular escape rhythms (slow ventricular tachycardia) (Fig. 2)
- First-degree AV nodal block
- Infrequent second-degree AV nodal block
- Sinus arrest less than 2 seconds
- Atrial fibrillation with slow ventricular heart rate (<160–180 bpm)
Development of atrial fibrillation in the intraoperative or postoperative period in dogs with normal underlying myocardial function is common, especially in dogs receiving anesthetic or pain control medications that increase vagal tone, such as opioids. Increased parasympathetic tone to atrial myocardium increases the heterogeneity of refractoriness, which predisposes to development of primary or lone atrial fibrillation. In contrast with atrial fibrillation in animals with significant heart disease and underlying atrial enlargement, primary atrial fibrillation tends to produce slow ventricular rates and is often self-limiting and spontaneously resolves once the offending drug is reduced or discontinued. The author has had success in converting acute perioperative atrial fibrillation with intravenous (IV) procainamide (see Table 1) in canine patients. Other drugs, such as IV amiodarone, might be more efficacious but are associated with a high risk of adverse side effects.\(^\text{10,11}\)

**Therapeutic Options**

Therapeutic options include supportive measures and interventions to address transient imbalances as well as administration of antiarrhythmic drugs. Included in the supportive measures and interventions are the following:

- Removal of offending drugs
- Correction of acid-base status
- Correction of electrolyte derangements
- Correction of hypoxemia
- Correction of low cardiac perfusion
- Correction of hemostatic disorders
- Correction of device placement

Fig. 2. Six-lead ECG from a dog following splenectomy (25 mm/s; 10 mm/mV). The ECG shows an accelerated (idioventricular) ventricular escape rhythm (*solid arrow*). This rhythm is differentiated from ventricular tachycardia by its slow rate (approximately 130 bpm) and the timing of its onset, which is more consistent with an escape beat than a premature beat. Note that the ectopic ventricular rhythm begins after a pause in the sinus rhythm (*bar*). The preceding episode of accelerated ventricular escape rhythm ends with a fusion beat (*dotted arrow*), which is a common finding in patients with this rhythm.
• Reduction of stress or pain
• Correction of inflammatory or septic processes

If drug-based antiarrhythmic therapy is initiated, as much as possible, the specific antiarrhythmic is chosen based on the suspected mechanisms of arrhythmogenesis and identification of critical proarrhythmogenic components, vulnerable parameters, and subcellular drug targets (Fig. 3). The goal of therapy in patients with compromised hemodynamics is to improve cardiac output and tissue perfusion and alleviate any signs of congestion rather than complete suppression of the offending arrhythmia. Overly aggressive drug administration based on the ECG rhythm alone can predispose to adverse side effects, including proarrhythmia. The goal of therapy in patients at risk for sudden death is to normalize the myocardial electrical environment and increase the threshold for ventricular fibrillation. A list of common drugs used for therapy for perioperative arrhythmias is presented in Table 1. Initiation of drug therapy is accompanied by monitoring of ECG rhythm and hemodynamic status. In instances in which the mechanism of arrhythmogenesis is unclear or could have a variety of causes, empiric drug therapy based on the ECG diagnosis alone is attempted.

MONITORING AND CLINICAL OUTCOMES

The optimal clinical outcome of perioperative arrhythmias is return to normal sinus rhythm; however, as previously mentioned, some arrhythmias are well tolerated and might not require any (further) treatment. Monitoring of patients receiving antiarrhythmic therapy can include any or all of the following:

• Continuous or frequent ECG monitoring
• Echocardiography
• Blood pressure measurement
• Respiratory rate and effort
• Lactate concentration
• Acid-base and electrolyte status
• Cardiac troponin-I concentration

In some patients, a single or limited number of IV boluses of antiarrhythmic agents might be sufficient to suppress important arrhythmias as transient imbalances resolve. In other patients, constant-rate infusions of agents might be needed in the perioperative period for more sustained control of arrhythmias, and, in some cases, longer-term continuation of oral antiarrhythmic therapy might be indicated beyond hospital discharge. Arrhythmias are often sporadic, and assessment of whether or not a drug is effectively controlling both clinical signs and ECG rhythm after discharge requires close monitoring by the owner as well as frequent ECG rechecks or 24-hour ambulatory ECG (Holter) monitoring. In patients that are recovering well in the postoperative period, gradual weaning of IV or oral antiarrhythmics is performed in the hopes that return to health and resolution of transient imbalances render long-term antiarrhythmic therapy unnecessary.

Cardiac troponin-I is released by cardiac myocytes in proportion to cellular damage and necrosis. Increased circulating concentrations have been reported in dogs and cats with a wide variety of both cardiac and noncardiac diseases, including cardiomyopathy, valve disease, myocarditis, pericardial effusion, GDV, pulmonary disease, trauma, heat stroke, and hemoabdomen. In patients with heart disease or systemic inflammation, increased cardiac troponin-I level is associated with decreased survival. In general, markedly increased or increasing cardiac troponin-I concentrations are seen as evidence of ongoing myocardial injury and potential for electrical
**Fig. 3.** Drug selection based on suspected mechanisms of arrhythmogenesis.
instability, whereas decreasing concentrations suggest an acute injury that is resolving. The half-life of cardiac troponin-I in the dog is short and serial (daily or weekly) monitoring might help inform the need for antiarrhythmic therapy; however, further studies of the relationship between cardiac troponin-I and outcome in patients with perioperative arrhythmias are needed.

**COMPLICATIONS AND CONCERNS**

Complications of antiarrhythmic therapy for perioperative arrhythmias can include hypotension, central nervous system signs, proarrhythmia, exacerbation of congestion, or gastrointestinal signs, depending on the specific antiarrhythmic agent. During therapy, continuous ECG monitoring and either continuous or frequent blood pressure monitoring is recommended. If transient imbalances are identified and addressed, frequent reassessment for their reoccurrence is performed. As the patient recovers from its operative procedure, gradual withdrawal of antiarrhythmic agents is

### Table 1

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dog Dose</th>
<th>Cat Dose</th>
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<tbody>
<tr>
<td>Atenolol</td>
<td>0.25–2.0 mg/kg PO q12–24 h</td>
<td>6.25–12.5 mg PO q12–24 h</td>
</tr>
<tr>
<td>Atropine sulfate</td>
<td>0.01–0.04 mg/kg IV, IM, SC, prn</td>
<td></td>
</tr>
<tr>
<td>Diltiazem</td>
<td>0.5–2 mg/kg PO q8 h; 0.1–0.2 mg/kg IV bolus, then 2–6 μg/kg/min IV CRI</td>
<td>1.0–2.5 mg/kg IV q8 h; 0.1–0.2 mg/kg IV bolus, then 2–6 μg/kg/min IV CRI. Sustained-release diltiazem: Dilacor XR: 30–60 mg PO q24 h</td>
</tr>
<tr>
<td>Esmolol</td>
<td>50–100 μg/kg IV bolus every 5 min (up to 500 μg/kg max), then 25–200 μg/kg/min CRI</td>
<td></td>
</tr>
<tr>
<td>Glycopyrrolate</td>
<td>0.005–0.01 mg/kg IV, IM; 0.01–0.02 mg/kg SC</td>
<td></td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>0.04–0.09 μg/kg/min IV; 10 μg/kg IM, SC q6 h</td>
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<tr>
<td>Lidocaine</td>
<td>2 mg/kg slowly IV, interosseous (double the dose if given intratracheally) up to 3 boluses, then 30–80 μg/kg/min CRI</td>
<td>0.25–0.75 mg/kg IV over 5 min (use with caution because seizures can develop)</td>
</tr>
<tr>
<td>Mexiletine</td>
<td>4–8 mg/kg PO q8–12 h</td>
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<tr>
<td>Procainamide</td>
<td>2 mg/kg IV over 3–5 min up to total dose of 15 mg/kg, then 25–50 μg/kg/min CRI; 10–30 mg/kg IM, PO q6 h</td>
<td>2–5 mg/kg PO q6–8 h</td>
</tr>
<tr>
<td>Propranolol</td>
<td>0.02–0.06 mg/kg IV over 5–10 min; 0.2–1.0 mg/kg PO q8 h</td>
<td>Cat &lt;4.5 kg: 0.02–0.06 mg/kg IV over 5–10 min; 2.5–5 mg PO q8–12 h. Cat &gt;4.5 kg: 5 mg PO 8–12 h</td>
</tr>
<tr>
<td>Quinidine gluconate</td>
<td>6–20 mg/kg PO, IM q6 h; 6–20 mg/kg PO q8 h with sustained-release products</td>
<td></td>
</tr>
<tr>
<td>Quinidine sulfate</td>
<td>5–10 mg/kg IV (very slowly)</td>
<td></td>
</tr>
<tr>
<td>Sotalol</td>
<td>1–2 mg/kg PO q12 h</td>
<td>10 mg PO q12 h</td>
</tr>
<tr>
<td>Verapamil</td>
<td>0.05 mg/kg slow IV (1–2 min) boluses given at intervals of 10–30 min (to effect) to a maximum cumulative dose of 0.2 mg/kg</td>
<td>0.05 mg/kg slow IV, may repeat twice as described for dog</td>
</tr>
</tbody>
</table>

**Abbreviations:** CRI, constant rate infusion; IM, intramuscular; PO, orally; prn, as needed; q, every; SC, subcutaneous.
attempted. Long-term antiarrhythmic therapy is seldom required in patients with normal underlying cardiac structure. Persistent, refractory, or worsening arrhythmias might signal the presence of underlying cardiac injury or disease, and further diagnostics, such as radiographs or echocardiography, should be considered.

SUMMARY

When assessing perioperative arrhythmias, it is important to obtain an accurate electrocardiographic diagnosis, assess patient hemodynamic status during the arrhythmia, and determine whether underlying primary cardiac disease is present. The decision on whether to treat a specific arrhythmia should be based on the presence or absence of hemodynamic signs and risk of sudden death. If antiarrhythmic therapy is deemed necessary, consider the likely mechanisms of arrhythmia, address transient imbalances that contribute to arrhythmia formation, and select antiarrhythmic agents based on mechanism of action and arrhythmia diagnosis. Whether or not treatment is initiated, continuous and careful monitoring of ECG rhythm and hemodynamics is advisable.

REFERENCES
