

# Managing Myocardial Contusion and Arrhythmias

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## KEY WORDS

- myocardial contusion
- traumatic myocarditis
- cardiac arrhythmia
- antiarrhythmic therapy
- trauma

Cardiac arrhythmias are commonly observed following trauma in animals. These arrhythmias may result from direct trauma to the heart. Cardiac injuries are usually a result of blunt trauma caused by motor vehicles. Myocardial contusion, perforation of the myocardial wall or interventricular septum, ruptured chordae tendineae, tearing of major vessels, cardiac tamponade, and pericardial rents can all occur with blunt trauma.<sup>1</sup> Direct injury to the cardiovascular system following penetrating trauma is less commonly recognized, although gunshot wounds, arrows, and other penetrating objects can cause similar cardiac pathology.

A diagnosis of myocardial contusion is difficult to confirm without direct examination of the heart. Bruising to the myocardium can result from direct injury or compression of the heart by surrounding structures. Myocardial contusion can lead to arrhythmia formation, and a large contusion can disrupt the contractile action of the myocardium. In the latter case, hypotension and congestive heart failure can occur. Congestive heart failure is unlikely to result from cardiac bruising unless massive contusion is present or excessive intravenous fluid support has been given to the patient. Myocardial contusions resolve slowly and are unlikely to cause long-term complications.

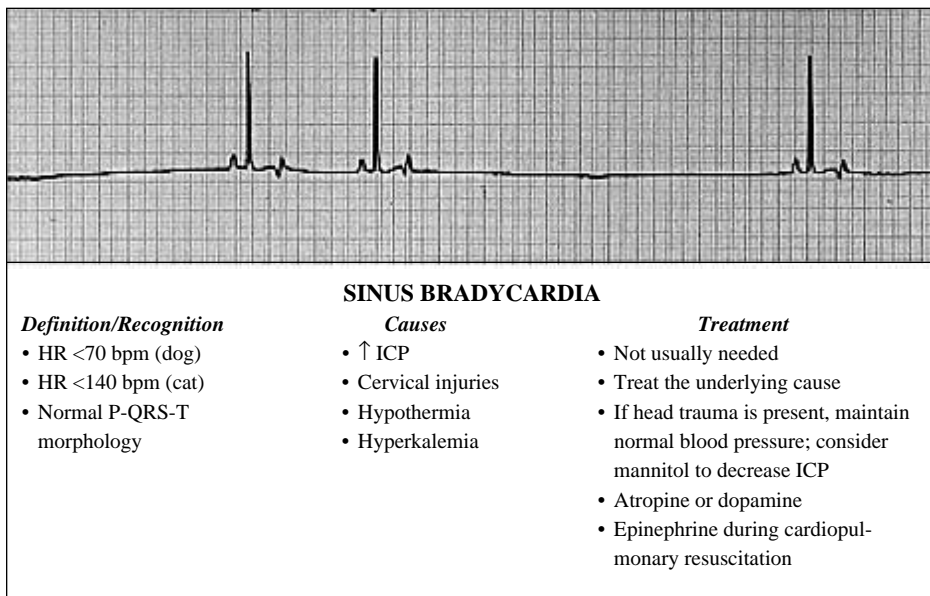
While the traumatic injuries described above may directly cause cardiac arrhythmia, several other causes of arrhythmias exist in this patient population. Electrolyte imbalance, hypoxemia, acid-base disturbance, anemia, sepsis, and concurrent neurologic injury<sup>1,2</sup> can all contribute to the development of cardiac arrhythmias. Dogs in particular are prone to developing cardiac arrhythmias in association with shock, especially

during the postresuscitation phase.<sup>2-4</sup> Necropsies done on these cases often identify small regions of myocardial necrosis, which presumably cause arrhythmia in many dogs and might explain the arrhythmias seen in dogs with trauma that exclusively involves the pelvis and rear limbs. Myocardial necrosis in these trauma cases has been proposed to be due to ischemia and reperfusion injury.<sup>5</sup> Many of the ventricular tachyarrhythmias are not evident at initial presentation but appear 2 to 72 hours after the traumatic incident, supporting a role for reperfusion injury in their genesis.

This article discusses arrhythmias that are frequently observed in traumatized patients and treatment considerations for each. Regardless of the type of arrhythmia, several clinical findings and therapeutic interventions should be considered for each animal with arrhythmia. Factors contributing to arrhythmogenesis include hypoxemia, tissue hypoxia, anemia, hypokalemia, and other electrolyte disturbances. Concurrent thoracic trauma is common, and hypoxemic animals should be treated with oxygen. Oxygen may even improve some arrhythmias in animals with only modest hypoxemia. Aggressive intravenous fluid support to maintain perfusion to all tissues is appropriate. In the trauma patient, a hematocrit of 20% to 25% may be sufficiently low to contribute to arrhythmia formation; transfusion often benefits these animals. Hypokalemia, which commonly occurs in anorexic trauma patients, is another treatable problem that can predispose to arrhythmia formation. Finally, pain can contribute to arrhythmia formation and analgesic medications are often underutilized in this patient population.

## SINUS BRADYCARDIA (Figure 1)

Although sinus bradycardia can be a normal rhythm in resting dogs and unstressed cats, this rhythm is uncommon in trauma patients unless the nervous system has been damaged. Fear, pain, anemia, hypoxemia, hypovolemia, and shock all tend to increase heart rate, which means that a slow heart rate is inappropriate in most trauma cases. Hypothermia causes a decrease in the rate of sinus node firing and can contribute to slowing of the heart rate, especially in cats. In cooler climates during the winter, trauma cases that are exposed to the elements may develop hypothermia and may be bradycardic. In premonitory animals, sinus bradycardia is often present just prior to cardiopulmonary arrest. Sinus bradycardia is often the first ar-



**Figure 1.** Electrocardiogram recorded from a dog using a transthoracic lead at 25 mm/sec and 1 cm/mV. The heart rate (HR) is approximately 30 bpm, and the rhythm is sinus bradycardia with sinus arrhythmia.

rhythmia identified during cardiopulmonary resuscitation.

Neurologic injury is the most common cause of a slow heart rate in trauma patients. While cervical injuries are recognized to cause bradycardia<sup>6</sup> (especially in anesthetized patients), the most common neurologic injuries causing sinus bradycardia are those that result in increased intracranial pressure (ICP). These animals usually manifest clear-cut evidence of neurologic injury with mental depression, focal neurologic deficits, hemorrhage from one or more sites on the head, and/or abnormalities of pupillary size.

Specific treatment of sinus bradycardia is rarely required. Treatment should be withheld if arterial pulse quality is good, mean arterial blood pressure is above 75 mmHg, mucous membrane color is pink, capillary refill time is acceptable, and the patient is not in imminent risk of cardiopulmonary arrest. Head-injured patients with sinus bradycardia and otherwise stable hemodynamic status are probably best served by appropriate treatment with oxygen, mechanical ventilation, hetastarch, hypertonic saline, mannitol, and/or corticosteroids. Hypothermic patients should be physically warmed and treated appropriately with warm intravenous fluids before drugs are administered to increase heart rate. Treatment with anticholinergic or sympathomimetic drugs is appropriate when cardiopulmonary arrest is imminent or hemodynamic instability is apparent. Use of an anticholinergic such as atropine (0.02–0.04 mg/kg IV or IM) or glycopyrrolate (0.011 mg/kg IV or IM) is usually the first-choice drug for sinus bradycardia not associated with car-

diopulmonary arrest. Sinus bradycardia that is unresponsive to anticholinergic drugs may be managed by intravenous infusion of catecholamines (i.e., dopamine, 5–20 µg/kg/min). Transvenous cardiac pacing can be used in the event that sinus bradycardia is unresponsive to the above therapies.

When sinus bradycardia is identified during cardiopulmonary resuscitation of the trauma patient, epinephrine is indicated. In the absence of a defibrillator, the standard dose or a low dose of epinephrine should be administered (0.02–0.05 mg/kg). If repeated dosing with the standard dose is ineffective (or if a defibrillator is available), the higher

epinephrine dose of 0.2 mg/kg can be used. The high dose of epinephrine is more likely to cause ventricular fibrillation than the low or standard dose.

### SINUS TACHYCARDIA (Figure 2)

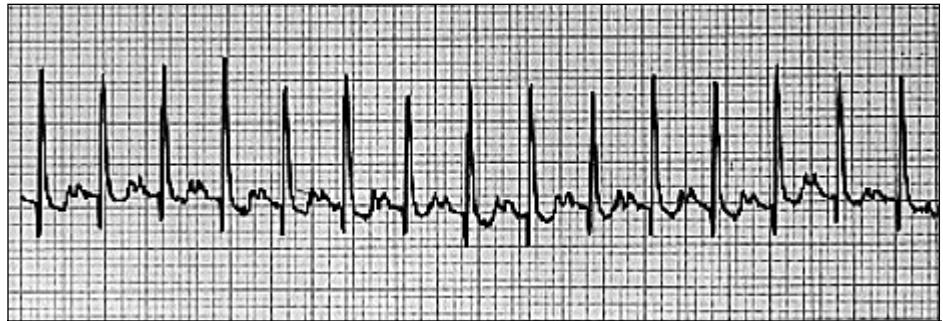
Sinus tachycardia is very common in traumatized dogs and cats. This cardiac rhythm is the appropriate cardiovascular response to hypovolemia, hemorrhagic or traumatic shock, pain, stress, and anemia.<sup>7</sup> The upper limit of heart rate for sinus rhythm in dogs is approximately 160 beats per minute (bpm), although many dogs with traumatic shock will have heart rates in excess of 200 bpm. One key in the trauma patient is to recognize this arrhythmia and distinguish it from supraventricular tachycardia. Sinus tachycardia should be appreciated as the appropriate arrhythmia in the setting of trauma and therefore is not an arrhythmia that requires specific therapy. The underlying cause of sinus tachycardia should be identified and treated. In most cases treatment with intravenous fluids, colloids, blood products, and, once blood pressure is stable, analgesics will lead to resolution of sinus tachycardia.

The differentiation of sinus tachycardia from supraventricular tachycardia can be difficult. Supraventricular tachycardia usually occurs at a faster rate than sinus tachycardia; however, there are no hard and fast criteria to differentiate the two based on heart rate alone. Because sinus tachycardia is the most common rhythm in trauma patients and because it resolves after successful treatment of shock, in most cases the distinction between the two only needs to be made when

tachycardia persists despite otherwise successful management of shock. A vagal maneuver can be helpful to distinguish these two arrhythmias. Both carotid sinus massage and ocular pressure can be performed to increase vagal tone, although I have had better luck with carotid sinus massage. This technique is accomplished by placing your fingers on the side of the neck at the level of the second cervical vertebra, along the lateral edge of the trachea near the jugular furrow. Compress the deep structures of the neck at this location against the vertebral body and massage in a cranial-to-caudal fashion. In response to a vagal maneuver, sinus tachycardia will often respond with a modest slowing of heart rate that returns to the prior heart rate once carotid massage is discontinued. In animals with supraventricular tachycardia, there is either no change in heart rate (unsuccessful vagal maneuver) or an abrupt termination of the arrhythmia with restoration of a much slower sinus rhythm.

### SUPRAVENTRICULAR TACHYCARDIA (Figure 3)

Supraventricular tachycardias in the trauma patient may result from atrial trauma or contusion, abrupt atrial stretch or dilatation resulting from rupture of chordae tendineae, hypoxemia, anemia, or electrolyte imbalance. Infrequent supraventricular premature depolarizations and nonsustained supraventricular tachycardia are uncommon problems in the trauma patient; when these arrhythmias occur, they rarely cause sufficient hemodynamic embarrassment to require antiarrhythmic therapy. Sustained supraventricular tachycardia in trauma patients may result from a spe-



#### SINUS TACHYCARDIA

##### Definition/Recognition

- HR >180 bpm (adult dog)
- HR >240 bpm (adult cat)
- Normal P-QRS-T complexes

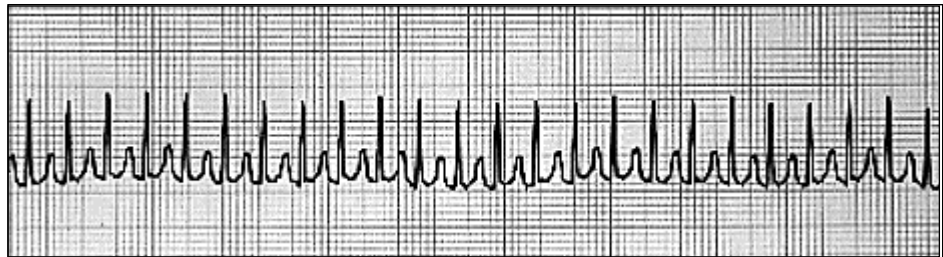
##### Causes

- Anemia
- Hypovolemia
- Shock
- Hypotension
- Pain/Fear

##### Treatment

- Specific treatment usually not necessary
- Treat the underlying cause
- Vagal maneuver to differentiate sinus tachycardia from supraventricular tachycardia

**Figure 2.** Lead II electrocardiogram recorded from a dog with traumatic injuries following an accident with a motor vehicle. Sinus tachycardia at a rate of 200 bpm is evident. Recorded at 25 mm/sec and 1 cm/mV.



#### SUPRAVENTRICULAR TACHYCARDIA

##### Definition/Recognition

- HR >180 bpm (adult dog)
- HR >240 bpm (adult cat)
- P wave often has different configuration than normal sinus P wave

##### Causes

- Atrial contusion
- Atrial stretch from ruptured chordae tendineae
- Ischemia
- Electrolyte imbalance
- Preexisting anomalous AV conduction (accessory pathway)

##### Treatment

- Vagal maneuver may abruptly terminate the arrhythmia
- Diltiazem 0.25 mg/kg slow IV bolus to maximum 0.75 mg/kg
- Esmolol, propranolol, and verapamil are also logical choices

**Figure 3.** Lead II electrocardiograms obtained from a dog with supraventricular tachycardia before (*top*) and after (*bottom*) administration of diltiazem. *Top:* The heart rate is 270 bpm, and the P waves are superimposed on the T waves. *Bottom:* Following administration of diltiazem the supraventricular tachycardia persists with an atrial rate of 270 bpm, but now every other P wave is blocked at the AV node (*arrows*) and the ventricular rate is 135 bpm. Recorded at 25 mm/sec and 1 cm/mV.

cific focal region of traumatic injury or from a reentrant atrioventricular (AV) nodal arrhythmia.

There appears to be a low risk for mortality directly due to supraventricular arrhythmias in the trauma patient, although they may contribute to hypoperfusion and slow patient recovery. In most cases the arrhythmia simply contributes to patient weakness and complicates overall patient management.

As discussed above, one of the potential clinical challenges is to differentiate sinus tachycardia from supraventricular tachycardia. Once shock has been treated, the appearance of sinus tachycardia in a previously stable animal is often a warning sign that hypotension, infection, pain, or another undiagnosed condition exists. Specific treatment of the cause is indicated, and antiarrhythmic therapy is not necessary. In contrast, supraventricular tachycardia often exists without a specific cause and requires specific antiarrhythmic therapy.

Antiarrhythmic therapy should be considered for animals with rapid supraventricular arrhythmias (rates above 260 bpm in the cat or above 200–220 bpm in the dog). As described above, a vagal maneuver may abruptly terminate supraventricular tachycardia and allow normal sinus rhythm to resume. In unstable animals or those with hemodynamically unstable and sustained (continuous) arrhythmias, intravenous therapy is preferred. Diltiazem can be administered in an initial 0.25 mg/kg IV bolus over 2 minutes. Subsequent 0.25 mg/kg boluses can be repeated at 15 minute intervals until conversion occurs or until a maximum dose of 0.75 mg/kg has been administered.<sup>2</sup> Alternatively, verapamil can be given intravenously in a series of 0.05 mg/kg boluses (slowly) up to a total dose of 0.15 mg/kg.<sup>8</sup> Propranolol can be administered intravenously at 0.02 to 0.06 mg/kg (slowly) every 8 hours; however, esmolol is a short-acting beta-blocker that is generally preferred over propranolol in the emergency setting.<sup>2</sup> Esmolol is given in incremental doses of 0.05 to 0.1 mg/kg boluses every 5 minutes up to a maximum dose of 0.5 mg/kg. Esmolol's effects are short-lived; if arrhythmia conversion does not occur, other drugs with negative inotropic properties (i.e., diltiazem or verapamil) can usually be safely given 30 minutes after esmolol administration.

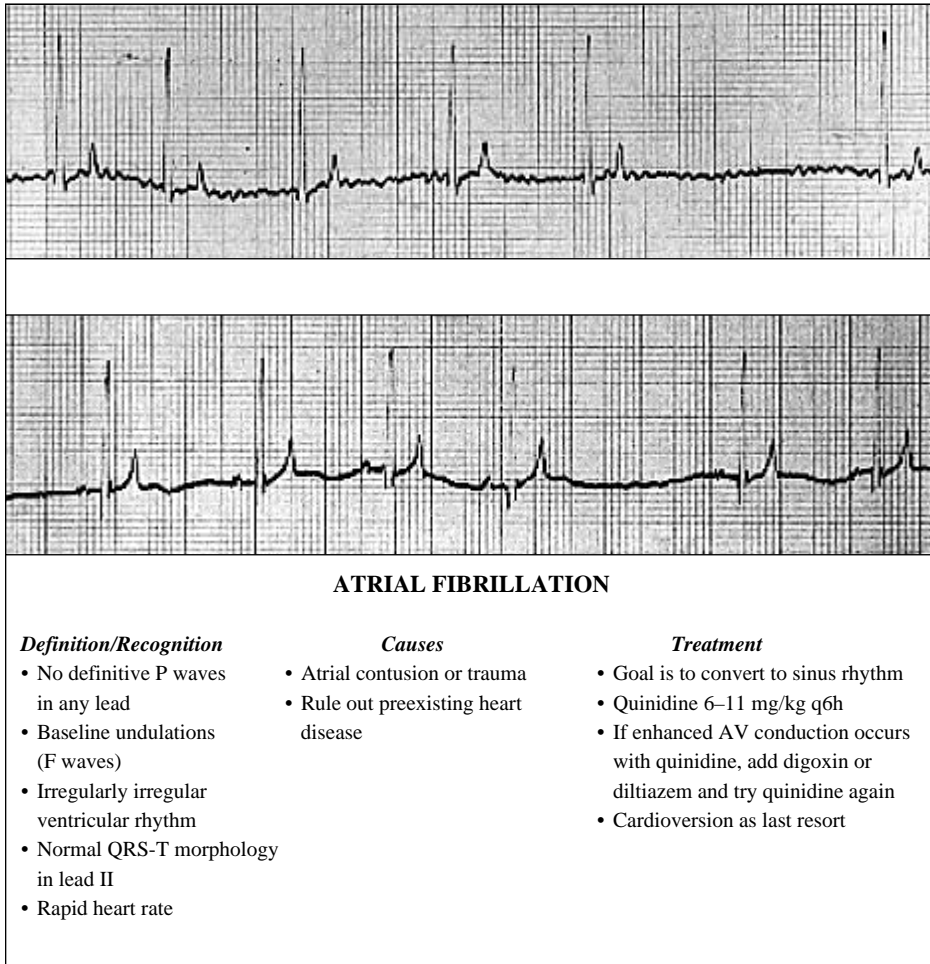
For stable individuals, oral administration of diltiazem or a beta-blocker may be sufficient. Unlike sustained supraventricular tachycardia associated with an accessory pathway, supraventricular tachycardia due to trauma is short-lived and, following initial treatment, the arrhythmia resolves and does not recur. Chronic therapy with oral antiarrhythmics is rarely required.

## ATRIAL FIBRILLATION (Figure 4)

Atrial fibrillation is extremely uncommon in cats in the absence of significant underlying cardiac disease. If it is identified in a cat with traumatic injuries, a high degree of suspicion should exist for concurrent underlying cardiac disease. Similarly, atrial fibrillation is uncommon in traumatically injured dogs weighing less than 20 kg. This arrhythmia is more common in dogs weighing more than 20 kg that have experienced significant thoracic trauma. In many of these cases, there is no preexisting cardiac disease and the arrhythmia results from myocardial contusion or as an arrhythmia induced at the time of the traumatic incident.

Atrial fibrillation is recognized by a lack of P wave, fine or coarse undulations in the baseline of the electrocardiogram (ECG), an irregular ventricular rhythm with a normal or upright QRS-T configuration in lead II, and typically a fast heart rate. Once shock has been treated, the heart rate often decreases to less than 160 bpm in dogs with traumatically induced atrial fibrillation.

The goal of treatment of atrial fibrillation in the trauma patient is conversion to sinus rhythm. In contrast to treatment of dogs with atrial fibrillation due to underlying cardiac disease, where conversion to sinus rhythm is uncommon and the goal is simply heart rate reduction, many dogs with atrial fibrillation of traumatic origin will convert to sinus rhythm. Conversion to sinus rhythm can occur spontaneously. It is unusual for atrial fibrillation to cause sufficient hemodynamic upset to necessitate immediate conversion. Since spontaneous conversion can occur during the first 48 hours after trauma, I recommend that pharmacologic or electrical conversion not be attempted until the dog has been stabilized and is recovering from other injuries. One may wish to exclude any preexisting cardiac disease by obtaining thoracic radiographs and echocardiography. Quinidine (6–11 mg/kg IM q6h) is usually the first choice for a pharmacologic attempt at conversion, and most dogs convert to sinus rhythm within the first 24 hours of therapy. Quinidine's vagolytic effect can increase AV conduction and lead to a rapid ventricular response. When this occurs, quinidine administration can be stopped and digitalis can be initiated to slow this accelerated AV conduction. Alternatively, diltiazem (0.5–1.5 mg/kg PO q8–12h) has been reported to convert atrial fibrillation to sinus rhythm in some dogs and can also be used to slow the rate of conduction through the AV node prior to a second attempt with quinidine. ECG synchronized cardioversion may be attempted if quinidine and diltiazem are unsuccessful in restoring sinus rhythm;



**Figure 4.** Electrocardiograms recorded from a dog with atrial fibrillation before (*top*) and after (*bottom*) quinidine administration. *Top:* The rhythm is irregular, and there are no P waves. Fibrillation waves are evident in the baseline of the ECG tracing. The heart rate is 70 bpm, much slower than the heart rate usually seen in dogs with atrial fibrillation due to advanced heart disease. *Bottom:* Following quinidine administration the dog is in sinus rhythm with a heart rate of 70 bpm. P waves are again evident, although the variable morphology of the P waves is due to a wandering pacemaker. Both electrocardiograms recorded at 25 mm/sec and 1 cm/mV.

however, this technique has some risk and requires heavy sedation or anesthesia to be performed safely.

### VENTRICULAR PREMATURE DEPolarIZATIONS AND VENTRICULAR TACHYCARDIA (Figure 5)

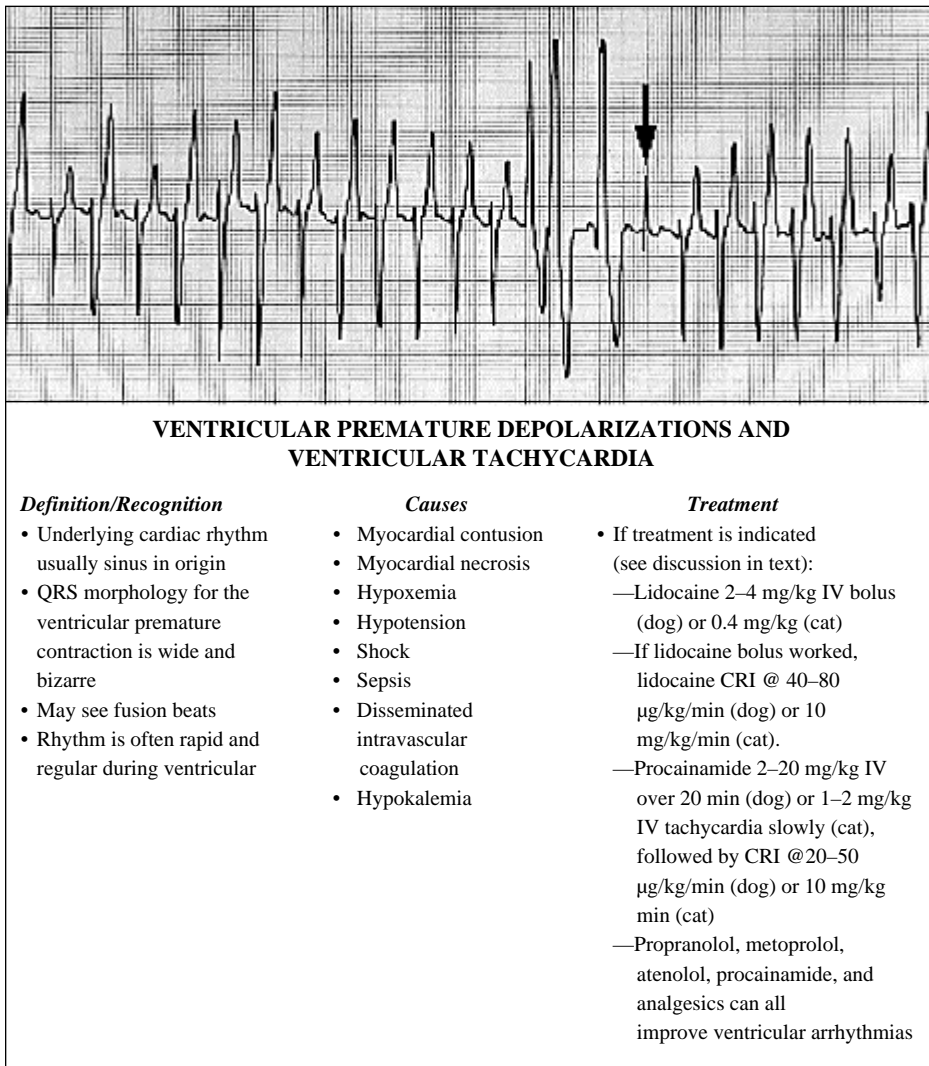
Ventricular arrhythmias are very common in dogs with traumatic injuries. Factors that contribute to the development of these arrhythmias include direct ventricular injury or myocardial contusion, electrolyte imbalance, hypoxemia, acid-base disturbance, anemia, and multifocal microscopic myocardial necrosis. Although the syndrome of ventricular arrhythmias in the canine trauma patient has often been termed “myocardial contusion” or “traumatic myocarditis,” many cases have neither myocardial contusions nor inflammatory myocarditis. Nevertheless, both of these terms

have been used to describe the syndrome of ventricular arrhythmias seen in dogs following a traumatic injury. In my practice, these arrhythmias are more common in medium- to large-breed dogs and are less commonly seen in dogs younger than 6 months of age. Ventricular arrhythmias are uncommon in cats with traumatic injuries.

There has been considerable debate regarding the risk of mortality due to these arrhythmias. Since these arrhythmias are often relatively benign and resolve within 72 hours in many animals, some clinicians have recommended that the best approach to management of these arrhythmias is to “turn off the ECG monitor.” However, ventricular fibrillation and cardiac arrest can occur in animals being monitored and/or treated for trauma-induced ventricular arrhythmias. It is my opinion that although the majority of these arrhythmias are benign, there is a small proportion of cases in which the arrhythmia is lethal and that these arrhythmias should not be ignored. Certainly, in most dogs with trauma-induced ventricular arrhythmias, the arrhythmia simply complicates the clinical

course, contributes to patient weakness, and is a source of distress for the clinician and owner.

Many ventricular arrhythmias in this setting do not require specific treatment. Correction of acidosis, electrolyte disturbance, anemia, and hypoxemia often improves overall patient status and reduces arrhythmia frequency. Analgesics may reduce arrhythmia frequency and should also be administered whenever indicated. No therapy is indicated for isolated ventricular premature depolarizations, no matter how frequently they occur, unless they are accompanied by clinical findings of syncope or collapse. The heart rate during ventricular arrhythmia can also factor into the decision of whether to initiate antiarrhythmic therapy. In many dogs, the heart rate during the arrhythmia is slow and ventricular arrhythmia occurs only during periods of slowed sinus node firing. In these cases the



**Figure 5.** Lead II electrocardiogram obtained from a dog 4 hours after initial treatment of traumatic injuries. Multifocal ventricular tachycardia is present at a rate of 200 bpm. A single P-QRS-T complex of sinus origin is evident (arrow). Several P waves are present in the baseline between the ventricular premature depolarizations. Recorded at 25 mm/sec and 1 cm/mv.

sinus rhythm is usurped by a ventricular rhythm whenever the sinus node firing rate falls below a critical heart rate. These ventricular rhythms often occur at a slow heart rate (100 to 150 bpm) and have been termed *idioventricular tachycardias* or *accelerated ventricular rhythms*. There are no data to support specific therapeutic recommendations for these accelerated ventricular arrhythmias; however, these arrhythmias are infrequently treated when the heart rate is less than 150 bpm, the patient is stable, and cardiopulmonary arrest does not seem imminent.

Treatment of ventricular tachycardia is usually indicated in animals with clinical signs resulting from the arrhythmia such as weakness, difficulty upon rising, mucous membrane pallor or delayed capillary refill time, hypotension, collapse, or syncope. Animals with sustained ventricular tachycardia (>15–30 seconds) are usually treated, especially when the heart rate dur-

ing the ventricular arrhythmia is in excess of 180 bpm. Dogs that are weak, that have severe concurrent respiratory tract injury, and that are thought to be at high risk of cardiopulmonary arrest are more likely to be treated for ventricular arrhythmias than are stable individuals.

Hemodynamically unstable ventricular tachycardia in the dog is usually first treated with a 2 mg/kg intravenous bolus of lidocaine. This bolus may be repeated up to a total dose of 8 mg/kg, although doses above 4 mg/kg are increasingly likely to result in either vomiting or seizures. If the animal has a beneficial response to lidocaine, a continuous rate infusion (CRI) should then be initiated at 40 µg/kg/min and increased as needed up to 80 µg/kg/min. In many cases, additional boluses of lidocaine may be required to control the arrhythmia until the infusion results in steady state blood levels of lidocaine. Serum potassium levels should be maintained in the normal range as many class I antiarrhythmic drugs are less effective in the face of hypokalemia. If the initial lidocaine boluses fail to im-

prove the arrhythmia, procainamide can be administered intravenously by CRI (20–50 µg/kg/min) or intramuscularly (6–15 mg/kg q4–6h). Although most dogs need antiarrhythmic drug therapy for only 2 to 4 days, one advantage of procainamide is that it can be continued orally if chronic antiarrhythmic therapy is deemed necessary. In animals that are unresponsive to class I antiarrhythmic drugs, the addition of a beta-blocker (propranolol, 0.2–0.4 mg/kg PO q8–12h, or metoprolol, 0.2–0.4 mg/kg PO q12h) should be considered if the patient has responded adequately to treatment for shock. Treatment with beta-blockers is inadvisable in animals that are receiving or may be in need of positive inotropic drugs such as dopamine or dobutamine as beta-blockade will negate the beneficial effects of these drugs.

Trauma-induced ventricular arrhythmias often are self-limiting and in most cases resolve within 3 to 10

days. The therapeutic endpoint is not necessarily total alleviation of arrhythmia; adequate therapy may be reduction of the heart rate to less than 140 bpm and alleviation of arrhythmias that result in hemodynamic compromise. In most cases, antiarrhythmic therapy can be discontinued within 48 to 72 hours. When arrhythmia persists, chronic oral therapy is initiated with either procainamide or a beta-blocker. In these cases, I instruct the owner to discontinue antiarrhythmic medications 24 hours prior to a recheck examination performed 7 to 10 days after hospital discharge. If a long lead II ECG is free from arrhythmia at that time, all antiarrhythmic medication is discontinued.

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