Myocardial Injury Secondary to Blunt Thoracic Trauma in Dogs: Diagnosis and Treatment*

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ABSTRACT: Blunt thoracic trauma to both humans and dogs may result in myocardial injury. Myocardial injuries commonly manifest as arrhythmias within the first 48 hours following a traumatic event. These injuries may be occult, or clinical signs may be delayed and are frequently overlooked. This article discusses both traditional and novel diagnostic methods used to detect myocardial injuries and provides guidelines for the treatment of myocardial injuries in dogs.

Although the true prevalence of myocardial injuries remains controversial, it is clear that they are often overlooked during the initial physical examination. The initiation of arrhythmias in dogs with myocardial injuries is thought to be caused by alterations in cation transport that result in lowering of the resting membrane potential of injured myocytes, making them susceptible to aberrant depolarization. This article focuses on an organized approach for diagnosis and treatment of myocardial injuries and arrhythmias in dogs with blunt thoracic trauma.

DIAGNOSIS

The diagnostic gold standard in the identification of cardiac injury remains the gross or histologic examination of the heart.1–3 The search for a single, noninvasive, sensitive, and specific diagnostic modality to identify myocardial injuries secondary to blunt thoracic trauma is an active area of scientific investigation. Currently, a combination of tests is used to diagnose traumatic myocardial injuries in dogs. These methods include electrocardiography (ECG), cardiac biomarkers, echocardiography, and computed tomography (CT).

Electrocardiographic abnormalities in dogs with blunt thoracic trauma may not be evident for up to 48 hours after injury.

Arrhythmias secondary to myocardial injury are often overlooked by intermittent electrocardiographic monitoring, and continuous monitoring should be considered in high-risk patients.

Troponins, cardiac-specific proteins, have been shown to be an effective biomarker of myocardial injury in dogs.

Treatment of myocardial injuries is currently aimed at maintaining optimal cardiac output and suppressing life-threatening arrhythmias.

*See companion article on p. 934.
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An understanding of the mechanism of injury, the awareness of associated injuries, and a high index of suspicion for myocardial injury are essential in making a diagnosis. In human medicine, the most commonly used tests to diagnose myocardial injury include electrocardiography, thoracic radiography, echocardiography, and serum myocardial isoenzyme/protein analysis.

Myocardial injury should be suspected in dogs involved in motor vehicle accidents and that have the following associated injuries: (1) fractures of the extremities, the spine, or pelvis; (2) external evidence of thoracic trauma; (3) radiographic evidence of chest trauma, such as pulmonary contusions, pneumothorax (Figure 1), hemothorax, diaphragmatic rupture, and rib/scapular fractures; and (4) neurologic injury. Lead II electrocardiography (ECG) should be performed on dogs with any of these injuries during the initial examination and repeated intermittently (i.e., every 12 to 24 hours). It is important to note that ECG abnormalities may not be apparent for up to 48 hours after blunt chest trauma in humans and dogs.

Continuous ambulatory ECG monitoring (Holter monitoring) was recently identified as a sensitive tool for the detection of arrhythmias in severely injured dogs. Snyder et al prospectively examined 30 traumatized dogs using Holter monitoring and found ventricular ectopy within 24 hours in 29 subjects, whereas ECGs performed on presentation detected ventricular ectopy in only four of the dogs.

Within the first 48 hours of injury, echocardiographic examination should be considered in severely traumatized dogs with a poor response to resuscitative efforts and evidence of thoracic injuries, even if there are no ECG abnormalities. In dogs, transthoracic echocardiography can identify and localize the structural and functional abnormalities of injured myocardium due to blunt chest trauma. The distinctive transthoracic echocardiographic features of regional myocardial injury secondary to blunt thoracic trauma in dogs include: (1) increased end-diastolic wall thickness; (2) impaired contractility, indicated by decreased fractional shortening; (3) increased echogenicity; and (4) localized areas of echolucency consistent with intramural hematomas. A recent prospective study used transthoracic echocardiography to identify myocardial injury in seven dogs with known thoracic trauma. Six of the dogs in this study had no echocardiographic abnormalities. One dog had a regional wall motion abnormality in the left ventricular free wall and decreased fractional shortening measurements that resolved when the examination was repeated 2 days later. Although use of transesophageal echocardiography is uncommon in veterinary practice, it has been shown to be more accurate than transthoracic echocardiography in the detection of thoracic cardiovascular injuries in humans with blunt chest trauma.

Other modalities used in the diagnosis of myocardial injury in humans include serum myocardial isoenzyme/protein analysis and nuclear cardiology. Nuclear cardiography can be used to simultaneously assess ventricular function and the direction and magnitude of cardiac shunts and to map myocardial perfusion. The benefits of nuclear cardiography include a short study acquisition time, noninvasiveness, and the ability to conduct the test without sedation. It appears that nuclear studies are more sensitive than thoracic radiography in evaluating the significance of cardiac injuries because functional changes of the myocardium often precede changes in cardiac size and shape.

Measurement of left ventricular ejection fraction using radionuclide ventriculography has been shown to correlate well with cardiac output measured by thermodilution, M-mode, and two-dimensional echocardiography in healthy anesthetized dogs. Disadvantages include the limited availability of nuclear studies, arrhythmias affecting image acquisition, and the fact that both first pass (image acquisition obtained from a radioactive bolus with a few cardiac cycles recorded) and gated studies (image acquisition obtained after equilibration of the radioactive bolus, synchronized with ECG, with hundreds of cardiac cycles recorded) must be performed to account for the overlap of the right and left sides of the heart. Historically, the isoenzyme used to identify myocardial necrosis in human medicine is creatinine phosphokinase–myocardial band (CPK-MB). Several human clinical studies have demonstrated CPK-MB isoenzyme measurement in traumatized patients is neither sensitive, specific, nor predictive of functional cardiac dysfunction.
abnormalities. In contrast, a recent metaanalysis of humans with blunt cardiac trauma found that abnormal CPK-MB, when coupled with abnormal ECG findings, correlated directly with cardiac complications in trauma patients that required treatment.

There have been several recent reports concerning the use of cardiac-specific proteins to diagnose myocardial injuries in humans. These proteins or troponins form a complex, which is located on the thin filament of the contractile apparatus in both striated and skeletal muscle tissues (Figure 2). Troponins consist of three proteins, each identified by a single letter (i.e., T, I, C). Troponins interact to regulate the force and velocity of muscle contraction by modulating the interaction of calcium with actin and myosin. The isoforms of the troponin proteins expressed in cardiac muscle are different from those in skeletal muscle. Current troponin testing is based on immunologic detection (monoclonal antibody) of the cardiac-specific isoforms of two of the three troponins (troponin T [cTnT] and troponin I [cTnI]). In both humans and dogs, the detection of troponins in the circulation occurs within hours of cardiac myocyte injury and abnormal serum elevations have been shown to be present for up to 7 days. The troponin structure is highly conserved across many differing species, allowing for canine application of immunologic tests currently in use at human health care facilities.

In two separate studies, O’Brien et al demonstrated high concentrations of cTnT in the canine heart. In a canine model of myocardial infarction, release of cTnT by injured myocytes resulted in 1,000- to 10,000-fold increases in serum concentrations of cTnT within 3 hours of injury. The elevations in cTnT were highly correlated to infarct size. Using this model, O’Brien et al also determined that cTnT was an effective biomarker in traumatic cardiac injury and puncture. Another study demonstrated similar elevations in cTnI using a canine heart model postinfarction but found that these elevations did not correlate well with subsequent infarct size. Schober et al measured cTnI, cTnT, and CPK-MB in 40 healthy dogs and found that these enzymes were not detectable in most patients in the study group. A recently published abstract described normal cTnI values (mean, 0.02 ng/ml; range, 0.0 to 0.07 ng/ml) for dogs. In addition to establishing a normal cTnI range for dogs, the study found elevated cTnI levels (>0.07 ng/ml) in five dogs with known cardiac disease or thoracic trauma, suggesting cTnI levels >0.07 ng/ml may be correlated with myocardial damage.

A prospective study of 71 human trauma patients in a level-one trauma center compared several diagnostic modalities and found that echocardiography, ECG, CPK-MB (>4%), and cTnT have poor sensitivity but high specificity in the diagnosis of myocardial injury (Table 1). A similar canine study evaluated the ability of cTnI, cTnT, CPK-MB, and ECG to detect myocardial injury in traumatized dogs. In this study, cTnI was found to be the most sensitive indicator of myocardial cell injury. Although these cardiac-specific markers appear to be extremely sensitive and specific for myocardial injuries as well as appearing to be of greater diagnostic value than CPK-MB, their clinical value remains controversial in the human literature and requires further investigation involving veterinary patients.

Figure 3 outlines a suggested diagnostic protocol for the detection of suspected myocardial injury in dogs. Although the diagnosis of myocardial injury remains difficult to confirm without direct examination of the heart, it should be suspected in traumatized animals that develop arrhythmias in the face of properly managed shock, pulmonary injuries, acid–base disturbances, and pain. Currently, Holter monitoring appears to be the most sensitive, least invasive, and most readily

![Figure 2](image-url) - Depiction of the microanatomical arrangement of troponin complex, actin, and tropomyosin.

<table>
<thead>
<tr>
<th>Diagnostic Method</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Echocardiogram (transthoracic or transesophageal)</td>
<td>0.12</td>
<td>0.98</td>
</tr>
<tr>
<td>ECG</td>
<td>0.38</td>
<td>0.93</td>
</tr>
<tr>
<td>CPK-MB (&gt;4%)</td>
<td>0.12</td>
<td>0.96</td>
</tr>
<tr>
<td>Troponin T (&gt;0.2 µg/L)</td>
<td>0.27</td>
<td>0.91</td>
</tr>
</tbody>
</table>

Figure 3—Diagnostic screening protocol for blunt cardiac injuries in canine trauma patients. (Modified from Mucha P: Blunt myocardial injury [myocardial contusion], in Cameron JL [ed]: Current Surgical Therapy, ed 6. St. Louis, Mosby, 1998, pp 1004–1008.)
available indicator of arrhythmias in dogs with suspected myocardial injuries. The lack of immediate Holter interpretation (rapid turnaround time) may limit the practical application of this modality to veterinarians. Other forms of continuous ECG monitoring, such as single patient monitors and telemetry, would likely provide a similar advantage over intermittent ECGs without the delays in interpretation encountered with Holter monitors. In the future, as more controlled studies are published, troponin testing in dogs may emerge as the most rapid, noninvasive, economical, and sensitive indicator of myocardial injury.

TREATMENT

Treatment of myocardial injuries is typically aimed at suppressing potentially life-threatening arrhythmias. Treatment of most arrhythmias is not recommended if the patient has good arterial pulse quality, mean arterial pressure greater than 75 mm Hg, pink mucous membranes, and capillary refill time ≤ 2 seconds and is not at risk for cardiopulmonary arrest. Antiarrhythmic therapy is recommended when patients are “at risk,” meaning those that have been properly stabilized (i.e., received adequate fluids, electrolytes, oxygen, pain control) yet still develop severe arrhythmias, such as multiformal premature ventricular complexes, ventricular tachycardia, and the R on T phenomenon (Figure 4). Treatment is also required when arrhythmias are accompanied by clinical evidence of decreased cardiac output, such as hypotension, weakness, pale mucous membranes, delayed capillary refill time, collapse, or syncope. Initiation of treatment is indicated when the detected arrhythmia is sustained (>15 to 30 seconds) and/or has a ventricular rate that exceeds 180 bpm in dogs.

Ventricular ectopy in traumatized dogs that result in hemodynamic instability is initially treated with a 2 mg/kg IV bolus of lidocaine. Lidocaine boluses may be repeated over 10 to 20 minutes until a cumulative dose of 8 mg/kg is given. If the therapeutic response is favorable to IV bolus therapy, an IV constant rate-infusion (CRI) of 40 to 80 µg/kg/min should be initiated. Additional boluses may be required to suppress the arrhythmia while steady state blood levels of lidocaine are achieved by the CRI. The high end of the recommended doses of lidocaine may cause vomiting or seizures; therefore, administration should be slowed or temporarily discontinued if these signs develop. When lidocaine fails to resolve ventricular ectopy, procainamide may be administered IV or IM (6 to 15 mg/kg q4–6h). Procainamide may also be administered as a CRI (10 to 40 µg/kg/min) or given orally (sustained-release formulation, 20 mg/kg tid) when appropriate. Hypotension and atrioventricular conduction block are serious potential side effects of procainamide administration.

Tocainide (20 mg/kg bid to tid PO) and mexiletine (4 to 8 mg/kg tid PO) may suppress arrhythmias unresponsive to other class I antiarrhythmics. A disadvantage of both tocainide and mexiletine is that oral
administration is required and may not be optimal in severely traumatized dogs. Side effects of tocainide, such as nausea, vomiting, and anorexia, occur commonly, whereas side effects associated with mexiletine, such as excitement or depression, are less frequently observed. Because these class I antiarrhythmics are less effective in patients with hypokalemia, serum electrolyte balance in animals unresponsive to these antiarrhythmics should be monitored. The addition of a β-blocker (e.g., propanolol, metoprolol, atenolol, sotalol) should be considered for the treatment of ventricular ectopy in traumatized dogs that have been appropriately treated for shock and are not receiving positive inotropic medications yet remain unresponsive to class I antiarrhythmics. Esmolol, an ultrashort-acting IV β-blocker, may be used to test the efficacy of β-blockers in treating ventricular arrhythmias that have not responded to other medications. Serious potential side effects, such as atrioventricular block, hypotension, bronchoconstriction, and decreased cardiac contractility, are associated with the use of β-blockers. Table 2 provides a summary of the medications used to treat ventricular ectopy in traumatized dogs.

Trauma-induced arrhythmias that do not fulfill the above guidelines for treatment are often self-limiting and, in most cases, resolve within 3 to 10 days. The therapeutic goal of these recommendations is not necessarily total alleviation of the arrhythmia; adequate therapy may be reduction of the heart rate (<140 bpm) or the return of hemodynamic stability. Although antiarrhythmic therapy can be discontinued within 48 to 72 hours in most cases, it is recommended that intermittent ECG monitoring continue up to 1 week after discharge. Before reexamination, antiarrhythmic medications should be discontinued for a minimum of 24 hours. Holter monitoring, if available, would be the most sensitive way to detect complete resolution of arrhythmias after discontinuing antiarrhythmic med-

Table 2. Medications Used to Treat Ventricular Arrhythmias In Dogs with Blunt Thoracic Trauma

<table>
<thead>
<tr>
<th>Drug</th>
<th>Classification</th>
<th>Route of Administration</th>
<th>Dosage</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procainamide</td>
<td>IA</td>
<td>IV</td>
<td>6–8 mg/kg slow IV, 25–40 µg/kg/min CRI</td>
<td>Nausea, hypotension, arrhythmias</td>
</tr>
<tr>
<td></td>
<td></td>
<td>IM</td>
<td>6–20 mg/kg q4–6h</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>PO</td>
<td>20 mg/kg tid, sustained-release</td>
<td></td>
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<tr>
<td>Lidocaine</td>
<td>IB</td>
<td>IV</td>
<td>2–4 mg/kg slow IV, repeat to a max of 8 mg/kg; 25–80 kg/min CRI</td>
<td>CNS excitement, seizures, nausea, hypotension, arrhythmias</td>
</tr>
<tr>
<td>Mexiletine</td>
<td>IB</td>
<td>PO</td>
<td>4–8 mg/kg tid</td>
<td>Depression, tremors, vomiting, hypotension</td>
</tr>
<tr>
<td>Tocainide</td>
<td>IB</td>
<td>PO</td>
<td>5–20 mg/kg tid</td>
<td>CNS excitement, seizures, nausea, hypotension, arrhythmias, tremors, weakness</td>
</tr>
<tr>
<td>Propranolol</td>
<td>II</td>
<td>IV</td>
<td>0.02–0.06 mg/kg slow IV, titrate to effect 0.2–0.4 mg/kg bid–tid</td>
<td>Bradycardia, AV block, hypotension, decreased cardiac contractility, bronchospasm</td>
</tr>
<tr>
<td>Metoprolol</td>
<td>II</td>
<td>PO</td>
<td>0.2–0.4 mg/kg bid</td>
<td>Bradycardia, AV block, hypotension, decreased cardiac contractility, bronchospasm</td>
</tr>
<tr>
<td>Esmolol</td>
<td>II</td>
<td>IV</td>
<td>0.1–0.5 mg/kg slow IV bolus; 20–50 µg/kg/min CRI</td>
<td>Bradycardia, AV block, hypotension, decreased cardiac contractility, bronchospasm</td>
</tr>
<tr>
<td>Sotalol</td>
<td>II–III</td>
<td>PO</td>
<td>0.5–2.0 mg/kg bid</td>
<td>Proarrhythmic, bradycardia, decreased cardiac contractility, depression, anorexia</td>
</tr>
</tbody>
</table>

*Please consult text for indications of treatment. AV = atrioventricular; CNS = central nervous system; CRI = constant-rate infusion.
ications. If Holter monitoring is not available, intermittent lead II ECG monitoring can be performed. Initially intermittent ECG monitoring should be performed every 12 to 24 hours posttrauma; however, as arrhythmias begin to resolve, ECGs can be performed less frequently. When arrhythmias are persistent, chronic oral therapy may be initiated. Patients with a suspected myocardial injury often require anesthesia for correction of other injuries. If a patient with a myocardial injury must undergo anesthesia, drugs should be selected that are least likely to induce arrhythmias (e.g., acepromazine, butorphanol, isoflurane, glycopyrrolate). Halothane, atropine, and the thiobarbiturates would be poor choices as they tend to exacerbate dysrhythmias and sensitize the heart to catecholamine-induced arrhythmias.

SUMMARY

The available literature regarding the significance of myocardial injuries secondary to blunt thoracic trauma is controversial. A literature search has revealed no prospective studies that investigate the need for therapeutic intervention of traumatic myocardial injury in dogs. Although the advent of newer, noninvasive technologies may assist in diagnosing these injuries, the search for a single noninvasive diagnostic modality to detect myocardial injury continues. Currently, the diagnosis of myocardial injuries relies on an organized approach that uses multiple diagnostic tests. Recently, Holter monitoring has been shown to be a sensitive, noninvasive indicator of arrhythmias in traumatized dogs and should be performed in patients with suspected myocardial injuries. Although the benefit of using Holter monitors to identify arrhythmias may be outweighed by the delay in interpretation of collected data, continuous “live” and intermittent ECG monitoring (single patient monitors and telemetry systems) can be used effectively for this purpose in canine trauma patients. In the future, the application of immunodiagnostics, such as the troponin assays, may assist in early identification of myocardial injuries in traumatized dogs.

REFERENCES


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**ARTICLE #3 CE TEST**

The article you have read qualifies for 1.5 contact hours of Continuing Education Credit from the Auburn University College of Veterinary Medicine. *Choose the best answer* to each of the following questions; then mark your answers on the postage-paid envelope in *Compendium*.

1. What current diagnostic test is considered the gold standard for confirming myocardial injuries?
   a. echocardiogram  
   b. ECG  
   c. gross or histopathologic examination of the heart  
   d. elevation of serum CPK-MB

2. ECG abnormalities secondary to myocardial injuries usually
   a. appear at presentation.  
   b. occur within 48 hours of injury.  
   c. resolve with fluid therapy.  
   d. are fatal.

3. Of the following, which is the most sensitive detector of arrhythmias in dogs suffering from myocardial injuries?
   a. Holter monitoring  
   b. thoracic radiography  
   c. intermittent ECG  
   d. physical examination
4. Which of the following is a distinctive echocardiographic feature of myocardial injury secondary to blunt thoracic trauma?
   a. decreased end-diastolic wall thickness
   b. increased fractional shortening
   c. decreased echogenicity
   d. localized areas of echolucency

5. A disadvantage of using nuclear cardiography to diagnose myocardial injuries is
   a. arrhythmias alter image acquisition.
   b. functional changes are detectable before structural changes.
   c. they are noninvasive and do not require sedation.
   d. a short study acquisition time.

6. Historically, which isoenzyme/protein is used to detect myocardial injuries?
   a. troponin T
   b. troponin I
   c. CPK-MB
   d. troponin C

7. The troponin complex regulates the force and velocity of muscle contractions by
   a. enhancing calcium release within the cell.
   b. modulating the interaction of calcium with actin and myosin.
   c. increasing the number of calcium-binding sites on the actin filament.
   d. altering myocyte membrane potential.

8. Treatment of arrhythmias secondary to myocardial injury is aimed at
   a. the complete resolution of the ECG abnormalities.
   b. decreasing cardiac output.
   c. the resolution of clinical signs related to decreased cardiac output.
   d. alleviating the need for oxygen supplementation.

9. Which of the following is not a common side effect of class I antiarrhythmics?
   a. seizures
   b. coagulation disorder
   c. vomiting
   d. hypotension

10. If an animal with myocardial injuries requires anesthesia, which of the following selections would be indicated?
   a. halothane
   b. isoflurane
   c. glycopyrrolate
   d. b and c