The Electrocardiographic Features of Acute Cardiac Tamponade

By Howard S. Friedman, M.D., Joseph A. Gomes, M.D., Anthony R. Tardio, M.D., and Jacob I. Haft, M.D.

SUMMARY
To test the hypothesis that the electrocardiogram of cardiac rupture is due to acute cardiac tamponade, 27 episodes of cardiac tamponade were produced in ten open-chest dogs. During continuous monitoring of the electrocardiogram and the arterial and venous pressures, 10–30 cc of autologous, heparinized blood, or one of several other solutions, were intermittently infused into the pericardial sac until no effective blood pressure was recorded. The characteristic electrocardiographic findings of acute cardiac tamponade were peaked P waves, decrease of QRS complex voltage, left axis deviation of the QRS complex, deep T wave inversions, and ST-segment change. With the appearance of electromechanical dissociation, there was a sudden, vagally-mediated bradycardia. Because these changes are similar to those observed at the time of cardiac rupture, it was concluded that the electrocardiographic manifestations of cardiac rupture could be attributed to acute cardiac tamponade.

Additional Indexing Words:
Cardiac rupture, Electromechanical dissociation, Vagus nerve
Bradycardia, Left axis deviation, Electrical alternans
Pericardial effusion, Depressor reflexes

The sudden appearance of electromechanical dissociation, bradycardia, and ST-segment and T wave change have been observed to occur at the time of cardiac rupture. Indistinguishable electrocardiographic findings have been described in aortic dissection with cardiac tamponade. To test the hypothesis that these changes are due to acute cardiac tamponade—a disorder for which a specific electrocardiogram has not previously been described—cardiac tamponade was produced in the dog during continuous hemodynamic and electrocardiographic monitoring. The results of these studies are reported here.

Methods
Ten mongrel dogs having an average weight of 16.8 kg were anesthetized with pentobarbital, 30 mg/kg. Each dog was intubated and ventilated with a Harvard respirator. Using cutdown techniques, end-hole polyurethane catheters were passed to the inferior vena cava via the right femoral vein and to the descending aorta via the right femoral artery. Pressures were measured continuously with Statham P23 db strain gauges and were recorded on a multichannel photographic recorder (Electronics for Medicine DR-8). Electrocardiograms were recorded at a frequency response of 0.1–200 Hz. Electrodes were inserted beneath the skin of the distal part of each extremity with the dog in a supine position. Bipolar limb and augmented, unipolar extremity leads were recorded before and intermittently during each experiment. Lead II was recorded continuously. Electrocardiographic tracings were obtained before and after the chest was opened. The electrocardiogram recorded immediately before each experiment when the chest was open was used as the control tracing with which subsequent records were compared. After opening the chest with a median sternotomy incision, a small puncture was made in the pericardium and a J-shaped, 8-F, polyurethane catheter was inserted into the pericardial sac and secured with a purse-string suture and several silk ties.

During continuous monitoring of the electrocardiogram and venous and arterial pressures, 10–30 cc of heparinized, autologous blood (14 experiments); saline (8 experiments); heparinized saline (2 experiments); 5% dextrose in water (2 experiments); and Hypaque-M 75% (1 experiment) were intermittently infused into the pericardial sac (at 30–60 sec intervals) until no effective blood pressure was recorded. In 13 experiments, solutions had a temperature of 37 degrees whereas in 14 experiments they were at room temperature. After removing fluid from the pericardial sac, further experiments were performed only after a 15 min recovery period, at a time when pressures, heart rate, and electrocardiogram had generally returned to control values. In six dogs, the vagi were isolated in the neck and secured with moistened umbilical tapes. When bradycardia appeared, these nerves were severed. In three dogs, the right vagus nerve was cut first and 10–15 sec later the left, whereas in the other three the left vagus was cut first. After transecting the vagi, at a time when fluid had been removed and the

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electrocardiogram and pressures had returned to control values, cardiac tamponade was again produced in five dogs.

Results

To produce electromechanical dissociation — that is, persistent electrical activity when effective blood pressure could no longer be recorded — 178 ± 55 cc of pericardial fluid (10.6 cc/kg dog wt) were required. Prior to the appearance of marked hypotension, heart rate did not significantly change (from 142 ± 22 to 133 ± 21) (table 1). When electromechanical dissociation developed in dogs with intact vagi, bradycardia abruptly appeared (65 ± 16 vs 142 ± 22, P < 0.001) (table 1) (fig. 1). Typically sinus bradycardia or sinoatrial block was first seen. As the heart rate slowed, atrioventricular (A-V) junctional and/or idioventricular escape rhythms ensued (fig. 1). In some dogs the first change of rhythm was the appearance of a junctional mechanism. Severing both vagi immediately reversed bradycardia (from 73 ± 12 to 140 ± 13, P < 0.001) (table 1) (fig. 1). Rapid evacuation of fluid from pericardial sac shortly after the appearance of bradycardia also reversed the slowing of heart rate (table 1) (fig. 2). However, recovery of heart rate was not as rapid as that seen following vagotomy. When cardiac tamponade was produced in dogs after the vagi had been cut, electromechanical dissociation was not accompanied by bradycardia (from 146 ± 14 to 140 ± 32) (table 2). A significant fall in heart rate was seen, however, after three minutes without an effective blood pressure (112 ± 38 vs 142 ± 22, P < 0.05), although this heart rate was significantly greater than that seen at the time of electromechanical dissociation with vagi intact (112 ± 38 vs 65 ± 16, P < 0.001) (tables 1 and 2). Moreover, a sinus mechanism persisted in dogs with severed vagi even when agonal bradycardias appeared. Ventricular fibrillation was observed in only one dog. It occurred, however, after cardiac tamponade had been released.

P Wave

P wave voltage was maintained or increased and P wave contour became peaked in 21 of 24 experiments in which electromechanical dissociation was produced. The appearance of an atrial or junctional rhythm before electromechanical dissociation developed obscured P wave changes in three dogs (fig. 3). The increment of P wave voltage recorded in nine experiments did not correlate with change of venous pressure (r = 0.23).

QRS Complex

At the time of electromechanical dissociation total QRS complex voltage was less than 50% of preinfusion

Table 1

<table>
<thead>
<tr>
<th>Experiment no.</th>
<th>Control</th>
<th>50% Fall of B.P.</th>
<th>EMD</th>
<th>Evac.</th>
<th>Cut Vagi</th>
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<tr>
<td>1</td>
<td>162</td>
<td>155</td>
<td>72</td>
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<td>7</td>
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<td>139 ± 21</td>
<td>131 ± 23</td>
<td>62 = 16*</td>
<td>136 = 28</td>
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<tr>
<td>14</td>
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<td>144</td>
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<td>149 ± 21</td>
<td>138 ± 15</td>
<td>73 = 12*</td>
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<td>Total</td>
<td>142 ± 22</td>
<td>133 ± 21</td>
<td>65 ± 16*</td>
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*Significantly different from control, P < 0.001. Values given are means ± standard deviations.

Abbreviations: B.P. = Blood pressure; EMD = Electromechanical dissociation; Evac. = Evacuation of fluid.
amplitude. Mean QRS axis, which was generally vertical before infusion, shifted leftward and superiorly (fig. 4). These changes were observed in all 24 experiments in which electromechanical dissociation was produced. Removal of fluid reversed both findings whereas cutting vagi had no effect on these changes. Electrical alternans of the QRS complex was observed transiently in one dog in which cardiac tamponade was produced with blood after vagotomy.

**T Wave and ST Segment**

The first change observed in 25 of 27 experiments was deep T wave inversions (T wave axis shifted rightward and superiorly). This finding was seen within 2–3 beats after the start of an infusion of fluid (fig. 5). T wave inversions were produced with all fluids infused irrespective of their temperature at the time of infusion. The most marked changes occurred after rapid infusion of large quantities of fluid (60 cc in 10 sec). T wave changes did not appear to correlate with the amount of fluid in pericardial sac at the time of infusion; that is, T wave inversions were equally marked when infusing into a pericardial sac that did not have any fluid as when infusing into one with large quantities of fluid already there. However, T wave changes were less marked when experiments were repeated several times in the same dog.

ST segment changes were observed in five experiments, four times ST segments becoming elevated and once depressed. These changes occurred only after prolonged hypotension (fig. 6). ST(J) junction elevations at times accompanied deep T wave inversions; however, this was not a consistent finding (fig. 5).

**Discussion**

A characteristic electrocardiogram has been described in cardiac rupture. Although this pattern has been attributed to acute cardiac tamponade, there has been no previous experimental study that has

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**Table 2**

<table>
<thead>
<tr>
<th>Experiment No.</th>
<th>Control</th>
<th>50% Fall of B.P.</th>
<th>E.M.D.</th>
<th>3 min.</th>
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<tr>
<td>20</td>
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<td>168</td>
<td>144</td>
<td>114</td>
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<tr>
<td>23</td>
<td>150</td>
<td>156</td>
<td>150</td>
<td>140</td>
</tr>
<tr>
<td>24</td>
<td>138</td>
<td>138</td>
<td>126</td>
<td>90</td>
</tr>
<tr>
<td>Mean ± Standard Deviation</td>
<td>146 ± 14</td>
<td>148 ± 19</td>
<td>140 ± 32</td>
<td>112 ± 38*</td>
</tr>
</tbody>
</table>

*Significantly lower than the control (total) heart rate *P* < 0.05.

Abbreviations: 3 min. = 3 minutes without an effective blood pressure; EMD = electromechanical dissociation.
ECG IN CARDIAC TAMPOONADE

rate was not found. Failure to repeat the finding of reactive tachycardia in the present study and the conflicting data in the literature suggest three possible explanations: 1) rapid induction of cardiac tamponade, as in the present study, might have been too fast for the development of compensatory tachycardia; 2) use of pentobarbital anesthesia, a drug with atropine-like properties, produced a control tachycardia that might have obscured normal reflex mechanisms; and 3) reflexes that produce bradycardia during electromechanical dissociation might have been partially

dealt specifically with the electrocardiographic features of cardiac tamponade. Using the open-chest dog, an experimental model was devised in which cardiac tamponade was produced during continuous recordings of pressure and electrocardiogram.

During the early phase of cardiac tamponade, at a time when marked hypotension occurred, heart rate did not change. However, when electromechanical dissociation was produced bradycardia abruptly appeared. Release of tamponade or bilateral cervical vagotomy reversed bradycardia. When electromechanical dissociation was produced following vagotomy, significant bradycardia did not occur for several minutes. Moreover, after vagotomy bradycardia was not accompanied by sinus arrest and escape rhythms. Despite slowing of heart rate, a sinus mechanism persisted, in some instances for more than five minutes.

In some studies during the early hypotensive phase of cardiac tamponade, tachycardia was observed, whereas in other studies a significant change in heart

Figure 2
Strips from a continuous record of venous and arterial pressures and lead II during the release of cardiac tamponade. As arterial pressure increases, there is a gradual acceleration of junctional rhythm (1-3) and then sinus capture (4).

Figure 3
Simultaneous arterial and venous pressures and lead II recorded during a period of electromechanical dissociation showing junctional rhythm followed by sinus capture. P wave is tall and peaked despite low QRS complex voltage.

Figure 4
Electrocardiograms recorded before cardiac tamponade (upper ECG) and after blood pressure had fallen by more than 80% (lower ECG). When compared to control record, ECG after infusion shows QRS complexes with a lower voltage and a mean axis that has rotated leftward and superiorly.
same electrocardiographic pattern has been observed in a patient who developed cardiac tamponade following aortic dissection. It is also been reported that the immediate relief of cardiac tamponade in a patient with cardiac rupture reverses bradycardia. 

Using an experimental model like the one in our study, found bradycardia frequently during what he called Phase II, a point at which small increments of pericardial fluid led to a precipitous decline of arterial pressure.

Immediate reversal of the bradycardia of cardiac tamponade after vagotomy and its delay once the vagi had been cut demonstrates the presence of a reflex arc that includes the vagus nerve. Vagally-mediated cardiac depressor reflexes have been previously described. Receptors for these reflexes have been identified in both atria, the left coronary artery, the myocardium, and the epicardium. Which one of these afferents is involved in the bradycardia of cardiac tamponade remains to be determined.

The P wave became peaked in lead II with the same or an increased voltage when compared to control tracings. These findings occurred despite a concomitant marked reduction of QRS complex voltage. In some experiments, however, the appearance of junctional rhythm obscured these findings. The P wave changes are like those reported in the human with right atrial overload. However, in cardiac tamponade pressure elevations are not limited to the right atrium. Pressure changes are at least as marked in the left atrium as in the right atrium. An alternative explanation for these P wave changes — other than right and/or left atrial overload — could be a rotation of mean P wave forces. If mean P wave axis shifted so that it were more parallel to lead II, P wave voltages would increase, or perhaps, not change, depending on the countervailing effect of the fluid itself. On the other hand, the P wave changes might be an artifact of the open-chest preparation.

A reduction of QRS complex voltage was observed in all experiments. Morgensen et al. observed a 15–38% fall in QRS complex amplitude in all cases of cardiac rupture in which satisfactory records were obtained. Morton et al. reported on a case of cardiac rupture in which a gradual decline in voltage of QRS complexes was found. Moreover, blood and the other solutions used are good conductors of electricity. These fluids, therefore, by their short-circuiting action, would be expected to reduce voltages recorded on surface electrocardiographic leads.

Left axis deviation of the QRS complex was another consistent finding in all experiments. Clinical reports have not described changes of QRS axis. The failure to observe axis shifts in cardiac rupture may be due to less mobility of the human heart, or on the other
hand, to the limitations of estimating QRS axis with a single bipolar monitor lead used in clinical reports. It is possible, however, that the development of left axis deviation is related to the use of an open-chest model.

Electrical alternans of the QRS complex was observed in one experiment. Electrical alternans is not an uncommon finding with large pericardial effusion, and it has been reported to occur transiently in a case of cardiac rupture with cardiac tamponade. Both left axis deviation and electrical alternans could be explained by cardiac displacement caused by pericardial effusion. Whereas with electrical alternans the cardiac position would vary with each beat like a rotary pendulum in a distended pericardial sac, with left axis deviation the heart would be fixed in a new position.

Deep, T wave inversions were observed shortly after the onset of an infusion of fluid into the pericardial sac. T wave changes were not related to the type of fluid used or its temperature. They were, however, most marked with rapid infusions into a noncompliant pericardial sac. Mean T wave axis shifted rightward and superiorly, in a direction opposite to that of the mean QRS axis. Hellerstein and Katz produced such T wave changes in the tissue surrounding areas of subepicardial injury; in some of their experiments these T wave changes preceded ST-segment elevations in the area of injury itself. These investigators referred to these T wave changes as "injury currents of repolarization" due to a delay in repolarization. The T wave changes observed in acute cardiac tamponade may reflect, therefore, a reversal of the normal sequence of repolarization. Whether these changes are due to myocardial ischemia resulting from reduced coronary flow or are the result of a direct pressure effect of the fluid on the epicardium is not clear. In previous reports of acute cardiac tamponade, peaked T waves and reversal of pre-existing T wave polarity have been found in some cases. In one of these reports, ST segment changes were also observed. Although ST segment changes were observed in five of our experiments, these developed after prolonged hypotension, and therefore, appear to be related to myocardial ischemia rather than to a direct effect of cardiac tamponade.

Thus, the present study establishes that electromechanical dissociation, bradycardia, reduction of QRS voltage, electrical alternans, and ST segment and T wave change — the electrocardiographic features of cardiac rupture — can all be duplicated in an experimental model in which cardiac tamponade alone is produced. Furthermore, this study confirms that the vagus nerve is involved in the bradycardia that occurs in cardiac tamponade. Determination of whether the other features observed in the dog — left axis deviation of the QRS axis and peaked P waves — are also present in the human during acute cardiac tamponade requires further investigation.

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