Electrocardiographic and Hemodynamic Changes in Experimental Right Ventricular Infarction

TE-CHUAN CHOU, M.D., NOBLE O. FOWLER, M.D., MARJORE GABEL, JOHANNA VAN DER BEL-KAHN, M.D., AND ELIZABETH J. FELTNER, B.A.

SUMMARY To investigate the electrocardiographic and hemodynamic changes in isolated right ventricular infarction, 0.25 ml or 0.5 ml of metallic mercury was injected into the right coronary artery of 14 closed-chest dogs. At autopsy, at least 60% of the right ventricle was necrotic in every dog. Hemodynamic observations were made in 11 and electrocardiographic mapping was performed in all 14 dogs. Right atrial pressure rose in 10 and left atrial pressure in nine of the 11 dogs; early right atrial pressure did not exceed left atrial pressure, but late right atrial pressure was greater in four dogs. Although cardiac output and blood pressure fell significantly, circulation was maintained.

Twelve of 14 dogs had transient ST-segment elevation in the right precordial leads, and 12 developed right bundle branch block. Abnormal Q waves or R waves of 1 mm or less appeared in the right precordial leads in 13 of the 14 dogs. Since right bundle branch block and abnormal Q waves in the right precordial leads have not been recognized as useful signs in human right ventricular infarction, further investigations are warranted to determine their value in clinical applications.

The diagnosis of right ventricular infarction is usually based on the hemodynamic findings.1–4 Noninvasive procedures such as radionuclide imaging and echocardiography are also used.5–6 Although the ECG is one of the most valuable tools in the diagnosis of myocardial infarction involving the left ventricle, only limited information is available about its application in right ventricular infarction. We previously reported transient ST-segment deviation in lead V1 in patients with acute right ventricular infarction associated with acute left ventricular inferior wall myocardial infarction.7 Similar changes in other right precordial leads have also been described.8–10 However, the effect of right ventricular infarction on the QRS complexes has not been systematically examined in man.

In humans, right ventricular infarction is almost always associated with inferoposterior myocardial infarction. The effect of right ventricular infarction alone on the hemodynamic findings is uncertain. Because of the anatomy of the coronary circulation in dogs, isolated right ventricular infarction may be induced experimentally with minimal or no left ventricular involvement. In this study, isolated right ventricular infarction was produced in closed-chest dogs by selective intracoronary injection of metallic mercury. The ECG changes were observed by precordial mapping and correlated with the pathologic findings. Hemodynamic observations were made to delineate the changes due to right ventricular infarction in the absence of significant coexisting left ventricular involvement.

Methods

Fourteen mongrel dogs that weighed 19–25 kg were studied. The experiments were performed under pentobarbital sodium anesthesia. Right ventricular infarction was produced by injecting 0.25 or 0.5 ml metallic mercury into the right coronary artery through a #7 Judkins catheter placed in its orifice under fluoroscopy. All 14 dogs had the ECG study and 11 were also monitored for hemodynamic changes. In the latter 11 dogs, a preparatory operation was performed 4–8 days before the experiment. Tygon catheters were placed in the left atrium and pericardial space through a left lateral thoracotomy in the fourth intercostal space. The catheters were secured by purse-string sutures and the pericardium was closed tightly. The tips of the catheters were aligned so that they were at the same horizontal level when the dog was lying on its right side. In three dogs, five small radiopaque beads were sutured on the epicardial surface to assist in outlining the borders of the anterior right ventricular free wall under fluoroscopy. Three beads were placed just to the left of the anterior descending branch of the left coronary artery, one bead was placed over the outflow tract of the right ventricle and one along the right atrioventricular groove.

Electrocardiographic Study

The ECGs were recorded with the dogs in the supine position. In addition to the six limb leads (leads I, II, III, aV1, aVr, and aVF), 48 precordial leads were obtained during each recording. The sites for the electrode placement were arranged in six horizontal and eight vertical rows (fig. 1). The adjacent leads were 2 cm apart. The six horizontal rows were designated as rows A, B, C, D, E, and F, with row A the most cephalad and located at the level of the third intercostal space at the sternal border. The eight vertical rows were equally divided by the midsternal line. The right and left precordial leads were represented by the initials R and L, respectively, with R1 and L1 indicating leads closest to the midsternal line. The precordial lead positions were marked by gentian violet and the same locations were used throughout the experiment. The leads were recorded sequentially with a needle electrode attached to the V lead terminal of the electrocardiograph.

A complete ECG, including the precordial map-

From the Division of Cardiology, Department of Medicine, and the Department of Pathology, University of Cincinnati College of Medicine, Cincinnati, Ohio.

Address for correspondence: Te-Chuan Chou, M.D., University of Cincinnati, Division of Cardiology, Mail Location 542, Cincinnati, Ohio 45267.

Received November 29, 1982; revision accepted March 3, 1983.

Circulation 67, No. 6, 1983.
solution of sodium bicarbonate was administered if needed to maintain the blood pH between 7.3 and 7.48.

Pathologic Study

Detailed pathologic examination of the heart was performed on all 14 dogs. The external surface of the heart was inspected for gross abnormalities and the distribution of the major extramural coronary arteries was determined. After adequate fixation in 10% buffered formalin, the four chambers of the heart were separated. The ventricular dissection was performed at the anterior and posterior margins of the ventricular septum at its junction with the free wall of the right and left ventricles.

The individual components of the heart, including the right and left ventricular free walls, the ventricular septum, and the right and left atria, were weighed. The amount of myocardial necrosis in each of the components was quantitated by making serial horizontal 0.5–1.0 cm-thick slices. The areas of necrosis in each slide was cut out, weighed and the sum total calculated as a percentage of the component examined. Microscopic examination was performed on each section whenever it was needed to verify the gross impression about the presence or absence of necrosis. Serial sections were also made of the sinoatrial and atrioventricular nodal regions as well as the area of the right bundle branch according to the method described by Lev and associates. 11

Results

Selective injection of metallic mercury into the right coronary artery under fluoroscopy was successful in all 14 dogs. In one dog, a small amount of the mercury escaped and was detected in the cerebral circulation by fluoroscopy. With the dogs supine, the mercury was seen mostly in the right upper half of the cardiac shadow. This area of distribution of the mercury was within the confines of the precordial mapping area in all dogs. In the three dogs in which radiopaque beads were implanted to assist in outlining the right ventricular borders, most of the mercury was distributed to the right of these beads (fig. 2).

One dog died 3 hours after the mercury injection, one after 4 hours and one after 24 hours. The other 11 dogs were killed by pentobarbital sodium overdose after 3–4 days.

Pathologic Findings

Examination of the epicardial surface of the 14 dogs revealed evidence of pericarditis in all, with considerable variations in the extent of the lesions. The three dogs that did not have preparatory thoracotomy had pericardial lesions limited to the areas overlying the infarcted myocardium. The extramural portion of the right coronary artery was clearly confined to the right ventricular surface in 10 dogs. In the other four dogs it was difficult to determine whether it supplied a small posterior descending branch because of the presence of pericarditis and myocardial necrosis in that area.

Hemodynamic Study

Hemodynamic investigation was performed on 11 dogs before and 1–2 hours after mercury was injected into the right coronary artery. The respiratory rate, left and right atrial pressures, pericardial pressure and aortic pressure were monitored and recorded, along with lead II of the ECG, on a direct-writing, strip-chart recorder. The right atrial catheter was introduced through the left femoral vein and the aortic catheter through the left femoral artery under fluoroscopy.

The cardiac output was determined serially in duplicate by thermodilution. The arterial PO2 was maintained at a level greater than 100 mm Hg and the PCO2, at 38–49 mm Hg by a ventilator if necessary, but not during ECG and hemodynamic observations. An i.v.
All hearts had evidence of acute myocardial infarction involving mostly the right ventricular free wall and right atrium. They had marked dilatation of the right ventricle and right atrium. The areas and extent of necrosis in the individual dogs are listed in table 1. The portion of the free wall of the right ventricle that was necrotic varied from 60% to 90%. The intraventricular septum did not have evidence of infarction in six dogs. In another six dogs, less than 5% of the septum was necrotic. The extent of the septal infarction was 15% and 20% in the remaining two dogs. The free wall of the left ventricle showed no or minimal (less than 5%) area of myocardial infarction in 12 dogs. In two dogs, about 10% of the free wall was necrotic. In the hearts that exhibited septal and left ventricular infarction, the necrosis was characteristically located in the posterobasal or apical regions. Infarction of the right atrium was found in the 13 dogs in which adequate examination was performed. The extent of necrosis varied from 40% to 95% of the right atrial wall. The left atrium had no or minimal evidence of infarction in 10 hearts and in four the area of necrosis varied from 10% to 30% of the left atrial free wall.

The sinoatrial node was necrotic in all 13 dogs in which the region was examined. The atrioventricular node was intact in 11; in two it was questionable. The right bundle branch was free from necrosis in all hearts, although two dogs had subendocardial infarction of the ventricular septum near the area of the right bundle branch.

Electrocardiographic Findings

The ECG changes after the injection of the mercury into the right coronary artery in the 14 dogs are summarized in table 2. In two dogs the ECG observation was limited to 150 minutes, but in 12 it was 24 hours or more. Shortly after the injection of the mercury, atrioventricular junctional rhythm developed in seven dogs. Among them, in four of the six dogs that survived for more than 24 hours, the junctional rhythm had persisted. Since the ventricular rate during the atrioventricular junctional rhythm was slower than the sinus rate preceding its development, it was probably an escape rhythm due to sinus bradycardia or sinus arrest. Ventricular tachycardia or ventricular fibrillation appeared in six dogs, who were treated with an i.v. bolus of lidocaine or direct-current countershock. Right bundle branch block developed in all but two of the 14 dogs, but persisted only in four of the 12 dogs that survived for more than 24 hours.

In the 11 dogs with prior thoracotomy, ST-segment elevation of 2 mm or more developed shortly after the injection of mercury into the right coronary artery in the right precordial leads in nine dogs. Two to 24 leads were involved. Five dogs had also ST elevation in the left precordial leads, but in none of them was such

<table>
<thead>
<tr>
<th>Dog</th>
<th>RVFW</th>
<th>VS</th>
<th>LVFW</th>
<th>RA</th>
<th>LA</th>
<th>SAN</th>
<th>AVN</th>
<th>RBB</th>
</tr>
</thead>
<tbody>
<tr>
<td>2321</td>
<td>60</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2324</td>
<td>90</td>
<td>15</td>
<td>10</td>
<td>60</td>
<td>30</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2325</td>
<td>85</td>
<td>&lt;5</td>
<td>&lt;5</td>
<td>60</td>
<td>0</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2326</td>
<td>80</td>
<td>0</td>
<td>0</td>
<td>40</td>
<td>&lt;5</td>
<td>+</td>
<td>-</td>
<td>?</td>
</tr>
<tr>
<td>2334</td>
<td>75</td>
<td>&lt;5</td>
<td>&lt;5</td>
<td>50</td>
<td>0</td>
<td>+</td>
<td>-</td>
<td>?</td>
</tr>
<tr>
<td>2337</td>
<td>70</td>
<td>0</td>
<td>0</td>
<td>45</td>
<td>0</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2338</td>
<td>85</td>
<td>0</td>
<td>&lt;5</td>
<td>60</td>
<td>10</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2349</td>
<td>75</td>
<td>&lt;5</td>
<td>&lt;5</td>
<td>65</td>
<td>&lt;5</td>
<td>+</td>
<td>-</td>
<td>?</td>
</tr>
<tr>
<td>2363</td>
<td>85</td>
<td>&lt;5</td>
<td>&lt;5</td>
<td>70</td>
<td>&lt;5</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2383</td>
<td>80</td>
<td>&lt;5</td>
<td>&lt;5</td>
<td>70</td>
<td>10</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2391</td>
<td>90</td>
<td>20</td>
<td>10</td>
<td>95</td>
<td>15</td>
<td>+</td>
<td>-</td>
<td>?</td>
</tr>
<tr>
<td>2472</td>
<td>80</td>
<td>0</td>
<td>&lt;5</td>
<td>85</td>
<td>5</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2500</td>
<td>85</td>
<td>0</td>
<td>0</td>
<td>85</td>
<td>&lt;5</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2501</td>
<td>80</td>
<td>&lt;5</td>
<td>&lt;5</td>
<td>85</td>
<td>&lt;5</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Abbreviations: RVFW = right ventricular free wall; VS = ventricular septum; LVFW = left ventricular free wall; RA = right atrium; LA = left atrium; SAN = sinoatrial node; AVN = atrioventricular node; RBB = right bundle branch.
change seen in more than seven leads. ST-segment depression of 2 mm or more occurred mostly in the left precordial leads except in three dogs in which several leads (10, 11 and 13, respectively) showed such change in the right precordium. Except for dogs 2324 and 2391, the ST-segment displacement subsided in most of the mapping area after 24 hours. However, in these two dogs a preparatory thoracotomy was performed before the acute experiment; therefore, the significance of the ST-segment changes in relation to the myocardial infarction after the acute event is difficult to determine. In seven dogs, a baseline ECG was obtained before thoracotomy. All seven had ST-segment elevation after surgery, although its distribution varied. The additional ST-segment elevation in the right precordial leads that appeared soon after the injection of mercury suggested that the surgical procedure did not interfere with the development of the signs of myocardial injury in these dogs.

Changes in the initial QRS forces were also observed in the 11 dogs with prior thoracotomy, especially in the tracings taken 24 hours after the mercury injection. A reduction of the R-wave amplitude of 5 mm or more was seen in each of the nine dogs that survived for 1 day or longer. This occurred predominantly in the right precordial leads. Q waves or decreased R waves with an amplitude of 1 mm or less developed in all but one of these nine dogs. With one exception, these were seen exclusively in the right precordial leads (fig. 3).

In dogs 2472, 2500 and 2501, who did not have prior thoracotomy, ST-segment elevation occurred in many of the right precordial leads soon after the mercury injection, but the displacement was transient in most. In two of the dogs, ST-segment elevation appeared in some of the left precordial leads in the later tracings without apparent explanation. A reduction of the R-wave amplitude of 5 mm or more was seen in all of the right precordial leads in all three dogs. The left precordial leads also showed such change, but in two dogs, fewer showed changes. Q waves or R waves of 1 mm or less developed in most right precordial leads and a few of the left precordial leads in all three dogs (fig. 3).

In summary, with few exceptions, the injection of mercury into the right coronary artery was followed by the appearance of ST-segment elevation and a reduction or loss of R waves in the right precordial leads, whether or not a prior thoracotomy had been performed. However, there was no apparent relation between the number of leads with such findings and the extent of the myocardial infarction found at autopsy. The serial ECG changes in dog 2391 are illustrated in figures 4–6.

**Hemodynamic Findings**

The mean right and left atrial pressures 5–10 minutes after injection of mercury into the right coronary artery increased significantly (table 3). In three dogs, peak right atrial pressure equalled peak left atrial pressure, but in none did right atrial pressure exceed left atrial pressure.

Final mean right atrial pressure (6.1 mm Hg) was similar to final mean left atrial pressure (6.4 mm Hg); right atrial pressure equalled left atrial pressure in three dogs and exceeded left atrial pressure in four.

Peak effect pericardial pressure significantly exceeded control pericardial pressure, −0.68 mm Hg vs
−3.0 mm Hg, but the final pericardial pressure was not greater than the control.

Mean cardiac output declined significantly between the control period and the acute peak. The mean final cardiac output was also significantly lower than the control cardiac output.

In the acute period, four of nine dogs showed only a borderline\(^2\) (10-mm Hg) inspiratory decline of systolic

**Figure 3.** The leads that developed Q wave or R wave of 1 mm or less after the injection of mercury into the right coronary artery (solid circles). The number of such leads in these 12 dogs that survived for more than 24 hours varied from 0 to 24. The leads in the right upper quadrant of the mapping area were most frequently involved. The average reduction of the amplitude of the R wave in these leads in the individual dogs ranged from 9.5 to 20.5 mm (average 13 mm).
arterial pressure, and only dog 2391 showed a definitely abnormal value (15 mm Hg).

Mean systemic arterial pressure did not change significantly; it was 142 mm Hg in the control period, 136 mm Hg during the peak hemodynamic effect and 130 mm Hg in the final period.

Discussion

Myocardial infarction involving the free wall of the right ventricle alone is rare in man but is present in 13–34% of autopsied hearts with transmural left ventricular infarction. The associated left ventricular infarction is almost always located in the inferior and posterior wall.

Until recently, right ventricular infarction was seldom recognized clinically. Its presence has important therapeutic implications. Patients with acute myocardial infarction and predominantly right ventricular dysfunction with low cardiac output may require fluid administration rather than fluid restriction, as in the case of left ventricular failure. In view of the incidence of right ventricular infarction found in autopsied hearts, it is likely many of the clinical cases are missed. Improvement in its detection would be highly desirable. It is also important to define the ECG and

**Figure 4.** ECG recorded immediately before the injection of mercury into the right coronary artery in dog 2391.

**Figure 5.** ECG recorded 30 minutes after the injection of mercury in dog 2391.
Electrocardiographic Findings

In 1949, Myers and associates reported the ECG changes in 18 autopsy cases of right ventricular infarction. They stated that none of the findings were distinctive of the right ventricular lesions. Since then, the ECG changes described in most of the patients with right ventricular infarction were those of the inferoposterior myocardial infarction. More recently, Erhardt and associates reported ST-segment elevation in a bipolar lead in nine patients with right ventricular infarction associated with inferior myocardial infarction.

The electrodes for the bipolar leads were placed on the right arm and in the fifth intercostal space in the right midclavicular line. In 11 cases of right ventricular infarction diagnosed by autopsy or hemodynamic findings, we found transient ST-segment elevation in one (lead V₁) or more of the conventional right precordial leads. Other studies suggested that the inclusion of lead V₄₃ or V₆₈ to V₆₉ may increase the sensitivity for the ECG recognition of right ventricular infarction. However, ST-segment elevation in the right precordial leads is not specific for right ventricular infarction. Coexisting anterior left ventricular myocardial injury or infarction, pericarditis or acute pulmonary embolism may also cause similar changes. Because of the transient nature of the ST-segment elevation in right ventricular infarction, this useful sign may be easily missed if the ECG is not recorded during the acute phase of the infarction. Furthermore, since the ST elevation only represents myocardial injury, specific signs for the diagnosis of right ventricular necrosis are needed. Cardenas and associates observed a loss of anterior QRS forces in some of the right precordial leads in patients who had inferoposterior myocardial infarction with right ventricular involvement. The incidence and specificity of such a finding in these patients were not mentioned.

The canine model of right ventricular infarction has advantages and disadvantages. In most dogs, the left coronary artery is dominant and its circumflex branch supplies the posterior descending artery. This was true in 10 of the 14 hearts we examined. In four, it was uncertain because of technical difficulties in the examination. Since the right coronary artery supplies almost exclusively the free wall of the right ventricle, its occlusion would result in essentially isolated right ventricular infarction. In 12 of our 14 dogs, myocardial necrosis was found in less than 5% of the interventricular septum and the free wall of the left ventricle. Most of the electrocardiographic changes may, therefore, be attributed to right ventricular infarction alone. In man, right ventricular infarction is almost always associated with inferior myocardial infarction of the left ventricle.

The morphology of the canine ECG complexes varies considerably with changes in body position. This variability is due to the instability of the dog heart in the mediastinum. During our studies, great care was taken to maintain the dog in the same supine position when the ECG was performed. The precordial mapping sites were marked and remained the same throughout the experiment. Only major changes in the ECG were considered significant. The T waves were not analyzed.

Medrano and de Micheli induced chemical necrosis in the posterior free wall of the right ventricle in 30 mongrel dogs by infiltration of 90% phenol solution. Nine precordial leads (lead V₁ to V₆₉) and three high abdominal leads were recorded. A reduction of R-wave amplitude or the appearance of Q waves was observed in leads V₆₈ to V₆₉ and the right-sided high abdominal leads. Similar results were achieved in the right precordial leads in dogs when the chemical necro-

![Figure 6. ECG recorded 72 hours after the injection of mercury in dog 2391.](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.67.6.1264)
TABLE 3.  Hemodynamic Changes in Experimental Right Ventricular Infarction

<table>
<thead>
<tr>
<th>Dog</th>
<th>Control</th>
<th>Peak effect</th>
<th>Final</th>
<th>Survival time (hours)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RAP (mm Hg)</td>
<td>LAP (mm Hg)</td>
<td>PP (mm Hg)</td>
<td>CO (l/min)</td>
</tr>
<tr>
<td>2321</td>
<td>0</td>
<td>2.5</td>
<td>-2</td>
<td>5.5</td>
</tr>
<tr>
<td>2324</td>
<td>-1.5</td>
<td>2</td>
<td>-1.5</td>
<td>3.1</td>
</tr>
<tr>
<td>2325</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>3.13</td>
</tr>
<tr>
<td>2326</td>
<td>0</td>
<td>0</td>
<td>-4</td>
<td>3.68</td>
</tr>
<tr>
<td>2334</td>
<td>-1</td>
<td>0</td>
<td>-4</td>
<td>4.7</td>
</tr>
<tr>
<td>2337</td>
<td>-1</td>
<td>3</td>
<td>-3</td>
<td>5.15</td>
</tr>
<tr>
<td>2338</td>
<td>-2.5</td>
<td>1</td>
<td>-4</td>
<td>3.5</td>
</tr>
<tr>
<td>2349</td>
<td>-1</td>
<td>0</td>
<td>-4</td>
<td>5.1</td>
</tr>
<tr>
<td>2363</td>
<td>-1.5</td>
<td>2.5</td>
<td>—</td>
<td>5.7</td>
</tr>
<tr>
<td>2383</td>
<td>-1.5</td>
<td>3.5</td>
<td>-4</td>
<td>2.92</td>
</tr>
<tr>
<td>2391</td>
<td>-0.5</td>
<td>4</td>
<td>—</td>
<td>2.78</td>
</tr>
</tbody>
</table>

n = 11  n = 11  n = 8  n = 11  n = 11  n = 11  n = 8  n = 11  n = 11  n = 11  n = 11  n = 7  n = 9

Mean  -1  2  -3  4.90  5.22*  7.68*  -0.68*  2.7*  6.1*  6.4*  -3.3  3.12*  Mean  -1  2  -3  4.90  5.22*  7.68*  -0.68*  2.7*  6.1*  6.4*  -3.3  3.12*

SD  1  2  2  1.06  2.76  3.40  0.65  0.71  2.9  3.32  1.9  1.40  SD  1  2  2  1.06  2.76  3.40  0.65  0.71  2.9  3.32  1.9  1.40

SEM  0  0  1  0.32  0.83  1.02  0.23  0.21  0.87  1.0  0.73  0.46  SEM  0  0  1  0.32  0.83  1.02  0.23  0.21  0.87  1.0  0.73  0.46

*p < 0.05 vs control.

Abbreviations: RAP = right atrial mean pressure; LAP = left atrial mean pressure; PP = intrapericardial pressure; CO = cardiac output.

A rhythm that survived for more than 24 hours. The arrhythmia had its anatomic counterpart in that the region of the sinoatrial node was necrotic in all of the 13 hearts that were examined pathologically in this area. In contrast, necrosis of the atrioventricular nodal region was not evident in 12 dogs and was questionable in the remaining two. Atrioventricular block was not observed in any dog. Both the ECG and pathologic findings are consistent with the fact that in dogs, the blood supply to the atrioventricular node comes from the dominant left circumflex instead of the right coronary artery, as in humans. In human right ventricular infarction, second- and third-degree atrioventricular block are quite common.7

Six of the dogs developed ventricular tachycardia or fibrillation. This high incidence is similar to that in human right ventricular infarction.7 Of particular interest is the development of right bundle branch block in 12 of the 14 dogs. In the 10 dogs that had right bundle branch block and survived for more than 24 hours, it persisted in four. At autopsy, none of the dogs had definite signs of necrosis along the course of the right bundle branch. Therefore, we may assume that the conduction defect was caused by the lesions in the free wall of the right ventricle and is similar to that induced in dogs by Medrano and de Michelli28 by incising the subdivisions of the right bundle branch. Observations made during cardiac surgery in man also suggested that damage of the more peripheral right ventricular Purkinje network may be an additional mechanism for the genesis of right bundle branch block.29,30

In humans, complete right bundle branch block occurs in 3–7% of patients with acute myocardial infarction.31 It is usually associated with anterior infarction.32 The anterior descending branch of the left coronary artery is usually the vessel involved and the
infarction includes the interventricular septum with ischemia or necrosis of the right bundle branch. Right bundle branch block is seldom seen in patients with isolated inferior infarction. When it is, additional causes may be suspected. The results of this study suggest that right bundle branch block may develop in myocardial infarction limited to the free wall of the right ventricle. One may speculate that it may also occur in patients with acute right ventricular infarction associated with inferior myocardial infarction of the left ventricle. Therefore, in patients with acute inferior infarction, the appearance of right bundle branch block could indicate coexisting right ventricular infarction. Since in most of the cases bundle branch block is transient, it may be missed unless the ECG is taken soon after the onset of the infarction. Further investigation to determine whether such a phenomenon occurs in humans appears justifiable.

The appearance of an abnormal Q wave on the ECG is one of the standard criteria for diagnosing myocardial infarction of the left ventricle. This sign has not been used or thought to be helpful for the recognition of right ventricular infarction. In the absence of right ventricular hypertrophy, the right ventricular potential is mostly masked by the normally dominant left ventricular potential. The R waves in the right precordial leads are usually quite small. The ratio of the left and right ventricular mass in dogs is similar to that in humans. However, tall R waves were recorded in the right precordial leads in our dogs in the supine position before myocardial infarction developed. The large amplitude of the right ventricular potential is probably due to proximity effect. Left precordial leads overlying the right ventricle recorded with the dogs in the right lateral decubitus position (results not included in this report) revealed much smaller R waves. The origin of these tall R waves in the right precordial leads is further substantiated by the reduction or disappearance of these R waves in association with the development of essentially isolated right ventricular infarction proved by autopsy. All of the dogs had a decrease of the R-wave amplitude in many of the right precordial leads 24 hours after the induction of the right ventricular infarction. All except one developed abnormal Q waves or decreased R waves of 1 mm or less. The number of precordial leads showing such changes, however, had no apparent relationship to the extent of right ventricular necrosis.

The present study is unique in that it shows the ECG changes that may occur with extensive right ventricular necrosis in the absence of significant necrosis of the left ventricle. The value of this experimental finding in the diagnosis of human right ventricular infarction is uncertain, but deserves further exploration. An ECG-pathologic correlation study in patients who have acute inferoposterior left ventricular infarction and a decrease or loss of R wave in the right precordial leads may provide useful information.

**Hemodynamic Findings**

Starr et al. extracted the right ventricle of dogs and found no rise of systemic venous pressure. Our study demonstrated a consistent rise in right atrial mean pressure after acute right ventricular infarction and is consistent with the right ventricular catherization study of Guiha et al. In our study, right atrial necrosis involved 40–95% of the right atrium and atrioventricular junctional rhythm was present in six of 11 dogs in the acute period and in four of nine in the final period. Although atrial pressures tended to fall when atrioventricular rhythm changed to sinus rhythm, five dogs with sinus rhythm in the final period had definite increases of right atrial pressure over the control. Loss of right atrial contractile power may well have been an important contributor to the consistent increase of right atrial pressure in these experiments.

Unlike Guiha et al., we found a significant increase of left atrial pressure. The dilated right ventricle may raise intrapericardial pressure sufficiently to increase the left-heart diastolic pressures; however, left atrial pressure rose in dogs 2324 and 2325, in which intrapericardial pressure did not rise. Atrioventricular junctional rhythm could raise left atrial pressure, but four of five dogs with sinus rhythm in the final period had elevated left atrial pressure. Only dogs 2324 and 2391 had more than 5% necrosis of the interventricular septum and only dogs 2324 and 2334 had more than 5% necrosis of the left ventricular free wall to explain elevated left atrial pressure. Finally, ischemic damage to the right ventricle might affect the function of a muscle bundle, which also is involved in left ventricular contraction.

In patients with acute right ventricular infarction, there is almost always accompanying inferior left ventricular infarction. Only two of our dogs had significant (> 10%) necrosis of the left ventricular free wall. In patients, the right atrial pressure tends to be more elevated (> 10 mm Hg in 10 of 11 patients) and cardiac output lower. Further, a clinical picture suggesting cardiac tamponade was found in nine of 12 patients: five of nine had pulsus paradoxus and two had Kussmaul's sign. In contrast, only two of our nine dogs had a definite paradoxical pulse in the late period and only two had right atrial pressure ≥ 10 mm Hg. The lower cardiac outputs and higher right atrial pressures generally present in patients are probably in part related to the greater necrosis of the left ventricle.

Goldstein et al. produced right ventricular infarction by injecting mercury into the right coronary artery of closed chest dogs. These authors also found that pericardial pressure rose significantly (by 2.9 mm Hg) after right ventricular infarction. Left ventricular transmural diastolic pressure, left ventricular diastolic volume, cardiac output, blood pressure and left ventricular stroke work decreased. If the pericardium was removed, both right ventricular and left ventricular diastolic transmural pressure increased and both left ventricular and right ventricular function improved. Our study was carried out with spontaneous respiration and with a normal negative intrapericardial and intrapleural pressure. Goldstein et al. ventilated the dogs with positive pressure; control intrapericardial pressures were positive, and the hearts were paced. Thus, the hemodynamic effects of right ventricular infarction...
upon the left ventricle might well be different in our study, in which systemic blood pressure did not fall significantly.

References
27. Guiha NH, Limas CJ, Cohn JN: Predominant right ventricular dysfunction after right ventricular destruction in the dog. Am J Cardiol 33: 254, 1974
Electrocardiographic and hemodynamic changes in experimental right ventricular infarction.

T C Chou, N O Fowler, M Gabel, J van der Bel-Kahn and E J Feltner

Circulation. 1983;67:1258-1267
doi: 10.1161/01.CIR.67.6.1258

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1983 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/67/6/1258