Right Ventricular Infarction: 
Role of the Moderator Band Artery in Determining Infarct Size

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SUMMARY We studied 19 patients with proximal right coronary artery occlusions associated with acute myocardial infarcts less than 30 days old. Right ventricular infarct size, determined as a percentage of right ventricular surface area, ranged from 0% to 29%. Correlation of 24 variables measuring infarct size, chamber size and coronary artery disease failed to demonstrate a significant correlation with the extent of right ventricular infarction. However, estimates of the degree of obstruction to potential collateral flow into the right coronary arterial system from the left anterior descending coronary artery, especially through the moderator band artery, showed a significant positive correlation with infarct size (p < 0.02). Among the five patients with massive (>25%) right ventricular infarction, four had significant (>75%) obstruction of the left anterior descending system, resulting in potentially impaired collateral blood flow; the other patient had normal coronary arteries and embolic occlusion of the proximal right coronary artery with contraction band necrosis. The study suggests that collateral flow to the right ventricular myocardium, especially through the moderator band artery, protects against massive infarction in the presence of proximal right coronary artery occlusion.

RIGHT VENTRICULAR INFARCTION may complicate left ventricular infarction.1-10 Except for Bean’s study,11 which found right ventricular involvement in one-third of the patients, the reported incidence of right ventricular infarction in various series is 5–18%.6 Isolated right ventricular infarction is relatively uncommon. In a study of heart disease in 2000 autopsy cases,1 right ventricular infarcts were found in 22 of 160 patients (13.8%) with infarcts; infarction involved the right ventricle exclusively in four. In the series of Insner and Roberts,7 a similar frequency (14%, 33 of 236 patients) was noted; however, right ventricular infarction occurred exclusively as a complication of posterior left ventricular infarction.

The reasons for the relative infrequency and marked variation in size of right ventricular infarcts are not well understood. Several factors have been postulated to protect the right ventricular myocardium from ischemic injury: a lower oxygen requirement of the right ventricle by virtue of its smaller muscle mass and lower intracavitary pressure; greater systolic coronary artery blood flow in the right ventricle; and more extensive potential collateralization of the right ventricle, including that provided by the left coronary arterial system. In the course of reviewing hearts with postmortem angiograms we were struck by the possible role of potential collateral flow to the right ventricle from the left anterior descending coronary artery, especially through the moderator band artery. The present study was undertaken to evaluate the protective role that this collateralization may play in prevention of massive right ventricular infarction in cases of proximal right coronary occlusion.

Materials and Methods

Patients listed in the autopsy files of The Johns Hopkins Hospital were included in the study if their heart had been studied after coronary arteriography and fixation in distention12 and they had an acute myocardial infarct (less than 30 days of age) caused by a lesion in the right coronary artery proximal to the acute marginal branch (segments 1 and 213).

Each patient’s clinical record was reviewed for age, sex, race, symptoms and duration of symptoms, date of apparent infarct and time relationship to death. All gross specimens of the heart and postmortem angiograms were reviewed. Measurements were made from the angiogram of the height (apex-to-base distance) and width (sagittal diameter) of the right ventricle, and the total surface area was calculated by assuming the ventricle to be one-fourth of a prolate spheroid.12 The dimensions of the right ventricular infarct were determined from direct measurements over the epicardial surface of the heart. Area was estimated by assuming that the infarct was an ellipse. Serial sections of the coronary arterial lesions responsible for the infarcts were examined. Additional histologic sections were prepared of a complete transverse section of the free wall of the right ventricle and of the inferior wall of the left ventricle.

The degree of coronary atherosclerosis was estimated from the postmortem arteriograms and given a semiquantitative grade: 0 = no visible obstruction to the coronary artery lumen, 1+ = one or several obstructive lesions less than 50% in major epicardial branches, 2+ = one obstructive lesion greater than 75% in a major epicardial branch, 3+ = two obstructive lesions greater than 75%, and 4+ = greater than 75% obstruction in all three major epicardial coronary artery systems. The obstruction to potential collateral flow from the left anterior descending to the right coronary artery system was similarly evaluated on a semi-
quantitative scale: 0 = no obstruction, 1+ = 50% or less proximal obstruction, 2+ = over 75% obstruction, 3+ = proximal 75% obstruction, and 4+ = proximal occlusion in the left anterior descending system. The angiograms were evaluated without knowledge of the extent of right ventricular infarction.

Results

Nineteen patients (11 men and eight women), ages 22–82 years (average 63 years), were studied. One to 10 days had elapsed since the first clinical evidence of myocardial infarction (average 5.7 days).

In these 19 patients, with proximal right coronary occlusion and acute myocardial infarcts less than 30 days of age, right ventricular infarct size, determined as a percentage of right ventricular surface area, ranged from 0% to 29% (fig. 1). The percentage of right ventricular infarction did not correlate with left ventricular infarct size, chamber size or coronary artery disease (table 1). However, there was a significant positive correlation of right ventricular infarct size with the degree of obstruction to potential collateral flow from the left anterior descending artery to the right coronary artery ($r = 0.54$, $p < 0.02$).

In the five patients who had massive (greater than 25%) right ventricular infarction, four had significant (greater than 75%) obstruction of the left anterior descending system proximal to the origin of the artery of the moderator band. In two of these four patients, obstruction of the first septal perforator branch of the left anterior descending coronary artery, which gives origin to the moderator band artery, was noted (fig. 2). The fifth patient had normal coronary arteries but had recently sustained an embolic occlusion of the proximal right coronary artery. Examination of the ischemic right ventricular myocardium in this patient revealed contraction band necrosis (fig. 3). In humans, this type of necrosis occurs when prompt reflow of blood occurs into areas of myocardium that have had a period of no

![Figure 1](https://example.com/figure1.png)

**Figure 1.** Comparison of a heart with proximal right coronary artery occlusion producing an infarct that has no right ventricular component (A and B) and a heart with massive right ventricular infarction (C and D). (A) The postmortem arteriogram shows nonfilling of the right coronary artery, which was caused by a proximal occlusion. The left coronary artery system is widely patent. There was a large inferior wall left ventricular infarct, but no right ventricular involvement. (B) Transverse section of the ventricles of a heart with proximal right coronary artery occlusion and an inferior wall infarct in the left ventricle (line over epicardium). The left coronary artery system was patent and the right ventricular myocardium was entirely spared, as confirmed by histologic examination. (C) Postmortem arteriogram of heart with proximal right coronary artery occlusion and severe obstructive atherosclerosis in the left system. There was massive right ventricular, as well as inferior septal left ventricular, infarction. (D) Transverse section of ventricles with a large left and right ventricular infarct (line over epicardium) complicated by a ventricular septal defect (arrow). The infarct was caused by a proximal right coronary artery occlusion. LV = left ventricle; IVS = interventricular septum; RV = right ventricle.
perfusion, suggesting that in this patient the right ventricular necrosis occurred from reflow, presumably derived at least in part from collateral circulation.

**Discussion**

This study suggests that right ventricular infarction occurs when there is obstruction to potential collateral flow from the left anterior descending coronary artery system, especially through the artery of the moderator band. This potential protective role of collateral flow to the right ventricular myocardium has been suggested by other investigators.

Farrer-Brown studied the myocardial vascular patterns in 52 human hearts. In 49, the right coronary artery supplied the right ventricular free wall, except for the anterior margin, which was supplied by the left anterior descending coronary artery. The posterior descending branch of the right coronary supplied the posterior wall of the right ventricle, as well as the posterior surface of the left ventricle, in a majority of hearts. The right marginal artery of the right coronary supplied the lateral wall of the right ventricle. Farrer-Brown suggested that the artery of the moderator band, a large branch of the left anterior descending coronary artery that passes through the moderator band and is up to 1 mm in diameter, may be an important source of blood supply to the anterolateral portion of the right ventricular free wall and that the moderator band artery provides a major source of flow to the anterior papillary muscle of the right ventricle.

Wade emphasized that in cases of right coronary artery occlusion, the right ventricle receives much of its blood supply from the left coronary artery, particularly through branches that traverse the septum and anterior third of the right ventricle. He stated that although right ventricular infarcts occur directly as a result of right coronary occlusion, preexisting lesions in the left anterior descending coronary artery play an important role in the pathogenesis.

**TABLE 1. Correlation of Selected Pathologic Variables with Size of the Right Ventricular Infarct**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Right ventricular infarct size</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0%</td>
</tr>
<tr>
<td>No. of pts</td>
<td>7</td>
</tr>
<tr>
<td>Heart weight (g)*</td>
<td>415</td>
</tr>
<tr>
<td>Coronary atherosclerosis grade (0-4 +)*</td>
<td>2.8 +</td>
</tr>
<tr>
<td>Obstruction right coronary artery*</td>
<td>80%</td>
</tr>
<tr>
<td>Left ventricular infarct size*</td>
<td>18%</td>
</tr>
<tr>
<td>Obstruction to potential collateral blood flow (0-4 +)†</td>
<td>1.1 +</td>
</tr>
</tbody>
</table>

*NS.
†r = 0.54, p < 0.02.

**FIGURE 2.** Two patients with large right ventricular infarcts caused by proximal right coronary artery lesions also had obstructive lesions in the first septal perforator branch of the left anterior descending coronary artery, which gives rise to the artery of the moderator band. (A) Postmortem coronary arteriogram showing the lesion at the upper right arrow. The moderator band artery originates from the septal perforator (lower left arrow). (B and C) Postmortem arteriogram and gross photograph of heart with massive right ventricular infarction. The obstructive lesion in the septal perforator is shown by the lower arrow in B. The moderator band artery is at the upper arrow. LV = left ventricle; RV = right ventricle; IVS = interventricular septum.
Ratliff and Hackel observed that isolated right ventricular infarcts are rarely seen at autopsy, except in cases of right ventricular hypertrophy associated with pulmonary hypertension. Peter et al. studied a porcine model of myocardial infarction and found that experimentally induced right ventricular hypertrophy rendered the right ventricle susceptible to infarction and impaired collateralization of the occluded right coronary artery in some cases. In pigs without right ventricular hypertrophy, occlusion of the right coronary artery consistently produces infarction of the posterior left ventricle and interventricular septum, but spares the right ventricle. Ratliff and Hackel interpret these findings as evidence that right ventricular hypertrophy impairs collateral blood flow to the right ventricular myocardium. Although an alternative interpretation is that right ventricular oxygen demand is increased with hypertrophy; impaired blood flow probably also plays a role. There was no significant correlation of right ventricular infarct size with right ventricular hypertrophy in our series, suggesting that factors other than ventricular thickness are determinants of relative right ventricular ischemia.

The presence of contraction band necrosis in one patient in our series is interesting in light of the proposed importance of collateral flow in protection of the right ventricular myocardium. This type of myocardial necrosis is typical of reperfusion. This patient had an embolic occlusion of the proximal right coronary artery but normal coronary arteries. Presumably, flow through the left coronary arterial system caused rapid reperfusion of the ischemic area and resulted in contraction band necrosis.

In conclusion, the results of our study support the concept that collateral flow from the left anterior descending coronary artery, especially through the moderator band artery, protects the right ventricle in cases of acute proximal right coronary artery occlusion.

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Comparison of Early Thallium-201 Scintigraphy and Gated Blood Pool Imaging for Predicting Mortality in Patients with Acute Myocardial Infarction

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SUMMARY The extent of abnormality in early thallium-201 and gated cardiac blood pool scintigrams has been reported to be useful for predicting mortality in patients with acute myocardial infarction (AMI). To compare the two techniques, 91 patients admitted consecutively with evident or strongly suspected AMI underwent both imaging studies within 15 hours of the onset of symptoms. Patients with pulmonary edema or shock were excluded. AMI developed in 84% of patients, and 6-month mortality for the entire group was 16%. A thallium defect score of 7.0 or greater (corresponding to at least a moderate reduction of activity involving 40% of the left ventricular circumference) identified a subgroup of 14 patients with 64% 6-month mortality rate. Similarly, a left ventricular ejection fraction of 35% or less identified a high-risk subgroup of 10 patients with a 6-month mortality of 60%. Mortality in the remaining patients was 8% for thallium score less than 7 and 11% for ejection fraction greater than 35%. The mortality rate was highest among patients who had concordant high-risk scintigrams (five of six, 83%), lowest in those with discordant low-risk studies (five of 64, 8%) and intermediate in those with discordant results (four of 11, 36%). Of a number of clinical variables, only the appearance of Q waves, peak creatine kinase > 1000 IU/l, and history of infarction were significantly associated with mortality. High-risk thallium or blood pool scintigraphic results were significantly more predictive and a thallium score of 7 or greater was more sensitive for detecting nonsurvivors than ejection fraction 35% or less at a similar level of specificity. Stepwise multiple logistic analysis showed that the thallium score was the best predictor of mortality, but that appearance of Q waves and ejection fraction were additive. Using these three variables, 11 patients were calculated to have a 50% or greater chance of dying and eight (73%) actually died, compared with six of 70 (9%) with a calculated chance of death of less than 50%. These results in a prospectively identified and consecutive group of patients support the value of early thallium and blood pool scintigraphy for separating high- and low-risk subgroups of hemodynamically stable infarct patients.

PATIENTS with acute myocardial infarction admitted in pulmonary edema or cardiogenic shock are at a high risk for early death. Those who demonstrate lesser degrees of hemodynamic compromise, in clinical class I or II, have a much better prognosis as a group, but among these patients is a subset with poor prognosis. Early identification of those at highest risk would allow optimal application of aggressive therapeutic interventions with the minimum exposure of low-risk patients to adverse effects. Initiation of treatment before hemodynamic deterioration should permit salvage of jeopardized, but not yet infarcted, myocardium with subsequent improvement in prognosis.

A number of approaches have been used for early identification of high-risk patients, including clinical classifications based on the degree of heart failure, invasive hemodynamic characterization, and multivariate equations using combinations of clinical variables. We previously reported the use of early thal-
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