Patterns of Coronary Compromise Resulting in Acute Right Ventricular Ischemic Dysfunction

Terry R. Bowers, MD; William W. O’Neill, MD; Mark Pica, BS; James A. Goldstein, MD

Background—Although proximal right coronary artery (RCA) occlusion is the culprit commonly responsible for acute right ventricular (RV) infarction (RVI), the severity of RV dysfunction ranges broadly. This study was designed to delineate the patterns of coronary compromise that determine the magnitude of RV ischemic dysfunction.

Methods and Results—In 125 patients with acute inferior myocardial infarction undergoing emergency angiography, the culprit infarct lesion was identified, RV branch flow assessed (TIMI flows and frame counts), and individual patient RV perfusion indices calculated by separately averaging the branch flows and frame counts, which were correlated with RV wall motion by ultrasound. RVI occurred in 53 (42%) patients, with the RCA as the culprit vessel and the lesion sufficiently proximal to compromise flow in at least one RV branch in all cases, thereby resulting in depressed RV perfusion (flow index, 0.7±0.2). In patients without RVI, the RCA was the culprit in 89%; the circumflex, in 11%. RCA culprits were proximal in 19% of such cases, with lack of RVI explained by preserved RV perfusion (flow index, 2.7±0.3; P=0.001) attributable to at least 1 patent RV branch, spontaneous reperfusion, or prominent collaterals. Overall, there was a strong correlation between RV perfusion and wall motion (Spearman correlation coefficient =0.79).

Conclusions—Proximal RCA occlusion compromising RV branch perfusion commonly results in RV ischemic dysfunction. In some cases with proximal RCA culprits, collaterals or spontaneous reperfusion preserve RV performance.

Key Words: ventricles ■ arteries ■ myocardial infarction ■ collateral circulation

Right ventricular (RV) infarction (RVI) associated with inferior-posterior left ventricular (LV) myocardial infarction contributes to higher in-hospital morbidity and mortality.1–10 Global RV performance is predominantly determined by the RV free wall (RVFW), which receives blood flow primarily from RV branches of the right coronary artery (RCA). Although RCA occlusion compromising RV branch flow is the culprit commonly responsible for RVI,2,4–10 some patients with proximal RCA occlusion develop minimal acute RV dysfunction, whereas in others, RVI occurs with more distal occlusions. Furthermore, in those with RVI, the severity of RV dysfunction ranges from mild contraction abnormalities to profound depression of RV performance. We hypothesized that the magnitude of RVFW dysfunction would correlate with the extent of RV branch flow impairment. The purpose of this study was to delineate the derangements of coronary anatomy that determine the severity of RV ischemic dysfunction.

Methods

Patients presenting to William Beaumont Hospital with acute inferior myocardial infarction (chest pain with ST elevation ≥1 mm in leads II, III, and aVF) underwent urgent 2D echocardiography before reperfusion to document the presence of RV ischemic dysfunction.5,8,11,12 Of 165 patients screened, 40 were excluded because of technically inadequate echocardiographic images, leaving 125 patients analyzed in this study who underwent emergency coronary angiography according to standard techniques.5,13–15

Assessment of Ventricular Function

Echocardiograms were analyzed according to previous methods.5,8,11,12 To assess global RV performance, RV area was measured at end-diastole and end-systole, and RV fractional area change was calculated. RVFW motion was analyzed in the apical 4-chamber view; the RVFW was divided into equal apical, middle, and basal segments, and a qualitative motion score (normal, 1; hypokinetic, 2; akinetic, 3; and dyskinetic, 4) was determined for each segment. Total RVFW motion score was calculated as the average of the RVFW segments. LV ejection fraction was determined by cineangiography.

Assessment of Coronary Anatomy and Flow

Detailed analysis of the infarct-related artery was performed, including quantitative calculation of percent stenosis and analysis of flow rates according to the Thrombolysis in Myocardial Infarction (TIMI) methodology.13–15 To characterize RVFW perfusion, the RCA was analyzed to determine the flow pattern in the RV branches. The location of the culprit lesion was noted relative to origin of the RV branches and defined as proximal (before the first major RV branch...
Statistical Methods

All data are expressed as mean±SD. Comparisons were made with use of the χ² test for categorical variables and a 2-tailed Student’s t test for normally distributed continuous variables. Comparisons between groups were made with a 2-tailed Student’s t test. For analysis of flow parameters and perfusion indices, comparisons between the RV infarction positive and negative groups were made for the continuous variables using a Wilcoxon rank test, a nonparametric approximation of the t test. Comparisons of average wall motion scores between the TIMI flow scores and TIMI frame count categories were performed with a Kruskal-Willis test, a nonparametric approximation of an unbalanced ANOVA based on ranks. The relationship between RV perfusion and RVFW motion index values versus the RV wall motion score with separate plots for the total wall motion score as well as the worst single segment score. Statistical significance was defined as P<0.05.

Results

Patient Characteristics

In all 125 patients, echocardiography documented LV inferior-posterior wall motion abnormalities. RV ischemic dysfunction was evident by ultrasound in 53 (42%) patients (RVFW motion score, 3.0±0.4), whereas in 72 (58%), RV function was normal (RVFW motion, 1.0±0.3; P<0.001). The groups were similar with respect to age (61±14 versus 60±14 years), sex (58% male versus 60% female), and baseline risk factors, including hypertension (51% versus 37%), diabetes mellitus (17% versus 20%), hypercholesterolemia (40% versus 37%), and smoking (40% versus 35%), as well as prior MI (15% versus 17%), coronary bypass surgery (2% versus 3%), PTCA (23% versus 18%), and times from symptom onset to presentation (2.2±3.4 versus 2.4±2.8 hours) and “door to reperfusion” (1.3 0.8 versus 1.5 0.7) (all P=NS). As in prior studies, patients with RV ischemic dysfunction suffered an increased incidence of systemic hypotension (aortic systolic pressure <90 mm Hg; 43% versus 3%; P<0.01), bradyarrhythmias (40% versus 16%; P<0.05), need for transvenous pacemakers (30% versus 13%; P<0.05), and sustained ventricular tachyarrhythmias (30% versus 7%; P<0.01).

Patterns of Coronary Compromise in Patients With RV Ischemic Dysfunction

In all (100%) patients with RV ischemic dysfunction, the RCA was the infarct-related vessel, and there was severe compromise of RVFW perfusion, as indicated by depressed RV branch flow index (0.7±0.2). The culprit lesion was proximal to all major RV branches in 38 (71%) cases, mid vessel in 12 (23%), and distal in 3 (6%) others (Table). There was impaired flow in at least 1 major RV branch in all (100%) cases, with diminished flow in all major RV branches in 51 (96%) patients (Table). Aggregate analysis of flow in all RV branches in patients with RV dysfunction documented impaired flow in 97% of all branches, with TIMI 0 flow in 56% of branches. In all 38 cases of proximal occlusion, no RV branches were patent (Figure 2).

Angiographic analysis provided explanations for RV ischemic dysfunction in patients with mid or distal culprit lesions. In all 12 patients with mid-RCA lesions, flow was impaired in at least 1 major RV branch attributable in all cases to a culprit stenosis interposed between 2 major RV branches and compromising flow in the more distal branch; in the 3 patients with distal lesions, RV branch flow impairment (Figure 3) was associated with branch stenosis or thrombus, which may have extended retrograde from an adjacent clot-laden lesion. In patients with RV dysfunction, left-to-right collaterals were evident by angiography in 20 (38%) cases. A Spearman correlation analysis confirmed a strong correlation in individual patients between reduction of RV perfusion index calculated both by TIMI flow grades and frame count methods and by impairment of RV wall motion (R=0.79, P<0.0001) (Figure 4). In those with RVI, there was impairment of RA branch flow in 9 (8%) patients (RA branch flow index,
Culprit Lesion, Impaired Flow, RV Branch Perfusion Index, and RVFW Motion Index

<table>
<thead>
<tr>
<th></th>
<th>RV(+) (n=53)</th>
<th>RVI(−) (n=72)</th>
<th>P</th>
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<tr>
<td>RCA culprit vessel</td>
<td>53 (100)</td>
<td>64 (89)</td>
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<tr>
<td>Circumflex culprit vessel</td>
<td>0 (0)</td>
<td>8 (11)</td>
<td>. .</td>
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<td>RCA culprit lesion location</td>
<td></td>
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<tr>
<td>Proximal</td>
<td>38 (71)</td>
<td>14 (19)</td>
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<tr>
<td>Mid</td>
<td>12 (23)</td>
<td>34 (48)</td>
<td>0.01</td>
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<tr>
<td>Distal</td>
<td>3 (6)</td>
<td>16 (22)</td>
<td>0.01</td>
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<tr>
<td>Patients with impaired flow in at least 1 RV branch</td>
<td>53 (100)</td>
<td>12 (18)</td>
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<tr>
<td>Patients with impaired flow in all RV branches</td>
<td>51 (96)</td>
<td>7 (11)</td>
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<tr>
<td>Patients with TIMI 3 flow in at least 1 RV branch</td>
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<tr>
<td>Patients with TIMI 3 flow in all RV branches</td>
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<td>52 (81)</td>
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<td>RV branch with impaired flow, %</td>
<td>97</td>
<td>9</td>
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<tr>
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<td>56</td>
<td>8</td>
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<tr>
<td>RA branch with impaired flow, %</td>
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<td>0.001</td>
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<td>RV branch perfusion index*</td>
<td>0.7±0.2</td>
<td>2.7±0.3</td>
<td>0.001</td>
</tr>
<tr>
<td>RVFW motion index</td>
<td>3.0±0.6</td>
<td>1.2±0.3</td>
<td>0.001</td>
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</tbody>
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Values are n (%), mean±SD, or percentage.

*RV branch perfusion index calculated with the use of TIMI flow grade.

0.8±1.0 versus 3.0±0.0 in those with RVI but with intact RA branches; P=0.005; all such cases were attributable to very proximal occlusions (Table). Because proximal occlusion was responsible for 29/51 (57%) of cases with RVI, the incidence of RA branch flow compromise with such proximal lesions was 31%. Perfusion to the LV branches of the RCA was severely compromised in all patients (LV branch flow, 0.6±0.2).

Patterns of Coronary Compromise in Patients Without RV Dysfunction

In those without RVI, the RCA was the infarct-related vessel in 64 (89%), whereas in 8 (11%) the left circumflex was the culprit. RV branch flow was preserved and was significantly greater compared with those with RV ischemic dysfunction (2.7±0.3 versus 0.7±0.2; P=0.001). In those with RCA culprits, the lesion was distal to all major RV branches in 16 (22%), mid RCA in 34 (48%), and proximal in 14 (19%). In those with distal occlusions, all (100%) RV branches were patent with intact flow (Table). Angiographic analysis provided potential explanations for patients with intact RV function despite proximal or mid RCA lesions. In those with mid RCA lesions, TIMI 3 flow was present in ≥1 major RV branches (Figure 5). In patients with proximal culprit lesions, angiography also demonstrated preserved RV branch perfusion; in 7 cases, there was TIMI 3 flow antegrade through a high-grade stenosis, consistent with spontaneous reperfusion (Figure 6). In 7 with proximal total occlusion, the absence of RV involvement appeared to be explained by prominent
left-to-right collaterals (Figure 7). Overall, however, the presence of collaterals was similar in those with and without RV dysfunction (38% versus 43%; \( P = \text{NS} \)). Compared with patients with RVI, those with preserved RV performance had significantly greater incidence of TIMI 3 flow in \( \geq 1 \) major RV branch (91% versus 4%; \( P = 0.001 \)) and in all major RV branches (81% versus 0%; \( P = 0.001 \); conversely, there was a lower incidence of impaired flow in at least 1 branch (18% versus 100%; \( P = 0.001 \)) and in all major RV branches (11% versus 96%; \( P = 0.001 \)). There was a strong correlation between maintenance of RVFW perfusion and preservation of RV wall motion (Figure 4). No patient without RVI developed RA branch flow compromise. Perfusion to the LV branches was similarly compromised in both groups (LV branch flow index, 0.5 \( \pm \) 0.2; \( P = \text{NS} \)).

**Discussion**

Observations from this study delineate the patterns of coronary compromise that determine the magnitude of ischemic RV dysfunction in patients with acute inferior myocardial infarction. The status of RV branch perfusion is the critical determinant of RV performance. Proximal RCA occlusion typically compromises RV branch perfusion, resulting in RV ischemic dysfunction, whereas distal RCA occlusions rarely do. Even under conditions of severe proximal culprit lesions, however, RV branch flow may be maintained by spontaneous reperfusion or collaterals, thereby preserving RV performance.

Based on previous clinical reports, proximal RCA occlusion is accepted as the culprit most commonly responsible for RVI.\(^1\)\(^-\)\(^{10}\) Some patients with proximal occlusions, however, develop minimal or no RV dysfunction, whereas in others, RV involvement develops in association with more distal occlusions. Furthermore, in those with RVI, there is a broad spectrum in the magnitude of RV dysfunction,\(^1,3,4,10\) the pathophysiological basis of which has not previously been elucidated.

Observations from the present study are consistent with and extend those of previous reports documenting an association between proximal RCA occlusion and RV dysfunction. The present findings now document a direct correlation between the extent of RV branch flow compromise and the...
magnitude of RV ischemic dysfunction. These observations also delineate the patterns of coronary compromise associated with preservation of RV performance, despite proximal RCA culprit lesions, and the development of RV ischemic dysfunction with more distal occlusions. In patients with RVI, the RCA was the culprit vessel, and RV branch perfusion to the RVRW was severely compromised in all cases, with the majority of such cases manifesting high-grade proximal RCA occlusions compromising flow to all major RV branches. Severe RV ischemic dysfunction was also seen, albeit less commonly, in patients with mid-vessel lesions; in all such cases, there was flow impairment in at least 1 major RV branch attributable to a culprit stenosis interposed between 2 RV branches with flow compromise in the more distal branch. Angiographic analysis provided potential explanations for the few patients with RV ischemic dysfunction associated with a distal culprit lesion, all of whom showed impairment of the RV branch flow that appeared to be attributable to thrombus extending retrograde from an adjacent distal culprit lesion.

Patients without RVI tended to have infarct-related lesions in the RCA that spared perfusion to the preponderance of major RV branches, thereby preserving RVFW perfusion. In some cases without RV involvement, the culprit lesion was in the left circumflex coronary artery, none of which manifested RVI. Although the majority of patients with RCA culprit vessels without RVI had mid or distal RCA culprit lesions, nearly 20% manifested culprit lesions proximal to the first major RV branch. Angiographic analysis provided the basis to explain preserved RV function in these patients. In some with proximal occlusions, intact RV function was explained by antegrade flow through a high-grade lesion, consistent with spontaneous reperfusion, a phenomenon reported in \(~10\%\) of patients with acute myocardial infarction.\(^\text{18}\) In others, preservation of RV performance despite proximal lesions was explained by prominent collaterals.

The present findings are consistent with and extend those of prior studies in experimental animals\(^\text{11,12,19,20}\) and in man,\(^\text{5,10,19,20}\) demonstrating that proximal RCA occlusion compromises RV branch perfusion, resulting in severe RV dysfunction. Selective RV branch compromise alone impairs RV function, even when the main body of the RCA and its distal LV branches are spared.\(^\text{8,19,20}\) Observations from experimental animal and human studies documenting the beneficial effects of reperfusion on recovery of RV performance further emphasize the importance of RV branch perfusion status.\(^\text{8,11,12}\) Until recently, RCA perfusion status was defined predominantly on the basis of flow in the main RCA and its LV branches only, largely ignoring the status of the RV branches. Studies in patients with acute RVI now demonstrate that successful complete reperfusion of the RCA, including flow restoration to the major RV branches, leads to immediate improvement in and later complete recovery of RV performance, which is associated with excellent clinical outcome.\(^\text{8}\) In contrast, failure to restore RV branch flow is associated with lack of recovery of RV performance and refractory hemodynamic compromise, leading to high in-hospital mortality, even if flow to the LV branches is restored. Given the documented salutary effects of RV branch reperfusion, it may reasonably be concluded from the present observations that reperfusion of RCA occlusions compromising RV branch flow exerts beneficial effects on RV performance and clinical outcome. Whether selective recanalization of compromised RV branches in patients with successful reperfusion of the main RCA and its LV branches but persistently occluded RV branches is efficacious requires further study.

We have previously demonstrated that very proximal RCA occlusions compromise RA and RV branches, resulting in ischemic RA dysfunction that exacerbates hemodynamic compromise associated with RVI owing to loss of the critical compensatory contribution of augmented RA contraction to RV performance.\(^\text{5,10,19,20}\) The present findings extend those prior findings, documenting that very proximal occlusions often compromise RA branch flow. Based on prior observations correlating RA branch flow and RA contractility, it is likely that compromised RA perfusion resulted in RA ischemic dysfunction in many such cases; however, lack of echocardiographic or hemodynamic data on RA function precludes such conclusions in the present study.

It is important to consider other limitations pertinent to the methods of this study. Although TIMI flow grade and frame count methodology are the accepted standard for angiographic assessment of coronary flow, these techniques measure angiographic flow rates, an approximation of true myocardial perfusion. The observed preservation of RV function under conditions of culprit lesions proximal to the RV branches appeared to be associated with spontaneous reperfusion or collaterals and is consistent with prior experimental animal and clinical studies of RV ischemia documenting prompt recovery of RV function after successful reperfusion and the protective role of collaterals.\(^\text{8,11,12,21}\) Although angiographic data were not available instantaneously at the initial onset of occlusion, it is nevertheless likely that in the context of initial presentation with inferior
ST elevation, spontaneous reperfusion explains the observed intact antegrade flow through proximal high-grade lesions. Given the more favorable oxygen supply-demand characteristics of the RV compared with the LV and the strength of observations supporting the notion that the preponderance of acutely dysfunctional RV myocardium is viable, it is not surprising that spontaneous reperfusion and collateral restoration of flow would exert prompt salutary effects on RV function.

The protective benefit of collaterals has previously been suggested to explain the lower risk of developing RV infarction in patients with acute inferior myocardial infarction and antecedent angina. Although collaterals may preclude RV ischemic dysfunction in patients with proximal RCA lesions, in the present study, however, the mere angiographic presence alone of collaterals did not necessarily preclude RV ischemic dysfunction. This discrepancy may be attributable at least in part to limitations in correlation between angiographic estimation of collateral flow and physiological myocardial perfusion. Proximal lesion angioplasty may result in compromise of RV branches, resulting in RV ischemic dysfunction despite an otherwise patent RCA and previously normal RV. Therefore, we cannot exclude the possibility that some patients in the present study may have suffered such selective branch compromise, which influenced RV function. Finally, although our observations support those of previous studies that emphasize the importance of the status of RV branch perfusion to RV performance, whether selective recanalization of persistently occluded or stenosed RV branches improves RV performance in patients with otherwise patent arteries will need to be clarified in future studies.

In summary, these findings demonstrate that in patients with acute inferior myocardial infarction, RV branch perfusion is the critical determinant of RV performance. Proximal RCA occlusion compromising flow to the major RV branches is the most common anatomic substrate resulting in RV ischemic dysfunction, whereas more distal occlusions rarely compromise RV performance. However, even in those with proximal RCA culprit lesions, spontaneous reperfusion and collaterals may maintain RV branch flow and thereby preserve RV function.

References

2. Isner JM, Roberts WC. Right ventricular infarction complicating left ventricular infarction secondary to coronary heart disease: frequency, locations, associated findings and significance from analysis of 236 necropsy patients with acute or healed myocardial infarction. Am J Cardiol. 1978;42:885–894.
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