Determinants of Hemodynamic Compromise With Severe Right Ventricular Infarction

James A. Goldstein, MD, Benico Barzilai, MD, Thomas L. Rosamond, MD, Paul R. Eisenberg, MD, and Allan S. Jaffe, MD

To elucidate determinants of hemodynamic compromise in patients with acute right ventricular (RV) infarction, we studied 16 patients with hemodynamically severe RV infarction by right heart catheterization and two-dimensional ultrasound. Severe RV systolic dysfunction, evident by ultrasound in all patients as RV dilatation and depressed RV free wall motion, was associated with a broad sluggish RV waveform, diminished peak RV systolic pressure (27.6±4.5 mm Hg), and depressed RV stroke work (4.6±2.4 g · m/m²). Paradoxical septal motion was consistently noted. In some cases, the septum bulged into the right ventricle in a pistonlike fashion and appeared to mediate systolic ventricular interaction through which left ventricular septal contraction contributed to RV pressure generation. RV diastolic dysfunction was indicated by elevated RV end-diastolic pressures (13.7±2.7 mm Hg), RV “dip and plateau,” equalization of diastolic filling pressures, and reversal of diastolic septal curvature toward the volume-deprived left ventricle. A prominent right atrial (RA) X and blunted Y descent, indicative of impairment of RV filling throughout diastole, were confirmed in all patients by their relation to RV systolic events. Patients manifested one of two distinct RA waveform morphologies differentiated by A wave amplitude and associated with disparate clinical courses. In eight patients, an RA W pattern was evident, characterized by augmented A waves; eight others manifested an M pattern constituted by depressed A waves. Compared with those with an M pattern, patients with a W pattern had higher peak RV pressures (29.6±3.8 versus 25.5±4.3 mm Hg, p<0.05), better cardiac output (3.4±0.3 versus 2.9±0.7 l/min, p<0.05), more favorable response to volume and inotropes, and less frequently required emergency revascularization for refractory shock (none versus five for those with an M pattern). Patients with a W pattern were more severely compromised if atrioventricular dyssynchrony developed and were more dramatically improved by restoration of physiological rhythm. Angiography in patients with depressed A waves demonstrated more proximal coronary obstruction leading to ischemic compromise of RA function, whereas in those with augmented A waves, the culprit lesion was proximal to the RV but distal to the RA branches. These results indicate that hemodynamic compromise in patients with RV infarction is exacerbated by decreased preload reserve that is dependent on atrial systole. The amplitude of the RA A wave, an indication of the status of RA function, is an important determinant of RV performance and hemodynamic compromise. (Circulation 1990;82:359–368)

Infarction of the right ventricle is common in patients with transmural inferoposterior myocardial infarction1–3 and may result in hemodynamic compromise despite adequate left ventricular (LV) function.2–5 In experimental animals, ischemic injury of the right ventricular (RV) free wall depresses RV systolic performance, which in association with diastolic ventricular interaction induced by acute RV dilatation and elevated intrapericardial pressure leads to deprivation of LV end-diastolic volume, resulting in reduced cardiac output and hypotension.6–9 Although the severity of hemodynamic derangements in patients with RV infarction is related to the extent of RV free wall dysfunction,2,5 some patients tolerate severe depression of RV contractility without hemodynamic compromise, whereas others manifest life-threatening low cardiac output despite similar extents of RV systolic impairment.2,3,5 Furthermore, the mechanisms by which RV pressure and stroke volume are generated in the absence of demonstrable RV free wall motion have not yet been delineated. To clarify factors that determine RV performance and the magnitude of hemodynamic

From the Washington University School of Medicine, St. Louis, Mo. Supported in part by National Institutes of Health SCOR in Ischemic Heart Disease grant HL-17646, Bethesda, Md. Address for correspondence: James A. Goldstein, MD, Cardiovascular Division, Washington University School of Medicine, 660 South Euclid Avenue, Box 8086, St. Louis, MO 63110. Received September 7, 1989; revision accepted March 13, 1990.
Impairment in patients with severe RV infarction, we studied 16 consecutive patients with hemodynamically severe RV infarction by right heart catheterization and two-dimensional ultrasound.

Methods

From January 1988 through January 1989, 16 consecutive patients admitted to the Cardiac Care Unit at Barnes Hospital, Washington University Medical Center, within 72 hours of onset of acute transmural inferoposterior myocardial infarction with hemodynamic compromise consistent with RV infarction were studied. Criteria for study included 1) electrocardiographic evidence of inferior ST elevation or Q waves with 2) hypotension (systolic blood pressure, \( \leq 95 \) mm Hg), elevated neck veins (jugular venous pressure, \( \geq 10 \) cm H_2O), and clear lungs without radiographic evidence of pulmonary venous congestion and 3) echocardiographic evidence of RV dilatation and RV free wall motion abnormalities.

The clinical indication for right heart catheterization was hypotension. Right atrial (RA), RV, pulmonary artery (PA), and pulmonary capillary wedge (PCW) pressures were measured with fluid-filled floatation catheters (American Edwards, Irvine, Calif.). A double-lumen catheter, with a proximal (RA) lumen (nominal frequency response, 35 Hz) and a distal lumen (RV or RA pressure frequency response, 25 Hz), was used in nine patients. In seven other patients, a triple-lumen catheter was used with a distal (PA) lumen (nominal frequency response, 17 Hz), an RV lumen (frequency response, 26 Hz), and a proximal (RA) lumen (frequency response, 20 Hz). The manometer and interconnect tubing were optimized for critical damping. The band-pass filters for the strip-chart recorder (Hewlett-Packard, Palo Alto, Calif.) was set at 250 Hz, and the photographic recorder (Hewlett Packard Instruments, Minneapolis, Minn.) was set at 500 Hz. The recording system (manometer, interconnect tubing, catheter, and strip-chart recorder) had a flat frequency response out to 40 Hz. Pressures were recorded simultaneously with the electrocardiogram at paper speeds of 25 or 50 mm/sec. The RA pressure waveform was recorded simultaneously with RV pressure in 10 patients and with PA pressure in the remaining six. In seven patients, simultaneous and superimposed RA and RV pressures were recorded (Hewlett Packard). Cardiac output was calculated by thermodilution and repeated in triplicate, and the results were averaged. Hemodynamic measurements were recorded immediately after placement of the right heart catheter. Blood pressure was measured by sphygmomanometer \((n=8)\) or through an arterial fluid-filled catheter \((n=8)\). Two-dimensional ultrasound studies (Hewlett-Packard) were performed within 2 hours of the right heart hemodynamic measurements. Hospital charts were analyzed to assess changes in hemodynamics over time and in the clinical courses of patients. Cineangiograms were reviewed to obtain results of LV and coronary angiography.

Measurement of Hemodynamic Parameters

Peak pressures and electrical mean pressure were measured from strip-chart and photographic recordings. Peak and mean pressures, slopes of the RA negative waves (X and Y), and areas of the A and V waves were determined by planimetry from the strip-chart and photographic recordings. The RA waveform components, timed by simultaneous electrocardiography and RV or PA recordings, were defined as follows. The A wave was the initial positive wave after the P wave of the electrocardiogram and just before RV/PA systolic pressure generation, the X descent was the first negative wave after the A wave, the V wave was the last positive deflection before the next A wave and coincident with peak systolic pressure, the Y descent was the negative wave after the V wave and just before the subsequent A wave, and the C wave, when present, was a positive wave after the A wave and occurring simultaneous with early systolic pressure generation. The presence of a C wave separated the X descent into an X component before and an \( X' \) descent after the C wave.

Echocardiographic Analysis

Echocardiograms were recorded on \( \frac{1}{2} \) -in. videotape and analyzed by two experienced echocardiographers (J.G. and B.B.). RV dilatation was designated as mild, moderate, or severe, and RV free wall motion abnormalities as normal, hypokinetic, akinetic, or dyskinetic. LV size and regional wall motion abnormalities were assessed similarly. The end-diastolic orientation of the inteventricular septum was assessed as normal, flattened, or frankly reversed curved. The presence or absence of paradoxical systolic motion of the septum anteriorly toward the RV cavity was noted.

Statistics

Group hemodynamic measurements were reported as mean \( \pm \) SD. Differences in measurements between groups were analyzed by unpaired \( t \) test. Significance was defined at the 95% confidence level.

Pertinent Considerations

The hemodynamic data reported in this study were obtained from measurements recorded with fluid-filled catheters. Care was taken to ensure proper catheter positioning by analysis of waveforms and fluoroscopy. Catheters were flushed and balanced before each recording. Catheter artifact from entrapment or excessive motion was unlikely because the right heart chambers were markedly dilated and RV contraction was depressed, conditions that tend to lessen the likelihood of these types of artifact. Furthermore, the RA and RV waveforms in the present study are morphologically similar in many respects to those reported in previous clinical studies of severe RV infarction\(^{10-13}\) and to those observed in experi-
mental models of acute RV dysfunction using high-fidelity micromanometer catheters.6–9,14 Waveform analysis was facilitated by timing with mechanical events by simultaneous RV or PA pressure as well as with electrical events by electrocardiography. Due to concern that fluid-filled catheters may not permit quantitation, particularly of subtle abnormalities, only clearly apparent qualitative distinctions have been emphasized. The statistics reported have been used only as descriptors of these qualitative observations. Furthermore, because our patient population included only patients with predominant severe RV infarction, caution must be used in extrapolating these results to patients with less severe RV infarction or with associated severe LV infarction.

Results

Clinical Course

All patients in this study presented with RV infarction complicated by hypotension severe enough to warrant hemodynamic evaluation with right heart catheterization. The initial episode of hypotension developed within the first 24 hours of infarction in 14 of 16 patients. Hypotension occurred initially in association with sinus bradycardia in 15 of 16 patients. Subsequent progression to advanced atrioventricular (AV) block occurred in nine patients who required temporary pacemakers. Ventricular demand (VVI) pacing was adequate in five in whom AV block was intermittent; however, in four other

patients, AV synchronous pacing was required. Six of 16 patients with hypotension and low cardiac output responded to initial treatment with fluids, atropine, and temporary pacing (Figure 1). Ten other patients manifested hypotension and low cardiac output refractory to these initial therapeutic measures; they required support with positive inotropic drugs and other interventions including intra-aortic balloon pumping (n=3), emergency coronary artery bypass graft surgery (n=4), or emergency percutaneous transluminal coronary angioplasty (n=1). Although all patients undergoing emergency surgical revascularization for cardiogenic shock had severe and protracted postoperative low cardiac output, all survived and were ultimately hemodynamically improved.

Five of the 16 patients died; in each of the five patients, volume expansion and cardiac stimulation with drugs initially elicited apparent hemodynamic stability. However, several days later, recurrent chest pain associated with anterior or lateral ischemic electrocardiographic changes heralded the onset of refractory cardiogenic shock. All of the five patients deteriorating in this fashion were elderly (71, 77, 80, 81, and 85 years old) and had contraindications to more aggressive intervention or revascularization.

Right Atrial Pressure

Mean RA pressure increased in all patients (mean RA pressure, 13.7±3.8 mm Hg), as did the mean amplitude of the RA A wave (16.5±4.1 mm Hg)
TABLE 1. Hemodynamic Parameters in Right Ventricular Infarction

<table>
<thead>
<tr>
<th>Age (yr)/sex</th>
<th>Initial rhythm/heart rate</th>
<th>AoP</th>
<th>CO</th>
<th>RA</th>
<th>RA A</th>
<th>X/Y</th>
<th>RVSP</th>
<th>RVEDP</th>
<th>RVDP</th>
<th>RVS</th>
<th>PCW</th>
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<tr>
<td>Patients with RA W pattern</td>
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<tr>
<td>80 F</td>
<td>SR/65</td>
<td>90</td>
<td>2.8</td>
<td>12</td>
<td>15</td>
<td>1.2</td>
<td>25</td>
<td>1.25</td>
<td>5.3</td>
<td>15</td>
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<tr>
<td>48 M</td>
<td>SB/45</td>
<td>70</td>
<td>3.6</td>
<td>10</td>
<td>13</td>
<td>3.1</td>
<td>27</td>
<td>1.8</td>
<td>8.2</td>
<td>10</td>
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<tr>
<td>61 M</td>
<td>2° AV/62</td>
<td>70</td>
<td>3.6</td>
<td>18</td>
<td>24</td>
<td>2.2</td>
<td>36</td>
<td>1.23</td>
<td>7.2</td>
<td>17</td>
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<tr>
<td>81 M</td>
<td>Junct/60</td>
<td>95</td>
<td>3.4</td>
<td>10</td>
<td>13</td>
<td>1.2</td>
<td>30</td>
<td>1.2</td>
<td>8.5</td>
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<tr>
<td>52 M</td>
<td>SB/52</td>
<td>85</td>
<td>3.3</td>
<td>11</td>
<td>14</td>
<td>1.3</td>
<td>26</td>
<td>1.27</td>
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<tr>
<td>36 M</td>
<td>SB/45</td>
<td>75</td>
<td>3.5</td>
<td>13</td>
<td>20</td>
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<td>1.5</td>
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<tr>
<td>46 M</td>
<td>SB/48</td>
<td>95</td>
<td>3.7</td>
<td>13</td>
<td>16</td>
<td>2.7</td>
<td>29</td>
<td>1.14</td>
<td>3.8</td>
<td>19</td>
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<tr>
<td>77 F</td>
<td>2° AV/58</td>
<td>80</td>
<td>2.7</td>
<td>15</td>
<td>19</td>
<td>1.3</td>
<td>30</td>
<td>1.14</td>
<td>2.0</td>
<td>16</td>
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| Patients with RA M pattern | | | | | | | | | | | |
| 74 M         | SB/52                      | 70  | 2.0| 13 | 15   | 3.7 | 21   | 1.08  | 1.0   | 14  |
| 71 M         | 3° AV/54                   | 75  | 3.1| 11 | 12   | 1.4 | 27   | 1.0   | 2.5   | 13  |
| 56 F         | 3° AV/50                   | 65  | 1.8| 25 | 26   | 1.8 | 25   | 1.0   | 1.4   | 19  |
| 85 M         | 2°/3° AV/45                | 85  | 3.5| 14 | 16   | 1.3 | 24   | 1.0   | 2.3   | 13  |
| 52 M         | SB/50                      | 90  | 3.2| 14 | 15   | 2.1 | 35   | 1.07  | 4.4   | 14  |
| 57 M         | SB/48                      | 90  | 3.7| 13 | 15   | 2.2 | 26   | 1.23  | 4.5   | 14  |
| 55 M         | 2° AV/50                   | 80  | 3.3| 10 | 12   | 1.3 | 22   | 1.29  | 5.0   | 12  |
| 76 F         | 2°/3° AV/40                | 85  | 2.7| 17 | 19   | 1.4 | 24   | 1.17  | 2.0   | 16  |
| Mean         | 51.5                       | 81.3| 3.2 | 13.7| 16.5| 1.8| 27.6 | 1.27  | 4.6   | 14.8|
| ±SD          | (±6.9)                     | (±9.6) | (±0.6) | (±3.8) | (±4.1) | (±0.8) | (±4.5) | (±2.7) | (±0.32) | (±2.4) | (±2.7) |

AoP, aortic systolic pressure; CO, cardiac output (l/min); RA, mean right atrial pressure; RA A, peak RA A wave pressure; X/Y, ratio of slope of X and Y descents; RVSP, peak right ventricular systolic pressure; RVEDP, right ventricular end-diastolic pressure; RVDP/RVDP, ratio of RVDP to mean right ventricular diastolic pressure; RVS, right ventricular stroke work (g·m/m²); PCW, pulmonary capillary wedge pressure; SB, sinus bradycardia; Junct, junctional rhythm; AV, atrioventricular block. Values for pressures are given in mm Hg.

(Table 1). The RA pressure was disproportionately elevated in comparison to the mean PCW pressure (14.8±2.7 mm Hg), and the ratio of the mean RA pressure to the mean PCW pressure increased (RA/PCW, 0.93±0.12; normal, <0.75). When the RA waveform components were timed by the electrocardiogram, the most prominent RA descent coincided with or followed the T wave in 13 of 16 cases and occurred just before the T wave in three, in all cases suggesting a diastolic Y descent (Figures 2–5). However, when the RA waveform components were timed with RV mechanical events via simultaneous RV or PA recordings, the most prominent negative RA wave occurred during peak ventricular systole, thereby confirming a prominent systolic X descent in all patients (Figures 2–5). In each case, the Y descent was comparatively blunted (ratio of X/Y slopes, 1.8±0.8, p<0.05). In 12 of 16 patients, a C wave separated the normally monophasic systolic descent into initial X and subsequent X' components (Figures 2–5).

In eight of 16 patients, the RA waveform manifested an M-shaped pattern characterized by a diminutive A wave followed by a brisk systolic X descent (or X' in presence of a C wave), a small V wave, and a blunted Y descent (Figures 2 and 3). In eight others, prominent A waves imparted a W-shaped pattern to the RA pressure tracing (Figures 4 and 5). The ratio of the peak A wave pressure to the mean RA pressure was 20% greater in patients with a W pattern compared with those with an M pattern (peak A pressure/mean RA pressure, 1.3±0.9 versus 1.1±0.05, p<0.05; Figure 1).

In patients manifesting C waves, the most prominent negative wave was the X' descent. In patients with a W pattern, the X component preceding the C wave was also prominent, whereas in patients with an M pattern, this X descent was comparatively blunted. In one patient with a W pattern, C wave and 2° AV block, atrial contraction was followed by an X descent in all beats but an X' descent in conducted beats only, suggesting that the X subcomponent reflects atrial relaxation (Figure 5). This X descent component was more exaggerated in patients with a W pattern (Figures 4 and 5) than in those with an M pattern (Figures 2 and 3).

**Right Ventricular Hemodynamics**

The RV systolic waveform was broad (mean RV systole duration, 0.34±0.5 seconds), with a narrow pulse pressure (mean, 16.3±4.5 mm Hg), a depressed upstroke, and delayed relaxation (Figures 2, 4, and 5). Peak RV systolic pressure for the entire group was 27.6±4.5 mm Hg (Table 1). Peak RVSP was less than 30 mm Hg in 11 patients and 25 mm Hg or less in six. In 11 patients, the RV systolic waveform exhibited a bifid morphology (Figures 2 and 5). RV end-diastolic pressure was elevated in all patients (mean, 13.7±2.7 mm Hg). A diastolic “dip and plateau” pattern in the RV waveform was present in 12
patients (Figure 5), and an exaggerated RV end-diastolic pressure rise was evident in some patients with augmented RA A waves (Figure 4). Equalization of the RV, RA, and PCW pressures occurred in 11 patients.

**Echocardiographic Features**

RV cavity enlargement and RV free wall dysfunction (Figure 6) were present in all patients. The RV free wall was judged to be hypokinetic in three, akinetic in 10, and dyskinetic in three patients. LV wall motion abnormalities were confined to the inferior wall in 13 patients (five hypokinetic and eight akinetic) and the inferolateral wall in three (all hypokinetic). Mild apical-septal wall motion abnormalities were present in two patients. Overall, qualitative global LV function appeared adequate in 14 patients and moderately depressed in two. Abnormal diastolic orientation of the interventricular septum was noted in all patients; septal flattening occurred in 10, and frank reversal of septal curvature was noted in six others (Figure 6). Paradoxical systolic septal...
motion was present in 14 patients, whereas in two cases, septal motion was difficult to assess due to technical limitations. In eight patients, the septum bulged dramatically into the akinetic right ventricle in systole in a pistonlike fashion (Figure 6).

Results of Cardiac Catheterization

Eleven patients underwent cardiac catheterization. One-vessel disease was documented in four patients, two-vessel disease in two, three-vessel disease in four, and normal coronary arteries in one. The right coronary artery was considered the infarct-related artery in nine patients, a dominant circumflex with a small hypoplastic right coronary artery was considered the infarct-related artery in one patient, and normal coronary arteries were observed after thrombolysis in one patient. In the nine patients in whom the right coronary artery was the culprit vessel, a high-grade right coronary artery stenosis proximal to the RV branches was present. The degree of right coronary artery stenosis was estimated as 85% in one patient, more than 95% in two, 99% in two, and total in four. Four patients had faint distal right coronary artery collateral flow from the left circulation; in one case, collateral filling of an RV branch was noted. LV cineangiography revealed inferoposterior LV wall motion abnormalities in all patients with lateral extension in two cases and mild apical-septal hypokinesis in two others. Overall LV ejection fraction ranged from 45% to 60% (mean, 52%).

Clinical-Hemodynamic Correlations

Patients manifested one of two distinct RA waveform morphologies differentiated by A wave amplitude and associated with disparate clinical courses (Figure 1). Patients with depressed A waves (M pattern) had more severe, protracted, and refractory hemodynamic compromise than patients with augmented A waves (W pattern). Seven of eight patients
with depressed A waves developed severe persistent low cardiac outputs despite intact AV conduction and treatment with inotropic agents. Patients with diminutive A waves had more severe depression of cardiac output (M pattern, 2.9±0.7 l/min; W pattern, 3.4±0.3 l/min, p<0.05), required higher dose and more prolonged inotropic support, and more frequently required intra-aortic balloon pumping (M pattern, three; W pattern, none) and emergency revascularization for refractory shock (M pattern, five; W pattern, none). Such patients also had more severe depression of peak RV systolic pressure (25.5±4.3 compared with 29.6±3.8 mm Hg, p<0.05) and RV stroke work (2.9±1.5 compared with 6.2±1.8 g m/m², p<0.05). In contrast, only three of eight patients with augmented A waves manifested refractory low cardiac output; in each, severe hemodynamic compromise was related to the development and persistence of AV dyssynchrony. Six patients with augmented RA A waves manifested a prominent rise in RV end-diastolic pressure with atrial systole (Figure 4), whereas patients with severely depressed RA A waves (M pattern) had a less prominent increase (Figure 2), as reflected in a lower ratio of RV end-diastolic pressure to mean RV diastolic pressure (W pattern, 1.43±0.4; M pattern, 1.11±0.1, p<0.05).

Angiographic correlates also were different between these two groups of patients. Obstruction was proximal to the major RA branches of the right or circumflex coronary artery in all six patients who had diminished A waves and were undergoing angiography. In contrast, the culprit stenosis was proximal to the RV branches but distal to the RA branches in four of five patients with augmented A waves (one patient had normal coronary arteries). In two patients with depressed A waves who required emergency surgical revascularization for refractory shock, gross observation at thoracotomy documented a dilated and akinetic right atrium consistent with RA ischemia or infarction.

**Discussion**

Observations from the present study demonstrate that hemodynamic compromise in patients with severe RV infarction results from profound depression of RV free wall contraction and associated severe RV diastolic dysfunction. When RV contraction is acutely depressed, RV systolic performance is dependent, in part, on LV septal contraction. The presence or absence of compensatory changes in RA function and the integrity of AV synchrony are important determinants of the hemodynamic impact of RV systolic and diastolic dysfunction. Precise quantitation of the magnitude of effect of each of these abnormalities will require confirmatory data from future studies.
Evidence for Diastolic Dysfunction

RV diastolic dysfunction was indicated by the elevated RV filling pressures, an RV dip and plateau pattern, and equalization of diastolic filling pressures in the RA, RV, and PCW pressures, all reflecting the effects of intrinsic RV myocardial stiffness and pericardial restraint.11,12 Judging from results of animal studies of RV infarction, equalized filling pressures result from diastolic ventricular interaction induced by RV dilatation, mediated by the reversed curved septum, and exacerbated by elevated intrapericardial pressure.6–9 Mean RA pressures were elevated in all patients. When RA waveform components were identified by timing with RV mechanical correlates, the RA waveform consistently demonstrated a prominent X but blunted Y descent, reflecting increased resistance to RV filling throughout diastole. Previous studies have characterized the Y descent as prominent in patients with RV infarction10–13 who are clinically similar to those described in this report. Patients with RV infarction manifest a spectrum of RA pressure morphologies that may be affected not only by the loading conditions and systolic and diastolic properties of the right heart chambers but also by arrhythmias and tricuspid incompetence. The RA waveforms observed in the present study are morphologically similar in appearance to those in patients with normal sinus rhythm reported in prior studies.10–12 However, by relating RA waveform components to mechanical events rather than to electrocardiographic criteria, our findings demonstrate that the predominant RA descent is systolic and therefore an X descent, whereas the diastolic Y descent is blunted. The distinction between X and Y descents may be confounded in some patients by AV dyssynchrony, the presence of both X and X' descents when a C wave is present, or significant tricuspid regurgitation, further emphasizing the importance of timing waveform components to mechanical correlates.

The diastolic abnormality described should not be unexpected. Ischemia and infarction increase intrinsic RV myocardial stiffness, the infarcted ventricle dilates at end systole, and ischemia impairs ventricular recoil, relaxation, and early AV filling.15 Therefore, early in diastole there is increased resistance to filling of the infarcted right ventricle, with progressive impedance to inflow as the right ventricle fills and ascends a noncompliant diastolic pressure-volume curve. Acute RV dilatation results in elevated intrapericardial pressure,6,7 which further contributes to the pattern of diastolic dysfunction reflected in the right atrium as a blunted Y descent and in the RV wave forms as a sharp rise to a plateau. These diastolic abnormalities contribute to a marked reduction in RV preload reserve, which further compromises RV performance.

The abnormal diastolic orientation of the interventricular septum seen in our patients also reflects altered RV diastolic properties. In all patients, the septum was flattened or reversed curved from the dilated right ventricle toward the left ventricle, indicative of abnormal RV compliance.15,16 Because pressure or volume overload of one ventricle can alter the compliance and filling of the contralateral ventricle,15,17–19 the septum may mediate as well as reflect diastolic dysfunction. This occurs in experimental RV dysfunction where similar diastolic interaction alters LV compliance and contributes to low cardiac output.6–9,14

Role of Right Atrial Function in Severe Right Ventricular Infarction

RA function was an important determinant of RV performance and hemodynamic stability in patients with severe RV infarction. RA pressure and the amplitude of the RA A wave are related to the interaction of total blood volume, venous tone, atrial contractility, tricuspid valve integrity, and RV compliance.20–24 Although the mechanisms determining RA A wave amplitude were not specifically defined in this study, two distinct RA waveform patterns were identified that were differentiated by A wave amplitude and associated with disparate clinical courses.

In one half of the patients, the A wave was markedly augmented, resulting in a W-shaped morphology in the RA pressure waveform, whereas diminutive A waves imparted an M-shaped morphology in the other half. Increased RA contractility, which would lead to augmented A waves, would be the expected response to increases in RA preload and afterload imposed by the stiff dilated right ventricle. Judging from the markedly different hemodynamic and clinical courses of patients according to the presence or absence of augmented A waves, this response constitutes an important hemodynamic compensatory mechanism. Augmented A waves, present when the coronary obstruction leading to RV infarction spared the RA blood supply, resulted in a greater rise in RV end-diastole pressure and was associated with higher peak RV systolic pressures, better cardiac outputs, less severe hypotension, and a better therapeutic response to volume and inotropes. Furthermore, such patients less frequently required urgent revascularization for refractory shock. However, as might be expected from loss of this atrial contribution,25–28 patients with augmented A waves were more severely compromised by AV dyssynchrony.

Patients with diminutive A waves tended to have lower peak RV systolic pressures, more severe low cardiac output, hypotension necessitating higher dose and more prolonged inotropic support, and more frequent urgent revascularization for refractory shock. Under the increased loading conditions imposed by the infarcted right ventricle, the lack of augmented RA A waves appears physiologically inappropriate and was related to more proximal infarct-related lesions compromising RA perfusion. At thoracotomy in two such patients, gross RV dilatation and akinesia were evident, indicative of RA ischemia or infarction. These findings indicate that enhanced RA contraction, reflected in augmented A waves, improves RV filling and performance,
whereas ischemic compromise of RA contractility, manifest as depressed A waves, contributes to low cardiac output in severe RV infarction. These observations have not previously been reported.29

**Right Ventricular Systolic Dysfunction**

Our data also provide new insights regarding RV systolic dysfunction in patients with RV infarction. Severe RV free wall contractile dysfunction on ultrasound was associated with a depressed and often bifid peak RV systolic pressure, delayed RV upstroke and relaxation, and severely depressed RV stroke work. These changes are similar to those observed in the left ventricle after acute ischemia.15,26 Under conditions of normal RV function and in experimental animal preparations of RV free wall dysfunction, left ventricular septal contraction contributes to RV systolic performance through systolic ventricular interaction mediated by the septum and reflected in a bifid RV pressure trace14 and bifid RV (+)-dP/dt.30–34 Judging from these data, the bifid systolic RV wave form observed may reflect the supportive effects of ventricular interaction. Paradoxical systolic septal motion, known to correlate with severe low cardiac output in clinical RV infarction,2 was consistently present in our patients. In several cases, the septum bulged into the akinetic right ventricle in systole in a pistonlike fashion, similar to the septal motion patterns observed in experimental RV dysfunction.14,34 This septal behavior likely contributes to RV systolic pressure generation and performance through mechanical displacement and primary right-sided septal contraction.

**Conclusion**

The findings in this study elucidate the importance of RV diastolic dysfunction to hemodynamic compromise in patients with severe RV systolic dysfunction due to RV infarction. Impaired RV filling throughout diastole, reflected in the RA pressure waveform as a blunted Y descent, decreases RV preload reserve and renders the right ventricle more dependent on atrial transport. In addition, RV diastolic dysfunction alters LV compliance through diastolic ventricular interaction. Under these conditions, RV systolic pressure generation and performance are dependent, in large part, on LV septal contractile contributions. Enhanced RA function, manifest as augmented RA A waves, is associated with improved RV performance, which contributes to hemodynamic stability. The loss of this enhanced atrial transport function, due to AV dys synchrony or ischemic depression of RA function, contributes to severe low cardiac output in patients with RV infarction.

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**References**

8. Tani M: Roles of the right predominant right ventricular free wall and ventricular septum in right ventricular performance and influence of the parietal pericardium during right ventricular failure in dogs. *Am J Cardiol* 1983;52:196–202
19. Glantz SA, Misbach GA, Moores WY, Mathey DG, Lekven J, Stowe DF, Parmley WW, Tyberg JV: The pericardium sub-
stallion affects the left ventricular diastolic pressure-volume relationship in the dog. Circ Res 1978;42:433–441
32. Oboler AA, Keefe JF, Gaasch WH, Banas JS, Levine HJ: Influence of left ventricular isovolumic pressure upon right ventricular pressure transients. Cardiology 1973;58:32–44

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