Sensitivity and Specificity of Hemodynamic Criteria in the Diagnosis of Acute Right Ventricular Infarction

José Lopez-Sendon, M.D., Isabel Coma-Canella, M.D., and Carlos Gamallo, M.D.

SUMMARY To test the sensitivity and specificity of hemodynamic criteria for acute right ventricular infarction (RVI), two groups of patients with anatomically proved acute myocardial infarction and hemodynamic monitoring were studied. Group A included 22 patients with acute RVI and group B, 38 with infarction confined to the left ventricle. In both groups, the closest relation between right atrial and pulmonary capillary pressures (RAP and PCP), as well as the presence of a severe noncompliant pattern (SNCP), were studied. A SNCP was defined as a y descent deeper than the x descent in RAP. RAP was equal to or higher than PCP in 10 patients from group A and in none from group B. In group B, a significant relation was found between RAP and PCP (r = 0.777, y = 0.43x + 0.18) (p < 0.05), and the 95% confidence limits could be calculated. Above these limits, a closer relation between RAP and PCP was only found in patients with RVI. However, six patients with RVI showed an RAP/PCP relation within 95% confidence limits of group B (sensitivity 72.7%, specificity 100%). A SNCP was present in 12 patients with RVI and only in one without RVI (p < 0.01) (sensitivity 54.5% and specificity 97.4%). When either criterion is present (close relation between RAP and PCP or SNCP), a high sensitivity (81.8%) and specificity (97.4%) can be achieved in the diagnosis of acute RVI.

RIGHT VENTRICULAR INFARCTION often represents an interesting diagnostic and therapeutic challenge. Its diagnosis is based on clinical, hemodynamic, echocardiographic and radionuclide data.1-18 The anatomic diagnosis of right ventricular necrosis can only be made by demonstrating an anomalous myocardial uptake of technetium pyrophosphate,10, 12, 17, 18 but there is an incidence of false-positive interpretations20 and usually right ventricular infarction is first suspected at bedside when hemodynamic alterations suggesting primary right ventricular dysfunction are present.4-6, 8, 11-14, 18 This situation is termed ischemic right ventricular dysfunction.18

The finding of a right atrial pressure equal to or greater than the left ventricular filling pressure has been suggested as the characteristic hemodynamic profile of right ventricular infarction,9 and this diagnosis has been confirmed in some patients at necropsy.3, 5, 6, 8, 11, 14, 18 This strict criterion is probably very specific,21 but the amount of hemodynamic data from patients with right ventricular infarction confirmed by autopsy is too small to permit definitive conclusions about its sensitivity. Moreover, the hemodynamics in patients in whom the diagnosis of acute right ventricular infarction was established at necropsy have not been reported. Isner and Roberts21 performed such an investigation, but hemodynamic studies were performed in only four patients with biventricular infarction, two of whom had an old right ventricular infarction.

We studied patients with a postmortem anatomic diagnosis of acute myocardial infarction in whom hemodynamic monitoring had been performed. We analyzed the hemodynamic alterations that may help to increase accuracy in the diagnosis of right ventricular infarction.

Materials and Methods

Sixty-one patients with acute myocardial infarction confirmed at postmortem examination in whom hemodynamic monitoring had been performed were studied. All had been admitted to the coronary care unit between August 1977 and December 1979. Patients with valvular heart disease, cardiomyopathy, pericardial disease or cardiac surgery were excluded.

Two groups were defined, based on the presence of acute right ventricular necrosis found at the postmortem examination. Group A included 22 patients with acute right ventricular necrosis. Group B included 38 patients without acute right ventricular necrosis. The remaining patient, without acute right ventricular necrosis, had evidence of an old posterior right ventricular infarction and was therefore excluded from a statistical evaluation.

Anatomic Study

Transverse sections of the heart were incubated in triphenyltetrazolium chloride solution22 to label or identify the necrotic areas and were then examined microscopically to make the histologic diagnosis of acute myocardial infarction. The extent of right ventricular necrosis was classified according to the method of Isner and Roberts21: grade I = necrosis of less than 50% of the right ventricular posterior wall; grade II = necrosis limited to but involving more than 50% of the posterior wall; grade III = necrosis of the posterior wall and less than 50% of the anterolateral wall; and grade IV = necrosis of the right ventricular posterior wall and more than 50% of the anterolateral wall. We also added a grade V: involvement of a portion of the anterolateral wall without posterior wall necrosis. The lungs were also examined to investigate the presence of chronic obstructive pulmonary disease and pulmonary embolism.

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### Table 1. Anatomic and Hemodynamic Findings in Group A (n = 22)

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<th>Pt</th>
<th>Acute LV</th>
<th>Acute RV</th>
<th>Old LV</th>
<th>Lung disease</th>
<th>Low output</th>
<th>Initial</th>
<th>Interventions or clinical events</th>
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<td>8/16</td>
<td>Dextran + dopamine</td>
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<td>IVS</td>
<td>COPD + APE</td>
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<td>Dextran + nitroprusside</td>
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<td>—</td>
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<td>13/25</td>
<td>AVB</td>
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</table>

*Intermittent

Abbreviations: LV = left ventricle; RV = right ventricle; RAP = right atrial pressure; PCP = pulmonary capillary pressure; P = posterior; A = anterior; IVS = interventricular septum; COPD = chronic obstructive pulmonary disease; APE = acute pulmonary embolism; CPE = chronic pulmonary embolism; IVSR = interventricular septal rupture; AVB = atioventricular block; PH = pulmonary hypertension; I, II, III, IV, V = location of right ventricular infarction (see text); Timing = time of diagnostic finding.

### Hemodynamic Study

Right-heart hemodynamic monitoring was performed in all patients for 1–10 days. At admission, a Swan-Ganz thermodilution catheter was inserted into the pulmonary trunk through a right antecubital vein. Right atrial (RAP), pulmonary trunk (PTP) and pulmonary capillary (PCP) pressures were recorded with a Hewlett Packard 1280 transducer on a Siemens-Elema Mingograph 34. The RAP wave form was carefully analyzed. The term “severe noncompliant pattern” was used when RAP showed an M or W wave form with the y descent deeper than the x, and “mild noncompliant pattern” when the y descent was equal to the x. Cardiac output was determined by thermodilution on an Edwards 9500 computer. In every case, at least three successive determinations were made, and the average value was calculated and standardized for body surface area to cardiac index.

A severe low-output syndrome was defined as a cardiac index lower than 2.2 l/min/m² with evidence of reduced tissue perfusion, such as mental impairment and oliguria or anuria. A complete hemodynamic evaluation was made at least every 8 hours. One to 40 hemodynamic studies were performed per patient (mean 13 ± 9).

### Interventions

Volume loading with Dextran-40 was performed in every patient with a basal PCP lower than 18 mm Hg. Other interventions included i.v. administration


### Table 1. (Continued)

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<thead>
<tr>
<th>RAP and PCP</th>
<th>Indicative of RVI timing</th>
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<td>Initial</td>
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<td>—</td>
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</tr>
<tr>
<td>15/16</td>
<td>—</td>
<td>1st day*</td>
</tr>
</tbody>
</table>

of diuretics or inotropic or vasoactive drugs.

Special attention was given to the initial relationship between RAP and PCP levels and to the presence of a noncompliant pattern in the RAP wave form, as well as to the relation between these measurements after therapeutic interventions or clinical events.

### Statistical Analysis

All statistical analyses were performed considering the initial determinations and those which showed the closest relation (minimal difference) between RAP and PCP levels. Fisher's exact test was applied to evaluate the difference in incidence of hemodynamic and anatomic data in each group. A gaussian distribution of RAP and PCP was demonstrated in both groups by the Kolmogorov-Smirnov test. The Pearson's correlation coefficient between RAP and PCP was calculated as well as the regression equation, its \( S_{yx} \) (standard error of the estimate) and the 95% confidence limits for each set of data.

### Results

#### Anatomic Findings

The locations of acute or healed myocardial infarction and pulmonary lesions for each patient are listed in tables 1 and 2. In group A, both right and left ventricular free walls and the interventricular septum were involved in all patients. The incidence of acute left posterior myocardial infarction was higher in group A (19 patients, 86.4%) than in group B (13 patients, 34.2%) \( (p < 0.01) \). The incidence of left anterior myocardial infarction was lower in group A (nine patients, 40.9%) than in group B (31 patients, 81.6%) (NS). Acute interventricular septal necrosis was found in all group A patients, and in 31 (81.6%) of group B patients (NS). The incidence of chronic obstructive pulmonary disease or pulmonary thromboembolism, both well-known causes of right ventricular dysfunction, was similar in group A (eight patients, 36.4%) and group B (13 patients, 34.2%) (NS).
### Table 2. Anatomic and Hemodynamic Findings in Group B (n = 38)

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<th>Pt</th>
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<tr>
<td>35</td>
<td>A + P + IVS</td>
<td>—</td>
<td>—</td>
<td>Yes</td>
<td>15/31 Nitroprusside</td>
<td>12/21 —</td>
</tr>
<tr>
<td>36</td>
<td>P + IVS</td>
<td>—</td>
<td>—</td>
<td>Yes</td>
<td>11/18 —</td>
<td>11/18 —</td>
</tr>
<tr>
<td>37</td>
<td>A</td>
<td>P</td>
<td>—</td>
<td>Yes</td>
<td>14/33 Nitroprusside</td>
<td>9/23 —</td>
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<tr>
<td>38</td>
<td>P + IVS</td>
<td>—</td>
<td>—</td>
<td>Yes</td>
<td>9/19 —</td>
<td>9/19 —</td>
</tr>
</tbody>
</table>

**Abbreviations:** See table 1.

### Hemodynamic Data

The incidence of severe low-output syndrome was very high in both group A (19 patients, 86.4%) and group B (27 patients, 71.1%), with no statistical difference between the groups.

### Relationship of RAP and PCP

Initial RAP and PCP values and measurement after interventions or relevant clinical events are shown in tables 1 and 2. Initial determinations of RAP and PCP values at admission and before treatment are
represented in figure 1. A more significant correlation could be demonstrated between these values in group B ($r = 0.824, p < 0.01$) than in group A ($r = 0.421, p < 0.05$), but no difference could be demonstrated between groups because of the wide dispersion in group A.

The minimum difference (closest relation) between RAP and PCP values recorded during the hemodynamic profile evolution of each patient is shown in figure 2A. Usually, RAP was closer to PCP in both groups when left ventricular failure was not present or when PCP was reduced after therapeutic interventions such as administration of i.v. nitroprusside or furosemide.

In group A, the RAP was equal to or higher than the PCP at some point in 10 patients. In six this occurred after dextran or dextran plus dopamine infusion, in one after nitroprusside administration, in two after dextran plus nitroprusside and in the other throughout the entire duration of the hemodynamic monitoring. In seven patients, RAP values were 1–5 mm Hg lower than PCP values. This relation was observed after relief of pulmonary congestion with nitroprusside or furosemide in three patients, coinciding with atrioventricular block in another three patients and after dextran administration in one. In five of these patients the RAP was greater than 10 mm Hg. In the remaining five patients from group A, the difference between RAP and PCP was greater than 5 mm Hg throughout hemodynamic monitoring.

In group B, RAP was usually lower than PCP. The exceptions were four patients with left ventricular rupture in whom RAP approached or equaled PCP values after cardiac tamponade. In these patients, the closest relation between RAP and PCP recorded before cardiac tamponade was considered for the statistical analysis. In only two patients without cardiac tamponade were RAP values found to reach 1–5 mm Hg below PCP values. RAP levels in both were less than 10 mm Hg and the difference between RAP and PCP

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Relation between right atrial pressure (RAP) and pulmonary capillary pressure (PCP) at admission and before treatment. AMI = acute myocardial infarction; RVI = right ventricular infarction.

![Figure 2](http://circ.ahajournals.org/)

**Figure 2.** (A) Closest relation between right atrial pressure (RAP) and pulmonary capillary pressure (PCP) in groups A and B. AMI = acute myocardial infarction; RVI = right ventricular infarction. (B) Linear regression equation and 95% (± 1.96 SyX) confidence interval.
increased when RAP exceeded 10 mm Hg.

When the hemodynamic measurements showing the closest relation between both pressures was considered, a significant correlation was found between RAP and PCP in group A ($r = 0.683, p < 0.05$) and in group B ($r = 0.777, p < 0.01$). The linear regression equations for groups A and B were calculated (group A: $y = 0.861x - 0.681$, and group B: $y = 0.432x + 0.178$), and are represented in figure 2B with the 95% confidence limits ($\pm 1.96 S_{xy}$). A closer relation between RAP and PCP than the upper confidence limit of group B would be indicative of right ventricular dysfunction not secondary to left ventricular failure, and could be expected to appear only in 2.5% of the patients without right ventricular infarction. The wide dispersion of group A values ($r = 0.683$) explains why six patients from this group showed a minimal difference between RAP and PCP values within the limits calculated for group B. Moreover, at admission and before therapeutic interventions, only six patients from group A exceeded these limits.

Noncompliant Pattern

The presence of a noncompliant pattern, its type and timing are indicated in tables 1 and 2. Twelve patients in group A (54.5%) had a severe noncompliant pattern. In five, the pattern was present throughout the duration of hemodynamic monitoring. In seven it was present only in some RAP tracings: in three at low heart rates, in one after volume overloading and in the other three with an aggravation of left ventricular failure. Three other group A patients had a mild noncompliant pattern. In one it was only present with low heart rates and in the other two after dextran administration. No form of noncompliant pattern could be demonstrated at any time in the seven (31.8%) remaining patients from group A. In group B, only one patient had the severe form of noncompliant pattern, coinciding with severe left ventricular failure and pulmonary hypertension. This patient had a massive left ventricular infarction without right ventricular involvement and an aneurysm of the interventricular septum that compressed the right ventricular cavity, greatly reducing it. The patient with an old right ventricular infarction also showed a severe form of noncompliant pattern coinciding with severe pulmonary hypertension. In two group B patients, the RAP wave form showed a sporadic, mild noncompliant pattern, in one of them only at low heart rates (50 beats/min). All the patients showing a noncompliant pattern except for the one with the aneurysm of the interventricular septum presented severe obstructive lesions (> 75%) in a dominant right coronary artery. The difference between the occurrence of a noncompliant pattern in the two groups was significant ($p > 0.01$).

The sensitivity and specificity of the different hemodynamic criteria in the diagnosis of right ventricular infarction is shown in table 3. When the criterion of a close relation between RAP and PCP is used the sensitivity is 72.7% and the specificity 100%. A severe noncompliant pattern has a sensitivity of 54.5% for a specificity of 97.4%. When either of these two criteria is present, a high sensitivity (81.8%) and specificity (97.4%) can be achieved.

Hemodynamic Patterns

Several hemodynamic patterns could be clearly differentiated in group A patients, considering the difference between systolic and diastolic pressures in the pulmonary artery, RAP/PCP relation and presence of the noncompliant pattern (table 4):

In three cases, the difference between systolic and diastolic pressure in the pulmonary artery was reduced (< 10 mm Hg), RAP was higher than PCP, and a severe noncompliant pattern was present (fig. 3A).

In seven cases, the pulmonary artery pressure difference was normal and RAP was equal to or higher than PCP (fig. 3B). In three patients the noncompliant pattern was severe and in three it was mild.

In six patients, the pulmonary artery pressure difference was normal and RAP was lower than PCP, but the relation between these measurements indicated right ventricular infarction, exceeding the limits found in patients without right ventricular involvement (fig. 3C). Four of the patients had a severe noncompliant pattern.

In two patients, the pulmonary artery pressure

<table>
<thead>
<tr>
<th>Hemodynamics</th>
<th>Sensitivity</th>
<th></th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Value</td>
<td>95% confidence limits</td>
<td>Value</td>
</tr>
<tr>
<td>RAP = PCP according to fig. 2B limits</td>
<td>72.7%</td>
<td>49.8–89.3%</td>
<td>100%</td>
</tr>
<tr>
<td>Severe NCP</td>
<td>54.5%</td>
<td>32.2–75.6%</td>
<td>97.4%</td>
</tr>
<tr>
<td>RAP = PCP according to fig. 2B limits, or severe NCP</td>
<td>81.8%</td>
<td>59.7–94.8%</td>
<td>97.4%</td>
</tr>
<tr>
<td>RAP $\geq$ PCP$^*$</td>
<td>45.5%</td>
<td>24.4–67.8%</td>
<td>100%</td>
</tr>
<tr>
<td>RAP $&gt; 10$ mm Hg and PCP $&lt; 15$ mm Hg</td>
<td>72.7%</td>
<td>49.8–89.3%</td>
<td>100%</td>
</tr>
</tbody>
</table>

*Criteria proposed in the literature.

Abbreviations: RAP = right atrial pressure; PCP = pulmonary capillary pressure; NCP = noncompliant pattern.
Table 4. Hemodynamic Patterns in Right Ventricular Infarction

<table>
<thead>
<tr>
<th>Hemodynamic pattern</th>
<th>PTP systolic-diastolic difference</th>
<th>RAP/PCP</th>
<th>NCP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Reduced (&lt;10 mm Hg)</td>
<td>RAP &gt; PCP</td>
<td>Present</td>
</tr>
<tr>
<td>2</td>
<td>Normal</td>
<td>RAP ≥ PCP</td>
<td>Present or absent</td>
</tr>
<tr>
<td>3</td>
<td>Normal</td>
<td>RAP &lt; PCP indicative of RVI</td>
<td>Present or absent</td>
</tr>
<tr>
<td>4</td>
<td>Normal</td>
<td>RAP &lt; PCP not indicative of RVI</td>
<td>Present</td>
</tr>
<tr>
<td>5</td>
<td>Normal</td>
<td>RAP &lt; PCP not indicative of RVI</td>
<td>Absent</td>
</tr>
</tbody>
</table>

Abbreviations: PTP = pulmonary trunk pressure; RAP = right atrial pressure; PCP = pulmonary capillary pressure; NCP = noncompliant pattern; RVI = right ventricular infarction.

difference was normal, RAP values were lower than PCP, and the relationship of these variables did not suggest right ventricular infarction. Both had a severe noncompliant pattern (fig. 3D).

In four cases, the pulmonary artery pressure difference was normal, the relation between RAP and PCP did not suggest right ventricular infarction and no form of noncompliant pattern was found during hemodynamic monitoring.

Relationship of Anatomic Involvement and Hemodynamics

No relation could be established between either the presence of noncompliant pattern or a close RAP/PCP relation at admission, and chronic obstructive pulmonary disease or pulmonary embolism (tables 1 and 2). At admission, seven of the eight group A patients with either chronic obstructive pulmonary disease or pulmonary embolism failed to present a RAP/PCP relation suggestive of primary right ventricular dysfunction, and only two showed any form of noncompliant pattern. Furthermore, group B patients with chronic obstructive pulmonary disease or pulmonary embolism showed a striking difference between RAP and PCP values, and no form of noncompliant pattern could be demonstrated in any of them.

The location and extension of the right ventricular necrosis are shown in table 1. Patients presenting the most severe hemodynamic alterations, such as severe low-output syndrome, RAP equal to or higher than PCP and severe noncompliant pattern, presented a highly variable degree of right ventricular involvement. However, patients without a severe noncompliant pattern and in whom the relation between RAP and PCP did not suggest primary right ventricular dysfunction presented with a grade I or grade V right ventricular infarction.

Discussion

Until recently, right ventricular infarction has been considered a clinical curiosity. The works of Starr et al. and others seemed to demonstrate that the right ventricle is not necessary for maintaining the normal dynamics of both circulations. However, some degree of right ventricular dysfunction, varying from contractility index alterations to severe hypotension and shock, has been found after right ventricular necrosis.

Right ventricular failure can be secondary to left ventricular dysfunction with pulmonary hypertension, but in the presence of acute myocardial infarction, when right ventricular dysfunction can not be attributed to left ventricular failure, right ventricular infarction can be strongly suspected.

Hemodynamics in Right Ventricular Infarction

In most clinical reports, right ventricular infarction is frequently accompanied by a low-output syndrome or shock. The incidence of severe low-output syndrome in the present study was very high in both groups, and the difference between them was not significant. Some patients with confirmed right ventricular involvement do maintain a normal cardiac output, so the presence or absence of low output is of little value in the diagnosis of right ventricular infarction.

Relationship of RAP to PCP

Some investigators postulate that a disproportionate increase of RAP in relation to PCP is suggestive of right ventricular infarction. However, the limits of this disproportion have either not been established or were proposed arbitrarily. On this basis, most authors estimate that the RAP should be equal to or higher than the PCP. This criterion has turned out to be highly specific. No false positives were recorded in our group of patients, but its sensitivity was low, including only 45% of the cases with anatomically confirmed acute right ventricular infarction (table 4). This rigid criterion can be extended if one takes into consideration the limits RAP can reach secondary to left ventricular dysfunction.

In our study, right ventricular dynamics were more consistently abnormal in patients with biventricular infarction. When infarction involved only the left ventricle, the alterations in RAP were more dependent on the degree of left ventricular dysfunction. A substantially better correlation between RAP and PCP was found in these group B patients ($r = 0.777$) ($p < 0.01$) and the confidence limits for a 95% sensitivity could be calculated (fig. 2B). Above these limits, a closer relation between RAP and PCP can be expected to appear.
only in 2.5% of the patients without right ventricular infarction. For practical purposes, RAP must be greater than PCP when PCP is below 10 mm Hg, can be 4 mm Hg below PCP when PCP reaches 15 mm Hg, may be 7 mm Hg lower than PCP when PCP exceeds 20 mm Hg, and can be as much as 14 mm Hg lower than PCP when PCP exceeds 30 mm Hg. Using these criteria, the sensitivity of diagnosis in right ventricular infarction reaches 73% and the specificity remains 100% (table 3).

In a previous report,18 we stated that for the diagnosis of right ventricular dysfunction, factors to be considered include an RAP above 10 mm Hg and either greater than, equal to or 1–5 mm Hg lower than PCP. This criterion has been proved to be accurate for the diagnosis of acute right ventricular infarction, and no false positives have been observed in this series (table 3).
Alterations in Right Ventricular Compliance

The presence of a noncompliant pattern in the right heart with a pressure wave form in right-heart cavities similar to that observed in constrictive pericarditis and restrictive myocardiopathies has already been described in right ventricular infarction, and probably is representative of the limited expansion of the damaged myocardium. In the present study, the sensitivity of the severe form of noncompliant pattern was found to be 54.5%. In the course of acute myocardial infarction, this severe form of noncompliant pattern is a highly specific sign of right ventricular infarction (table 4) and may be the only hemodynamic sign leading to suspicion of right ventricular involvement when left ventricular dysfunction predominates, in which case a close relation between RAP and PCP would be no longer evident.

When either of the two criteria is present, a close relation between RAP and PCP or a severe noncompliant pattern, sensitivity increases to 81.8% and specificity remains close to 100% (table 3). Some cases are nevertheless overlooked because all the proposed criteria refer to ischemic right ventricular dysfunction, a condition that may be absent in some patients with right ventricular infarction. One must consider that the patients we studied were by definition (autopsy) particularly ill; therefore, more surviving patients would present a less significant degree of right ventricular dysfunction, and in these same patients the sensitivity of the proposed criteria would be lower.

Effect of Stress

Ischemic right ventricular dysfunction can sometimes only be manifest under cardiac interventions such as epinephrine administration, volume loading, or rapid atrial pacing. According to the diagnostic criteria herein proposed, only six (27.3%) group A patients showed an RAP/PCP relationship indicative of right ventricular infarction at the first hemodynamic evaluation. Three stress conditions have proved to be of great value in demonstrating right ventricular dysfunction clearly not secondary to left ventricular failure (table 1).

Volume Loading

In six group A patients, a close relation between RAP and PCP indicative of right ventricular infarction was observed only after dextran administration. In three patients, a noncompliant pattern was also observed in these same conditions. Volume loading is especially interesting in patients with low RAP and PCP values, because in this situation in both groups of patients the relationship between these two measurements is similar (fig. 2B).

Left Ventricular Failure

In two group A patients with left ventricular dysfunction and passive pulmonary hypertension, the severe form of noncompliant pattern was observed only with aggravation of left ventricular failure. Only one similar case in the absence of right ventricular involvement was found in group B. On the other hand, when left ventricular failure was present, a closer relation between RAP and PCP was always observed in both groups after the administration of i.v. vasodilators or diuretics with the consequent reduction of PCP values. That is, while producing a severe noncompliant pattern, a close relation between RAP and PCP is prevented by the development of pulmonary capillary hypertension.

Bradycardia

Bradycardia is an interesting spontaneous stress situation in right ventricular infarction. Probably, low heart rate acutely aggravates right ventricular ischemia in patients with a critical obstructive lesion of the right coronary artery, producing an acute reduction in right ventricular contractility and compliance. Bradycardia is poorly tolerated in patients with right ventricular infarction and decreased compliance, and a significant decrease in cardiac output may be observed, perhaps as the result of an ineffective Frank-Starling mechanism. A close relation between RAP and PCP or a severe noncompliant pattern was observed only during low heart rates in three patients from group A. In group B, bradycardia was followed by neither of these two hemodynamic patterns, except in one patient presenting a severe right coronary artery stenosis, in whom a mild form of noncompliant pattern appeared at low heart rates (50 beats/min).

Hemodynamic Patterns

In this study, a wide hemodynamic profile was observed, and several hemodynamic patterns could be distinguished in biventricular infarction. The presence of one or the other is probably related to the relative degree of right and left ventricular dysfunction in each patient (table 4).

In the most severe form of dysfunction, the right ventricle practically does not generate pressure, as indicated by a reduced difference between the systolic

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**Figure 3.** Hemodynamic patterns in acute right ventricular infarction. (A) Type 1. Pulmonary trunk pressure (PTP) is reduced, right atrial pressure (RAP) is higher than pulmonary capillary pressure (PCP) and severe noncompliant pattern is present. (B) Type 2. The PTP difference is normal and RAP exceeds PCP. In this patient, a severe noncompliant pattern is present. (C) Type 3. PTP difference is normal, and RAP is slightly lower than PCP. In this patient, the noncompliant pattern is absent. (D) Type 4. PCP is higher than RAP and a severe noncompliant pattern is the only finding to suspect right ventricular infarction.
and diastolic pulmonary pressures. RAP is higher than PCP and closely resembles pulmonary trunk pressure. In this situation, low-output syndrome and severe noncompliant pattern are invariably present, and the RAP is higher than PCP (fig. 3A).

In the more common and perhaps characteristic pattern, the right ventricle generates pressure with a normal difference between systolic and diastolic pulmonary pressures, and RAP equals or exceeds PCP (fig. 3B). In these patients, low-output syndrome and noncompliant pattern are very common.

Some patients show a RAP slightly below the PCP for which many authors have chosen to exclude the diagnosis of right ventricular infarction (fig. 3C). However, the relation between these two measurements is suggestive of right ventricular infarction according to the limits presented in this study.

In some patients, the diagnosis of right ventricular infarction is only supported by the presence of a noncompliant pattern (fig. 3D). In these patients, the close relation between RAP and PCP characteristic of ischemic right ventricular dysfunction is lost, probably because the dysfunction of the left ventricle is more severe than that of the right ventricle.

Another pattern occurs, particularly when right ventricular necrosis is small (grade I) or involves only the anterior wall of the right ventricle (grade V). In this situation, primary right ventricular dysfunction is absent, and the hemodynamic diagnosis of right ventricular infarction is impossible. This situation is probably more frequent in survivors and the diagnosis of right ventricular involvement may be of little practical importance.

Relation Between Anatomic Involvement and Hemodynamics

No definite conclusions can be drawn as to the relation between the extent of right ventricular necrosis and the functional impairment, because a precise quantification of right ventricular necrosis was not made; the classification proposed by Isner and Roberts is only semiquantitative and does not take into account the necrosis extent from apex to base. We believe that severe right ventricular dysfunction may coexist with variable degrees of right ventricular involvement. Conversely, all patients with normal right ventricular function had a small right ventricular infarction. Right ventricular ischemia may have contributed to the functional impairment in patients with grade I right ventricular infarction, and the involvement of the anterior wall of the right ventricle has little significance in right ventricular dynamics.

Differential Diagnosis

The specificity for biventricular infarction using any of the aforementioned criteria found in the literature is very high (table 3). However, the diagnosis is based upon predominant right ventricular dysfunction, which is not pathognomonic for this condition, and must only be reached once other causes of right ventricular failure, such as pulmonary embolism, chronic obstructive pulmonary disease, interventricular septal rupture or cardiac tamponade, have been ruled out.

Chronic obstructive pulmonary disease or pulmonary embolism can produce right ventricular dysfunction, so some authors have excluded patients with this condition in the diagnosis of right ventricular infarction. However, for RAP to increase, severe pulmonary hypertension must be present, a situation in which a gradient between diastolic pulmonary pressure and PCP would be noticeable.

A severe noncompliant pattern, on the other hand, has been described in myocarditis, constrictive pericarditis, after right ventriculotomy, in cases of severe tricuspid regurgitation, and in valvular heart disease with atrial fibrillation. It is always absent in cardiac tamponade.

In the present study, a severe intermittent noncompliant pattern was found in a patient without right ventricular involvement. In this patient, who presented with severe pulmonary hypertension secondary to left ventricular failure, an aneurysm of the interventricular septum that almost completely obliterated the right ventricular cavity was found at necropsy. It is reasonable to suppose that during maximal left ventricular filling pressure elevation, the right ventricular cavity became nearly occluded, leading to a situation of severe impairment of right ventricular compliance. To our knowledge, no similar case has been reported, but such a cause of noncompliant pattern must be taken into account when considering the differential diagnosis of right ventricular infarction in the presence of severe left ventricular failure.

Acknowledgment

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