

# Right Ventricular Infarction

## KEY POINTS

- ▶ Right ventricular infarction is rarely isolated; it is usually part of an inferior infarction.
- ▶ The syndrome of right ventricular infarction is that of low output and hypotension.
- ▶ Right ventricular infarction is responsible for some cardiogenic shock cases and a larger proportion of hypotensive inferior infarction cases.
- ▶ Bedside physical diagnosis and electrocardiographic findings are strongly suggestive in most cases.
- ▶ Right ventricular wall motion abnormalities correlate with the site of occlusion and the coronary anatomy.
- ▶ Particular pathophysiologic issues:
  - ▶ Prominent sensitivity of the ischemic right ventricle to preload
  - ▶ Ventricular interdependence may develop or be provoked.
- ▶ Particular management strategies depend on the individual patient's status and response:
  - ▶ Optimal range of volume status
  - ▶ Inotropic support
  - ▶ Reperfusion

The recognition of right ventricular infarction is relatively recent and dates to the 1930s—a patient with the typical symptoms of inferior territory infarction (pale, diaphoretic, ashen, bradycardic, hypotensive) and cardiogenic shock with elevated jugular venous pressure and no dyspnea (shock from right-sided heart failure).

The setting of right ventricular infarction is indeed that of acute inferior infarction in the great majority of cases, although uncommonly (<3%), right ventricular infarction may be isolated (i.e., without recognized inferior involvement of the left ventricle).<sup>1</sup> The probability of right ventricular infarction rises in proportion to the size of the inferior infarction and the degree of hemodynamic compromise. The described incidence of right ventricular infarction has ranged widely (14% to 84%) according to the diagnostic technique used and the patient's profile. Right ventricular infarction is recognizable clinically in one seventh of hemodynamically stable inferior infarctions, two thirds of hypotensive inferior infarctions, most inferior infarctions with cardiogenic shock, and one tenth of anterior infarction cases. At autopsy, two thirds of inferior infarctions have pathologic evidence of associated right ventricular infarction.

Right ventricular infarction is associated with all lesions and other complications of right coronary artery occlusion, including bradyarrhythmias and atrioventricular (AV) block; all forms of myocardial rupture, including right ventricular rupture and right ventricular papillary muscle rupture; true and false aneurysms; and right ventricular thrombi. The occurrence of

right ventricular infarction has a major influence on the perioperative course of patients with ventricular septal defects and is a prominent determinant of hemodynamics and of mortality.

Right ventricular infarction is prominently associated with a higher incidence of clinical events and risk, and it is an independent predictor of prognosis after acute inferior myocardial infarction. A series of right ventricular infarctions documented bradyarrhythmias in 40%, ventricular tachycardia (>15 beats) and ventricular fibrillation in 30%, and hypotension in 43% (12 of 23 responded to volume and bradycardia treatment, 11 of 23 required inotropes).<sup>2,3</sup> From a meta-analysis of six studies of right ventricular infarction involving 1200 patients, the odds ratio of death was 3.2; of cardiogenic shock, 3.2; of atrioventricular block, 3.4; and of ventricular tachycardia and fibrillation, 2.7.<sup>4</sup> The SHOCK registry established that the mortality of cardiogenic shock due to right ventricular infarction (55%) differed little from that of cardiogenic shock due to left ventricular infarction (59%), despite lesser incidence of anterior infarction, younger age, and greater incidence of single-vessel disease.<sup>5,6</sup> The mere sign of ST elevation in  $V_4R$  establishes a 30% in-hospital mortality.

Relevant coronary anatomy is illustrated in Figure 6-1 and Table 6-1. The coronary anatomy usually responsible for right ventricular infarction is an occlusion of a proximal dominant right coronary artery before acute marginal branches (Fig. 6-2). The relative risk of right ventricular infarction with occlusion of a proximal dominant right coronary artery before acute marginal



**Figure 6-1.** Coronary anatomy and right ventricular perfusion. UPPER IMAGES, The posterior descending (interventricular) coronary artery and its blood supply to the lower third of the interventricular septum and the medial posterior right and left ventricles. MIDDLE IMAGES, Right coronary perfusion of the right ventricle through acute marginal branches and the posterior descending (interventricular) artery. The acute marginal branches supply the free wall of the right ventricle. Hence, proximal right coronary occlusion (before the acute marginal branches) results in extensive right ventricular dysfunction, segmentally involving the free wall (lateral) and posterior walls of the right ventricle as well as the inferior septum. LOWER IMAGES, The LAD delivers, through diagonal branches to either side of the anterior interventricular groove, perfusion to the medial anterior right and left ventricles. LAD occlusion will therefore result in failure of the anterior right ventricle and the anterior two thirds of the interventricular septum. Septal function contributes significantly to right ventricular function.

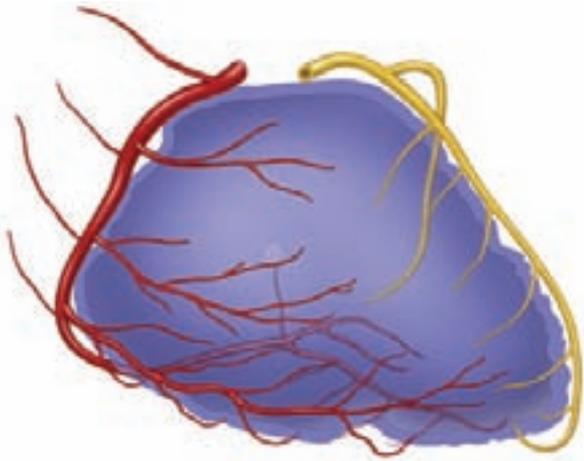
**Table 6-1.** Branches of the Right Coronary Artery

Right Coronary Branches	Supplies
Artery to the sinoatrial (SA) node	SA node in 60%
Conus branch	RVOT, may collateralize to LAD
Atrial branches	RA
Acute marginal branches	RV lateral wall
Posterior descending (interventricular) artery	
Septal perforators	Inferobasal septum
Diagonal branches to the LV	Posteromedial LV, posteromedial papillary muscle
Diagonal branches to the RV	Posteromedial RV
Distal (ongoing) right coronary artery	
Artery to the atrioventricular (AV) node	AV node in 90%
Posteroventricular branches	Posterior LV
Posterolateral branches	Posterolateral LV

LAD, left anterior descending coronary artery; LV, left ventricle; RA, right atrium; RV, right ventricle; RVOT, right ventricular outflow tract.

branches is 3.0 [1.2-8.1] ( $P = .02$ ), although *not all proximal right coronary artery occlusions result in right ventricular infarction.*<sup>7</sup> Uncommonly, an occlusion of a dominant left circumflex artery, of an isolated acute marginal branch alone, of a nondominant right coronary artery, or of the left anterior descending (LAD) coronary artery results in a recognized right ventricular infarction (Fig. 6-3). An angiographically significant LAD coronary lesion (75% have more than 75% stenosis) is common in large, clinically severe right ventricular infarctions due to occlusion of a dominant right coronary artery.<sup>8,9</sup> A proximal dominant right coronary occlusion interrupts blood flow through the acute marginal branches to the lateral and inferior walls of the right ventricle—resulting in right ventricular dysfunction of a sufficient extent to be recognized in most cases. Interestingly, only half of proximal right coronary occlusions produce right ventricular infarction, suggesting resistance of the (nonhypertrophied) right ventricle to ischemia and potentially of collateralization from the left coronary artery in an important proportion of cases. The relevance of right ventricular thebesian veins to right ventricular oxygen supply is controversial.<sup>1</sup>

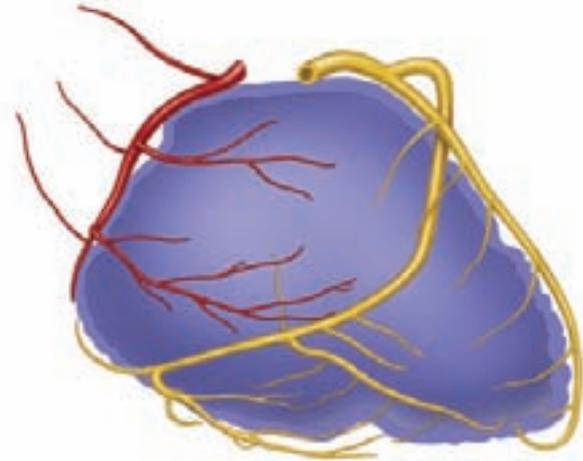
Although the most prominent right ventricular infarctions are the consequence of right coronary artery occlusions, 10% of LAD-generated infarctions have pathologically and clinically apparent right ventricular infarction.<sup>10</sup>



**Figure 6-2.** Blood supply to the right ventricle. The wall of the right ventricle is depicted as a pale blue rim and the cavity as a darker space. The normal vascular anatomy that supplies the right ventricle is composed of the conus branch that perfuses the RVOT region and that may evolve to collateralize to the LAD; acute marginal branches, of which there are usually two or three angiographically apparent branches that perfuse the lateral-free wall of the right ventricle; small diagonal branches that arise from the LAD anteriorly to supply the medial anterior wall of the right ventricle and often its apex; and small diagonal branches that arise from the posterior descending coronary artery that supply the medial posterior aspect of the right ventricle. The inferior septum, which contributes to right ventricular systolic and diastolic function, is supplied by inferior septal branches of the posterior descending coronary artery (not shown) as the anterior superior septum is similarly supplied by septal perforator branches by the LAD, which also contribute to right ventricular function or dysfunction. Atrial branches also arise from the right coronary artery, and the artery to the sinoatrial node is usually the first branch of the right coronary artery. The artery to the atrioventricular node arises in most patients about 1 cm distal to the “crux” of the right coronary–posterior descending artery. The posteromedial papillary muscle is perfused by branches of the right coronary from the posterior descending coronary artery or posteroventricular branches off the ongoing distal right coronary artery. The right coronary artery supplies a large number of structures within the heart, and the permutations of cardiac dysfunction are greater in right coronary infarction than in infarction caused by either other coronary artery.

## DIFFERENCES OF RIGHT VENTRICULAR INFARCTION AND LEFT VENTRICULAR INFARCTION

- Right-sided heart failure versus left-sided heart failure
- Prominent need for adequate right ventricular preload
- Pericardium-mediated ventricular interdependence is an active phenomenon in right ventricular infarction because the right ventricle is more prone to rapid dilation.
- Recoverability of the right ventricle is greater and faster than that of the left ventricle.
- Preinfarction angina protects against right ventricular infarction.
- Right atrial pressure mechanical function is important and may be lost in atrial fibrillation, heart block, and atrial infarction.
- Right ventricular hypertrophy increases right ventricular ischemia.



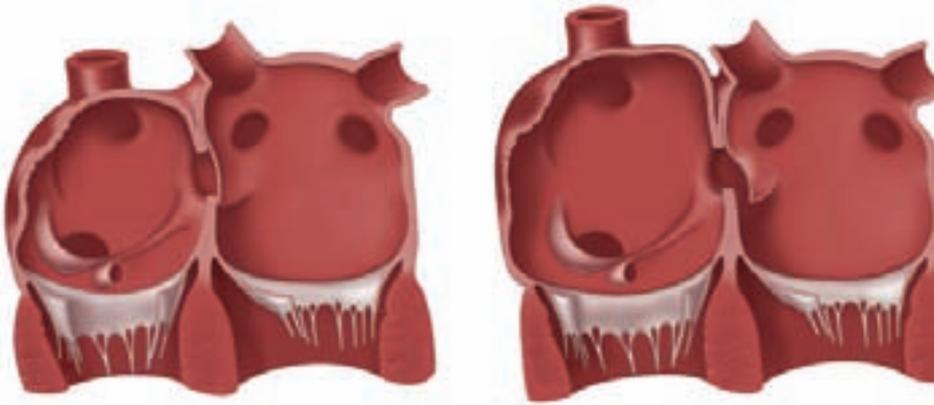
**Figure 6-3.** In  $\leq 10\%$  of patients, the left circumflex coronary artery supplies perfusion to a larger extent of the right ventricle than usual, through posterior diagonal branches off the posterior descending coronary artery and from acute marginal branches of an ongoing circumflex after the crux.

## COMPLICATIONS OF RIGHT VENTRICULAR INFARCTION

- Low output
- Shock (usually with little pulmonary congestion)
- Right ventricular papillary muscle rupture ( $1/20$  of papillary muscle ruptures)
- Right ventricular free wall rupture ( $1/7$  of myocardial ruptures)
- Patency of a foramen ovale with right-to-left shunting (hypoxemia unresponsive to supplemental oxygen) (Fig. 6-4)
- Pulmonary embolism of a right ventricular apical thrombus
- Pacer perforations
- Pericarditis

## THE NORMAL RIGHT VENTRICLE

The stroke volume of the right ventricle is the same as that of the left ventricle, but the right ventricle pumps against a lesser arterial pressure (one fifth of systemic) and resistance (one tenth of systemic); hence, the right ventricle in its usual state is a less forceful pump, having one sixth of the muscle mass, having one third of the wall thickness, and performing one fourth of the stroke work of the systemic ventricle. The right ventricle receives systemic venous blood at lower diastolic filling pressures than does the left ventricle and also with greater respiratory fluctuations in filling pressure. The normal right ventricle functions despite variations in preload. In contradistinction, the ischemic right ventricle is highly sensitive to •• despite variations in preload. The pattern of contraction of the right ventricle differs from that of the left ventricle, which contracts radially and longitudinally. The right ventricular free wall contracts toward the interventricular septum, with also some longitudinal shortening. The interventricular septum normally pushes into the right ventricle, contributing to right ventricular function.



**Figure 6-4.** LEFT IMAGE, Normal atrial size and anatomy. In about one quarter of individuals, there is patency of the foramen ovale. RIGHT IMAGE, Enlargement of the right side of the heart as a consequence of right ventricular infarction. The right atrium is dilated, and a foramen ovale is both stretched open by the chamber dilation and pushed open by a right atrial to left atrial gradient due to the right-sided heart failure and general absence of equally prominent left-sided heart failure in the context of right ventricular infarction.

## THE INFARCTED RIGHT VENTRICLE

Smaller infarctions of the right ventricle, such as those due to LAD occlusion, result in little hemodynamic disturbance. Infarctions of the right ventricular lateral wall and posterior wall, as occur with an occlusion of the right ventricle proximal to most of the acute marginal branches, would usually result in disturbed hemodynamics. The ischemic right ventricle is prominently dependent on preload as a means to maintain filling of the chamber that has less compliant walls from the ischemia. To achieve adequate filling, right atrial contraction augments to adapt to right ventricular ischemic noncompliance. This is apparent by an increase in the right atrial A-wave magnitude and  $x$  descent prominence.<sup>11</sup> As stroke volume falls from right ventricular systolic failure, heart rate rises to maintain cardiac output. A larger proportion of right ventricular ejection is effected by septal contraction, as free wall contraction fails because of ischemia.

Right-sided heart failure, when it is due to right ventricular infarction, results in a low-output, hypotension syndrome. There is rarely a prominent concurrent amount of left-sided heart failure because the right ventricular infarction reduces filling of the left side of the heart. The transmural distending pressure of the left ventricle (diastolic pressure minus the intrapericardial pressure) is profoundly influenced by right-sided heart failure and dilation. The intrapericardial pressure and the right-sided heart diastolic pressures are nearly identical and very low. Left ventricular filling (transmural distending pressure) is normally approximately 10 mm Hg minus 0 mm Hg; whereas in right ventricular infarction, the left ventricular filling (transmural distending pressure) is markedly reduced (approximately 10 mm Hg minus 10 mm Hg). Hypotension, due to underfilling of the left side of the heart, is the most obvious manifestation of right ventricular infarction.

The thin-walled right ventricle and atrium are prone to early dilation. Significant dilation will challenge the acute pericardial (volume) reserve. Excessive right-sided heart dilation will achieve a compressive phenomenon within the pericardial space, elevating diastolic pressures. The dilated right-sided chambers will compete for space with the left-sided chambers. Maneuvers that further augment right-sided heart filling, such as volume loading, will actually compromise left-sided heart filling within the finite intrapericardial space.<sup>12</sup>

Factors that impair right atrial systolic function may lead to a prominent fall in right ventricular function. The development of atrial fibrillation and complete heart block typically result in prompt hypotension. Right atrial infarction, which is difficult to

diagnose, has the same detrimental effect as atrial fibrillation.<sup>13</sup> The more proximal the occlusion of the right coronary artery, the greater the likelihood of right atrial branch loss and right atrial infarction, which is apparent as a lack of an increase in the right atrial A wave and  $x$  descent and an M pattern of the right atrial waveform, whereas a mid right coronary occlusion without extensive right atrial branch loss and without significant right atrial infarction is associated with an augmented right atrial A wave and  $x$  descent and a W pattern of the right atrial waveform.<sup>11</sup>

Septal contraction is an important component of right-sided heart function in right ventricular infarction because the right ventricular free wall is not contributing to contraction and may be dyskinetic, detracting from systolic function. In most individuals, the extent of concurrent septal infarction is small because the posterior septal perforators from the posterior descending coronary artery supply normally only the basal posterior septum and some proportion of the mid posterior septum. In individuals in whom the coronary anatomy generates a larger septal infarction, the compromising effect on right-sided heart systolic function will be larger.

Right ventricular ischemic or infarcting myocardium exhibits a steeper pressure:volume (length:tension) relationship and is therefore more sensitive to variation in filling.<sup>11</sup> Use of venodilators, such as nitroglycerin, or diuretics will deprive the right ventricle of venous return and preload, and nitroglycerin intolerance is a common occurrence in right ventricular infarction.

Elevation of right atrial pressure (from right ventricular infarction) will enable right-to-left shunting in those with patency of the foramen ovale. Dilation of the right atrium from right ventricular failure will enlarge the foramen ovale, augmenting shunt flow.

The systolic function of the right ventricular free wall recovers faster and more completely than does that of the left ventricular inferior wall,<sup>2</sup> although not all cases recover enough or fast enough to elude shock and death. The reasons that the right ventricle demonstrates greater recoverability are not well understood. The lesser right ventricular myocardial demand (less mass, pressure), the presence of collaterals from the left coronary system, the balanced pattern (systolic and diastolic) of right coronary blood flow versus left coronary blood flow (diastolic predominant), and potentially the oxygen supply through right ventricular thebesian veins may contribute to the fact that the right ventricle is more likely to stun than to infarct. Preinfarction angina reduces the risk of right ventricular infarction and its

**Table 6-2.** Preinfarction Angina Decreases the Risk of Right Ventricular Infarction and Complications

		With Angina (N = 62)	Without Angina (N = 51)
ST elevation V <sub>4</sub> R	27%	71%	<.001
Complete AV block	11%	33%	.04
Hypotension or shock	8%	53%	<.001
In-hospital death	5%	10%	.26

From Shiraki H, Yoshikawa T, Anzai T, et al: Association between preinfarction angina and a lower risk of right ventricular infarction. *N Engl J Med* 1998;338:941-947.

complications, presumably because of ischemic preconditioning and collateral recruitment (Table 6-2).<sup>7</sup> Less than half of proximal dominant occlusions produce recognized right ventricular infarction. The extent of recovery of the right ventricle has prompted some to propose that the right ventricle experiences ischemia rather than infarction.

## CLINICAL PRESENTATIONS OF RIGHT VENTRICULAR INFARCTION

- Hypotensive inferior infarction
- Inferior infarction, cardiogenic shock
- Inferior infarction with venous distention, Kussmaul sign
- Inferior infarction, blood pressure intolerant of nitroglycerin
- Larger biomarker rise than anticipated from inferior infarction
- Greater amount of hypotension than expected from a first infarct with a small or moderate creatine kinase rise

Differential diagnosis of myocardial infarction with prominent hypotension but without proportionally as much pulmonary edema:

- Underfilling of the left ventricle
  - Hypovolemia, may be from medications
  - Right ventricular infarction
- Vasodepression
  - Excessive effect of medications
  - Medication-induced anaphylaxis
  - Vagal vasodepressant state
- Tamponade
- Ventricular septal rupture

## DIAGNOSIS OF RIGHT VENTRICULAR INFARCTION

### Physical Diagnosis

- 2 Right ventricular infarction can be diagnosed at the bedside. The combination of jugular venous pressure and >8 cm and a Kussmaul sign is 88% sensitive and 76% specific for right ventricular

infarction. The triad of hypotension, venous distention, and clear lung fields in the setting of inferior infarction is 96% specific for right ventricular infarction but only 25% sensitive.<sup>14</sup> Hypovolemia and hypervolemia obscure jugular venous findings of right ventricular infarction.

### Electrocardiography

Electrocardiography (ECG) is useful for the diagnosis of right ventricular infarction, as long as the baseline ECG recording is normal and if ECG is performed very early in the infarction. Several ECG patterns have been advanced. Right-sided chest leads (V<sub>4</sub>R) demonstrating more than 0.5 mm or more than 1 mm of ST elevation suggest right ventricular infarction. Use of 0.5 mV or mm is more sensitive but less specific; 1 mm of ST elevation 0.08 second after the J point is seen in most (>80%) cases and has a high pathologic correlation with right ventricular infarction (100%).<sup>15</sup> A significant caveat is that the finding of ST elevation in V<sub>4</sub>R is evanescent, lasting only 24 to 48 hours and normalizing in half of cases within 10 hours. False-positives are also common and may be generated by pulmonary embolism, pericarditis, acute anteroseptal ST elevation myocardial infarction (STEMI), and anteroseptal aneurysm. ST elevation in V<sub>1</sub> through V<sub>4</sub> is associated with pure right ventricular infarction.<sup>16</sup> ST elevation in standard limb lead III more than in standard limb lead II is 97% sensitive and 70% specific.<sup>17</sup>

Other common, clinically important but not diagnostic ECG findings in right ventricular infarction include sinus bradycardia, sinus tachycardia, atrial fibrillation (seen in up to one third of cases),<sup>18</sup> atrioventricular block and third-degree block, and right bundle branch block. Right bundle branch block has been noted in up to 48% of cases of right ventricular infarction and is associated with a poor prognosis.<sup>19,20</sup>

### Echocardiography

Echocardiography is useful to corroborate findings of right ventricular systolic dysfunction and to identify or to exclude associations and complications of inferior infarction or of the right ventricular infarction itself. Older echocardiographic and nuclear signs of right ventricular infarction were simply right ventricular dilation and overall systolic dysfunction. With improved imaging, assessment of regional right ventricular systolic function is now possible and useful because it correlates with the right coronary anatomy and the location of occlusion. The posterior wall of the right ventricle is most commonly affected by right ventricular infarction because it is the most distal right coronary territory.<sup>21</sup> Combined right ventricular lateral and posterior wall motion abnormalities are produced by more proximal right coronary artery occlusions and are the typical findings of hemodynamically significant right ventricular infarction. In the context of inferior infarction, right ventricular akinesis or hypokinesis (segmental) is 83% sensitive and 93% specific for right ventricular infarction.<sup>22</sup>

Right ventricular wall motion abnormalities do occur in other disease states, such as pulmonary hypertension, pulmonary embolism, and trauma, and after poor pump protection. Views that assist with right ventricular regional wall motion assessment are listed in Table 6-3. Echocardiographic findings in right ventricular infarction are summarized in Table 6-4.

**Table 6-3.** Echocardiographic Views That Assist with Right Ventricular Regional Wall Motion Assessment

	Segment of the Right Ventricle	Vascular Supply	If Wall Motion Assessment Suggests Site of Occlusion
Echocardiographic view			
Parasternal long-axis view	RVOT	Conus branch	Very proximal RCA
Parasternal short-axis view	Anterior RV	Diagonal branches off the LAD	LAD
	Lateral wall	Acute marginal branches	Before one (mid RCA) or all acute marginal (proximal RCA) branches
	Posterior wall	Diagonal branches off the PDA/PIV	PDA/PIV or anywhere proximal to PDA/PIV, including dominant left circumflex artery
RV inflow view			
Apical 4-chamber view	Lateral wall	Acute marginal branches	Before one (mid RCA) or all acute marginal (proximal RCA) branches
Subcostal long-axis view	Lateral wall	Acute marginal branches	Before one (mid RCA) or all acute marginal (proximal RCA) branches
Subcostal long-axis view	Posterior wall	Diagonal branches off the PDA/PIV	PDA/PIV or anywhere proximal to PDA/PIV, including dominant left circumflex artery
	Lateral wall	Acute marginal branches	Before one (mid RCA) or all acute marginal (proximal RCA) branches
	Anterior RV	Diagonal branches off the LAD	LAD

LAD, left anterior descending coronary artery; PDA/PIV, posterior descending (interventricular) artery; RCA, right coronary artery; RV, right ventricle; RVOT, right ventricular outflow tract.

## Radionuclide Angiography

The diagnosis of right ventricular infarction by radionuclide technique is possible by use of the combination of overall depression of right ventricular systolic function (<40%; normal range is 45% to 75%) and regional right ventricular systolic dysfunction.<sup>1</sup> Technetium pyrophosphate scanning is specific but not sensitive.<sup>1</sup>

## Cardiac Magnetic Resonance

Cardiac magnetic resonance (MR) is able to image the increased myocardial water (edema) by T2 weighting and by gadolinium contrast enhancement.<sup>25</sup> Cardiac MR SSFP sequences elegantly depict right and left ventricular regional wall motion.

## Hemodynamics, Cardiac Catheterization, and Angiography

Right-sided heart catheterization is seldom necessary for diagnosis of right ventricular infarction, but waveforms are revealing of the physiology. As described before, as long as right atrial infarction has not occurred, right atrial systolic function (Starling forces) is recruited to enable filling of a noncompliant right ventricle, resulting in an augmented A wave and x descent of the right atrial pressure and a W pattern. When significant right atrial infarction has occurred, the right atrial A wave is therefore diminished, as is the x descent, and the right atrial waveform has an M pattern.<sup>12</sup> Pericardial restraint will also contribute to the M pattern ("square root" or "dip and plateau" pattern) typical of severe and larger right ventricular infarction with impaired right ventricular compliance and pericardial restraint.<sup>21</sup> The x descent predominance can also be seen. The right ventricular systolic waveform

becomes broad and sometimes bifid, reflecting lesser dP/dt (upslope from systolic failure), impaired relaxation (reduced -dP/dt), and septal bulge into the right ventricle.

The most prominent pressure waveform of right ventricular infarction is the combination of diastolic failure (elevated right atrial and right ventricular diastolic pressure) and systolic failure (lower right ventricular systolic pressure). Thus, a right atrial pressure:pulmonary capillary wedge pressure (RAP:PCWP) above 0.86 (normal <0.6) is 82% sensitive and 97% specific for right ventricular infarction in the setting of inferior infarction.<sup>26</sup>

## MANAGEMENT

Management principles are based on the following:

- Optimize right ventricular preload
  - Optimize volume status (central venous pressure [CVP] of 15 mm Hg)
  - Maintain atrioventricular synchrony by maintaining or restoring sinus rhythm (if in atrial fibrillation) or sequential pacing for complete heart block
- Reduce right ventricular afterload
  - Normalize left atrial pressure
  - Consider use of nitric oxide
- Inotrope stimulation of stunned right ventricular tissue
- Maintain arterial pressure
  - Vasopressor support
  - Intra-aortic balloon counterpulsation (IABP) use
- Reperfusion (early)
- In case of intractable circulatory failure, consider:
  - Assist device
  - Transplantation

It is important to achieve adequate preload for the context of right ventricular infarction. Alleviation or avoidance of underfilling is beneficial, and optimization of the right ventricular diastolic pressure may improve the cardiac output. In many patients, judicious volume infusion corrects hypotension.

**Table 6-4.** Echocardiographic Findings in Right Ventricular Infarction

Right ventricular dilation
Right ventricular segmental systolic dysfunction
Reduced motion of the right atrioventricular groove due to reduced right ventricular longitudinal contraction
Right ventricular dilation (+ small left ventricle = interdependence)
Signs of elevated right atrial pressure
Right atrial dilation
Inferior vena cava dilation and reduced collapsibility
Interatrial septal deviation to the left side
Signs of elevated right ventricular diastolic pressure, low pulmonary artery diastolic pressure
Complete (presystolic) pulmonic valve opening <sup>23</sup>
Flow across a patent foramen ovale from the right to the left side (color Doppler flow mapping or agitated saline study)
Tricuspid regurgitation
Right ventricular papillary muscle rupture
Reduced cardiac output or index
Right ventricular systolic pressure not elevated, due to right ventricular systolic failure
Premature (post A wave) pulmonary valve opening (right ventricular noncompliance)
Right ventricular thrombus
Right atrial thrombus <sup>24</sup>
Right ventricular pseudoaneurysm
Complications of associated inferior wall or inferior septal infarction
Wall motion abnormalities
Aneurysms
False aneurysms
Intramycardial hematoma
Septal rupture
Papillary muscle rupture
(Nonrupture) mitral insufficiency

**Table 6-5.** Effect of Volume Loading on Experimental Right Ventricular Infarction

	Experimental RVMI	Volume Loading
RVSP	-25%	+28%
RVEF %	-57%	+67% (stroke work)
Aortic pressure	-36%	+35%
Cardiac output	-32%	+35%

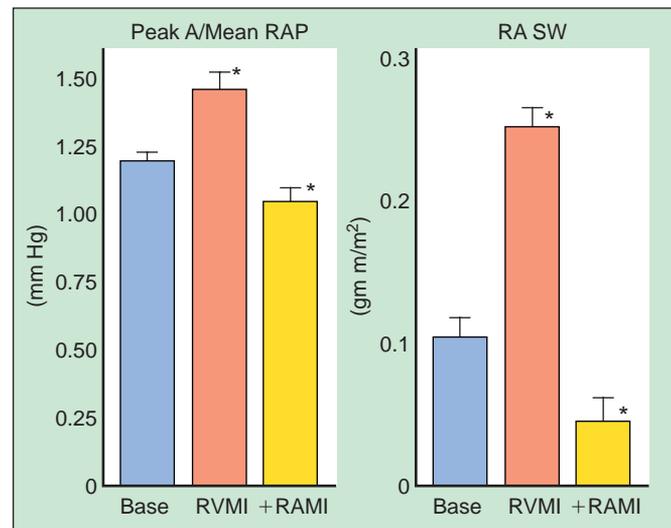
RVEF, right ventricular ejection fraction; RVMI, right ventricular myocardial infarction; RVSP, right ventricular systolic pressure.  
 From Goldstein JA, Vlahakes GJ, Verrier ED, et al: Volume loading improves low cardiac output in experimental right ventricular infarction. *J Am Coll Cardiol* 1983;2:270-278.

However, excess volume loading, in addition to congesting the venous vasculature and potentially compromising organ (especially hepatic) function, leads to overfilling of the right side of the heart and, because of ventricular interdependence, increases left-sided heart diastolic pressures while decreasing left-sided heart volumes, stroke volume, and cardiac output. Furthermore, experimental canine models of right ventricular infarction establish that volume loading in the context of intact pericardium increases right ventricular infarction twofold to threefold compared with pericardiectomy, probably by reducing right ventricular perfusion gradient (Table 6-5; Figs. 6-5 to 6-7).<sup>27,28</sup>

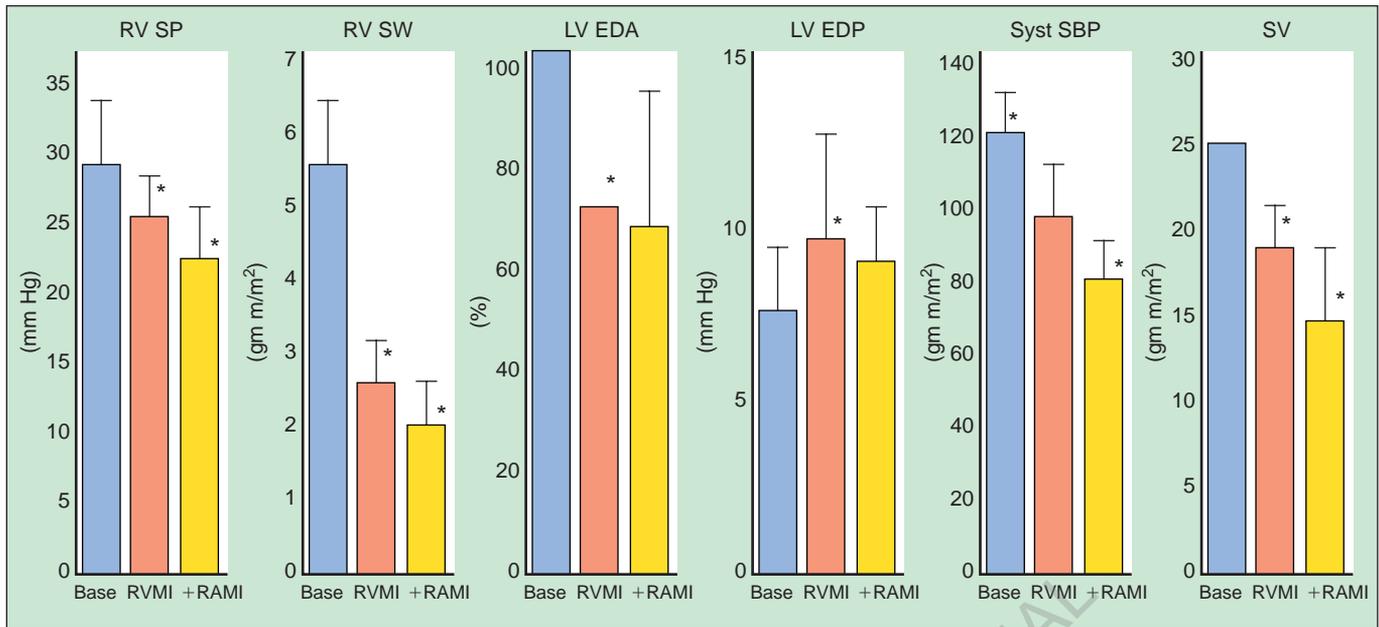
Above a right atrial pressure of 15 mm Hg, volume loading does not further improve cardiac index or mean arterial blood pressure, but inotropic stimulation (5 to 10 µg/min/m<sup>2</sup> dobutamine) does (Figs. 6-8 and 6-9).<sup>29</sup>

A small series demonstrated the need and the benefits of maintaining atrioventricular synchrony; atrial pacing at the same rate increased the stroke volume by a mean of 42%.<sup>30</sup> Observational data suggest that when reperfusion can be established, the mean right atrial pressure nearly normalizes (Fig. 6-10),<sup>31</sup> and right ventricular systolic function (ejection fraction) may also nearly normalize.<sup>32</sup> Unsuccessful percutaneous coronary intervention (PCI) is associated with absence of improvement of right ventricular systolic function, persistence of hypotension and low cardiac index, and significantly higher mortality (Fig. 6-11).<sup>2</sup> Among patients in whom PCI was successful, the percentage with persistent hypotension and low cardiac output is significantly less, as is mortality.<sup>2</sup> The effect of successful PCI on right ventricular systolic function, right atrial pressure, and cardiac index is demonstrable at 1 hour after the procedure and is yet further •• at 1 and 3 days (Figs. 6-12 to 6-14).<sup>2</sup> The percentage of cases in which right ventricular systolic function has normalized at 1 month is significantly greater after successful reperfusion (Fig. 6-15).<sup>2</sup>

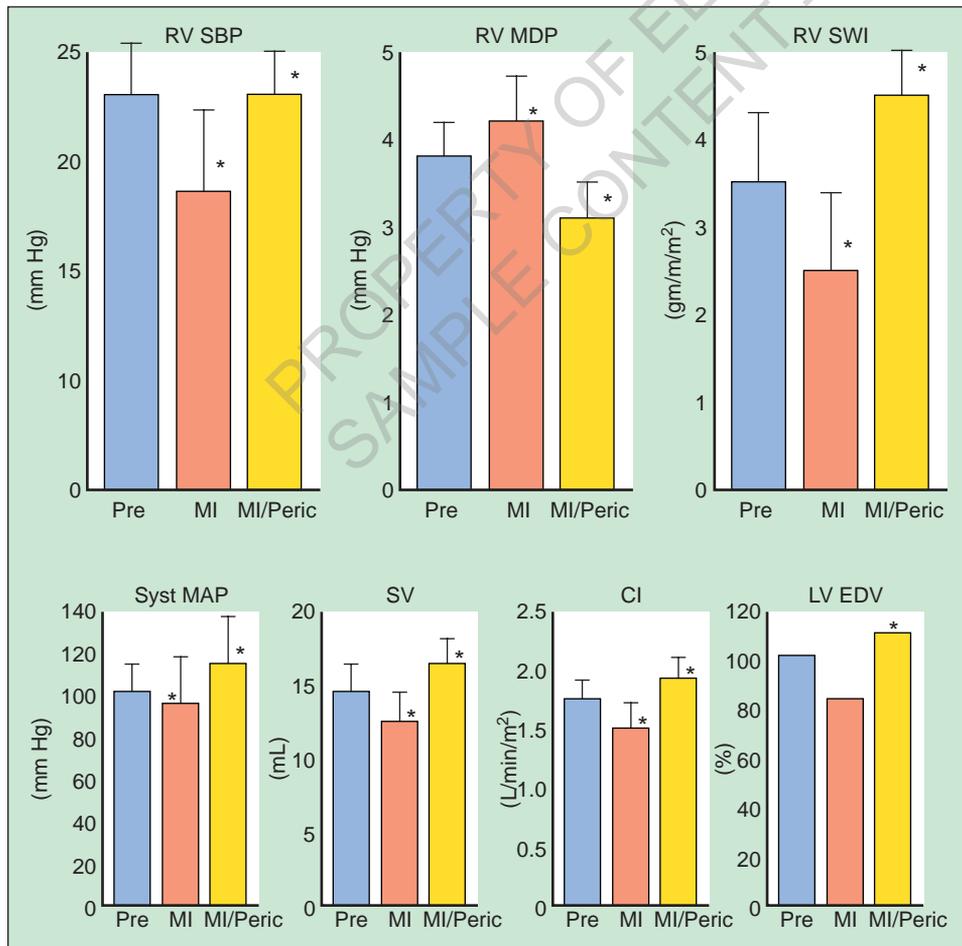
Although PCI has not been shown in large prospective trials to reduce mortality in right ventricular infarction, the



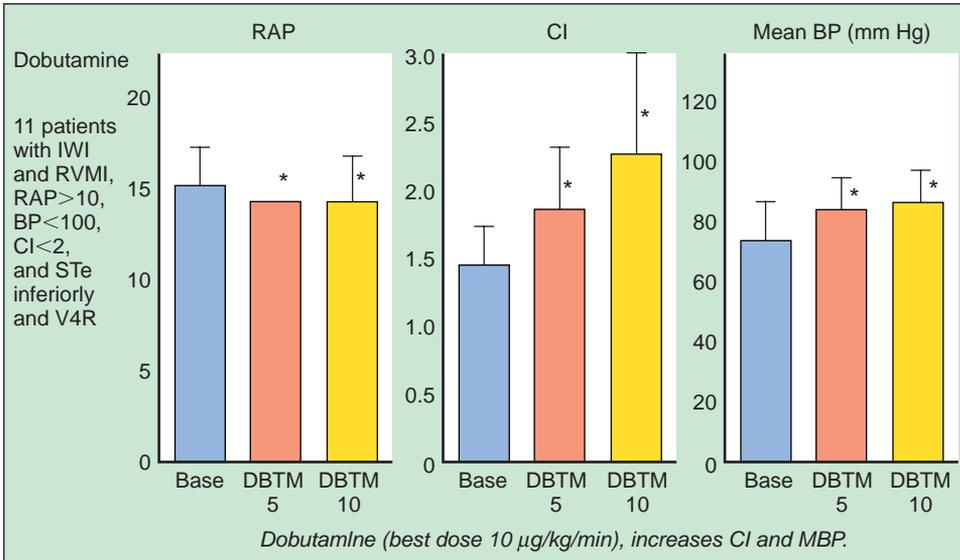
**Figure 6-5.** Experimental right ventricular (RV) infarction increases right atrial (RA) function (stroke work [SW]) because of Starling recruitment. Right atrial infarction results in right atrial mechanical failure. MI, myocardial infarction; RAP, right atrial pressure. (Data from Goldstein JA, Vlahakes GJ, Verrier ED, et al: The role of right ventricular systolic dysfunction and elevated intrapericardial pressure in the genesis of low output in experimental right ventricular infarction. *Circulation* 1982;65:513-522.)



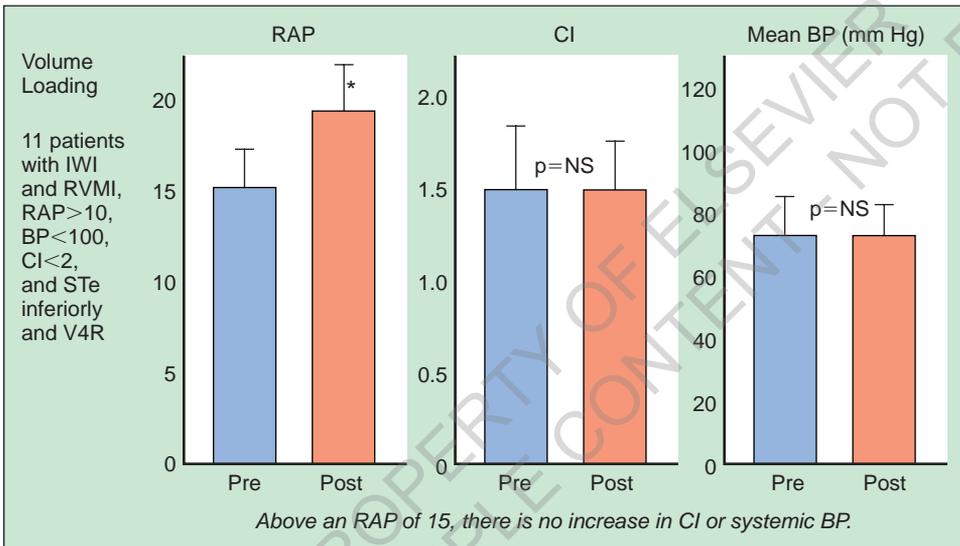
8  
9 **Figure 6-6.** Experimental right atrial infarction worsens right ventricular (RV) systolic function in right ventricular infarction and results in underloading of the left ventricle and worsened arterial pressure. LV EDA, ●●; LV EDP, left ventricular end-diastolic pressure; SBP, systolic blood pressure; SP, stroke power; SV, stroke volume; SW, stroke work. (Data from Goldstein JA, Vlahakes GJ, Verrier ED, et al: The role of right ventricular systolic dysfunction and elevated intrapericardial pressure in the genesis of low output in experimental right ventricular infarction. *Circulation* 1982;65:513-522.)



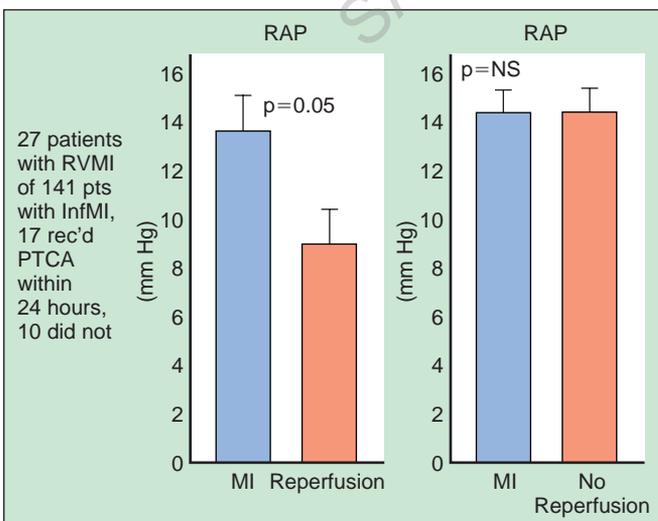
10 **Figure 6-7.** In experimental right ventricular infarction, hemodynamic indices are improved by pericardiotomy, revealing that pericardium-mediated ventricular interdependence contributes to left-sided heart underloading and output. CI, cardiac index; LV EDV, left ventricular end-diastolic volume; MAP, mean arterial pressure; MDP, mean diastolic pressure; RV, right ventricle; SBP, systolic blood pressure; SWI, stroke work index; SV, stroke volume. (Data from Goldstein JA, Vlahakes GJ, Verrier ED, et al: The role of right ventricular systolic dysfunction and elevated intrapericardial pressure in the genesis of low output in experimental right ventricular infarction. *Circulation* 1982;65:513-522.)



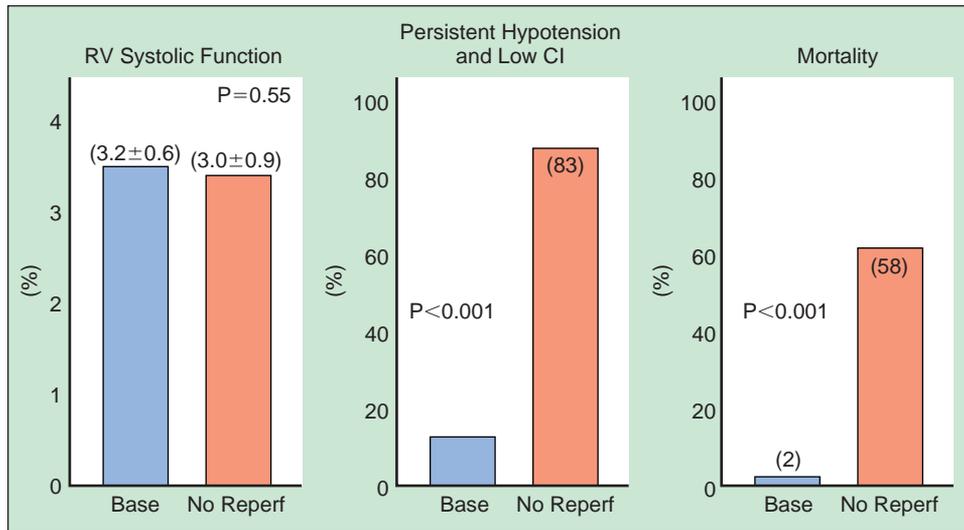
**Figure 6-8.** In patients with right ventricular infarction and inferior left ventricular infarction optimized with right atrial pressure above 10 mm Hg but still hypotensive and with low output, inotropic doses of dobutamine lower right atrial pressure (RAP) and increase cardiac index (CI) and arterial blood pressure. IWI, inferior wall infarction; RVMI, right ventricular myocardial infarction. (Data from Ferrario M, Poli A, Previtali M, et al: Hemodynamics of volume loading compared with dobutamine in severe right ventricular infarction. *Am J Cardiol* 1994;74:329-333.)



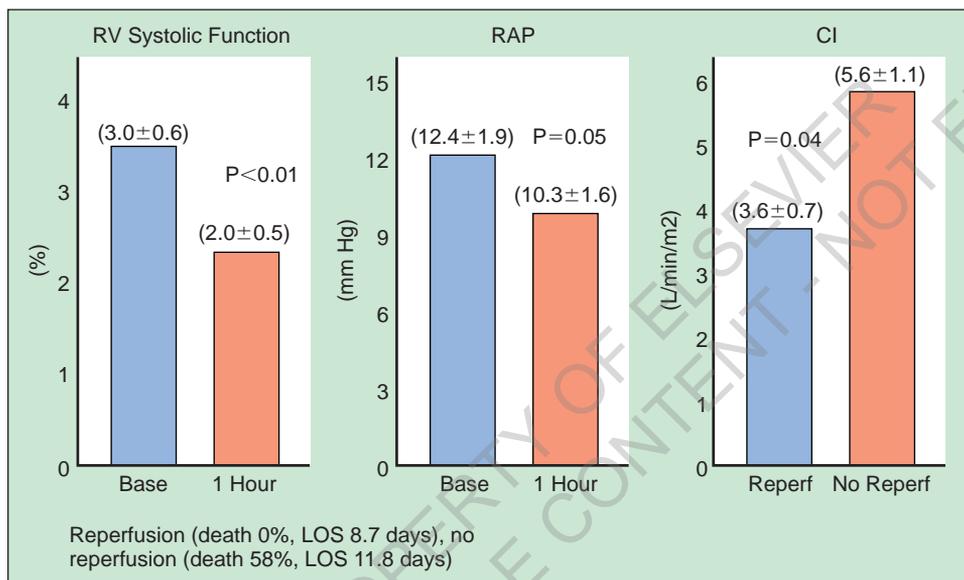
**Figure 6-9.** In patients with right ventricular infarction and inferior left ventricular infarction already with optimal right ventricular filling pressures, further volume loading does not improve arterial hemodynamics. CI, cardiac index; IWI, inferior wall infarction; RAP, right atrial pressure; RVMI, right ventricular myocardial infarction. (Data from Ferrario M, Poli A, Previtali M, et al: Hemodynamics of volume loading compared with dobutamine in severe right ventricular infarction. *Am J Cardiol* 1994;74:329-333.)



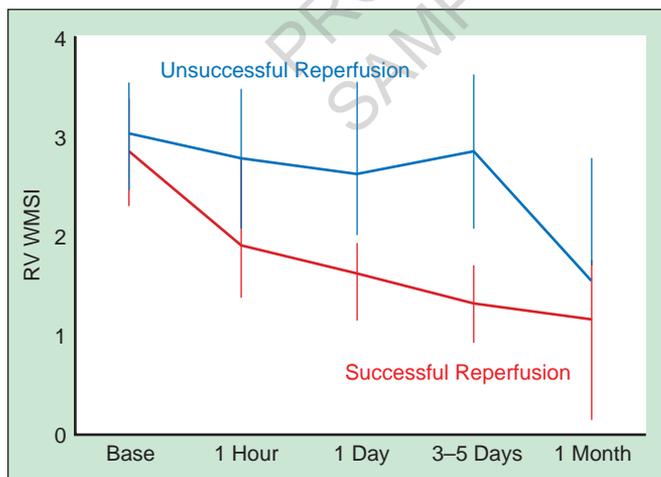
**Figure 6-10.** Successful reperfusion reduces mean right atrial pressure (RAP). PTCA, percutaneous transluminal coronary angioplasty; RVMI, right ventricular myocardial infarction. (Data from Kinn JW, Ajluni SC, Samyn JG, et al: Rapid hemodynamic improvement after reperfusion during right ventricular infarction. *J Am Coll Cardiol* 1995;26:1230-1234.)



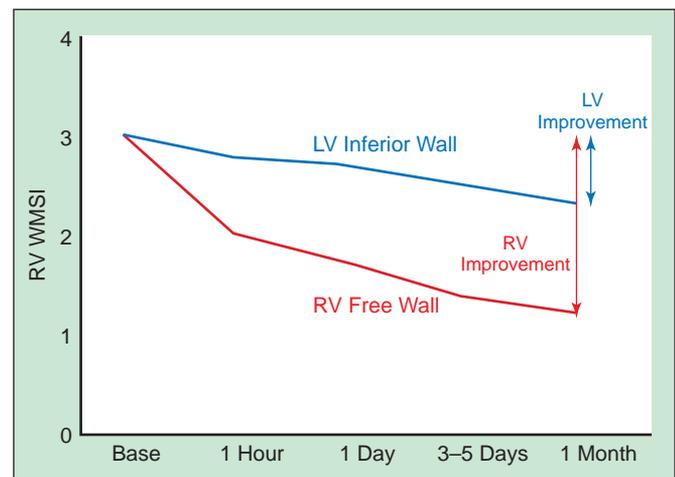
**Figure 6-11.** Unsuccessful PCI is associated with absence of improvement of right ventricular (RV) systolic function, persistence of hypotension and low cardiac index (CI), and significantly higher mortality. (Data from Bowers TR, O'Neill WW, Grines C, et al: Effect of reperfusion on biventricular function and survival after right ventricular infarction. N Engl J Med 1998;338:933-940.)



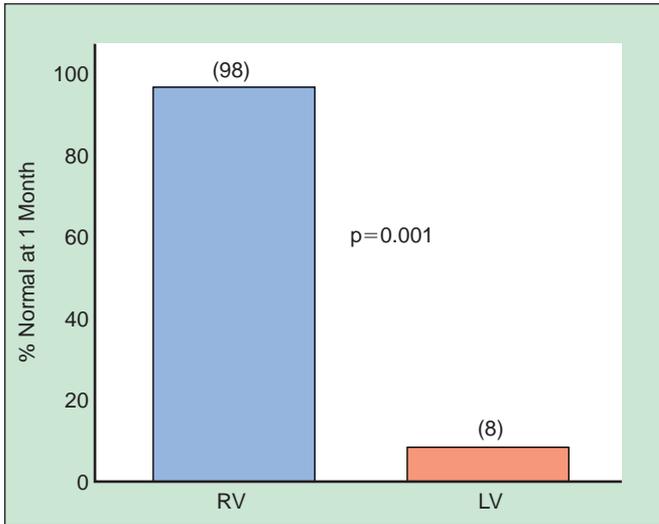
**Figure 6-12.** PCI, effective in establishing reperfusion, is associated with improved right ventricular (RV) systolic function, lower right atrial pressure (RAP), and improved cardiac output and index (CI). LOS, length of stay. (Data from Bowers TR, O'Neill WW, Grines C, et al: Effect of reperfusion on biventricular function and survival after right ventricular infarction. N Engl J Med 1998;338:933-940.)



**Figure 6-13.** Successful PCI leads to early improvement (fall in right ventricular wall motion score index [RV WMSI]) in right ventricular function, whereas PCI unsuccessful in achieving reperfusion is associated with delayed recovery. (From Bowers TR, O'Neill WW, Grines C, et al: Effect of reperfusion on biventricular function and survival after right ventricular infarction. N Engl J Med 1998;338:933-940.)



**Figure 6-14.** The right ventricle (RV) recovers more and faster than does the inferior wall of the left ventricle (LV). RV WMSI, right ventricular wall motion score index. (From Bowers TR, O'Neill WW, Grines C, et al: Effect of reperfusion on biventricular function and survival after right ventricular infarction. N Engl J Med 1998;338:933-940.)



**Figure 6-15.** At 1 month after infarction, almost all survivors have normal right ventricular (RV) systolic function, but few have normalized left ventricular (LV) systolic function. (Data from Bowers TR, O'Neill WW, Grines C, et al: Effect of reperfusion on biventricular function and survival after right ventricular infarction. *N Engl J Med* 1998;338:933-940.)

available evidence favors its use, particularly early use, and suggests that it is underused.

Therapy for right ventricular infarction cases should be individualized. General principles of therapy are summarized in Table 6-6.<sup>33</sup>

**Table 6-6.** General Principles of Therapy for Right Ventricular Infarction Cases

Type of Case	Therapy
Asymptomatic	Avoid diuretics and vasodilators that may precipitate low output and hypotension.
Symptomatic low-output state and normotensive with right atrial pressure or pulmonary capillary wedge pressure <15 mm Hg	Add fluid to increase the pulmonary capillary wedge pressure to 15-18 mm Hg. If cardiac output does not increase adequately, add a vasodilator. If the cardiac output still does not increase adequately, add dobutamine or amrinone. Reperfusion therapy should be considered.
Symptomatic low-output state and normotensive with right atrial pressure or pulmonary capillary wedge pressure >15 mm Hg	Add intravenous dobutamine or amrinone. Additional vasodilator with fluid therapy support may be added. Reperfusion therapy should be considered.
Cardiogenic shock	Sustain blood pressure with dopamine; additional dobutamine may increase cardiac output. Consider right ventricular assist or pulmonary artery counterpulsation for select cases. Reperfusion therapy should be strongly considered.

## OUTCOMES

The in-hospital mortality of right ventricular infarction is, as expected, graded according to the severity of the hemodynamic disturbances (Killip I: 5% mortality; Killip IV, cardiogenic shock: 55% mortality) and the occurrence of complications or associated lesions. The in-hospital mortality of inferior infarction without right ventricular infarction is 6%; with right ventricular infarction, 31%; and with right ventricular infarction and cardiogenic shock, 50% to 60%.<sup>34</sup>

The long-term survival of patients discharged from the hospital is good. Most do recover a large proportion of right ventricular systolic function and therefore have their prognosis determined by the degree of postinfarction left ventricular function and the extent and severity of coronary artery disease. However, postinfarction persistence of right ventricular dysfunction is an independent predictor of the development of heart failure and of death.<sup>35</sup>

### CASE 1

#### History

- ▶ 83-year-old man with chronic renal insufficiency, presenting with 9 hours of ischemic chest pain

#### Physical Examination

- ▶ BP 110/60 mm Hg, HR 48 bpm, RR 12/min
- ▶ Venous distention to the angle of the jaw
- ▶ Normal S<sub>1</sub>, S<sub>2</sub>; S<sub>4</sub> present
- ▶ No murmurs or rubs
- ▶ Extremities warm, mentation normal

#### Management

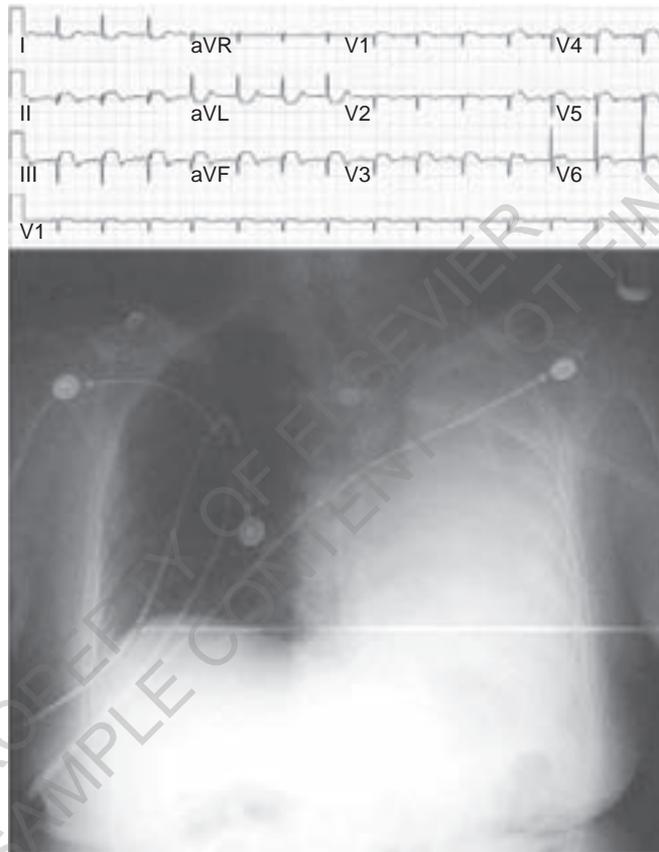
- ▶ Avoidance of preload-reducing maneuvers
- ▶ As the neck veins were indicative of adequate preload, no volume was given.
- ▶ As the output was clinically adequate, no inotropes were given.

#### Evolution and Outcome

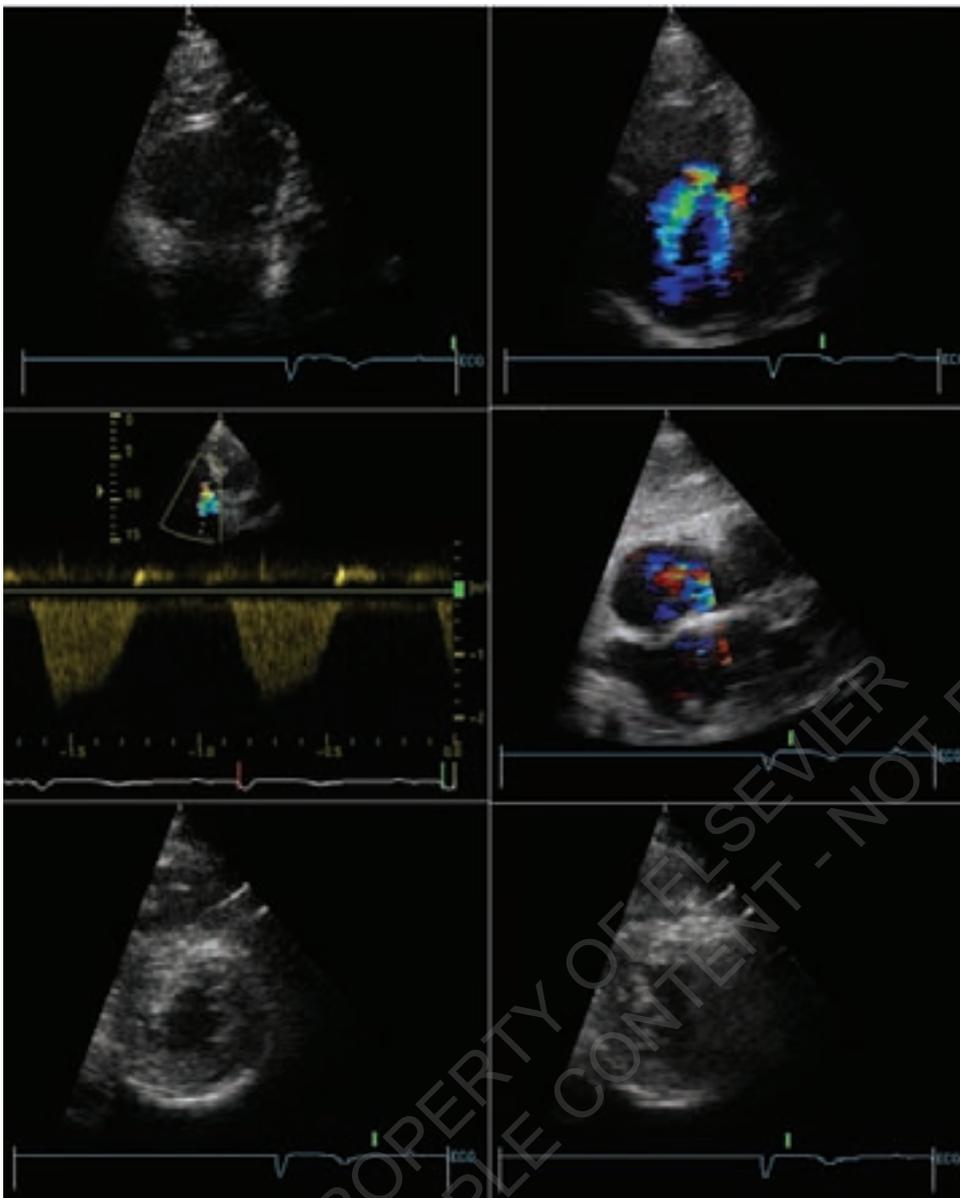
- ▶ Right-sided heart failure resolved after 3 hours.
- ▶ The remainder of the hospital course was uneventful: no recurrent pains, no reversible defect on perfusion scanning.
- ▶ Well at follow-up

#### Comments

- ▶ Right ventricular myocardial infarction diagnosed by elevated neck veins in the context of an acute inferior STEMI; echocardiography corroborative
- ▶ No major hemodynamic disturbances other than mild hypotension
- ▶ Early spontaneous recovery of right ventricular systolic function



**Figure 6-16.** TOP, ECG shows sinus rhythm, inferior ST elevation, and inverted T waves. Lateral ST elevation and inverted T waves suggest acute inferolateral infarction. BOTTOM, Chest radiograph shows right lung field, no pulmonary edema, and large left pleural effusion.



**Figure 6-17.** Transthoracic echocardiography. TOP ROW, Apical 4-chamber views oriented to the right. There is dilation and “sphericalization” of the right ventricle, consistent with infarction and systolic dysfunction. There is moderate tricuspid insufficiency due to the right ventricular systolic dysfunction. MIDDLE LEFT, Tricuspid regurgitation spectral profile. Right ventricular systolic pressure:  $4 \times V^2 + \text{RAP} = 13 + 15 = 28$  mm Hg. The right ventricular systolic pressure is low for a typical myocardial infarction case with shock, and the diastolic pressure (the right atrial pressure) is disproportionately high (50% of the systolic pressure). The delayed velocity rise (slope) of the tricuspid regurgitation is consistent with impaired right ventricle dP/dt. MIDDLE RIGHT, Right atrial and right ventricular dilation. There is marked systolic bulging of the right atrium due to right atrial pressure exceeding that of the left atrium. BOTTOM ROW, Diastolic (LEFT) and systolic (RIGHT) views. There is diastolic flattening of the interventricular septum due to elevation of the right ventricular diastolic pressure from the right ventricular infarction.

## CASE 2

### History

- ▶ 75-year-old man presenting with 3 syncopal episodes; no chest pain
- ▶ Found by EMS to be in third-degree aortic valve block, BP 55/– mm Hg
- ▶ Mechanical ventilation and vasopressors started at community hospital
- ▶ Transferred for a pacer

### Physical Examination

- ▶ BP 90/60 mm Hg, HR 55 bpm
- ▶ Cool extremities
- ▶ Head trauma from syncope, 5-cm laceration across the forehead

- ▶ Venous distention to the angle of the jaw
- ▶ Quiet heart sounds
- ▶ No gallops, rubs, or murmurs
- ▶ CK 3000, creatinine 300

### Management

- ▶ Temporary pacer inserted, with poor capture
- ▶ High-dose vasopressors and inotropes used
- ▶ IABP inserted

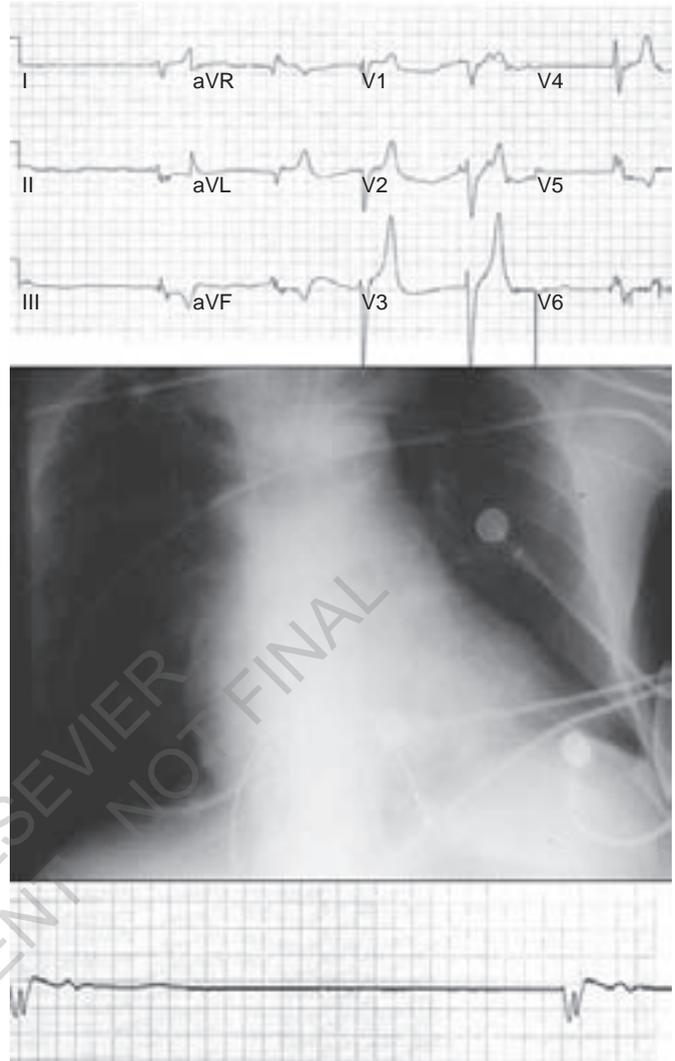
### Outcome

- ▶ Arrived in shock, remained in shock
- ▶ Died within an hour of arrival, within 4 hours of presentation to the community hospital

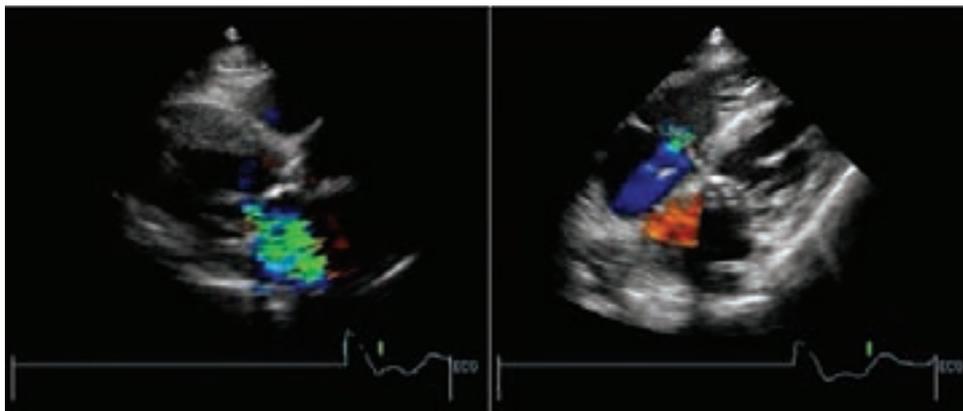
- ▶ Died despite the preceding measures and before angiography could be obtained

**Comments**

- ▶ Massive right ventricular infarction associated with inferior infarction
- ▶ Cardiogenic shock in the context of an inferior STEMI is usually due to right ventricular infarction, as it was here.
- ▶ Acute right-sided heart failure (venous distention and hypotension) is usually due to tamponade, pulmonary embolism, or right ventricular myocardial infarction.
- ▶ The difficulty in achieving capture with the pacer was likely due to the right ventricle's infarction.
- ▶ Pulseless electrical activity likely resulted from progression of the right ventricle's failure, worsened by acidosis; right ventricle perforation, after infarction or due to the pacer.
- ▶ An unfortunate example of the real risk that some right ventricular infarctions confer.



**Figure 6-18.** TOP, ECG shows complete heart block with an irregular wide complex ventricular rhythm. There is no pacemaker capture due to right ventricular infarction. MIDDLE, Chest radiograph shows normal-sized cardiopericardial silhouette, no pulmonary edema. BOTTOM, ECG on arrival shows asystole with ventricular escape beats.



**Figure 6-19.** Transthoracic echocardiography. LEFT IMAGE, Parasternal long-axis view. Contracting anterior septum, akinesis of the posterior wall. Prominent jet of mitral regurgitation. RIGHT IMAGE, Subcostal view (right ventricle free wall akinesis). Right ventricular and atrial dilation and tricuspid insufficiency.

CASE 3	
<b>History</b>	<ul style="list-style-type: none"> <li>▶ 79-year-old man collapsed at home</li> <li>▶ Recent prolonged air travel</li> <li>▶ Various chest pains the day before</li> </ul>
<b>Physical Examination</b>	<ul style="list-style-type: none"> <li>▶ Awake, intubated, very weak, cool extremities</li> <li>▶ BP 80/50 mm Hg, HR 110 bpm, tachypneic</li> <li>▶ Venous distention to the angle of the jaw, S<sub>3</sub></li> <li>▶ No murmurs or rubs</li> <li>▶ CK 500</li> <li>▶ Oliguric</li> </ul>
<b>Clinical Impression</b>	<ul style="list-style-type: none"> <li>▶ Although there was a high index of suspicion of pulmonary embolism, CT scanning was normal, as were leg Doppler studies.</li> <li>▶ The patient was ventilated for airway management.</li> <li>▶ Transesophageal echocardiography was performed because transthoracic images were poor.</li> </ul>

**Management**

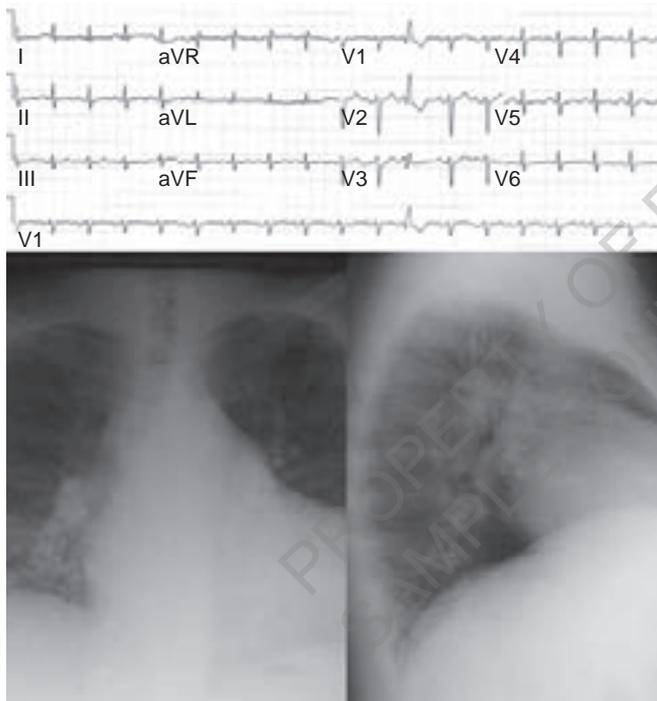
- ▶ Avoidance of preload reduction
- ▶ With pulmonary artery catheter guidance, the CVP was kept at 15 to 18 mm Hg.
- ▶ Dobutamine was given to increase cardiac output.
- ▶ Hemodynamics were acceptable with this regimen, and his hemodynamics spontaneously normalized on day 5.

**Outcome**

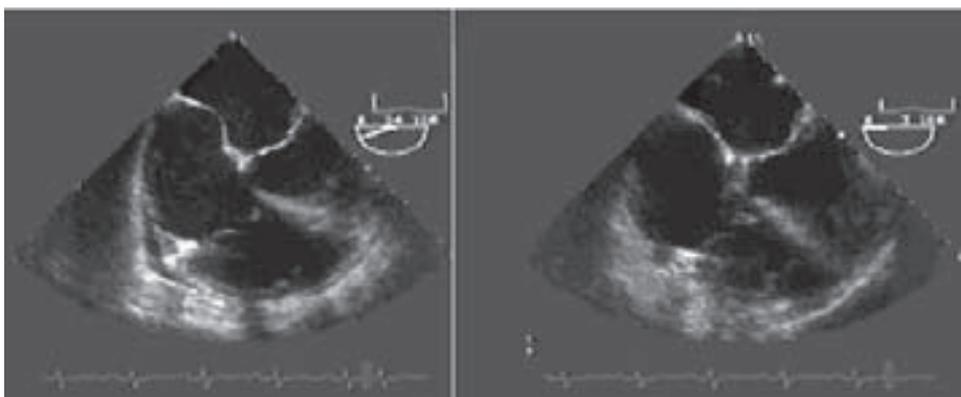
- ▶ Discharged angina and heart failure free

**Comments**

- ▶ Large submassive right ventricular infarction
- ▶ Late presentation
- ▶ Volume optimizing and inotropes corrected the hypotension and low output.
- ▶ Spontaneous recovery
- ▶ Postdischarge echo showed normalized right ventricular systolic function.



**Figure 6-20.** TOP, ECG shows sinus tachycardia with one premature ventricular contraction and nonspecific repolarization abnormalities. BOTTOM, Chest radiograph shows cardiomegaly and mild pulmonary edema.



**Figure 6-21.** Transesophageal echocardiography images show marked right atrial and right ventricular dilation. The left-sided heart chambers are small because of underloading. Interatrial septal bulging to the left side suggests that right atrial pressure is greater than left atrial pressure. The right ventricular free wall is severely hypokinetic or akinetic, despite inotropic support. There is no pericardial effusion.

**CASE 4****History**

- ▶ 54-year-old man developed indigestion at 5 PM
- ▶ At 7 PM, experienced a cardiac arrest on the street
- ▶ 20 minutes of bystander CPR, defibrillation by EMS

**Physical Examination**

- ▶ Deeply comatose, basal skull laceration
- ▶ BP 80/50 mm Hg, HR 100 bpm, ventilated
- ▶ Marked venous distention
- ▶ S<sub>1</sub>, S<sub>2</sub> normal
- ▶ S<sub>4</sub>
- ▶ No murmurs or rubs

**Impression and Evolution**

- ▶ Although there was a high index of suspicion of pulmonary embolism, CT scanning and leg Doppler studies were normal.
- ▶ The patient was ventilated for airway management.
- ▶ Transesophageal echocardiography was performed because transthoracic images were poor.

**Management**

- ▶ Avoidance of preload reduction
- ▶ With pulmonary artery catheter guidance, the CVP was kept at 15 to 18 mm Hg.

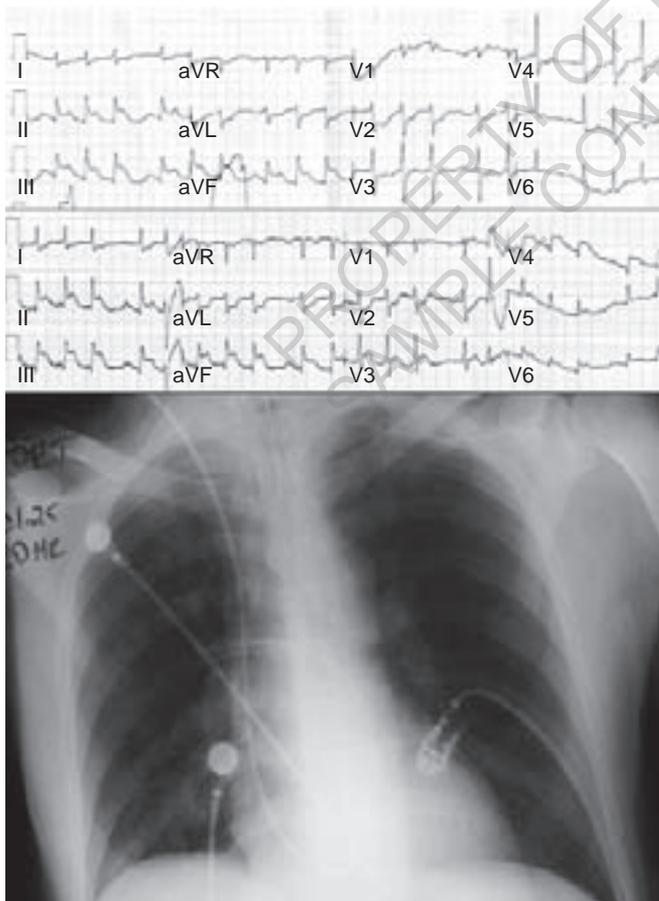
- ▶ Dobutamine was given to increase cardiac output.
- ▶ Hemodynamics were acceptable with this regimen, and his hemodynamics spontaneously normalized on day 5.

**Outcome**

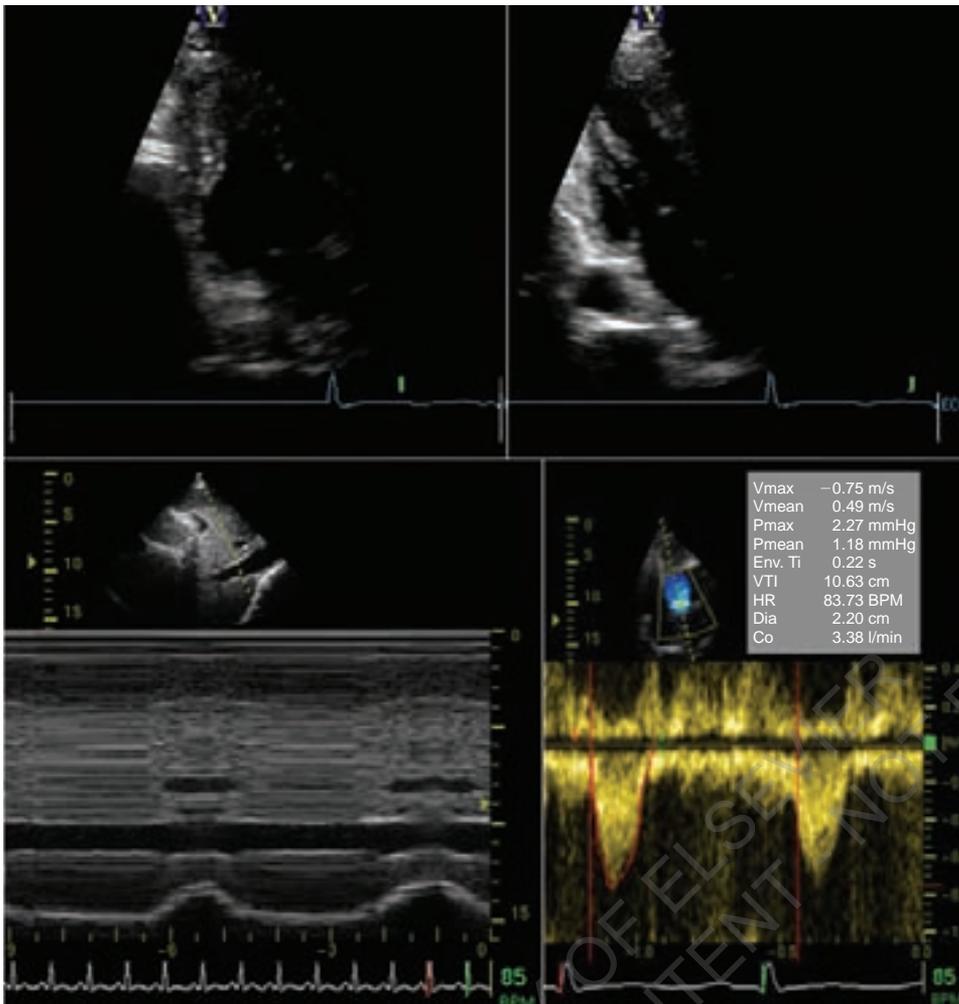
- ▶ Arrived in shock, remained in shock
- ▶ After 5 days, hemodynamic therapy was no longer needed, but ventilatory support was continued because of persistent neurologic impairment.
- ▶ Eventually (60 days later), he died of sepsis while still on a ventilator.

**Comments**

- ▶ Large right ventricular infarction complicating inferior STEMI
- ▶ Initial hypotension due to right ventricular myocardial infarction and recent cardiac arrest
- ▶ Hemodynamic resuscitation was straightforward.
- ▶ Clinical outcome was determined by neurologic sequelae of the out-of-hospital cardiac arrest, not cardiac performance.



**Figure 6-22.** TOP, ECG shows atrial fibrillation, inferior ST elevation (II, III, aVF), and septal and lateral ST depression, suggesting acute inferior myocardial infarction with septal ischemia or “reciprocal” pattern. MIDDLE, Right-sided ECG shows atrial fibrillation with inferior ST elevation. There is more than 1 mm of ST elevation in V<sub>4R</sub>. BOTTOM, Chest radiograph shows normal cardiopericardial silhouette and clear lung fields. There is no pulmonary edema because of the underloading of the left side of the heart.



**Figure 6-23.** Transthoracic echocardiography. TOP ROW, Apical 3-chamber views in systole (LEFT) and diastole (RIGHT). There is dyskinesia of the basal inferior wall. The two bodies of the posterior papillary muscle are intact. BOTTOM LEFT, Subcostal M-mode study of the inferior vena cava. Caval (inferior vena cava) dilation without respiratory collapse = elevated central venous pressure. BOTTOM RIGHT, Left ventricular outflow tract pulsed wave Doppler sampling: reduced VTI (10.6 cm; normal, 18 to 22 cm) consistent with severely reduced stroke volume.

### CASE 5

#### History

- ▶ 62-year-old man presented to a community hospital with 9 hours of chest pain
- ▶ Received tPA at hour 10, but pain continues, as do ST elevations

#### Physical Examination

- ▶ Ventilated, BP 85/50 mm Hg, HR 110 bpm
- ▶ Prominent venous distention
- ▶ S<sub>1</sub>, S<sub>2</sub> normal
- ▶ No gallops, rubs, or murmurs
- ▶ Chest clear

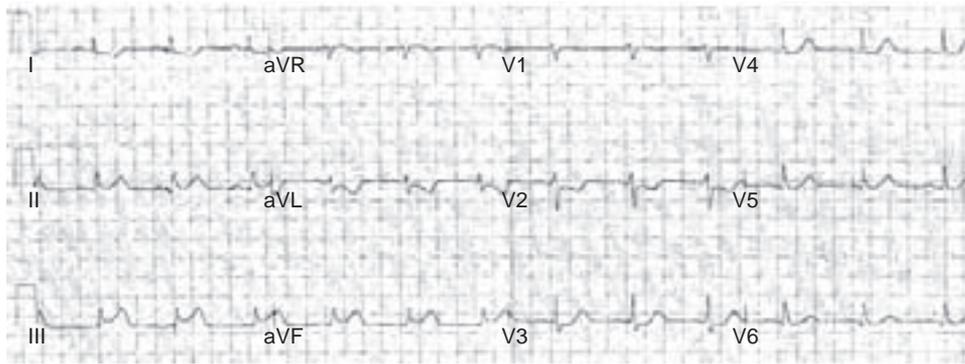
#### Impression and Evolution

- ▶ Inferior myocardial infarction with large right ventricular myocardial infarction
- ▶ Hypoxemia due to large amount of shunting through a patent foramen ovale (PFO)

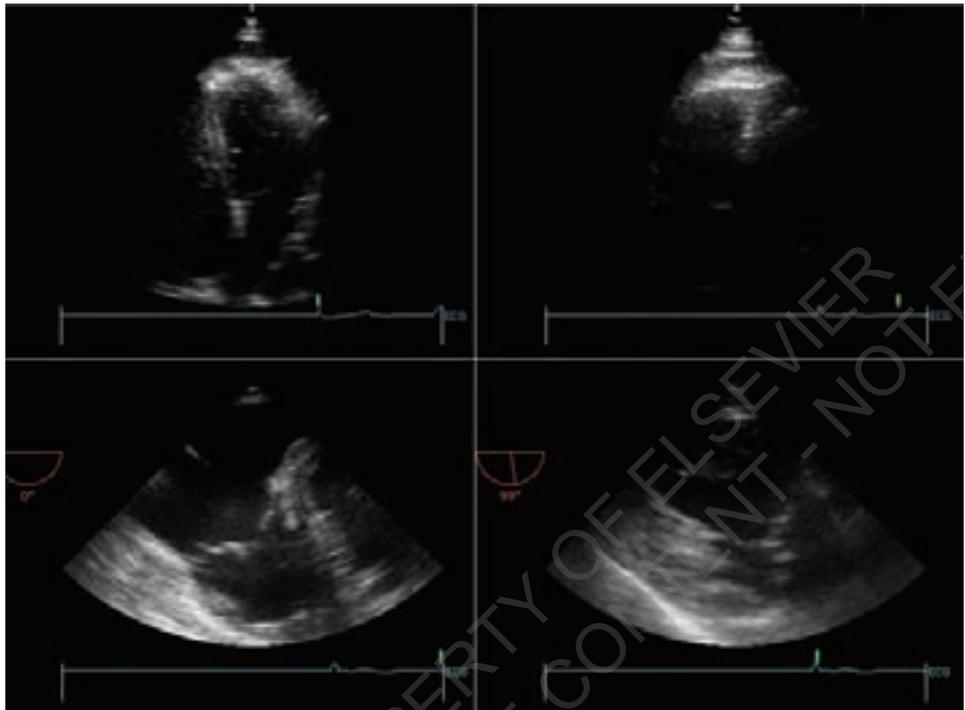
- ▶ The ventilator time became protracted because of persistent elevation of the CVP (18 mm Hg) and shunting through the PFO. Maintenance of adequate oxygenation was difficult.
- ▶ There was no significant left ventricular failure (PCWP 13 mm Hg).
- ▶ PFO closure with a device was considered, but first it was thought worth exploring whether occlusion of the PFO would really increase the oxygenation.

#### Comments

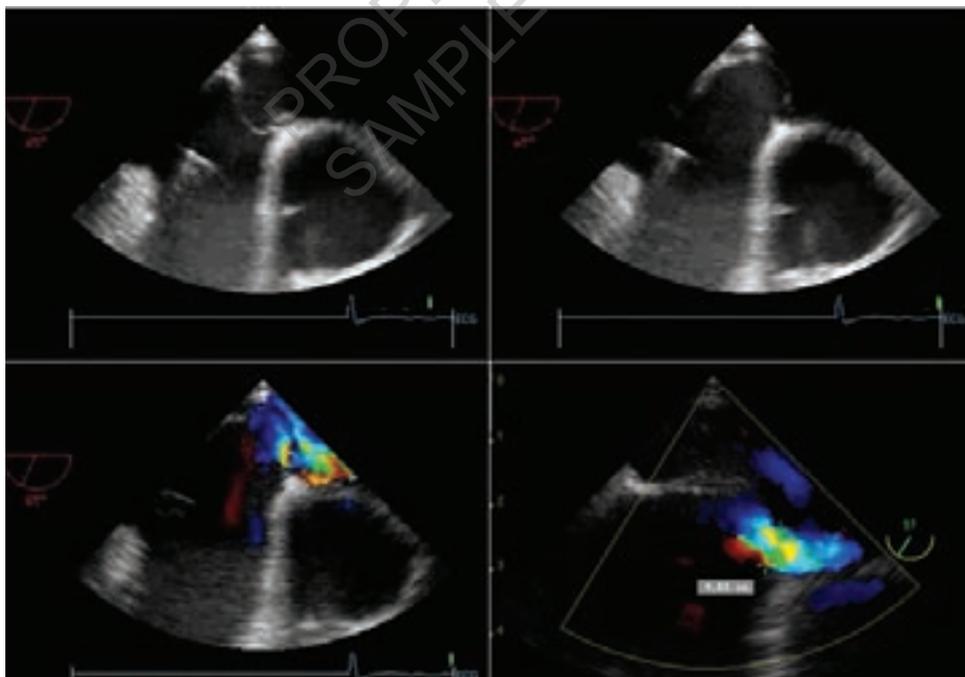
- ▶ Right ventricular infarction complicating inferior STEMI
- ▶ Right-to-left shunting through a PFO contributing to arterial hypoxemia
- ▶ Consideration of percutaneous closure of the PFO was given because the time course on the ventilator was becoming prolonged, but fevers precluded.
- ▶ Eventual recovery of right ventricular systolic function; concurrent with this was recovery of diastolic function as seen by spontaneous closure of the PFO and hypoxemia resolving.
- ▶ Postdischarge echo showed only mild left ventricular and right ventricular systolic function.



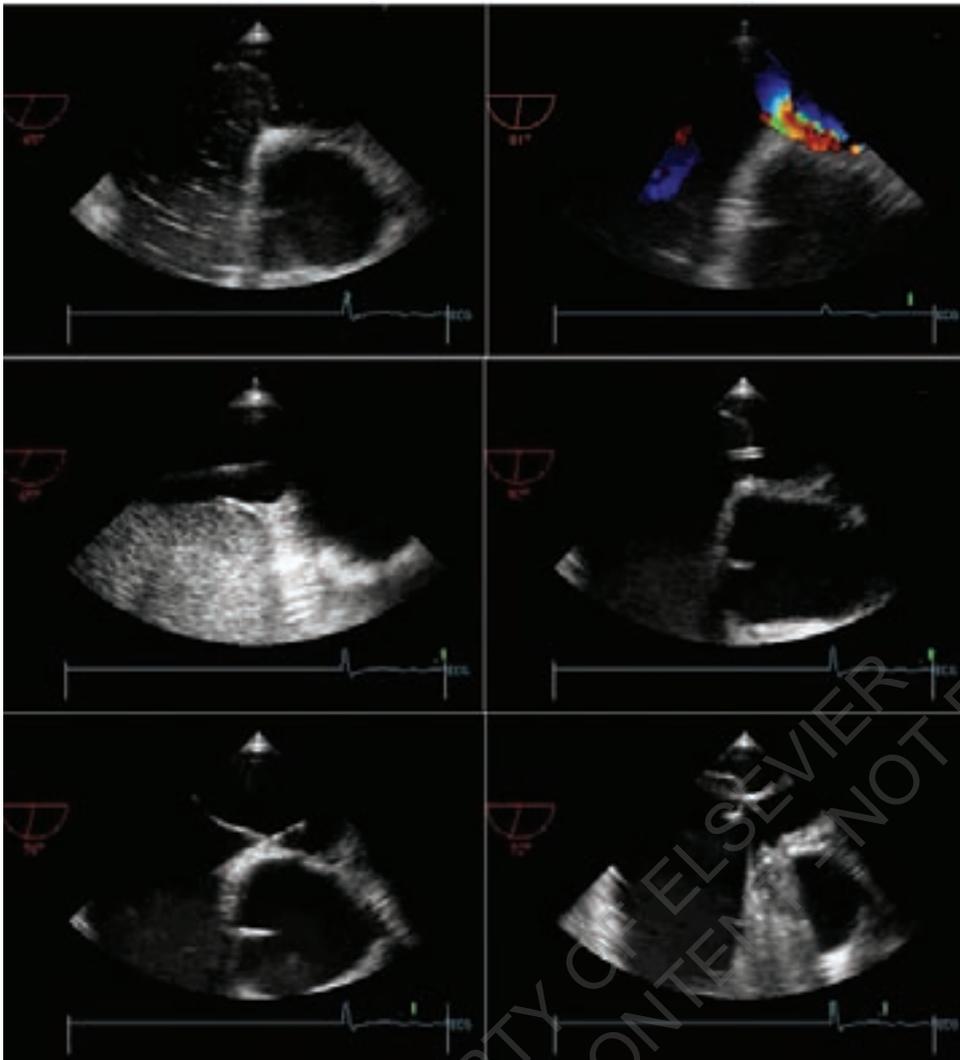
**Figure 6-24.** ECG shows sinus rhythm, ST elevation in II and aVF consistent with acute inferior infarction, and ST depression in V<sub>2</sub> and early R-wave transition, suggestive of acute infarction of the contiguous posterior wall.



**Figure 6-25.** TOP IMAGES, Transthoracic echocardiography. BOTTOM IMAGES, Transesophageal echocardiography. TOP LEFT, The left ventricle on this plane appears normal. TOP RIGHT, The right ventricle is substantially dilated and “sphericalized.” BOTTOM LEFT, The right atrium is prominently dilated, as is the coronary sinus, because of the elevation of the right atrial pressure. BOTTOM RIGHT, The anterolateral papillary muscle is intact.



**Figure 6-26.** Transesophageal echocardiography. TOP LEFT, The interatrial septum is mobile, and there is flow across it because of the elevation of the right atrial pressure, above that of the left atrial pressure, and the presence of a patent foramen ovale. TOP RIGHT, A catheter is being advanced across the interatrial septum. BOTTOM LEFT, A wire has been advanced across the interatrial septum. BOTTOM RIGHT, A balloon has been inflated across the interatrial septum, resulting in prominent reverberations.



**Figure 6-27.** Transesophageal echocardiography. TOP LEFT, View of the right and left atria. The interatrial septum is revealing a patent foramen ovale. A saline injection has resulted in marginal contrast effect in the right atrium. Two bubbles have crossed the PFO into the left atrium. TOP RIGHT, Color flow mapping depicts the right-to-left flow at the atrial level. MIDDLE LEFT, Inferior vena cava in long axis as it enters the right atrium. A guide wire is seen extending to the inferior vena cava:right atrial junction. MIDDLE RIGHT, A catheter (note the near and far wall echoes) has been advanced over the guide wire and has just traversed the PFO. BOTTOM LEFT, The guide wire has been advanced through the PFO into the left atrium. BOTTOM RIGHT, A balloon has been inflated within the left atrium and has filled the PFO. There are near and far wall artifacts from the balloon. With inflation of the balloon within the left atrium and occlusion of the PFO by the balloon, the arterial oxygenation increased.

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