CURRENT CONCEPTS

RIGHT VENTRICULAR INFARCTION

JACK W. KINCH, M.D., AND THOMAS J. RYAN, M.D.

THE description of right ventricular myocardial infarction appeared more than 60 years ago,[1] yet for decades it was not considered an important clinical entity, in large part because of studies in animals in which experimentally induced isolated right ventricular damage caused no substantial change in systemic venous pressure, pulmonary pressure, or cardiac output.[2-4] Twenty years ago Cohn and coworkers[5] published their classic report on right ventricular infarction as a distinct clinical entity. The condition is now recognized to occur in nearly half of all inferior myocardial infarctions.

NORMAL PHYSIOLOGY

The right ventricle has the same cardiac output as the left ventricle, yet it has one sixth as much muscle mass and performs one fourth the stroke work,[6,7] because the pulmonary vascular resistance is one tenth of the systemic resistance.[7] Although the two ventricles are very different geometrically, their interdependence is mediated by the shared interventricular septum and the surrounding pericardium.[8]

Coronary blood flow to the right ventricle is unique in that it occurs during both systole and diastole, in the absence of right ventricular hypertrophy.[9] The right coronary artery provides the predominant blood flow, supplying the lateral wall through the acute marginal branches; in the majority of patients, it also supplies the posterior wall and posterior interventricular septum through the posterior descending artery. The anterior wall of the right ventricle is supplied by the conus artery and by branches of the left anterior descending artery.[10]

PATHOGENESIS OF RIGHT VENTRICULAR INFARCTION

Typically, right ventricular infarction occurs when there is an occlusion of the right coronary artery proximal to the acute marginal branches, but it may also occur with an occlusion of the left circumflex artery in patients who have left-dominant coronary circulation.[11,12] Although less common, occlusion of the left anterior descending artery may also result in infarction of the anterior right ventricle.[12]

The incidence of right ventricular infarction in association with left ventricular myocardial infarction ranges from 14 to 84 percent, depending on the population studied and the pathologic criteria.[12-16] In early studies right ventricular infarction occurred exclusively in patients with concurrent transmural posterior interventricular infarction, and there was no right ventricular involvement in patients with anterior myocardial infarctions. Subsequently, Cabin and colleagues[16] found a 13 percent incidence of right ventricular involvement in 97 patients with anterior myocardial infarction.

Isolated right ventricular infarction accounts for less than 3 percent of all cases of infarction but may result in considerable morbidity.[12,13,17,18] It is associated with atherosclerotic disease of the acute marginal vessels or of a nondominant right coronary artery,[12,17] but it may occur in the absence of coronary disease when substantial right ventricular hypertrophy is present.[19] The presence of right ventricular hypertrophy increases the susceptibility of the right ventricle to ischemia.[14,15,20,21]

Many inferior myocardial infarctions result from an occlusion of the right coronary artery proximal to the acute marginal coronary arteries, yet in half of such occlusions there is no evidence of right ventricular involvement. Several theories have been advanced to explain this discrepancy.[22] The right ventricle has a more favorable oxygen supply–demand ratio than the left ventricle, a difference that is attributed to a lower oxygen requirement resulting from its smaller muscle mass, as well as improved oxygen delivery due to the biphasic nature of coronary blood flow during both systole and diastole. In addition, the right ventricle receives more extensive collateral flow from the rich left-to-right collateral system. Whether the right ventricular myocardium is perfused directly from the right ventricular cavity by the thebesian veins is still the subject of debate.[22]

PATHOPHYSIOLOGY OF RIGHT VENTRICULAR INFARCTION

The severity of the hemodynamic derangements associated with right ventricular infarction is related not only to the extent of right ventricular ischemia and consequent right ventricular dysfunction, but also to the restraining effect of the pericardium and the resulting interaction between the ventricles. Experimentally induced infarction of the right ventricle in dogs with an intact pericardium results in acute right ventricular dilatation and elevation of intrapericardial pressure caused by pericardial constraint.[23,24] There is also a reduction in right ventricular systolic pressure, left ventricular end-diastolic size, cardiac output, and aortic pressure, as well as equalization of the right and left ventricular diastolic pressures. These hemodynamic abnormalities improve when the pericardium is incised.[23,25]

It was initially postulated that, as right ventricular systolic function diminishes, the pressure gradient between the right atrium and the left becomes the driving force for pulmonary perfusion.[5] This mechanism seemed consistent with the clinical observations

From the Evans Memorial Department of Clinical Research, Section of Cardiology, Department of Medicine, Boston University Medical Center, Boston. Address reprint requests to Dr. Ryan at the Section of Cardiology, Boston University Medical Center, 88 E. Newton St., Boston, MA 02118.

that the ischemic right ventricle is particularly sensitive to alterations in both preload and afterload.[8,26-28] More recently, Goldstein and colleagues[29] have demonstrated the importance of an active right ventricular systolic pressure wave caused by left ventricular septal contraction that bulges in piston-like fashion into the right ventricle, generating systolic force sufficient for pulmonary perfusion. The loss of this compensatory mechanism with concomitant left ventricular infarction, particularly when the interventricular septum is involved, may result in further hemodynamic deterioration in patients with right ventricular infarction.[30] Augmented atrial contractility is necessary to overcome the increased myocardial stiffness associated with right ventricular infarction.[29] Factors that impair filling of the noncompliant right ventricle, such as intravascular volume depletion due to the use of diuretics and nitrates or any diminution in atrial function caused by concomitant atrial infarction or the loss of atrioventricular synchrony, are likely to have profoundly adverse effects on hemodynamics in patients with large right ventricular infarctions.[25,27,29] Interventricular forces, left ventricular function, and pericardial constraining factors are key determinants of the pathophysiology of right ventricular infarction.

Diagnosis

Because of the pathophysiology of right ventricular infarction, its management differs substantially from the routine management of left ventricular infarction. Early, accurate diagnosis is imperative. Since hemodynamically important right ventricular infarction typically occurs in patients with an acute inferior myocardial infarction, suspicion is warranted in any patient presenting with such an infarction.

The Physical Examination

The clinical triad of hypotension, clear lung fields, and elevated jugular venous pressure in a patient with an inferior infarction is virtually pathognomonic for right ventricular infarction. Although quite specific, this triad has a sensitivity of less than 25 percent.[31] Distended neck veins alone have been shown to be 88 percent sensitive and 69 percent specific for right ventricular infarction.[31] Caution must be exercised in relying on such findings, since they are readily masked by volume depletion and because the physical and hemodynamic signs of right ventricular infarction often emerge only after volume loading.[32] Kussmaul's venous sign (distention of the jugular vein on inspiration), a feature of constrictive pericarditis, has also been shown to be highly sensitive and specific for right ventricular infarction.[32,33] Other physical findings may include right ventricular gallops, tricuspid regurgitation, and atrioventricular dissociation.

Hemodynamic Measurements

Cohn and colleagues described the hemodynamic alterations associated with substantial right ventricular infarction[5]; in 6 of 78 patients with inferior infarctions, they found that the right atrial pressure was significantly elevated and exceeded the pulmonary-capillary wedge pressure. Lopez-Sendon and coworkers[34] reported that a right atrial pressure greater than 10 mm Hg and within 1 to 5 mm Hg of the pulmonary-capillary wedge pressure had a sensitivity of 73 percent and a specificity of 100 percent in identifying right ventricular infarction in 60 patients with acute myocardial infarction. These investigators also reported that a pattern of severe noncompliance in the right atrial wave form (defined as a y descent greater than the x descent) was indicative of right ventricular infarction.[34] In subsequent studies Goldstein and colleagues related right atrial wave-form components to mechanical events rather than to electrocardiographic criteria and demonstrated that the predominant atrial descent is systolic (x descent) and the diastolic (y) descent is blunted, reflecting an increased resistance to diastolic filling.[29]

The right ventricular pressure tracing has been described as broad and often bifid in right ventricular infarction, reflecting a depressed upstroke and delayed relaxation, with associated septal contraction contributing to the bifid wave form.[29] A "dip and plateau" pattern in the diastolic pressure curve has been widely observed and reflects decreased compliance as well as pericardial constraining forces.[29,35-37]

Electrocardiography

The electrocardiogram was generally believed to be unhelpful in identifying right ventricular infarctions until Erhardt and coworkers described the value of a right precordial lead (CR_{4R}) in patients with autopsy-proved right ventricular infarction.[38] A 1-mm ST-segment elevation in this lead was 70 percent sensitive and 100 percent specific for right ventricular infarction. Other investigators have used ST-segment elevations or the presence of Q waves in various right precordial leads to diagnose the condition (Table 1).[38-50] Right bundle-branch block and complete heart block are the most frequent conduction abnormalities associated with right ventricular infarction.[51] ST-segment elevation in lead V_{4R} remains the most predictive electrocardiographic finding for right ventricular infarction (Fig. 1). It is important to recognize the transient nature of ST-segment elevation. In one series, 48 percent of the patients had resolution of electrocardiographic changes within 10 hours of the onset of symptoms.[42] Thus, it is imperative to record the electrocardiogram through the accessory right precordial leads as early as possible.

Echocardiography

Echocardiography has become a useful tool in the diagnosis of right ventricular infarction. The initial M-mode studies demonstrated right ventricular dilatation and an increased ratio of right ventricular to left ventricular end-diastolic dimensions in patients with right ventricular infarction.[52] The introduction of two-dimensional echocardiography has allowed a better quantitative assessment. Abnormal findings include right ventricular dilatation, right ventricular wall asynergy, and abnormal interventricular septal mo-

Table 1. Electrocardiographic Findings in Patients with Right Ventricular Infarction.*

Study	Date	No. of Patients	Finding			Gold Standard	Sensitivity (%)	Specificity (%)	Positive Predictive Value (%)
			Qualitative	Quantitative	Lead				
Erhardt[39]	1974	21	ST-segment elevation	1.0 mm	CR_{4R}†	Postmortem examination	83	89	91
Erhardt et al.[38]	1976	18	ST-segment elevation	1.0 mm	CR_{4R}	Postmortem examination	70	100	100
Candell-Riera et al.[40]	1981	42	ST-segment elevation	0.1 mV	V_{4R}	Hemodynamic measures or TPS	93 83	68 71	59 68
Croft et al.[41]	1982	33	ST-segment elevation	0.1 mV	V_{4R}–V_{6R} V_{4R} V_2 V_1	TPS or RVG	90 70 10 20	91 91 87 91	82 78 25 40
Braat et al.[42]	1983	67	ST-segment elevation	1.0 mm	V_{3R} V_{4R} V_{5R} V_{6R}	TPS	69 93 90 83	97 95 92 92	95 93 90 89
Klein et al.[43]	1983	110	ST-segment elevation	0.5 mm	V_{4R}	Postmortem examination or RVG + echocardiography, TPS, or hemodynamic measures	83	77	70
Braat et al.[44]	1984	42	ST-segment elevation	1.0 mm	V_{4R}	Angiography‡	100	87	92
Morgera et al.[45]	1984	28	ST-segment elevation ST-segment elevation Q waves	0.5 mm 1.0 mm	V_{4R} V_{4R} V_{4R} V_{4R}–V_{3R}	Postmortem examination	76 57 94 78	86 100 88 100	94 100 100 94
Lopez-Sendon et al.[46]	1985	43	ST-segment elevation	0.5 mm	V_{4R} V_{3R} V_1 V_2	Postmortem examination	100 79 43 36	68 68 46 27	67 — — —
			Q waves		V_{4R}		79	40	—
Lew et al.[47]	1986	68	ST-segment depression (V_2) ST-segment elevation (aVF)	50%	V_2 and aVF	Angiography	79	91	82
Andersen et al.[48]	1989	24	ST-segment elevation	1.0 mm	III>II§	Postmortem examination	63	88	91
Zehender et al.[49]	1993	200	ST-segment elevation	1.0 mm	V_{4R}	Postmortem examination, TPS, angiography, or hemodynamic measures	88	78	79

*Reprinted, with modifications, from Isner.[50] TPS denotes technetium-99m pyrophosphate scintigraphy, and RVG radionuclide ventriculography.

†Lead is at fifth intercostal space, midclavicular line.

‡Occlusion of right coronary artery proximal to the free wall branch.

§ST-segment elevation in lead III exceeds elevation in lead II.

tion caused by a reversal of the transseptal pressure gradient due to the increased right ventricular end-diastolic pressure (Fig. 2).[53-55]

The short-axis view has been shown to have the highest sensitivity (82 percent), with a specificity ranging from 62 percent to 93 percent for hemodynamically important right ventricular infarction.[56,57] Interatrial septal bowing toward the left atrium, indicative of an increased right atrial–left atrial pressure gradient, is an important prognostic marker in right ventricular infarction. Patients with this finding have more hypotension, more heart block, and higher mortality than patients without it.[58] Doppler echocardiography is particularly helpful in detecting such complications of right ventricular infarction as tricuspid regurgitation and ventricular septal defect. Doppler echocardiography[59] can also detect premature opening of the pulmonic valve, which indicates a noncompliant right ventricle.

Nuclear Imaging

Radionuclide ventriculography is considered the gold standard for estimating the right ventricular ejection fraction and is also useful in detecting wall-motion abnormalities. Abnormal right ventricular function in patients with inferior myocardial infarction has been demonstrated by both first-pass and gated blood-pool scanning,[60,61] but because of the wide range of normal values reported for the right ventricular ejection fraction (35 to 75 percent), a reduction in this measure alone is not particularly specific for right ventricular infarction.[62] Dell'Italia and coworkers demonstrated that detection by radionuclide ventriculography of a low right ventricular ejection fraction in the presence of wall-motion abnormalities had a 92 percent sensitivity and an 82 percent specificity for identifying hemodynamically important right ventricular infarction.[32] They also showed that technetium-99m pyrophosphate scintigraphy, though it had a high specificity (94 percent), was only 25 percent sensitive.

Complications

Shock, though uncommon in patients with right ventricular infarction, is the most serious complication of this condition. A high degree of heart block or com-

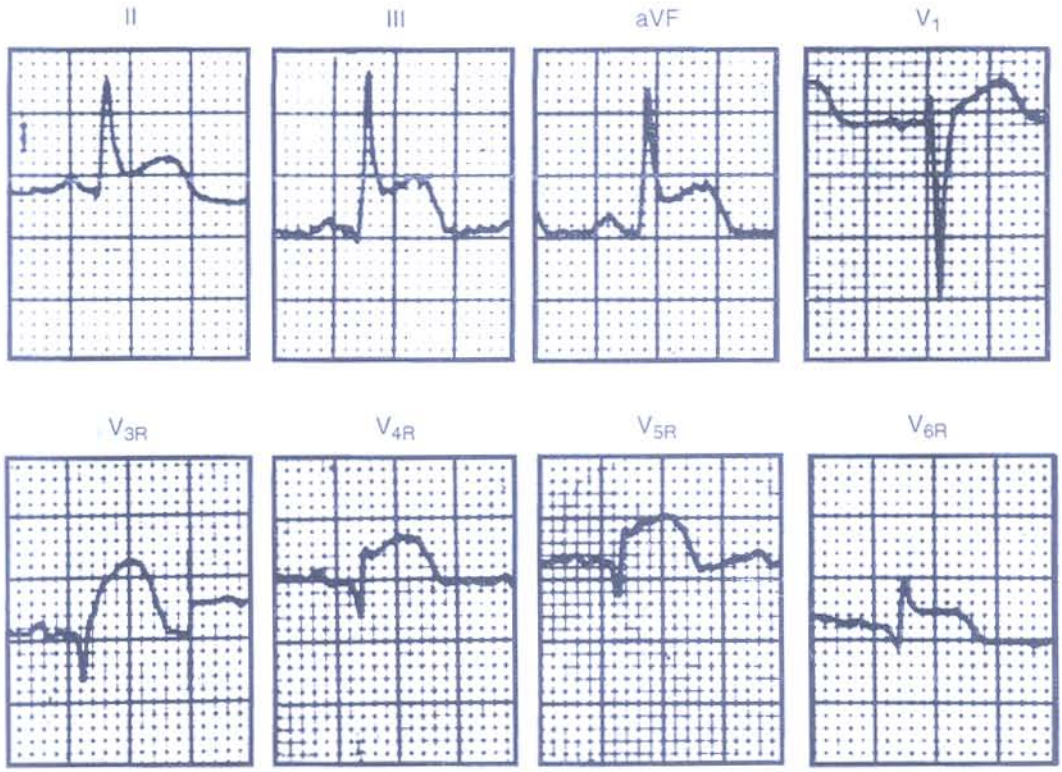

Figure 1. An Electrocardiogram from a Patient with an Inferior Myocardial Infarction and Right Ventricular Infarction.
ST-segment elevation is evident in leads II, III, and aVF, with the associated right ventricular myocardial infarction indicated by Q waves and ST-segment elevation in the right precordial leads (V_{3R} through V_{6R}).

plete block indicates a poor prognosis in patients with inferior myocardial infarction,[63] and mortality may be increased only among patients who also have right ventricular infarction.[64] The development of high-degree atrioventricular block has been reported to occur in as many as 48 percent of right ventricular infarctions.[65] Atrial fibrillation may occur in up to one third of patients with right ventricular infarction, presumably because of concomitant atrial infarction or right atrial dilatation.[66] Right ventricular infarction is not only associated with an increased incidence of ventricular arrhythmias but is also a risk factor for arrhythmias during right-heart catheterization.[49,67] Ventricular septal rupture may occur in patients with right ventricular infarction and transmural posterior septal infarction. Other complications of right ventricular infarction include right ventricular thrombus formation and subsequent pulmonary embolism, tricuspid regurgitation, and a high incidence of pericarditis, presumably due to the frequent transmural injury of the relatively thin-walled right ventricle.[13,68] A unique complication is the development of a right-to-left shunt through a patent foramen ovale,[69] which should be suspected in patients who have hypoxemia that is not responsive to the administration of oxygen.

Treatment

The strategy for the treatment of right ventricular infarction includes early maintenance of right ventricular preload, reduction of right ventricular afterload, inotropic support of the dysfunctional right ventricle, and early reperfusion (Table 2). The necessity of maintaining right ventricular preload differentiates the treatment of right ventricular infarction from that of predominantly left ventricular infarction. Because of their influence on preload, drugs routinely used in the management of left ventricular infarctions, such as nitrates and diuretics, may reduce cardiac output and produce severe hypotension when the right ventricle is ischemic. Volume loading with normal saline alone often resolves the accompanying hypotension and improves cardiac output.[25,35] In some cases, however, volume loading only further elevates the right-sided filling pressure, with no improvement in cardiac output.[70] The right ventricular dilatation may further compromise left ventricular output through pericardial restraining effects. Although volume loading is a critical first step in the management of hypotension associated with a right ventricular infarction, inotropic support (in particular, the use of dobutamine hydrochloride) should be initiated as well if the cardiac output fails to improve after several liters of fluid have been given.

Another important factor in sustaining adequate right ventricular preload is the maintenance of atrioventricular synchrony. Several investigators have shown that atrioventricular sequential pacing in patients with complete heart block leads to a significant increase in cardiac output and reversal of shock when ventricular pacing alone has no benefit.[71] When atrial fibrillation occurs, prompt cardioversion should

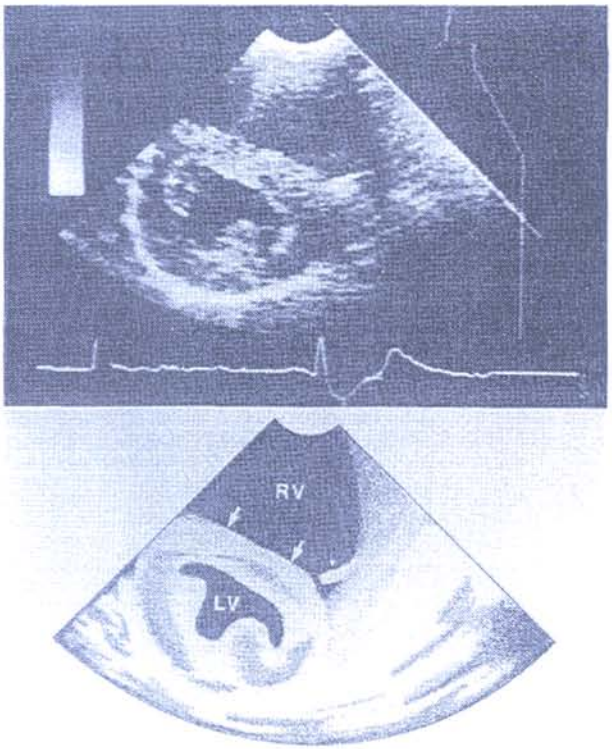

Figure 2. Echocardiogram (Short-Axis View) Obtained One Hour after Embolization of the Right Coronary Artery with Mercury in a Dog.

The image shows right ventricular enlargement and paradoxical interventricular septal displacement toward the left ventricle at end diastole as a result of the elevated right ventricular end-diastolic pressure (large arrows). The small arrow indicates the monitoring catheter. RV denotes right ventricle, and LV left ventricle. Reproduced from Sharkey et al.[54] with the permission of the publisher.

be considered in order to restore atrioventricular synchrony at the earliest signs of hemodynamic compromise. When left ventricular dysfunction accompanies right ventricular infarction, the right ventricle is further compromised because of the increased right ventricular afterload and reduction in stroke volume.[26,28] In such circumstances, the use of afterload-reducing agents such as sodium nitroprusside or an intraaortic counterpulsation device is often necessary to "unload" the left ventricle and subsequently the right ventricle.

A beneficial role of fibrinolytic therapy in right ventricular infarction has been suggested by studies in which patients with successful reperfusion had better right ventricular ejection fractions and less complete heart block than those in whom reperfusion was not successful.[72,73] In the second phase of the Thrombolysis in Myocardial Infarction trial, there was a reduction in the incidence of right ventricular infarction among patients with inferior myocardial infarctions who had patent infarct-related arteries, as compared with the rate in those whose arteries remained occluded; this finding suggests that successful thrombolysis may prevent right ventricular infarction.[74] Primary angioplasty has been used to treat right ventricular infarction, and in one report it resulted in rapid hemodynamic improvement.[75] The evidence suggests that reperfusion is beneficial in patients with right ventricular infarction; although this technique has not yet been shown to reduce mortality in such patients, it should be considered in the initial management of right ventricular infarction.

Prognosis

It is generally believed that patients with right ventricular infarction have a favorable prognosis. When inferior myocardial infarction is complicated by right ventricular infarction, however, the in-hospital mortality may be as high as 31 percent, as compared with 6 percent for patients with inferior myocardial infarction and no right ventricular involvement.[49] There are conflicting data on the long-term prognosis. Several studies have found that right ventricular dysfunction after a myocardial infarction is an independent risk factor for higher long-term mortality.[76,77] Others have not demonstrated any difference in long-term mortality between patients with and without right ventricular infarction,[49,74] suggesting that long-term outcome depends on the degree of concomitant left ventricular dysfunction.[78] In the vast majority of survivors of right ventricular infarction, manifestations of right ventricular dysfunction return to normal. Echocardiographic[56] and serial nuclear studies[32,43] demonstrate full resolution of a depressed right ventricular ejection fraction and wall-motion abnormalities. Clinical and hemodynamic recovery eventually occurs even in patients whose right ventricular function remains depressed for weeks or months. This return to normal may be due to the amelioration of concomitant left ventricular dysfunction, resulting in a reduction in right ventricular afterload, or to a gradual stretching of the pericardium with amelioration of its restraining effect.[28]

Summary

Right ventricular infarction complicates up to half of inferior left ventricular infarctions. The term represents a spectrum of disease from mild, asymptomatic right ventricular dysfunction to cardiogenic shock, and it includes transient ischemic myocardial dysfunction as well as myocardial necrosis. Right ventricular

Table 2. Treatment Strategy for Right Ventricular Infarction.

Maintenance of right ventricular preload
Volume loading (intravenous normal saline)
Avoidance of nitrates, diuretics, and morphine sulfate
Maintenance of atrioventricular synchrony
Atrioventricular sequential pacing for complete heart block
Prompt cardioversion for atrial fibrillation
Inotropic support
Dobutamine hydrochloride (if cardiac output does not respond to volume loading)
Reduction of right ventricular afterload (in the presence of left ventricular dysfunction)
Intraaortic balloon counterpulsation device
Vasodilators (sodium nitroprusside)
Reperfusion
Thrombolytic agents
Direct angioplasty

infarction is associated with considerable morbidity and mortality, and its presence defines a high-risk subgroup of patients with inferior left ventricular infarction. Diagnosis of this condition requires a high degree of suspicion based on clinical findings and the early recording of the electrocardiogram through right precordial leads, as well as elevated right-sided filling pressures out of proportion to left-sided filling pressures. The proper management of right ventricular infarction requires sustaining adequate right ventricular preload with volume loading and maintenance of atrioventricular synchrony, reduction of right ventricular afterload (particularly when left ventricular dysfunction is present), and inotropic support of the right ventricle. Early reperfusion with fibrinolytic therapy or direct angioplasty is also warranted. Survivors of right ventricular infarction generally have a restoration of normal right ventricular function with resolution of hemodynamic abnormalities.

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