RIGHT ventricular infarction is common in inferior or infero-posterior wall myocardial infarction, it is rarely encountered in patients with anterior wall myocardial infarction. Isolated right ventricular myocardial infarction, in the absence of left ventricular myocardial infarction, is rare and the incidence is only 3% to 4% in the autopsy studies. However, the frequency of right ventricular myocardial infarction, in association with left ventricular infarction, is more frequent and the incidence in the post-mortem hearts of patients dying of acute myocardial infarction ranges from 14% to 44%. In patients with inferior-posterior wall myocardial infarction, the involvement of the free wall of the right ventricle can be documented in up to 40% of patients. The anatomic severity of right ventricular myocardial infarction is traditionally graded into four types. In Type I, less than 50% of the inferior wall of the right ventricle is involved, in addition to the inferior wall of the left ventricle and the posterior-inferior part of the interventricular septum. In Type II right ventricular myocardial infarction, more than 50% of the right ventricular inferior wall is involved. In Type III, in addition to the inferior wall of the right ventricle, less than 50% of the anterior wall of the right ventricle is involved. In type IV, there is extensive necrosis of the free wall of the right ventricle in addition to its inferior wall. The hemodynamic abnormalities or low-output state are likely to occur in Type III and Type IV right ventricular myocardial infarction. Types I and II right ventricular myocardial infarction may occur from occlusion of the distal right coronary artery or posterior descending coronary artery or from occlusion of the distal portion of the left circumflex coronary artery. Types III and IV right ventricular myocardial infarction, however, almost always occur due to total occlusion of the proximal portion of the right coronary artery and occlusion is usually located proximal to the right ventricular branches.

Diagnosis

The clinical diagnosis (Table 1) of right ventricular myocardial infarction depends on the awareness of the fact that right ventricular myocardial infarction is frequent in patients with:

1. Evidence for associated inferior or posterior myocardial infarction.
2. Evidence for right ventricular failure in patients with mild or no left ventricular failure.
4. Electrocardiogram: ST segment elevation in the lead V4R or leads VI-V3 in the presence of electrocardiographic evidence for evolving inferior wall myocardial infarction; ST segment depression in the leads VI-V2 less than 50% of ST segment elevation in leads AVF and I1.
5. Radionuclide scintigraphy: technetium-99m pyrophosphate uptake by the free walls of the right ventricle; reduced right ventricular ejection fraction (less than 40%) with regional wall motion abnormalities detected by radionuclide ventriculography.
6. Two-dimensional echocardiography: dilated poorly contracting right ventricle with regional wall motion abnormalities.
7. Hemodynamics: right atrial pressure equal to or greater than 10 mm Hg and right atrial to pulmonary capillary wedge pressure equal to or greater than 0.86.

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inferior-posterior wall myocardial infarction and rare in patients with anterior wall myocardial infarction. For clinical purposes, appropriate investigations should be performed in all patients with inferior wall myocardial infarction for the diagnosis of associated right ventricular myocardial infarction.

Right ventricular myocardial infarction may be clinically silent without any manifestations of right ventricular failure. Combinations of hypotension, elevated jugular venous pressure, and clear lung fields occur only in patients with severe right ventricular myocardial infarction.3 However, in all patients with suspected right ventricular myocardial infarction, it is important to search for the clinical features suggestive of right ventricular failure, that is, elevated jugular venous pressure and right ventricular S3 gallop. If these findings are detected in the absence of overt left ventricular failure, such as pulmonary edema and left ventricular S3 gallop, one should suspect the presence of right ventricular myocardial infarction. An obvious Kussmaul's sign, defined as lack of decrease or an actual increase in the jugular venous pressure during the inspiratory phase of respiration, appears to be highly specific for the diagnosis of right ventricular myocardial infarction.4 However, Kussmaul's sign may not be appreciated in many patients. The recording of the jugular venous pulse may demonstrate a Y descent greater than or equal to the X descent but such abnormal wave forms are rarely recognized at the bedside. Significant pulsus paradoxus is also a relatively uncommon finding in patients with right ventricular myocardial infarction. These findings, however, reflect the hemodynamic abnormalities of effusive constrictive pericarditis and suggest that the impairment in the ventricular filling characteristics of pericardial tamponade or constrictive pericarditis may occur. Tricuspid regurgitation in the absence of clinical evidence for significant pulmonary arterial hypertension also suggests the presence of significant right ventricular myocardial infarction.

The electrocardiogram is very useful for the diagnosis of acute right ventricular myocardial infarction.5-7 An ST segment elevation of more than 1 mm in lead VI and V4R in a patient with electrocardiographic evidence of inferior wall myocardial infarction is very suggestive of right ventricular myocardial infarction. When the magnitude of ST elevation in leads V4R is at least 0.5 mm, a sensitivity of 83% and a specificity of 77% have been reported for the diagnosis of right ventricular myocardial infarction. ST segment elevations may extend from V3R to V7R in some patients with extensive right ventricular myocardial necrosis. With marked right ventricular dilatation and its anterior displacement, ST segment elevations can be observed in the anterior precordial leads extending from leads VI to V5 or V6. These ST segment elevations in anterior precordial leads in patients with inferior wall myocardial infarction do not necessarily indicate additional anterior wall infarction. In right ventricular myocardial infarction, associated with inferior wall infarction, the magnitude of Q waves in leads III is usually greater than the magnitude of Q wave in lead A VF. When the magnitude of ST segment depression in leads VI and V2 is less than 50% of the magnitude of ST segment elevation in leads II, III and A VF, right ventricular dysfunction is frequently observed.

Myocardial scintigraphy with technetium-99m pyrophosphate is an accurate method for the diagnosis of acute right ventricular necrosis. The uptake of the radionuclide in the area of the right ventricle has been reported to occur in approximately 40% of patients with acute inferior wall myocardial infarction.2 However, technetium-99m pyrophosphate images are not usually before 24 hours of onset of myocardial infarction.

Radionuclide ventriculography and echocardiography are useful noninvasive investigations to assess right and left ventricular systolic function. In right ventricular myocardial infarction, right ventricular ejection fraction is usually less than 40% and there is also regional dyskinesia or akinesis.7-9 Two- dimensional echocardiography also provides useful information concerning the right ventricular functional derangements in patients with right ventricular myocardial infarction. Dilatation of the right ventricle with impaired systolic function and dyskinesia or akinesis of the right ventricular wall segments strongly suggests right ventricular myocardial infarction. Echocardiography also reveals wall motion abnormality of the inferior wall of the left ventricle. Echocardiography is also very useful
the differential diagnosis of cardiac tamponade and right ventricular myocardial infarction, as the altered hemodynamics in right ventricular myocardial infarction may be very similar to those in tamponade.10-12

Hemodynamic abnormalities in right ventricular myocardial infarction are variable and are related to the extent of right ventricular necrosis and severity of associated left ventricular dysfunction.13-16 The predominant right ventricular myocardial infarction causes a disproportionate elevation of right atrial pressure compared to the rise in pulmonary capillary wedge pressure, and frequently the ratio of right atrial to pulmonary capillary wedge pressure is equal to or greater than 86. If the right atrial pressure is equal to or greater than 10 mm Hg and if the ratio of right atrial to wedge pressure exceeds 86, the sensitivity and specificity of these hemodynamic abnormalities for the diagnosis of right ventricular myocardial infarction are 82% and 97%, respectively. This hemodynamic profile, however, is not universally present and it has been suggested that the volume expansion can unmask the hemodynamics of right ventricular myocardial infarction. In the presence of old or recent left ventricular myocardial infarction, the expected relation between right atrial and pulmonary capillary wedge pressure may not be observed and pulmonary capillary wedge pressure may remain significantly higher than the right atrial pressure, even in the presence of significant right ventricular myocardial infarction. With marked right ventricular dilatation following right ventricular myocardial infarction, the hemodynamic abnormalities may be similar to those of effusive constrictive pericarditis. The equalizations of right and left ventricular end diastolic pressures and a “dip in plateau” type of right ventricular diastolic pressure pulse may be observed. An increase in intrapericardial pressure and the constraining effect of the pericardium, appear to be the mechanism of hemodynamic abnormalities similar to those of effusive constrictive pericarditis.

Complications of Right Ventricular Myocardial Infarction

Bradyarrhythmias and low-output state are the two major complications of right ventricular infarction. The incidence of sinus bradycardia, second-degree and third-degree atrioventricular (A V) block, is significantly higher in right ventricular infarction. The incidence of complete A V block may be as high as 20% and may produce hypotension and low-output state. The loss of timed atrial contribution in ventricular filling appears to be the predominant mechanism of decreased stroke volume and cardiac output. The increase in heart rate by ventricular pacing alone does not substantially increase stroke volume or cardiac output. In fact, ventricular pacing may induce a further reduction in stroke volume and cardiac output associated with hypotension. Thus, in patients who develop A V block, A V sequential pacing is desirable to maintain adequate ventricular filling and to increase cardiac output.17

Although right ventricular necrosis is common in patients with inferior wall myocardial infarction, only about 10% of patients develop cardiogenic shock or severe low-output state. The mechanism of decreased cardiac output in right ventricular myocardial infarction is decreased left ventricular preload. In dogs with induced isolated right ventricular myocardial infarction, left ventricular transmural pressure (left ventricular diastolic pressure minus intrapericardial pressure) declines where right ventricular transmural pressure increases. Similarly, left ventricular diastolic volume decreases when right ventricular dimensions increase. It appears that a number of interacting mechanisms contribute to decreased left ventricular preload and hence decreased left ventricular forward stroke volume. A decrease in right ventricular stroke volume appears to be one of the major determinants for decreased left ventricular preload. Following isolated right ventricular infarction in dogs, decreased right ventricular stroke work index is associated with increased right ventricular transmural pressure and volume, suggesting a significant depression of pump function. The magnitude of reduction of right ventricular stroke volume is dependent on the residual right ventricular Sterling function. In isolated right ventricular myocardial infarction in dogs, volume expansion with intravenous fluid therapy can be associated with a substantial increase in right ventricular stroke volume and stroke work index when there is also an increase in right ventricular transmural pressure and diastolic
The improved right ventricular Sterling function is associated with increased right ventricular stroke volume and an increase in left ventricular preload as evident from increased left ventricular transmural pressure and left ventricular diastolic volume. It should be emphasized, however, that if right ventricular diastolic pressure is already significantly elevated, intravenous fluid does not increase right ventricular diastolic volume or its preload and therefore there may not be any improvement in right ventricular pump function. This lack of increase in ventricular volume during volume expansion is primarily due to the constraining effect of the pericardium. In addition to impaired contractile function of the right ventricle due to right ventricular myocardial necrosis, increase in right ventricular afterload and its resistance to ejection also contributes to impaired right ventricular pump function. Following right ventricular infarction, right ventricular endsystolic and end-diastolic volumes increase which causes an increase in wall stress. The wall stress is one of the major components of afterload. The higher the afterload, more impairment of its pump function will occur associated with a decrease in its stroke volume. In the presence of intact pericardium with right ventricular dilatation, there is an increase in intrapericardial pressure which is associated with a passive increase in left ventricular end-diastolic pressure. Thus, there is also an increase in left atrial and pulmonary venous pressure. An increase in pulmonary venous pressure is associated with an obligatory increase in pulmonary artery pressure which thus offers a greater resistance to right ventricular systolic ejection. Pulmonary hypertension, therefore, is another contributing factor for impaired right ventricular pump function and reduction in its stroke output.

Left ventricular filling may also be compromised due to increased intrapericardial pressure. In experimental isolated right ventricular infarction, the intrapericardial pressure increases as well as an increase in right ventricular and left ventricular diastolic pressures. The intrapericardial right ventricular and left ventricular diastolic pressures also become similar, that is, equalization of the diastolic pressures. The mechanism of equalization of the diastolic pressures is the increase in intrapericardial pressure because when the pericardium is removed, equalization of the diastolic pressures is no longer present. These findings suggest that relatively non-elastic pericardium exerts a constraining effect on ventricular filling and contributes to lower left ventricular preload. Acute right ventricular dilatation encroaches upon the relatively fixed intrapericardial volume, resulting in increased intrapericardial pressure. The orientation and function of the intraventricular septum is also an important determinant of the changes in left ventricular compliance and preload. When intrapericardial pressure increases due to the constraining effect of pericardium, the intraventricular septum shifts towards the left ventricle end-diastole which further decreases left ventricular preload. In experimental right ventricular infarction in dogs, the intraventricular septum is considerably flattened. The right ventricle is enlarged and the left ventricular cavity is decreased in size because of this septal shift. Impaired right ventricular systolic function decreases right ventricular stroke volume due to the loss of propelling effect of the intraventricular septum. Right atrial ischemia and tricuspid regurgitation also decreases right ventricular forward stroke volume. Right atrial ischemia is associated with ineffective right atrial contraction which, in turn, decreases right ventricular filling. In the presence of tricuspid regurgitation, right ventricular forward stroke volume decreases in proportion to the severity of tricuspid regurgitation. It is apparent that a number of interacting functional and hemodynamic abnormalities resulting from right ventricular myocardial infarction impair left ventricular filling and decrease systemic output.

**Therapeutic Approach**

The rational therapeutic approach should be directed to increase left ventricular preload to correct low output state. Enhanced left ventricular filling is expected with the removal of the constraining effect of the pericardium. In experimental right ventricular infarction in dogs, following the removal of the pericardium, the left ventricular stroke volume and cardiac output
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increases. However, pericardial decompression as a therapeutic approach to improve systemic output in patients with acute right ventricular myocardial infarction has not been confirmed in clinical studies and remains generally an impractical therapy. The other physiologic mechanisms that can potentially augment right ventricular stroke volume are (1) increased right ventricular preload or filling pressure by Frank Starling mechanisms; (2) decrease right ventricular afterload; (3) increased right ventricular contractile function; and (4) to improve right ventricular contractile function by relieving myocardial ischemia.

For the relief of right ventricular ischemic, early reperfusion therapy to recanalize the infract related artery should always be considered. In patients with hypotension, low cardiac output, and cardiogenic shock, primary angioplasty of the infract related artery is preferable to intravenous thrombolytic therapy. Without establishing adequate flow in these patients, recovery of right ventricular systolic function does not occur sufficiently to reverse the low-output state.

In experimental right ventricular myocardial infarction in dogs, volume loading with intravenous fluid administration improved right ventricular stroke work which was associated with an increased transmural pressure and diastolic volume. This improvement in right ventricular pump function by the Frank Starling mechanism was associated with an increase in left ventricular transmural pressure and volume and there was an increase in systemic output. In patients with right ventricular myocardial infarction, the response to volume expansion depends on the initial right ventricular and left ventricular filling pressures and the severity of right ventricular systolic dysfunction. When a marked right ventricular dilatation is present and when its transmural pressure is already significantly elevated (right atrial pressure exceeding 10 to 15 mm Hg), a further increase in right ventricular volume and its filling pressure during volume loading is not accompanied by any significant increase in stroke volume. In these circumstances, the right ventricle operates in the relatively flat portion of the function curve. Also during volume expansion in these patients, right or left ventricular volume usually does not increase and there is only an increase in right atrial and pulmonary capillary wedge pressure, suggesting a further reduction in left ventricular compliance. Thus, in patients with already elevated right atrial and pulmonary capillary wedge pressure, intravenous fluid therapy is not indicated. In some patients, intravenous fluid therapy is only likely to be effective to increase systemic output when the right atrial and pulmonary capillary wedge pressure are normal.

Several vasodilators have been used in an attempt to reduce pulmonary artery pressure to decrease resistance to right ventricular ejection. Initially, sodium nitroprusside was found to be effective in improving systemic output in some patients with right ventricular infarction. However, vasodilator therapy may cause further hypotension. Also, the magnitude of improvement in cardiac output and right ventricular ejection fraction with nitroprusside therapy is much less compared to that with inotropic agents such as dobutamine. During vasodilator therapy with nitroprusside or nitroglycerin, there is also a reduction in right and left ventricular preload which may further compromise systemic output. During vasodilator therapy, intravenous fluid therapy is frequently necessary to maintain adequate right and left ventricular preload. It needs to be recognized that vasodilators, like nitroprusside and nitroglycerin, not only decrease venous return by venous pulling but also decrease systemic vascular resistance. Thus, if systemic output does not increase appropriately, severe and unacceptable hypotension may occur.

Positive inotropic agents such as dobutamine and dopamine have been found be effective in improving right ventricular ejection fraction and its stroke volume and increases left ventricular preload. Dobutamine, a predominantly beta, adrenergic receptor agonist, causes a substantial increase in right ventricular ejection fraction and systemic output. Dopamine, a dopaminergic receptor agonist, along with beta, and alpha agonist property, also improves cardiac output and systemic hemodynamics in patients with right ventricular myocardial infarction. However, dopamine can potentially increase pulmonary artery pressure and therefore right ventricular ejection resistance which may curtail the
magnitude on increase in right ventricular stroke volume due to its inotropic effect. It needs to be emphasized that beta adrenergic agonists are likely to increase myocardial oxygen consumption and therefore the potential exists for enhancing myocardial ischemia. Also, excessive tachycardia may result which may compromise right and left ventricular filling. Recently, intravenous phosphodiesterase inhibitors, such as amrinone and milrinone, have been used to improve right ventricular systolic function and to decrease pulmonary artery pressure. It has been shown that with phosphodiesterase inhibitors, right ventricular ejection fraction can increase along with an increase in right ventricular stroke volume. There is also a suggestion to indicate that left ventricular diastolic function may also improve with phosphodiesterase inhibitors. It should be emphasized, however, that phosphodiesterase inhibitors can also produce systemic vasodilatation and decrease systemic vascular resistance. Thus, if there is no proportional increase in cardiac output, one can expect hypotension with phosphodiesterase inhibitors. In some patients, a combination of dobutamine and phosphodiesterase inhibitors may be necessary to maximize the increase in cardiac output. It is clear that in patients with low output state and hypotension following right ventricular myocardial infarction, hemodynamic monitoring is essential to assess the response to therapy. The therapy should be individualized and also changed if needed, depending on the response to a specific therapy. The guidelines for management of low output state in patients with right ventricular myocardial infarction are summarized in Table 2.

**Conclusion**

Right ventricular infarction is common in patients with inferoposterior myocardial infarction. Diagnosis can be made in most patients by bedside examination and noninvasive investigations. A V sequential pacing is recommended in patients with A V block. Volume loading is usually not effective in increasing cardiac output in patients with elevated ventricular pressure. Inotropic agents appear to be more effective in these circumstances.

**References**


