# Right Ventricular Dysfunction After Thrombolysis in Patients with Right Ventricular Infarction

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*Background:* **Right ventricular (RV) infarction is frequently associated with highest risk of death and major complications. Doppler echocardiography can be useful in the diagnosis of RV involvement. The goal of this study was to evaluate Doppler echocardiography features associated with RV involvement and a poor prognosis.**

*Methods:* **Two-dimensional Doppler echocardiography was performed before and after thrombolysis in 108 consecutive patients with an RV infarction. The bedside examination was performed before and 2 to 3 hours after thrombolytic therapy, and repeated after 1 and 7 days. All patients underwent coronary angiography after 20 days, and the perfusion of the coronary-related artery (> thrombolysis in myocardial infarction [TIMI] 3 grade) was evaluated.**

*Results:* **Patients were divided into 2 groups according to the recovery of global and regional RV function after thrombolytic therapy. In the group of patients who showed a normalization or improvement of RV wall motion (as assessed by RV wall motion score index), we found a TIMI grade III perfusion in 78% of patients. The analysis of interatrial septal motion and interventricular septal motion showed a normalization in all reperfused patients. Major complication and deaths were more frequent in patients with echocardiographic findings of RV dysfunction persisting after thrombolytic therapy.**

*Conclusion:* **In patients with RV infarction treated with thrombolysis, persistent RV dysfunction is associated with a higher risk for the development of major cardiac complications and death.** (J Am Soc Echocardiogr 2000;13:655-60.)

## INTRODUCTION

Right ventricular (RV) infarction complicates up to half of inferior left ventricular (LV) infarctions.[1] It is associated with a high risk of in-hospital death and major complications.[2,3] The management and prognosis of RV infarction differs substantially from LV infarction.[4] The depressed RV function induces RV failure and low cardiac output.[5,6] Right ventricular infarction results from the occlusion of the right coronary artery proximal to the marginal branches.[7] The effect of reperfusion in RV infarction is somewhat controversial. Some authors have reported the recovery of RV function only after successful reperfusion,[8,9] whereas other authors observed improvement even in the absence of reperfusion.[10,11]

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The goal of our study was to evaluate the effect of thrombolytic therapy and of reperfusion on RV dysfunction evaluated by Doppler echocardiography.

## METHODS

### Patients

Included in this prospective study were 108 consecutive patients with RV infarction who were admitted to the intensive coronary care unit of our hospital from January 1994 to January 1998. The initial diagnosis of RV infarction was based on typical chest pain lasting more than 30 minutes and values greater than 2 times normal in aspartate transamine (30 U/I) and creatine phosphokinase (170 U/l) activity within 24 hours after admission. Electrocardiographic changes of ST-segment elevation of > 0.1 mV and/or Q waves in V4-V6R leads were also required.

### Study Protocol

In all patients, a standard 12-lead electrocardiogram and right precordial leads (V3-V6R) were recorded within 10

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**Table 1** Clinical characteristics of subjects

| Clinical characteristics | Right ventricular infarction |
|---|---|
| Number of patients | 108 |
| Mean age (y) | 65 ± 9 |
| Men / women | 71 / 37 |
| Heart rate (bpm) | 66 ± 12 |
| Systolic blood pressure (mm Hg) | 104 ± 19 |
| Previous myocardial infarction | 3 |
| Hypertension | 20 |
| Diabetes | 29 |

**Table 2** Imaging and Doppler echocardiography findings in subjects before thrombolytic therapy

| Echocardiographic data | Group A | Group B | *P* |
|---|---|---|---|
| RV end-diastolic diameter (cm) | 2.9 ± 3.4 | 3.0 ± 3.6 | NS |
| LV end-diastolic diameter (cm) | 4.1 ± 3.2 | 4.1 ± 2.8 | NS |
| RV wall motion score | 3.0 ± 0.7 | 3.2 ± 0.7 | NS |
| Mean RAP | 16.8 ± 2.5 | 19.2 ± 4.1 | NS |
| TR flow velocity ($m/s^{-1}$) | 2.9 ± 0.3 | 3.0 ± 0.7 | NS |

*RV,* Right ventricular; *NS,* not significant; *LV,* left ventricular; *TR,* tricuspid regurgitation; *RAP,* right atrial pressure.

hours from the onset of symptoms. ST-segment deviations were assessed 0.04 seconds after the J point in all 16 leads. An RV infarction was diagnosed if there were clinical, electrocardiographic, hemodynamic, and radiologic criteria. The hemodynamic alterations that were considered highly indicative of an RV infarction included right atrial pressure that was significantly elevated and exceeded the pulmonary-capillary wedge pressure.[6] Right atrial pressure was measured by catheter in 60 patients and was calculated from echocardiography in all patients.

### Coronary Angiography

The coronary angiography was considered indicative of an RV infarction if there was an occlusion of the right coronary artery proximal to the acute marginal branches.[12] The severity of stenosis and the extent of flow were determined and graded according to the thrombolysis in myocardial infarction (TIMI) classification.[13] Successful reperfusion was defined as a residual stenosis less than 50% and restoration of TIMI grade 3 flow in the main right coronary artery, its left ventricular branches, and all major RV branches.

### Doppler Echocardiography

On admission, an echocardiogram was obtained for all patients before and 2 to 3 hours after thrombolytic therapy was started. Echocardiograms were repeated after 1 day and 1 month. Echocardiographic and Doppler examinations were performed with a Hewlett-Packard 1500 (Andover, Mass) echocardiographic system with a 2.5-MHz transducer. The following parameters were recorded.

- Right and left ventricular end-diastolic diameters were recorded in the supine position with the same M-mode cross section in the parasternal long-axis view. Normal values were defined as less than 25 mm for the RV diameter, greater than 37 mm (maximal value 54 mm) for the LV diameter and an RV diameter of less than half the LV diameter.[14,15]
- Right ventricular function was assessed with the ejection fraction and the wall motion score. The RV free wall was divided into 3 segments, and the motion of each segment was scored on a scale of 1 to 4 (1, normal; 2, hypokinetic; 3, akinetic; 4 dyskinetic).[16]
- Interventricular septal motion was analyzed from the parasternal long-axis and apical 4-chamber views and was considered abnormal if there was paradoxical wall motion of the septum.
- Interatrial septal motion was analyzed from the subcostal and apical 4-chamber views and was considered abnormal if a bowing toward the left atrium was recorded.[17]
- From the hepatic vein flow velocity, the following parameters were measured: peak velocity and time-velocity integral of the systolic, diastolic, and atrial reversal waves. The duration of the atrial reversal wave was measured from the beginning to the end of the atrial reversal wave. The inspiratory change was measured from the 2-dimensional subcostal view, from which a percent collapse index was obtained. The inspiratory change, commonly referred to as a sniff test, was used frequently to estimate right atrial pressure.[18]
- Peak velocity of tricuspid regurgitant flow was recorded from parasternal short-axis and apical 4-chamber views.[19]

### Prognostic Evaluation

All patients were followed for 1 year. Complications occurring during the in-hospital and follow-up periods were determined for all patients. The major in-hospital complications evaluated were hypotension (systolic blood pressure < 100 mm Hg), cardiogenic shock (systolic blood pressure < 90 mm Hg for 30 minutes), second- and third-degree atrioventricular block, and death. During follow-up, patients were evaluated every month by phone contact and every 2 months by a clinical visit.

### Statistical Analysis

Data are expressed as mean ± SD. Comparisons were made with use of the chi-square test for categorical variables and a 2-tailed student *t* test for continuous variables. Analysis of variance was used to compare serial echocardiographic data. A probability (*P*) value of < .05 was considered significant.


Figure 1 Apical 4-chamber view of a patient with a right ventricular infarction. The right side of the heart is dilated; tricuspid regurgitation was moderate.

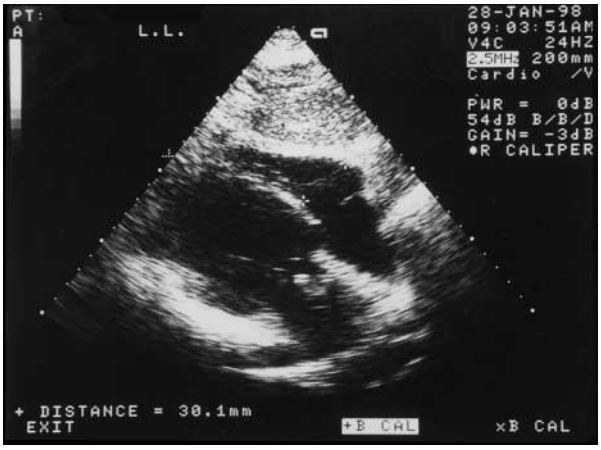


Figure 2 Subcostal 4-chamber view of a patient with right ventricular infarction after 1 month. The right ventricle is slightly dilated; the right ventricular ejection fraction and the right ventricular score are improved compared with the acute phase.

## RESULTS

Clinical data of the patients are shown in Table 1. The patients arrived at the hospital a mean of 1.6 ± 3.5 hours after the onset of symptoms.

### Echocardiographic Features Before Thrombolysis

Before thrombolysis, all patients had an increase in the RV end-diastolic diameter (Figure 1). Left ventricular end-diastolic diameter was within normal range in all patients except 3 who had a previous myocardial anterior infarction (Table 2). In all patients, analysis of the right regional wall motion showed severe RV dysfunction, with a mean score of 3.2 ± 0.7 with paradoxical septal motion. An inverted interatrial septal convexity was observed in all patients (Figure 2).

Nighty-four patients (87%) had a tricuspid regurgitation detectable by Doppler. In 70 of these patients, flow velocity was increased, compatible with mild to moderate elevation of RV systolic pressure (regurgitant jet > 2.5 m/s). The maximal pressure gradient was 65.8 ± 27.2, but most patients had an increased mean right atrial pressure (Table 2).

### Echocardiographic Variables After Thrombolysis

Marked changes in echocardiographic variables were seen after thrombolytic therapy in a group of 83 patients (group A). The RV diameter decreased significantly and RV function recovered promptly. As RV function recovered, LV function showed a concomitant improvement (Figure 3). Interventricular septal motion normalized in all patients of Group A. The shape and convexity of the interatrial septum normalized in all patients of Group A, suggesting a decrease in mean right atrial pressure that was confirmed by calculations (from 16.8 ± 2.5 to 8.3 ± 3.4; $P < .01$). After 1 month, RV dimension and RV wall motion were normal in 98% of patients in group A. On the contrary, a group of 25 patients showed a persistence of echocardiographic signs of RV dysfunction after thrombolytic therapy (group B). These patients showed a persistent, severe RV dysfunction at 24 hours (RV size from 30 ± 3.6 cm to 29 ± 3.3 cm; $P$ = not significant, and RV wall-motion score from 3.2 + 0.7 to 2.9 + 0.4) (Figure 3). The tricuspid regurgitant flow persisted after thrombolytic therapy in all patients, and mean right atrial pressure remained elevated. After 1 month, 78% of patients of group B had a recovery of RV function with a reduction of RV size and an increase in wall motion score.

### Angiographic Findings

Coronary angiography was performed in all patients after 24 ± 5 days from the onset of acute myocardial infarction (range 7 to 35 days). Single-vessel disease was documented in 67 patients, 2-vessel disease in 37, and 3-vessel disease in 4. The right coronary artery was involved in all cases. Reperfusion of the right main coronary artery and its LV branches was observed in 80 patients in group A and 5 patients in group B ($P < .01$).

### Prognostic Impact of Right Ventricular Infarction

The prevalence of major complications and the death rates during the in-hospital phase of the study

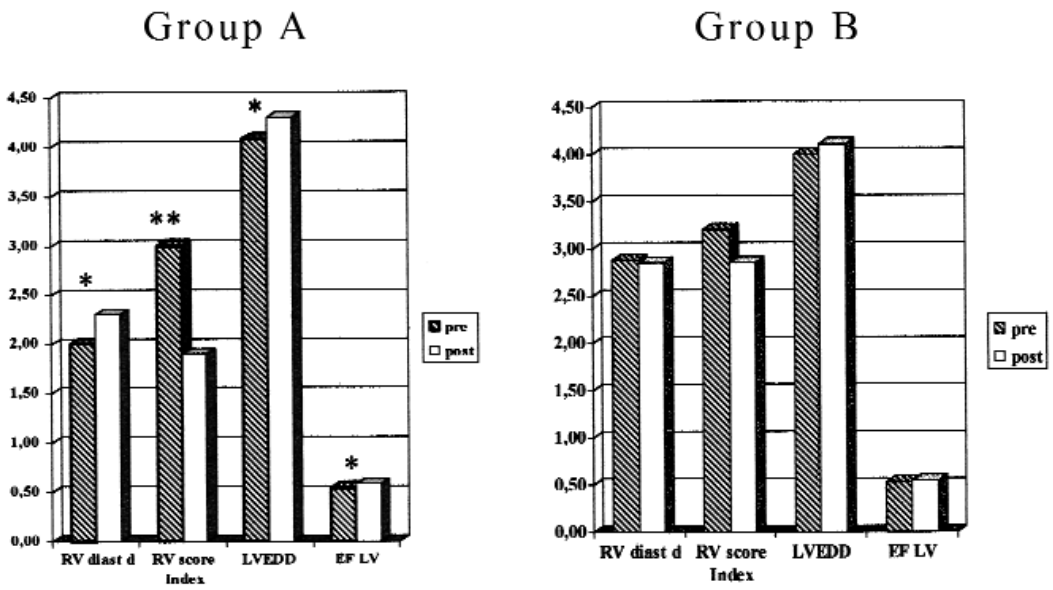


Figure 3 Bar graph showing different parameters before and after thrombolysis in the 2 groups of patients. *Group A,* Patients who showed a normalization or improvement of RV wall motion (as assessed by RV wall motion score index); *group B,* patients with echocardiographic findings of RV dysfunction persisting after thrombolytic therapy; *RV,* right ventricular; *LVEDD,* left ventricular end-diastolic diameter; *EF LV,* left ventricular ejection fraction.

and after discharge from the hospital were as follows. Hypotension occurred in 32 (29%) of 108 patients with RV infarction.The incidence of second- and third-degree atrioventricular block was 58 (54%) out of 108 patients.

Adverse events were rare in the patients from group A.The overall in-hospital mortality rate of the 108 patients with RV myocardial infarction was 12%. Of the 13 patients who died, 9 were from group B and had occlusion of the right coronary artery ($P < .01$). Seventy-eight (94%) patients of group A survived and were discharged from the hospital. At 1 month, all were alive; at 6 months, 68 patients were alive. The mortality (9 [36%] of 25 patients] and incidence of second- or third-degree AV block (18 [72%] of 25 patients] were higher in patients of group B. Of the 14 patients of group B who were discharged, 1 died within 1 month, and 3 more died before 6 months.

## DISCUSSION

We report a significant early reversal of RV dilatation and dysfunction in 76.8% of patients with RV infarction treated with thrombolysis who were successfully reperfused.

Many recent reports stress that the occlusion of the right coronary artery proximal to the major right branches results in RV dysfunction.[12,13,20] The majority of these studies focused their attention on the reperfusion of the LV branches of the right coronary artery, ignoring the affect on RV branches. In a recent report, Bowers et al[16] suggested that complete reperfusion led to a striking immediate improvement of RV function followed by a complete recovery. In a previous article, we evaluated the effect of thrombolysis on right atrial and ventricular pressures with the use of Doppler echocardiography in patients with RV infarction.[14] A group of patients developed a rapid restoration of RV function and the reduction of pulmonary pressures.

In the present study, we evaluate the relation between reperfusion as assessed with coronary angiography and the recovery of RV function as assessed by echocardiography. The echocardiographic changes seems to be related to the decrease in pulmonary artery pressure, which suggests that this improvement in RV function is related to the success of reperfusion therapy. The pathophysiology of pulmonary hypertension remains unclear.The hypokinetic right ventricle should not be able to generate hypertension. We suppose that increased pulmonary artery pressure depends on elevated right atrial pressure. In patients with a recovery of RV function, right atrial pressure drops and the pulmonary pressure decreases independently from tri-

cuspid regurgitation velocity. On the contrary, patients with persistent RV dysfunction had an elevated mean right arterial pressure that influenced the pulmonary pressure. Patients who had persistent RV dysfunction had higher prevalence of right coronary occlusion. This group had higher rates of mortality and complication during the acute phase of myocardial infarction. At baseline, the RV dysfunction was similar in patients who were later successfully or unsuccessfully reperfused, but the clinical outcome was better in patients who were successfully reperfused and promptly recovered right atrial function. Similarly, the LV function was intact in both group of patients, suggesting that the negative outcome depended mostly on RV dysfunction. As compared with the left ventricle, the right ventricle is protected from ischemia because it requires less oxygen because of its small muscular mass and workload. Most of the oxygen is provided by extensive collateral vessels in addition to the direct diffusion of oxygen from the ventricular cavity.[1,21] These factors may limit the size of the RV infarct but do not prevent the hemodynamic failure that results from RV involvement. Reperfusion induced a prompt recovery of ventricular function, a reduction of RV size, an increase in the RV wall motion score index, and a reduction of the tricuspid regurgitant flow, reflecting a reduction in pulmonary pressures.

### Limitation of the Study

Our conclusions are based on the evaluation of coronary angiography performed after thrombolysis. The diagnosis was made with the use of clinical, electrocardiographic, and echocardiographic features. For a better evaluation, angiography should be performed before and after thrombolysis. Bowers and coworkers[16] evaluated reperfusion after percutaneous transluminal coronary angioplasty in their control study.

### Conclusions

In this study, we related the echocardiographic evolution of RV infarction and the angiographic aspects. The persistence of echocardiographic findings of RV dysfunction and abnormal motion of the interventricular and interatrial septum is related to the prognosis of patients with RV infarction. The mortality rate and the incidence of major complications during the in-hospital phase of the study and after discharge were higher in patients with RV dysfunction after thrombolytic therapy who did not experience reperfusion of the related artery.

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