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# Cardiogenic Shock Caused by Right Ventricular Infarction

## A Report From the SHOCK Registry

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<b>OBJECTIVES</b>	The purpose of this study was to determine the characteristics and outcomes of patients with acute myocardial infarction (MI) complicated by cardiogenic shock due to predominant right ventricular (RV) infarction.
<b>BACKGROUND</b>	Although RV infarction has been shown to have favorable long-term outcomes, the influence of RV infarction on mortality in cardiogenic shock is unknown.
<b>METHODS</b>	We evaluated 933 patients in cardiogenic shock due to predominant RV (n = 49) or left ventricular (LV) failure (n = 884) in the SHould we emergently revascularize Occluded coronaries for Cardiogenic shock? (SHOCK) trial registry.
<b>RESULTS</b>	Patients with predominant RV shock were younger, with a lower prevalence of previous MI (25.5 vs. 40.1%, p = 0.047), anterior MI, and multivessel disease (34.8 vs. 77.8%, p < 0.001) and a shorter median time between the index MI and the diagnosis of shock (2.9 vs. 6.2 h, p = 0.003) in comparison to patients with LV shock. In-hospital mortality was 53.1% versus 60.8% (p = 0.296) for patients with predominant RV and LV shock, respectively, and the influence of revascularization on mortality was not different between groups. Multivariate analysis revealed that RV shock was not an independent predictor of lower in-hospital mortality (odds ratio 1.07, 95% confidence interval 0.54 to 2.13).
<b>CONCLUSIONS</b>	Despite the younger age, lower rate of anterior MI, and higher prevalence of single-vessel coronary disease of RV compared with LV shock patients, and their similar benefit from revascularization, mortality is unexpectedly high in patients with predominant RV shock and similar to patients with LV shock. (J Am Coll Cardiol 2003;41:1273–9) © 2003 by the American College of Cardiology Foundation

Despite more recent aggressive reperfusion and revascularization strategies, patients with myocardial infarction (MI) complicated by cardiogenic shock continue to have a relatively poor prognosis (1,2). The majority of patients with this condition have severe left ventricular (LV) dysfunction

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secondary to acute MI underlying their clinical presentation (3). However, a small subset of patients in shock has significant right ventricular (RV) dysfunction in isolation or in combination with LV dysfunction (1,3). Although RV infarction, which complicates acute inferior MI in approximately 50% of patients (4), has been thought to have a favorable long-term prognosis (5,6), some series report a

significant increase in early mortality in patients with acute inferior MI and RV involvement (7). However, few data are available describing the influence of RV infarction on mortality for patients in cardiogenic shock. Accordingly, to determine the characteristics and outcomes of patients with acute MI complicated by cardiogenic shock due to RV infarction, we compared patients in shock with predominant RV and LV dysfunction in a large registry associated with the SHould we emergently revascularize Occluded coronaries for Cardiogenic shock? (SHOCK) trial.

## METHODS

**SHOCK trial registry.** The SHOCK trial was a randomized comparison of early percutaneous or surgical revascularization versus initial medical stabilization (including thrombolytic therapy and intra-aortic balloon counterpulsation, where appropriate) for patients with acute MI complicated by cardiogenic shock (2). Patients with suspected cardiogenic shock either ineligible for participation or eligible but not randomized were entered into a registry which began in April 1993 and ended enrollment in August 1997. Additional screening of 188 patients from September 1997 to November 1998 identified nine additional RV shock patients. A local discharge diagnosis of acute MI plus

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## Abbreviations and Acronyms

CK	= creatine kinase
ECG	= electrocardiographic
LAD	= left anterior descending coronary artery
LV	= left ventricular
MI	= myocardial infarction
RCA	= right coronary artery
RV	= right ventricular
SHOCK	= Should we emergently revascularize Occluded coronaries for Cardiac shock?

cardiogenic shock (diagnosis-related group 410 and 785.51) constituted criteria for entering the registry. Patients were enrolled from 36 centers, initiated in staggered fashion, including 24 centers in the U.S. ( $n = 576$ , 62%), 5 centers in Canada ( $n = 197$ , 21%), 4 centers in Belgium ( $n = 54$ , 6%), and 1 center each in Australia, New Zealand, and Brazil ( $n = 106$ , 11%). The institutional committee on human research approved the study protocol at each center.

**Patient sample.** This report is based on 933 patients with cardiogenic shock due to predominant RV failure or primary LV failure, a subset of the 1,378 patients with cardiogenic shock complicating acute MI who were prospectively registered. The study group included 49 patients (5.3%) with predominant RV failure and 884 patients (94.7%) with predominant LV failure.

**Definitions.** "Isolated" RV shock was an exclusion for enrollment into the randomized trial and was assessed at each participating site on the basis of available data, which included clinical, electrocardiographic (ECG), hemodynamic, and echocardiographic findings. Predominant LV failure was designated as the etiology of cardiogenic shock when none of the following major shock categories was indicated: isolated RV shock, mechanical cause, tamponade, previous severe valvular heart disease, shock resulting from a cardiac catheterization laboratory complication, excess pharmacologic therapy, or noncardiac cause. Creatine kinase (CK) values reported are the highest recorded (based on three or more measures in 71.3% of patients). The ECG location of the infarct was defined as follows (8):  $V_1$  through  $V_4$  anterior; II, III, aVF inferior;  $V_5$ ,  $V_6$  apical; I, AVL lateral;  $V_1$ ,  $V_2$  posterior.

**Data collection.** Data were abstracted from the medical record by the SHOCK study coordinators, who were centrally trained to complete standard study report forms. Patient characteristics, MI characteristics, hemodynamics, procedure use, and vital status at discharge were recorded. Cardiac catheterization and angioplasty reports were sent to the Clinical Coordinating Center for abstraction and completion of the standard form. Of the 49 patients with predominant RV shock, 31 underwent coronary angiography and 23 angiograms were obtained for review by two independent angiographers.

**Statistical analysis.** All analyses compared a group of 49 patients with predominant RV shock to a group of 884

**Table 1.** Clinical Characteristics

	RV Shock (n = 49)	LV Shock (n = 884)	p Value
Mean age (yrs)	64.5 $\pm$ 12.0	68.5 $\pm$ 12.1	0.031
Age <75 yrs (%)	81.6	67.5	0.041
Male (%)	53.1	63.6	0.171
Hypertension (%)	52.2	51.7	1.00
Diabetes (%)	30.4	32.8	0.872
Smoking (%)	46.2	51.5	0.623
Elevated lipids (%)	66.7	40.2	0.022
Previous MI (%)	25.5	40.1	0.047
Congestive heart failure (%)	11.1	19.8	0.178
Renal insufficiency (%)	11.1	10.7	0.809
Peripheral vascular disease (%)	11.1	18.8	0.447
Previous CABG (%)	2.1	10.1	0.077
Previous PTCA (%)	6.5	6.7	1.00

CABG = coronary artery bypass graft surgery; LV = left ventricular; MI = myocardial infarction; PTCA = percutaneous transluminal coronary angioplasty; RV = right ventricular.

patients with primary LV shock complicating MI. The distributions of demographic, clinical, angiographic, and treatment variables were compared. Fisher exact test was used to compare the distribution of unordered categorical factors. Student  $t$  test was used to compare the means of normally distributed variables, and the Wilcoxon test was used to compare the distributions of ordinal and non-normally distributed variables. The Mantel-Haenszel test for linear trend (9) was used to compare the distribution of the number of diseased vessels for the two groups. Logistic regression was used to determine whether RV shock was an independent risk factor for in-hospital mortality. Kaplan-Meier estimates of in-hospital survival were used for the survival figure, and the log-rank test was used to compare survival distributions. All hypothesis testing was two-sided, and a  $p$  value  $<0.05$  was considered significant. All statistical analyses were conducted using the Statistical Analysis System (version 6.12, SAS Institute Inc., Cary, North Carolina) and S-Plus for Windows (Statistical Sciences Inc., Seattle, Washington).

**RESULTS**

**Clinical characteristics.** Patients with predominant RV shock were younger than patients with LV shock, although coronary artery disease risk factor profile, with the exception of hyperlipidemia, was similar between the two groups (Table 1). There was a lower incidence of previous MI for patients with predominant RV shock, although the prevalence of other comorbid conditions such as renal insufficiency, peripheral vascular disease, and previous revascularization was similar to that in patients with LV shock.

**Characteristics and location of the infarct.** Although the proportion of patients transferred from other hospitals to the SHOCK registry site was similar in the two groups, the median time between the index MI and the diagnosis of shock was shorter in patients with predominant RV shock in comparison to patients with LV shock (2.9 vs. 6.2 h,  $p = 0.003$ ). There was no difference in the median highest total

**Table 2.** Characteristics and Location of the Infarct

	RV Shock (n = 49)	LV Shock (n = 884)	p Value
Transfer admission (%)	36.7	43.2	0.459
MI to shock (h)*	2.9	6.2	0.003
Highest total CPK (U/l)*	2,359	1,940	0.795
CPK/ULN*	14.7	8.9	0.201
Thrombolytic therapy (%)	51.0	34.4	0.021
ECG location			
Anterior (%)	10.6	58.8	< 0.0001
Inferior (%)	85.1	34.4	< 0.0001
Posterior (%)	40.4	17.3	< 0.001
Lateral (%)	23.4	31.9	0.259
Apical (%)	6.4	9.6	0.612
Multiple locations (%)	55.3	48.4	0.372
	n = 17	n = 300	
LV ejection fraction post shock (%)†	41.9 ± 15.7	30.0 ± 12.6	0.002

\*Median; †LV function measured using echocardiography 59%, LV angiography 36%, radionuclide ventriculography 5%.

CPK = creatine phosphokinase; ECG = electrocardiographic; LV = left ventricular; MI = myocardial infarction; RV = right ventricular; ULN = upper limit of normal.

CK level between groups. Patients with predominant RV shock were treated with thrombolytic therapy more often than were patients with LV shock (51.1% vs. 34.4%,  $p = 0.021$ ). As expected, very few patients with predominant RV shock had an anterior MI (10.6%) in contrast to high rates for patients with LV shock (Table 2). Most patients with predominant RV shock had an inferior or posterior MI. Of note, LV ejection fraction, available in 34% of patients, was significantly higher in patients with predominant RV shock compared with LV shock ( $41.9\% \pm 15.7\%$  vs.  $30.0\% \pm 12.6\%$ ,  $p < 0.002$ ).

**Hemodynamic characteristics.** Right heart catheterization was performed in just over 60% of patients at a median time of 3.3 (1.0, 7.6) h after the onset of shock (Table 3). Of note, 96% of patients were treated with vasopressors (predominant RV shock,  $n = 34$ , 100% vs. LV shock,  $n = 633$ , 95.7%,  $p = 0.390$ ) and 70.6% were treated with inotropes (predominant RV shock,  $n = 34$ , 55.9% vs. LV shock,  $n = 633$ , 71.4%,  $p = 0.080$ ). Right atrial pressure was significantly higher and systolic pulmonary artery pressure signif-

icantly lower in patients with predominant RV shock, although mean pulmonary capillary wedge pressure, cardiac output, and cardiac index were similar in the two groups. Furthermore, there was no difference in systolic blood pressure ( $88 \pm 23$  vs.  $88 \pm 23$  mm Hg,  $p = 0.456$ ), diastolic blood pressure ( $52 \pm 15$  vs.  $53 \pm 17$  mm Hg,  $p = 0.314$ ), or lowest systolic blood pressure recorded ( $69 \pm 18$  vs.  $68 \pm 16$  mm Hg,  $p = 0.819$ ) in the two groups, although heart rate ( $85.4 \pm 25.7$  vs.  $95.2 \pm 25.8$  beats/min,  $p = 0.042$ ) was lower in patients with predominant RV shock in comparison to LV shock.

**Angiographic characteristics.** As expected, in the subset of patients undergoing coronary angiography (Table 4), the right coronary artery (RCA) was more likely to be the infarct artery in patients with predominant RV shock and the left anterior descending artery (LAD) was more likely to be the infarct-related artery in patients with LV shock. In addition, patients with predominant RV shock were more likely to have single- and double-vessel disease and less likely to have triple-vessel disease than patients with LV shock.

**Table 3.** Hemodynamic Characteristics\*

	RV Shock	LV Shock	p Value
Right heart catheterization (%)	61.2	64.4	0.649
n = 49,884			
Right atrial pressure (mm Hg)	23.0 ± 9.9	14.2 ± 7.4	0.0001
n = 17,276			
Systolic pulmonary artery pressure (mm Hg)	35.0 ± 7.3	41.1 ± 12.8	0.045
n = 21,341			
Diastolic pulmonary artery pressure (mm Hg)	22.2 ± 6.3	23.9 ± 8.0	0.370
n = 21,343			
Pulmonary capillary wedge pressure (mm Hg)	23.1 ± 11.2	23.6 ± 8.6	0.339
n = 30,534			
Cardiac output (l/min) n = 16,282	3.8 ± 1.6	3.9 ± 1.6	0.774
Cardiac index (l/min/m <sup>2</sup> ) n = 16,408	1.9 ± 0.6	2.1 ± 0.8	0.776
Right atrial/pulmonary capillary wedge pressure ≥0.8 (%)	70.6	23.6	0.0001
n = 17,275			

\*Measurements obtained on sympathomimetic amines and/or intra-aortic balloon support.

LV = left ventricular; RV = right ventricular.

**Table 4.** Angiographic Characteristics

	RV Shock	LV Shock	p Value
Infarct-related artery (%)	n = 23	n = 417	< 0.001
Left anterior descending	0.0	44.1	
Left circumflex	0.0	14.2	
Right coronary	95.7	27.1	
Left main	4.3	6.7	
Saphenous vein graft	0.0	7.9	
Number of vessels diseased (%)	n = 23	n = 518	< 0.001
One	65.2	22.2	
Two	30.4	20.7	
Three	4.4	57.1	
Left main (%)	8.0	16.3	0.402

LV = left ventricular; RV = right ventricular.

**In-hospital treatment.** There was no difference in the use of intra-aortic balloon counterpulsation (49.0 vs. 52.6%,  $p = 0.661$ ) between patients with predominant RV and LV shock, respectively. However, patients with predominant RV shock were more likely to be treated with coronary angioplasty (49.0 vs. 32.8%,  $p = 0.029$ ). Rates of coronary bypass surgery (10.2 vs. 15.4%,  $p = 0.415$ ) were similar.

**In-hospital outcomes.** The incidence of reinfarction (8.8 vs. 8.1%,  $p = 0.751$ ) and recurrent ischemia (12.1 vs. 18.9%,  $p = 0.490$ ) were similar for the two groups. Of note, in-hospital mortality was 53.1% for patients with predominant RV shock and 60.8% in patients with LV shock,  $p = 0.296$  (Fig. 1). Multivariate analysis of in-hospital mortality, with adjustment for age, the time between the index infarction to the onset of cardiogenic shock, coronary angioplasty, thrombolytic therapy, and anterior MI revealed that predominant RV shock was not an independent predictor of in-hospital death (odds ratio 1.07, 95% confidence interval 0.54 to 2.13). The Kaplan-Meier estimates of in-hospital survival for patients with predominant RV and LV shock are shown in Figure 2.

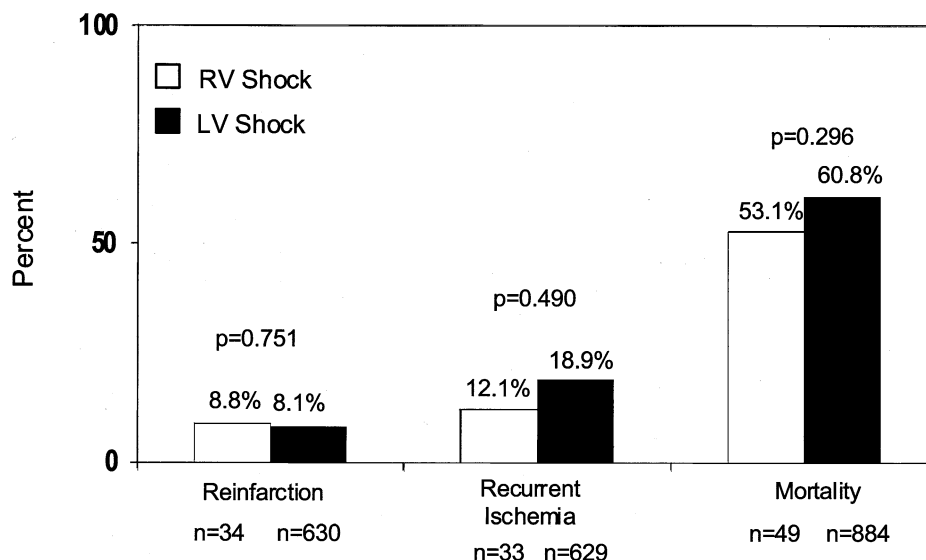
**In-hospital revascularization.** The influence of revascularization on mortality for patients with predominant RV and LV shock is depicted in Figure 3. Mortality for patients undergoing coronary angioplasty, coronary bypass surgery, or any revascularization procedure was not different in the two groups. Although mortality was higher for patients not selected for revascularization, there was no statistical difference in in-hospital mortality (65.2 vs. 78.3%,  $p = 0.195$ ) in those patients with predominant RV and LV shock.

## DISCUSSION

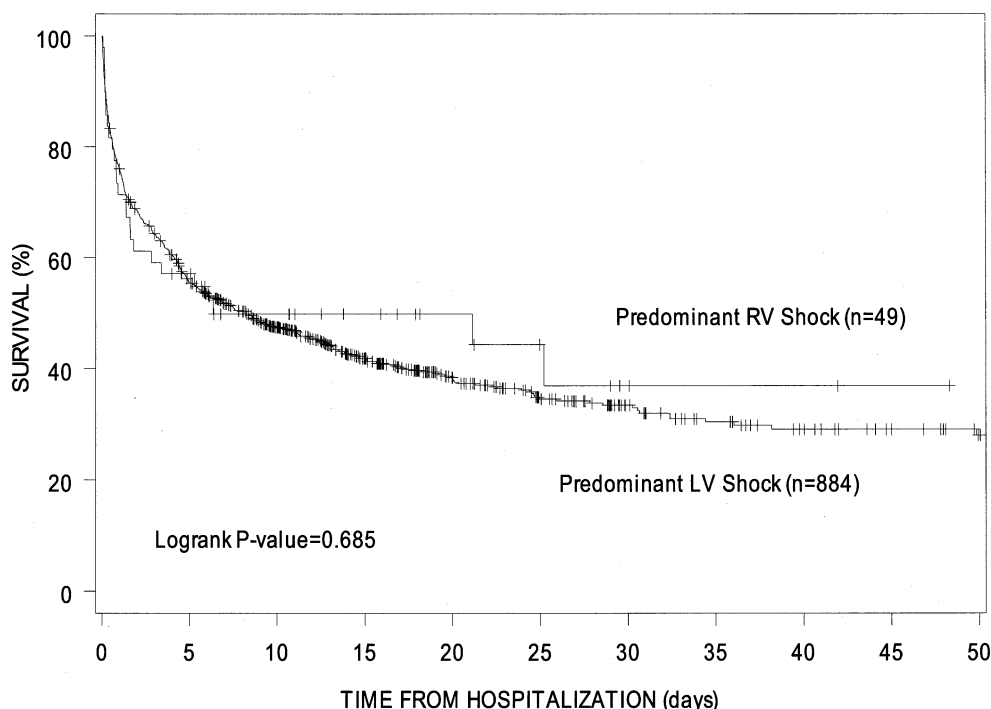
**Clinical characteristics.** The striking finding of this study is the similarly poor outcomes despite the younger age and different infarct location in patients with predominant RV shock compared with LV shock. Previous studies have noted a more favorable prognosis for patients with inferior MI in comparison to anterior MI (10,11). This observation may extend to patients with predominant RV shock where the degree of LV dysfunction is usually less than with LV shock. Therefore, it is disappointing that patients with predominant RV shock had a similar in-hospital mortality to patients with LV shock despite their seemingly lower risk profile, suggesting that the hemodynamic consequences of RV shock significantly impact early mortality.

Right ventricular infarction has been recognized as a clinical syndrome (12,13) and, as such, the diagnosis of predominant RV shock was made using clinical and non-invasive findings at each site. The hemodynamic data, high incidence of the right coronary as the infarct artery, and relatively preserved LV function serve to confirm the accurate diagnosis in this study.

**Hemodynamic characteristics.** The severity of the hemodynamic abnormalities associated with RV infarction is related to the extent of RV ischemia and consequent RV dysfunction as well as to the restraining effect of the

**Figure 1.** In-hospital outcomes in patients with predominant right ventricular (RV) and left ventricular (LV) shock.

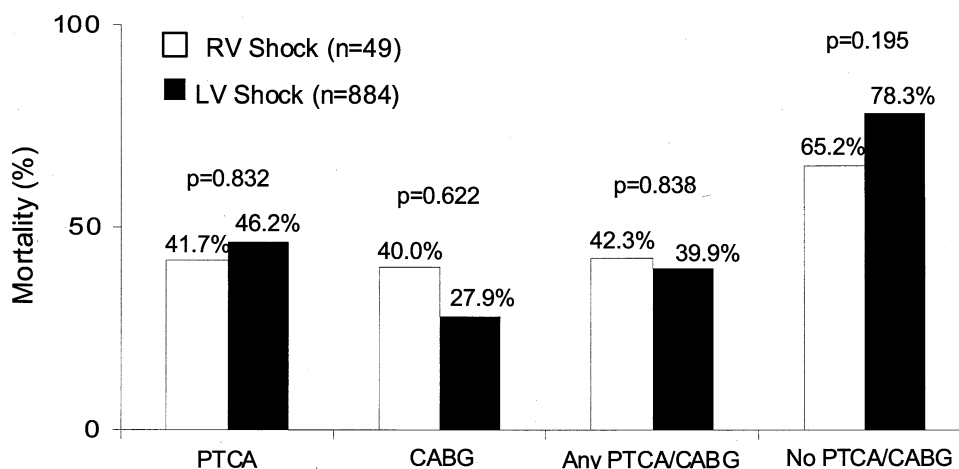




**Figure 2.** In-hospital survival curves for patients with predominant right ventricular (RV) and left ventricular (LV) shock truncated at 50 days. In-hospital survival rates were 46.9% for patients with predominant RV shock and 39.2% for patients with LV shock.

pericardium (14), LV function, and ventricular interdependence. It has now been shown both experimentally (15) and clinically (16) that the intact LV may assist RV ejection by LV septal contraction causing a bulging into the RV which generates an active RV systolic pressure wave and systolic force sufficient for pulmonary perfusion. Loss of this mechanism with concomitant LV infarction, particularly when the interventricular septum is involved, may lead to further hemodynamic deterioration in patients with RV infarction. Furthermore, augmented atrial contraction is necessary to overcome the stiffness of the ischemic RV, and factors that impair RV filling (intravascular volume depletion, concomitant atrial infarction, loss of atrioventricular synchrony)

may severely compromise hemodynamics and result in cardiogenic shock. In the present study, it was expected that right atrial pressure would be significantly higher (although a right atrial pressure of 23 mm Hg suggests excess volume loading) and pulmonary artery pressure significantly lower in patients with predominant RV shock in comparison to LV shock. The similarity in pulmonary capillary wedge pressure is noteworthy. It is consistent with the systemic hypotension and LV dysfunction that occurs even when RV infarction is responsible for shock and supports previous reports noting that the hemodynamic consequence of RV dysfunction is the result of a critical interaction between both ventricles with interventricular septal shift into the LV



**Figure 3.** Mortality for patients with predominant right ventricular (RV) and left ventricular (LV) shock undergoing coronary artery bypass graft surgery (CABG) and percutaneous transluminal coronary angioplasty (PTCA).

(17). In comparison to the LV, the RV is poorly adapted to compensate for the increase in afterload, with its large surface area and thin free wall, and this may explain the rapid hemodynamic compromise and earlier onset of hypotension and shock in patients with predominant RV shock (18). The lower heart rate in patients with RV shock is likely related to the lesser degree of LV dysfunction, although sinus node dysfunction, more vagal tone, or right atrial stretch may play a role. However, cardiac output and cardiac index would be expected to be similar for patients with predominant RV or LV shock once shock ensues.

**Angiographic characteristics.** Proximal occlusion of the RCA resulting in anterolateral RV infarction secondary to occlusion of the acute marginal arteries has been documented in patients with inferior MI and RV infarction (19). However, pathologic studies show that the posterior RV wall (usually in association with LV posterior and posteroseptal infarction) is most frequently involved in RV infarction, and this is consistent with the observation of RV infarction after occlusion of a left dominant circumflex artery or occlusion of the distal RCA (17,20,21). Right ventricular infarction has been associated with both occlusion of a nondominant RCA and significant RV hypertrophy that increases the susceptibility of the RV to ischemia (22). It is unclear why all proximal RCA occlusions are not associated with RV infarction, but the lower oxygen requirement of the RV, increased coronary blood flow during systole, increased collateral flow from the left coronary artery, and diffusion of oxygen from intracavitary blood through the thin wall of the RV have been implicated (23). In the present report, it is not surprising that in 96% of patients with predominant RV shock, the RCA was the infarct artery. In the 4% of patients with a left main infarct artery, the left coronary artery likely supplied collateral flow to a diseased or occluded RCA. It is of interest to note, however, that RV infarction occurs in less than 10% of patients with anterior MI. In this setting, RV infarction is attributed to RV dysfunction caused by LV infarction and the secondary increase in pulmonary artery pressure and RV afterload (24), and to compromise of the LAD collateral flow to the acute marginal arteries. However, an RV current of injury (ECG ST-segment elevation in  $V_2$  to  $V_3$ ) may play a role in the diagnosis of anterior MI. The absence of the LAD as the infarct artery in this study supports the latter hypothesis. The lower prevalence of previous MI in patients with predominant RV shock is consistent with a lower prevalence of multivessel disease in comparison with patients with LV shock.

**In-hospital revascularization.** The increased use of coronary angioplasty in patients with predominant RV compared with LV shock likely reflects the associated prevalence of multivessel disease in each group. Similar to the overall registry (3) and randomized trial (2), revascularization was associated with lower mortality in both groups. For Registry patients, this is in part due to a lower risk profile for patients selected for these treatments. There was no difference in the

impact of revascularization on mortality between patients with predominant RV or LV shock.

**In-hospital mortality.** It is generally believed that RV infarction is associated with a relatively favorable prognosis (25). However, when inferior MI is complicated by RV involvement, in-hospital mortality is significantly higher than in the absence of RV involvement (5), and when RV infarction results in cardiogenic shock, mortality would be expected to increase even further. Notwithstanding, it is still surprising that in this series of patients, mortality for patients with RV shock was similar to that for patients with LV shock, which suggests that RV shock defines a particularly high-risk subset of shock patients. In previous studies (26,27), mortality rates were lower when RV involvement was present than when the low cardiac output was due solely to LV dysfunction.

**Clinical implications.** Although studies differ concerning the influence of RV infarction on long-term prognosis, it is clear that in the majority of survivors, the clinical (and echocardiographic/radionuclide) (28-30) manifestations of RV dysfunction return to normal. Optimal management aimed at support of the RV and reversal of RV ischemia is essential and currently consists of maintenance of adequate RV preload with volume loading (although excess volume further compromises RV function), preservation of RV synchrony, reduction in RV afterload (particularly when LV dysfunction is present), and inotropic support of the RV (31-33). Several studies also suggest a role for early reperfusion with fibrinolytic therapy (34,35) or primary angioplasty (36,37). Furthermore, early recognition of predominant RV infarction in the pathogenesis of cardiogenic shock is critically important to ensure not only that appropriate treatment is instituted but that therapies that may be problematic (nitrates, morphine) are avoided. Our study suggests that in-hospital mortality for patients with predominant RV shock is unexpectedly high. Strategies to improve outcome in this subset of patients at high risk are needed.

**Study limitations.** These data should be interpreted with caution, because potentially confounding variables influencing mortality are not equally distributed between groups in a registry setting. In addition, the diagnosis of "isolated" (predominant) RV shock was based on local judgment at each site and not confirmed centrally. However, RV infarction is recognized as a clinical syndrome (26,38,39), and when present, served as an exclusion for entry into the SHOCK trial. The difference in hemodynamics, infarct artery, and LV function reported herein lends support to the accurate diagnosis of RV shock in this Registry.

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