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Right Ventricular Dysfunction and Risk of Heart Failure and Mortality After Myocardial Infarction

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OBJECTIVES The aim of this study was to determine the prognostic value of right ventricular (RV) function in patients after a myocardial infarction (MI).

BACKGROUND Right ventricular function has been shown to predict exercise capacity, autonomic imbalance and survival in patients with advanced heart failure (HF).

METHODS Two-dimensional echocardiograms were obtained in 416 patients with left ventricular (LV) dysfunction (ejection fraction [LVEF] ≤40%) from the Survival And Ventricular Enlargement (SAVE) echocardiographic substudy (mean 11.1 ± 3.2 days post infarction). Right ventricular function from the apical four-chamber view, assessed as the percent change in the cavity area from end diastole to end systole (fractional area change [FAC]), was related to clinical outcome.

RESULTS Right ventricular function correlated only weakly with the LVEF ($r = 0.12$, $p = 0.013$). On univariate analyses, the RV FAC was a predictor of mortality, cardiovascular mortality and HF ($p < 0.0001$ for all) but not recurrent MI. After adjusting for age, gender, diabetes mellitus, hypertension, previous MI, LVEF, infarct size, cigarette smoking and treatment assignment, RV function remained an independent predictor of total mortality, cardiovascular mortality and HF. Each 5% decrease in the RV FAC was associated with a 16% increased odds of cardiovascular mortality (95% confidence interval 4.3% to 29.2%; $p = 0.006$).

CONCLUSIONS Right ventricular function is an independent predictor of death and the development of HF in patients with LV dysfunction after MI. (J Am Coll Cardiol 2002;39:1450–5) © 2002 by the American College of Cardiology Foundation

The extent of left ventricular (LV) dysfunction is associated with an adverse prognosis in patients with heart failure (HF) and survivors of acute myocardial infarction (MI) (1–3). In patients with moderate or advanced HF, right ventricular (RV) dysfunction has been shown to predict a reduced exercise capacity (4), autonomic imbalance (5) and shortened survival (6–12). Nevertheless, the significance of RV dysfunction after MI is less clear (13–18).

The Survival And Ventricular Enlargement (SAVE) trial demonstrated that patients with LV dysfunction after MI, who were randomized to receive captopril, had improved survival and a decreased incidence of HF and MI (19). During the enrollment phase of the SAVE trial, an echocardiographic substudy was prospectively designed to determine the importance of LV enlargement on outcome, as well as the influence of captopril on LV size and the patient's clinical course (20,21). We studied the patients enrolled in the SAVE echocardiographic substudy to determine the prognostic value of RV function in predicting survival and the development of HF or MI in patients with LV dysfunction, but free of HF, after MI.

METHODS

Study group. In the SAVE trial, 2,231 patients with LV dysfunction (ejection fraction [LVEF] ≤40%), who were free of right or left HF after MI, were randomized to receive either captopril or placebo. Inclusion and exclusion criteria, and the details of patients' characteristics, have been previously described (19). Twenty-four of the original 45 centers participated in the echocardiographic ancillary study. Patients with LVEF ≤40%, but without signs of HF, and who were 21 to 80 years old, were included in the study between 3 and 16 days after MI. The echocardiographic substudy consisted of 512 patients who underwent two-dimensional echocardiography at a mean time of 11.1 ± 3.2 days after MI (baseline) (20,21). Right ventricular images of sufficient

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Abbreviations and Acronyms

FAC = fractional area change
HF = heart failure
LV = left ventricle or ventricular
LVEF = ejection fraction
MI = myocardial infarction
RV = right ventricle or ventricular
SAVE = Survival And Ventricular Enlargement trial

quality for quantitative analysis of RV function were available in 416 patients.

Left ventricular analysis. All two-dimensional echocardiograms were submitted to the core laboratory at the Brigham and Women's Hospital for assessment of technical quality and suitability for quantitative analysis (20). Left ventricular size was assessed by measuring the LV cavity areas at end diastole and end systole. Left ventricular function was assessed as the percent change in the cavity area from end diastole to end systole. The infarct location was estimated by determining the location of akinesia or dyskinesia, identified qualitatively on the echocardiogram (20). In addition, LVEF was determined at baseline by radionuclide ventriculography in all patients enrolled.

Right ventricular analysis. Right ventricular function was assessed quantitatively, by echocardiographic analysis, as the percent change in the cavity area from end diastole to end systole. The RV free wall and septal endocardium were digitized manually in the apical four-chamber view, utilizing a custom-designed echocardiographic analysis program. End diastole was identified by the onset of the R wave on the simultaneously recorded electrocardiogram. End systole was identified as the smallest RV cavity size just before tricuspid valve opening. The RV free wall was traced from the base to apex, and the RV areas were calculated from the average of three measurements. Right ventricular fractional area change (FAC) was calculated by using the following formula: (end-diastolic area − end-systolic area)/end-diastolic area (22). All measurements were performed by the same observer (Dr. Zornoff), who had no knowledge of the outcome data.

Normal RV function was determined by analysis of 50 patients who were identified as having normal echocardiograms (left and right), through a search of the laboratory records at the Non-Invasive Laboratory at Brigham and Women's Hospital. We defined abnormal RV function as RV FAC <2 SD below the mean value (<32.2%). The intraobserver reproducibility of the RV FAC measurement was assessed by the primary reader performing two sets of RV FAC measurements in 38 randomly selected patients, in a blinded fashion. The correlation coefficient (r) between the two assessments was 0.94 (coefficient of repeatability = 6.5%, by the Bland and Altman method). The interobserver variability was assessed by a second experienced physician who digitized and traced the RV areas in 16 randomly selected patients; the correlation coefficient between the two RV FAC measured was 0.83 (coefficient of repeatability = 8.8%, by the Bland and Altman method).

Statistical methods. To assess differences between the groups, we used the Student *t* test for continuous variables and the chi-square test for categorical variables. Logistic regression was used to assess the relationship between RV dysfunction and clinical outcome. The Kaplan-Meier method was used to analyze the time to death and to evaluate the crude effect of RV dysfunction. We used a Cox proportional hazards ratio model (multivariate analysis) to assess the relationship between baseline features and time-dependent outcomes. This model included the following variables known to influence the outcome after MI: age, gender, diabetes mellitus, hypertension, previous MI, LVEF (by either echocardiography or nuclear imaging techniques), cigarette smoking, infarct size and treatment assignment. Because the linearity assumption was not met in the Cox regression model for the aforementioned continuous variables, we recoded them as dichotomous variables. The interaction between LV and RV function was tested explicitly. A p value <0.05 was considered statistically significant. Statistical analyses were performed using STATA software, version 7 (Stata Corp., College Station, Texas).

RESULTS

The RV FAC in the normal study group ranged from 33.7% to 66% (mean 46.53 ± 7.18%) and was normally distributed. Thus, RV dysfunction in this study was defined as RV FAC ≤32.2% (2 SD below the mean value for normal subjects). The RV FAC in the SAVE population ranged from 11.6% to 67% (mean 41.5 ± 10.5%) and was also normally distributed. Right ventricular dysfunction was present in 79 patients (19%).

The baseline characteristics of the patients with and without RV dysfunction are shown in Table 1. There were no significant differences between the groups in terms of age, gender distribution, frequency of diabetes, hypertension or Killip class. The proportion of patients with a previous MI was significantly higher in the RV dysfunction group, and LVEF, as assessed by both radionuclide ventriculography and echocardiography, was significantly lower in the RV dysfunction group.

The RV FAC correlated only weakly, albeit significantly, with LVEF ($r = 0.12$, $p = 0.013$). In addition, RV function varied by infarct location (Table 2). Patients with an inferior infarct location had a higher incidence of RV dysfunction than those with an anterior infarct location, and RV FAC was slightly lower in patients with an inferior infarct location versus other locations (39.5 ± 11% vs. 41.2 ± 10%; $p = 0.01$).

Right ventricular function and outcome. For the composite end point of death or HF, the mean duration of follow-up was 671 days in the RV dysfunction group and 946 days in the group without RV dysfunction. During an

Table 1. Baseline Characteristics of Patients With and Without Right Ventricular Dysfunction After Myocardial Infarction

	No RV Dysfunction (n = 337)	RV Dysfunction (n = 79)	p Value
Age (yrs)	58.5 ± 11.1	60.3 ± 10.6	0.185
Diabetes	17.5%	24.1%	0.18
Male	80.4%	83.5%	0.523
Hypertension	32.9%	27.8%	0.383
Previous MI	27.9%	50.6%	< 0.0001
LVEF (%)	31.5 ± 5.9	29.8 ± 7.3	0.0282
LV FAC (%)	29.1 ± 5.8	26.3 ± 6.5	0.0003
Current smoking	41.5%	35.4%	0.320
Killip class ≥2	34.1%	38.0%	0.518
Thrombolytic use	40.1%	29.1%	0.071
Infarct size* (%)	33.0 ± 11	36.8 ± 14	0.030
Inferior infarct	28.8%	39.2%	0.070
RV FAC (%)	45.2 ± 7.8	25.8 ± 4.8	By design

*Wall motion abnormalities/ventricular cavity, as assessed by echocardiography. Data are presented as the mean value ± SD or percentage of patients.

LVEF = left ventricular ejection fraction; LV FAC and RV FAC = left and right ventricular fractional area change, respectively; MI = myocardial infarction, assessed by radionuclide ventriculography and echocardiography; RV = right ventricular.

average 2.6-year follow-up period, 79 patients died (67 from cardiovascular causes), and 82 patients were either admitted to the hospital or required open-label captopril for the management of HF. The combined end point of cardiovascular death or HF occurred in 130 patients (31.2%). A recurrent MI occurred in 53 patients (12.7%). The relationship between RV FAC and outcome after MI is shown in Table 3. Patients with RV dysfunction demonstrated higher total mortality, cardiovascular mortality and HF than patients without RV dysfunction. In contrast, there were no differences in the incidence of recurrent MI between the two groups. The Kaplan-Meier survival curves for patients with and without RV dysfunction are shown in Figure 1.

In a Cox proportional hazards model, RV dysfunction was a univariate predictor of total mortality, cardiovascular mortality and HF (each $p < 0.0001$) but not recurrent MI ($p = 0.715$). After adjusting for age, gender, diabetes mellitus, hypertension, previous MI, LVEF (by both nuclear imaging and echocardiography), infarct size, cigarette smoking and treatment assignment, RV dysfunction remained an independent predictor of cardiovascular mortality, total mortality and HF (Table 4). Infarct location did not modify the relationship between RV function and outcome. Considering RV function as a continuous variable, each 5% decrease in RV FAC was associated with a 16% increased odds of cardiovascular mortality (95% confidence interval [CI] 4.3% to 29.2%; $p = 0.006$).

Table 2. Relationship Between Infarct Location and Right Ventricular Dysfunction

Infarct Location (n = 415)	RV Dysfunction (n = 79)	RV FAC
Anterior (n = 276)	16.7%	42.5 ± 10%
Inferior only (n = 70)	22.9%	40.1 ± 10%
Anterior and inferior (n = 52)	26.9%	38.7 ± 11%
Any inferior (n = 128)	24.2%	39.5 ± 11%
Other (n = 17)	11.8%	41.2 ± 8.5%

FAC = fractional area change; RV = right ventricular.

DISCUSSION

Left ventricular function is a known predictor of cardiovascular morbidity and mortality after MI (1–3). The results of this study demonstrate that RV FAC is also an independent predictor of a poor outcome, including death and the development of HF, in patients with LV dysfunction after an acute MI. Thus, quantitative assessment of RV function, often neglected clinically, may further stratify this high-risk population.

Right ventricular function and outcome. Right ventricular dysfunction has been associated with an adverse outcome in patients with HF (4–12) and may be secondary to long-term exposure of the RV to chronic elevation of left-sided pressures. The importance of RV function in patients with acute MI is less clear. In a small study of 34 patients with clinically evident HF and coronary artery disease, RVEF was a predictor of mortality (13). In contrast, there was no relationship observed between RV function and one-year mortality in a study of 423 patients, many of

Table 3. Relationship Between Right Ventricular Dysfunction and Outcome

Outcome	No RV Dysfunction (n = 337)	RV Dysfunction (n = 79)	OR for RV Dysfunction (95% CI)
Death	14.5%	38.0%	3.6 (2.1–6.2)
CVD	12.2%	32.9%	3.5 (2.0–6.3)
HF	17.2%	30.4%	2.1 (1.2–3.7)
Death or HF	26.7%	50.6%	2.8 (1.7–4.7)
Recurrent MI	13.3%	10.1%	0.7 (0.3–1.6)

CVD = cardiovascular death; CI = confidence interval; HF = heart failure; MI = myocardial infarction; OR = odds ratio; RV = right ventricular.

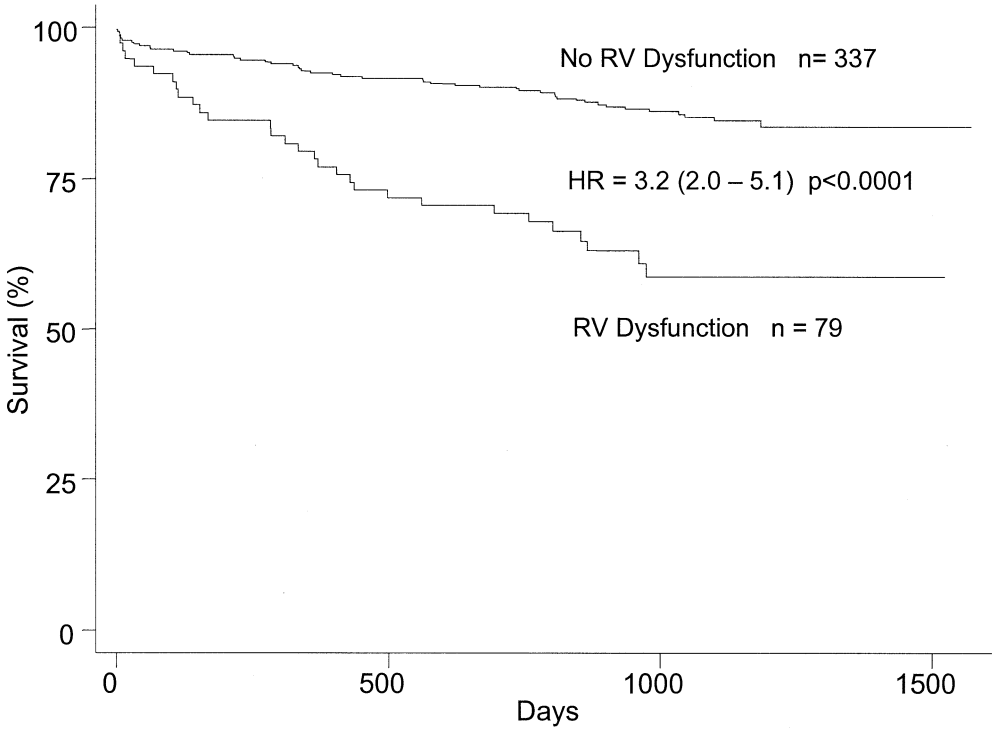

Figure 1. Cumulative percent survival of patients with and those without right ventricular (RV) dysfunction (fractional area change <32.2% or >32.2%). HR = hazard ratio.

whom had normal LV function (18). Furthermore, in the TIMI-II trial (17), in which all patients received reperfusion, a RV wall abnormality was detectable in only 5% of patients with MI (n = 1,110) and was not associated with increased mortality in the year after hospital discharge. However, only patients with inferior infarcts were included. In the present study, in which all patients had LV dysfunction without HF, by study design, RV function independently predicted mortality and the development of HF. These data suggest that RV alterations can occur in a close relationship to the LV alterations that accompany MI, an observation that is supported by experimental evidence that RV hypertrophy can develop very early after a large MI (23).

Etiology of RV dysfunction in MI. A number of possible etiologies have been proposed to explain RV dysfunction after acute MI. Left ventricular dysfunction is known to be an important precursor of RV dysfunction (24), and the RV is extremely sensitive to changes in afterload, a major determinant of which is left atrial pressure (25). The greater prevalence of RV dysfunction in patients with a previous MI (50.6%) suggests that RV function may be a sensitive integrator of left atrial pressure over time. Nevertheless, our study, as well as others, suggests that RV function is largely independent of LV function (15,18), and that RV dysfunction may be more dependent on the location and extent of infarction than on the extent of LV dysfunction (26).

Right ventricular infarction or ischemia in the setting of an inferior infarction could account for at least some of the RV dysfunction seen in the present study. Right ventricular infarction complicates ~50% of cases of acute inferior MI and is a predictor of major complications and mortality (27,28). In this cohort, RV dysfunction was more frequent in patients with an inferior MI. Nevertheless, the majority of RV dysfunction (69%) in our study occurred in the absence of inferior involvement and is therefore likely to be due to other mechanisms. Finally, there is evidence from other studies that RV function may recover to a great extent after acute MI, suggesting that RV myocardial stunning may also be implicated in the pathophysiology of RV dysfunction after MI (16,29).

Although patients with long-standing HF often develop pulmonary hypertension as an effect of elevated left-sided pressures, our study suggests that RV function is largely independent of LV systolic function, which in turn suggests a substantial variability in the response of the RV to LV dysfunction. It is unknown whether pre-existing pulmonary parenchymal disease, vascular disease or diastolic LV dysfunction might contribute to RV dysfunction after MI, although these factors may partly explain some of this variability.

Study limitations. We estimated RV function as the percent change in RV cavity area. Although there has been no clear consensus by echocardiographers regarding the quantitative measurement of RV function, we and others have used this measure in other studies (9,30). Because of the complexity of RV geometry, volumetric approaches to RV function have been problematic. Although assessment of RV function from the apical four-chamber view is a relatively simple technique, like all echocardiographic techniques, it is dependent on image quality. Although a true

Table 4. Multivariate Regression Assessing Risk of Various Outcomes

	Hazard Ratio (95% CI); p Value			
	Cardiovascular Mortality (n = 67)	**HF (n = 82)**	**Total Mortality (n = 79)**	**Death or HF (n = 130)**
Age ≥60 years	2.60 (1.45–4.65); 0.001	1.79 (1.11–2.89); 0.018	2.40 (1.43–4.04); 0.001	1.67 (1.14–2.44); 0.008
Male gender	1.28 (0.63–2.59); 0.481	0.59 (0.35–0.99); 0.047	1.10 (0.59–2.05); 0.752	0.72 (0.46–1.10); 0.137
Diabetes	0.91 (0.51–1.62); 0.746	1.78 (1.09–2.91); 0.021	0.91 (0.52–1.56); 0.719	1.38 (0.91–2.07); 0.120
Hypertension	2.01 (1.22–3.34); 0.006	1.78 (1.13–2.80); 0.012	1.65 (1.03–2.63); 0.036	1.67 (1.17–2.39); 0.005
Previous MI	1.66 (1.00–2.76); 0.048	2.07 (1.30–3.28); 0.002	1.48 (0.93–2.37); 0.096	1.48 (1.03–2.14); 0.034
Current smoking	0.88 (0.49–1.55); 0.652	1.25 (0.77–2.04); 0.362	0.96 (0.58–1.60); 0.893	1.08 (0.74–1.59); 0.664
Infarct segment length (>33%)	1.65 (0.93–2.92); 0.083	1.98 (1.20–3.26); 0.007	1.34 (0.81–2.23); 0.248	1.66 (1.12–2.47); 0.011
Treatment (captopril)	0.97 (0.59–1.60); 0.925	0.58 (0.36–0.93); 0.025	1.24 (0.79–1.96); 0.341	0.98 (0.68–1.40); 0.925
Thrombolytic use	0.75 (0.41–1.37); 0.352	1.22 (0.76–1.96); 0.405	0.75 (0.43–1.29); 0.300	0.85 (0.57–1.27); 0.450
LVEF <31% (by nuclear imaging)	2.09 (1.23–3.55); 0.006	2.03 (1.27–3.25); 0.003	1.82 (1.13–2.93); 0.013	1.86 (1.28–2.70); 0.001
LV FAC <28.5% (by echocardiography)	2.46 (1.26–4.77); 0.008	1.71 (1.01–2.86); 0.043	1.98 (1.12–3.48); 0.017	1.72 (1.14–2.61); 0.009
RV dysfunction (RV FAC <32.2%)	2.51 (1.49–4.22); <0.0001	2.31 (1.39–3.85); 0.001	2.57 (1.59–4.16); <0.0001	2.22 (1.50–3.30); <0.0001

CI = confidence interval; HF = heart failure; other abbreviations as in Table 1.

volumetric validation of this method has not been performed, the results of this analysis may be seen as a biologic validation of this measure.

We cannot exclude the possibility that assessing RV function at a different post-infarction time point would reveal a different relationship between RV function and outcome. It is likely that changes in RV function occur in the first few weeks after MI, as do changes in LV function. Although RV function at the time of randomization was a good predictor of outcome in these patients, we cannot specifically address the predictive value of RV function at different periods after MI.

The results of this study need to be considered in light of the unique patient population in SAVE because only patients with LVEF ≤40% were included. Therefore, we cannot simply extrapolate these data to patients with normal LV function after MI. Another potential limitation of our study is that we do not have data on the occurrence of RV infarction, and we concede that RV infarction probably accounts for some of the RV dysfunction in this cohort, although given the preponderance of anterior infarcts, we do not believe this could explain the majority of cases of RV dysfunction.

Conclusions. Assessment of LV function has become standard practice after MI, as this measure is a well-recognized predictor of subsequent morbidity and mortality. In contrast, quantitative clinical assessment of the RV after MI is uncommon. The present study suggests that RV function is an independent predictor of mortality and the development of HF in patients with known LV dysfunction. Thus, the estimation of RV function after MI may be warranted in the standard assessment of post-infarct patients with LV dysfunction.

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