

Implication of right ventricular dysfunction on long-term outcome in patients with ischemic cardiomyopathy undergoing coronary artery bypass grafting with or without surgical ventricular reconstruction

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Objective: Whether right ventricular dysfunction affects clinical outcome after coronary artery bypass grafting with or without surgical ventricular reconstruction is still unknown. The aim of the study was to assess the impact of right ventricular dysfunction on clinical outcome in patients with ischemic cardiomyopathy undergoing coronary artery bypass grafting with or without surgical ventricular reconstruction.

Methods: Of 1000 patients in the Surgical Treatment for Ischemic Heart Failure with coronary artery disease, left ventricular ejection fraction 35% or less, and anterior dysfunction, who were randomized to undergo coronary artery bypass grafting or coronary artery bypass grafting + surgical ventricular reconstruction, baseline right ventricular function could be assessed by echocardiography in 866 patients. Patients were followed for a median of 48 months. All-cause mortality or cardiovascular hospitalization was the primary end point, and all-cause mortality alone was a secondary end point.

Results: Right ventricular dysfunction was mild in 102 patients (12%) and moderate or severe in 78 patients (9%). Moderate to severe right ventricular dysfunction was associated with a larger left ventricle, lower ejection fraction, more severe mitral regurgitation, higher filling pressure, and higher pulmonary artery systolic pressure (all $P < .0001$) compared with normal or mildly reduced right ventricular function. A significant interaction between right ventricular dysfunction and treatment allocation was observed. Patients with moderate or severe right ventricular dysfunction who received coronary artery bypass grafting + surgical ventricular reconstruction had significantly worse outcomes compared with patients who received coronary artery bypass grafting alone on both the primary (hazard ratio, 1.86; confidence interval, 1.06-3.26; $P = .028$) and the secondary (hazard ratio, 3.37; confidence interval, 1.36-8.37; $P = .005$) end points. After adjusting for all other prognostic clinical factors, the interaction remained significant with respect to all-cause mortality ($P = .022$).

Conclusions: Adding surgical ventricular reconstruction to coronary artery bypass grafting may worsen long-term survival in patients with ischemic cardiomyopathy with moderate to severe right ventricular dysfunction, which reflects advanced left ventricular remodeling. (J Thorac Cardiovasc Surg 2015;149:1312-21)

See related commentary pages 1322-3.

In patients with heart failure (HF), right ventricular (RV) systolic dysfunction has been associated with decreased exercise capacity^{1,2} and a poor clinical outcome³⁻⁷ when compared

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Abbreviations and Acronyms

CABG	= coronary artery bypass grafting
CAD	= coronary artery disease
CV	= cardiovascular
EF	= ejection fraction
HF	= heart failure
LV	= left ventricular
RV	= right ventricular
RVFAC	= right ventricular fractional area change
STICH	= Surgical Treatment for Ischemic Heart Failure
SVR	= surgical ventricular reconstruction
TAPSE	= tricuspid annular plane systolic excursion

with patients who have preserved RV function. However, the small numbers of patients described in previous studies of RV dysfunction severely limit an assessment of the prevalence of RV dysfunction in patients with HF. Moreover, the clinical implications of RV dysfunction in patients with ischemic cardiomyopathy who undergo coronary artery bypass grafting (CABG) with or without surgical ventricular reconstruction (SVR) have not been clearly defined. The Surgical Treatment for Ischemic Heart Failure (STICH) trial⁸ provides a unique opportunity to assess the importance of RV dysfunction in this clinical situation. In the STICH trial, the Echocardiography Core Laboratory (Mayo Clinic, Rochester, Minn) provided a baseline echocardiographic evaluation of structural, functional, and hemodynamic parameters of both the left and right ventricles. The STICH trial tested 2 clinically unresolved and relevant hypotheses in patients with coronary artery disease (CAD) and reduced left ventricular (LV) ejection fraction (EF). The SVR hypothesis (Hypothesis 2) of STICH randomized 1000 patients with anteroapical dysfunction to CABG with SVR versus CABG alone to test the hypothesis that in patients with LV EF 35% or less, CAD amenable to CABG, and anterior LV dysfunction, the addition of SVR improves survival free of subsequent hospitalization for cardiac cause in comparison with CABG alone.⁸ The concept and technique of surgical ventricular restoration have been well described by Dor and colleagues.⁹ The primary outcome of this population has been reported by Jones and colleagues,¹⁰ and the description of clinical characteristics has been reported by Zembala and colleagues.¹¹ Patients randomized to Hypothesis 2 were followed for a median of 48 months. Only 4 of the 1000 patients withdrew consent for follow-up, and 6 patients were lost to follow-up.

The present study sought to examine the prevalence of RV dysfunction in those 1000 patients to determine the relationship between RV dysfunction and other parameters of cardiac structure and function measured by echocardiography. We also examined the interaction of RV dysfunction with treatment on short- and long-term survival in these patients.

MATERIALS AND METHODS**Study Population and Patient Selection**

Among the 2136 patients enrolled in the STICH trial with an LV EF 35% or less and CAD amenable to CABG, 1000 patients with anteroapical dysfunction for whom adding an SVR operation to CABG was reasonable but not required were randomized to CABG versus CABG + SVR. Of the 1000 patients enrolled, 866 had a baseline echocardiogram rated as fair to excellent quality (excellent for textbook quality, good for clear definition of RV walls from multiple views, and fair for good definition of RV walls from limited views) for qualitative assessment of RV function by the Echocardiography Core Laboratory (Figure 1).

Echocardiography Study

Baseline echocardiography was obtained within 3 months before enrollment by clinical sites and sent to the Echocardiography Core Laboratory, where each study was initially analyzed by a research sonographer blinded to randomized treatment assignment and clinical outcomes using American Society of Echocardiography guidelines¹² and with a second over-read by a physician. Details of the methodology used for echocardiographic analysis have been published.¹³

Right Ventricular Function Assessment

RV function was assessed prospectively by visual interpretation and categorized as normal, mild, moderate, or severe dysfunction. The appreciation of the overall mechanical function of the RV was mainly based on the extent of RV free wall segmental motion, wall thickening, RV cavity size, and subjective assessment of RV area change (normal >50%, mild 30%-50%, moderate 20%-30%, and severe <20% from diastole to systole). RV assessment was derived from the parasternal long-axis, apical 4-chamber, and subcostal views. This assessment was based on visual assessment by an experienced Echocardiography Core Laboratory physician.¹⁴

Once the results of the impact of RV function by visual assessment were known, 40 patients in each group (normal, mild, and moderate dysfunction) and all 21 patients with severe RV dysfunction were sent for blinded post hoc calculation of RV fractional area change (RVFAC). RVFAC was calculated from apical 4-chamber views as $([RV \text{ end-diastolic area} - RV \text{ end-systolic area}] / RV \text{ end-diastolic area})$ by a research sonographer with no knowledge of the patients' clinical or other echocardiography data.

Statistics

Clinical and echocardiographic characteristics were described using means and standard deviations for continuous variables and frequencies and percentages for categorical variables. Because of the limited number of patients with moderate and severe RV dysfunction, these 2 groups have been combined and analyzed as 1 moderate/severe subgroup. Comparisons of patients across 3 different levels of RV dysfunction (ie, normal, mild, or moderate/severe) were performed using Kruskal-Wallis nonparametric analysis of variance on continuous and ordinal variables. Group comparisons of nominal categorical variables were performed using the conventional chi-square test or Fisher exact test. The prognostic effect of RV dysfunction on the short-term end point of death within 30 days after surgery was tested using the logistic regression model. The effects of the 3 levels of RV dysfunction on the long-term end points of (a) death or cardiovascular (CV) hospitalization and (b) all-cause mortality and relative risks were assessed using the Cox regression model. Event-rate estimates in each RV dysfunction group for each long-term end point were calculated using the Kaplan-Meier method. The logistic regression model and Cox regression model were also used to assess the interaction of RV dysfunction and treatment (CABG vs CABG + SVR). Testing of the independent prognostic effect of RV dysfunction and testing of the interactive effect of RV dysfunction and treatment were performed after adjusting for LV EF and other key prognostic factors identified from previous modeling analyses of the STICH SVR hypothesis patient data.

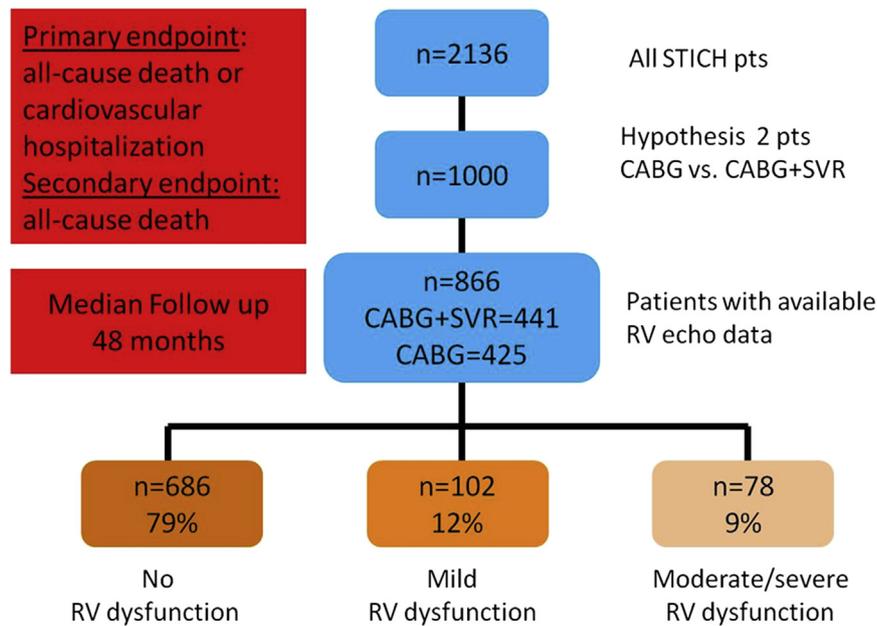


FIGURE 1. Chart illustrating the design of STICH patient selection for RV function analysis. CABG, Coronary artery bypass grafting; RV, right ventricular; STICH, Surgical Treatment for Ischemic Heart Failure; SVR, surgical ventricular reconstruction.

RESULTS

Patients

The study patients (n = 866) consisted of 739 men (85%) and 127 women (15%) with a mean age of 62 ± 10 years randomized to CABG alone (n = 425) and to CABG + SVR (n = 441) (Figure 1). At baseline, patients with

moderate to severe RV dysfunction had more advanced HF, had a higher percentage with atrial fibrillation, had a lower percentage with prior myocardial infarction, had higher levels of creatinine and blood urea nitrogen, required more diuretic therapy, and walked shorter distances in the 6-minute walk test (Table 1).

TABLE 1. Baseline clinical characteristics of the analyzed cohort of patients (N = 866) categorized by right ventricular dysfunction status

Clinical and laboratory variables	Total cohort N = 866	No RV dysfunction N = 686	Mild RV dysfunction N = 102	Moderate/severe RV dysfunction N = 78	P value*
Age, y	61.6 ± 9.8	61.9 ± 9.7	60.2 ± 10.2	60.7 ± 10.6	.246
BMI, kg/m ²	27.4 ± 4.4	27.6 ± 4.3	27.2 ± 4.9	26.6 ± 5.2	.159
Diabetes (%)	289 (33.4)	218 (31.8)	41 (40.2)	30 (38.5)	.148
Chronic renal insufficiency, n (%)	73 (8.4)	53 (7.7)	10 (9.8)	10 (12.8)	.270
Hypertension, n (%)	502 (58.0)	402 (58.6)	59 (57.8)	41 (52.6)	.592
Atrial fibrillation, n (%)	98 (11.3)	65 (9.5)	17 (16.7)	16 (20.5)	.003
Prior MI, n (%)	754 (87.1)	611 (89.1)	87 (85.3)	56 (71.8)	≤.001
Diuretics (loop/thiazide), n (%)	511 (59.0)	382 (55.7)	65 (63.7)	64 (82.1)	≤.001
Diuretics (K ⁺ sparing), n (%)	325 (37.5)	236 (34.4)	48 (47.1)	41 (52.6)	.001
Statin, n (%)	668 (77.1)	544 (79.3)	73 (71.6)	51 (65.4)	.008
Beta-blocker, n (%)	745 (86.0)	603 (87.9)	77 (75.5)	65 (83.3)	.003
Aspirin, n (%)	663 (76.6)	540 (78.7)	68 (66.7)	55 (70.5)	.012
Creatinine (mg/dL)	1.1 ± 0.4	1.1 ± 0.3	1.2 ± 0.7	1.3 ± 0.5	.001
BUN (mg/dL)	28.6 ± 20.1	26.7 ± 16.5	31.6 ± 27.0	38.8 ± 29.7	.001
Systolic BP (mm Hg)	120.7 ± 17.7	121.7 ± 17.7	117.9 ± 17.0	115.1 ± 16.8	.001
NYHA class III, n (%)	373 (43.1)	282 (41.1)	51 (50.0)	40 (51.3)	≤.001
NYHA class IV, n (%)	46 (5.3)	26 (3.8)	4 (3.9)	16 (20.5)	≤.001
Heart rate (beats/min)	72.5 ± 13.3	71.6 ± 12.9	75.8 ± 16.2	75.8 ± 11.7	≤.001
6-min walk distance (m)	347.2 ± 120.5	351.8 ± 116.5	355.8 ± 124.9	285.4 ± 137.2	.001

BMI, Body mass index; BP, blood pressure; BUN, blood urea nitrogen; MI, myocardial infarction; NYHA, New York Heart Association; RV, right ventricular. *P value differences between RV dysfunction groups.

TABLE 2. Baseline echocardiographic characteristics of the analyzed cohort of patients (N = 866) categorized by right ventricular dysfunction status

Echocardiography variables	Total cohort N = 866	No RV dysfunction N = 686	Mild RV dysfunction N = 102	Moderate/severe RV dysfunction N = 78	P value*
LVEDV (mL)	225 ± 69	220 ± 68	234 ± 69	256 ± 68	<.001
LVESV (mL)	161 ± 60	155 ± 58	172 ± 60	199 ± 61	<.001
LVED index (mL/BSA)	117 ± 35	114 ± 34	123 ± 32	137 ± 40	<.0001
LVES index (mL/BSA)	84 ± 31	80 ± 30	90 ± 29	107 ± 35	<.0001
EF (%)	29 ± 8	30 ± 7	27 ± 7	22 ± 6	<.0001
Sphericity index	1.49 ± 0.19	1.50 ± 0.19	1.47 ± 0.18	1.45 ± 0.16	.063
Global hypokinesis, n (%)	84 (9.7)	62 (9.1)	12 (11.8)	10 (12.8)	.433
Wall motion score index	2.22 ± 0.33	2.18 ± 0.33	2.32 ± 0.30	2.44 ± 0.24	<.001
Inferior basal WMSI	2.08 ± 0.80	2.02 ± 0.81	2.21 ± 0.75	2.43 ± 0.70	<.001
Apical WMSI	2.87 ± 0.45	2.88 ± 0.46	2.85 ± 0.34	2.90 ± 0.42	.489
E DT (ms)	184 ± 54	192 ± 53	161 ± 45	140 ± 40	<.001
E/A	1.33 ± 0.91	1.17 ± 0.75	1.82 ± 1.08	2.36 ± 1.28	<.001
E/E' septal	17 ± 9	16 ± 8	21 ± 10	27 ± 17	<.001
E/E' lateral	14 ± 10	13 ± 8	16 ± 11	20 ± 21	.059
Diastolic filling pattern	2.91 ± 0.77	2.80 ± 0.74	3.4 ± 0.78	3.46 ± 0.79	<.001
MR severity	1.11 ± 0.95	0.99 ± 0.85	1.38 ± 1.03	1.85 ± 1.22	<.001
LA volume (mL)	82 ± 29	78 ± 27	89 ± 30	101 ± 30	<.001
TR velocity (m/s)	2.83 ± 0.55	2.73 ± 0.50	2.97 ± 0.57	3.12 ± 0.63	<.001
PASP (mm Hg)	42 ± 15	39 ± 14	45 ± 14	52 ± 16	<.001
Stroke volume (mL)	66 ± 19	68 ± 18	61 ± 20	53 ± 17	<.01
Cardiac output (mL)	4519 ± 1384	4634 ± 1399	4236 ± 1227	3699 ± 1115	<.01
Cardiac index (mL)	2334 ± 708	2382 ± 722	2253 ± 602	1924 ± 563	≤.001

BSA, Body surface area; DT, deceleration time; E, early diastolic velocity; E/A, early/late velocity ratio; E/E', early mitral/annular velocity ratio; EF, ejection fraction; LA, left atrial; LVED, left ventricular end-diastolic; LVEDV, left ventricular end-diastolic volume; LVES, left ventricular end-systolic; LVESV, left ventricular end-systolic volume; MR, mitral regurgitation; PASP, pulmonary artery systolic pressure; RV, right ventricular; TR, tricuspid regurgitation; WMSI, wall motion score index. *P value differences between RV dysfunction groups.

Prevalence of Right Ventricular Dysfunction and Its Association

RV function was normal in 686 patients (79%), mildly reduced in 102 patients (12%), and moderately to severely reduced in 78 patients (9%) (Figure 1). Of the patients sent for post hoc analyses of RVFAC, measurements could be performed reliably in only 20 normal patients, 25 patients with mild RV dysfunction, 22 patients with moderate RV dysfunction, and 16 patients with severe dysfunction. RVFAC was 50.1% ± 9.1%, 34.6% ± 5.5%, 27.5% ± 4.9%, and 17.6% ± 3.7%, respectively ($P < .001$). Two-dimensional, Doppler, and tissue Doppler echocardiography data in the 3 groups of patients defined by RV function as (1) normal, (2) mild, and (3) moderate to severe dysfunction are shown in Table 2. Both LV end-diastolic and LV end-systolic volumes increased progressively with increasing RV dysfunction (Table 2 and Figure 2). In parallel, LV EF was progressively reduced with worsening of RV dysfunction. All LV diastolic function and filling parameters (E/A ratio, deceleration time, E/e, and left atrial volume index) were progressively worse with more severe RV dysfunction, indicating higher LV filling pressure with worsening RV dysfunction. Mitral regurgitation was more severe and Doppler-derived pulmonary artery systolic pressure was higher in patients with moderate to severe

RV dysfunction compared with the patients with normal RV function or mild dysfunction (Table 2 and Figure 3).

Prognostic Role of Right Ventricular Dysfunction

The prognostic effect of RV dysfunction (independent of treatment) was significant for the short-term outcome of 30-day mortality ($P = .023$) and the long-term outcome of death or CV hospitalization ($P = .022$). The relationship with long-term mortality did not achieve conventional significance ($P = .070$) (Table 3). The nature of these relationships is illustrated with Kaplan–Meier estimates of event rates by degree of RV dysfunction (Figure 3), where the highest event rates (worst outcomes) are observed in the patients with moderate to severe RV dysfunction. However, the relationship of RV dysfunction with each of the clinical outcomes considered was no longer significant after adjusting for LV EF and even less so after adjusting for LV EF plus the other prognostic clinical and echocardiographic factors (Table 3).

Impact of Right Ventricular Dysfunction on the Treatment Effect of Coronary Artery Bypass Grafting Versus Coronary Artery Bypass Grafting + Surgical Ventricular Reconstruction

For patients with normal or mildly reduced RV function, the outcomes of CABG alone compared with

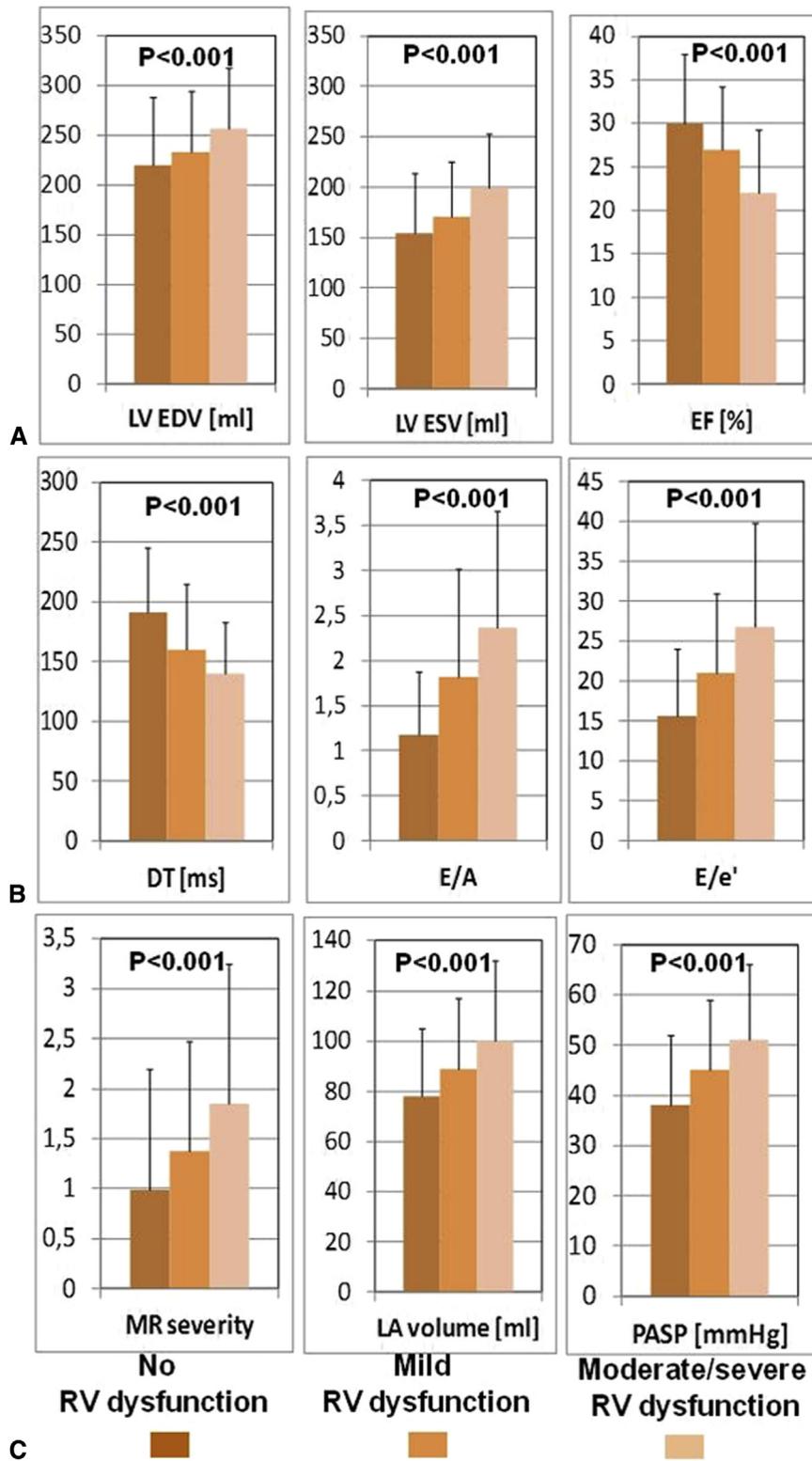


FIGURE 2. Associations between degree of RV dysfunction. A, LV remodeling expressed by LV EDV, ESV, and EF. B, LV diastolic properties represented by DT, E/A, and E/e'. C, Left atrial remodeling expressed by MR severity, left atrial volume, and pulmonary artery systolic pressure (all differences among RV dysfunction subgroups are significant, nonparametric Kruskal–Wallis test). DT, Deceleration time; EF, ejection fraction; LA, left atrial; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; MR, mitral regurgitation; PASP, pulmonary artery systolic pressure; RV, right ventricular. E/A, early/late velocity ratio; E/e', early mitral flow/early annular velocity ratio.

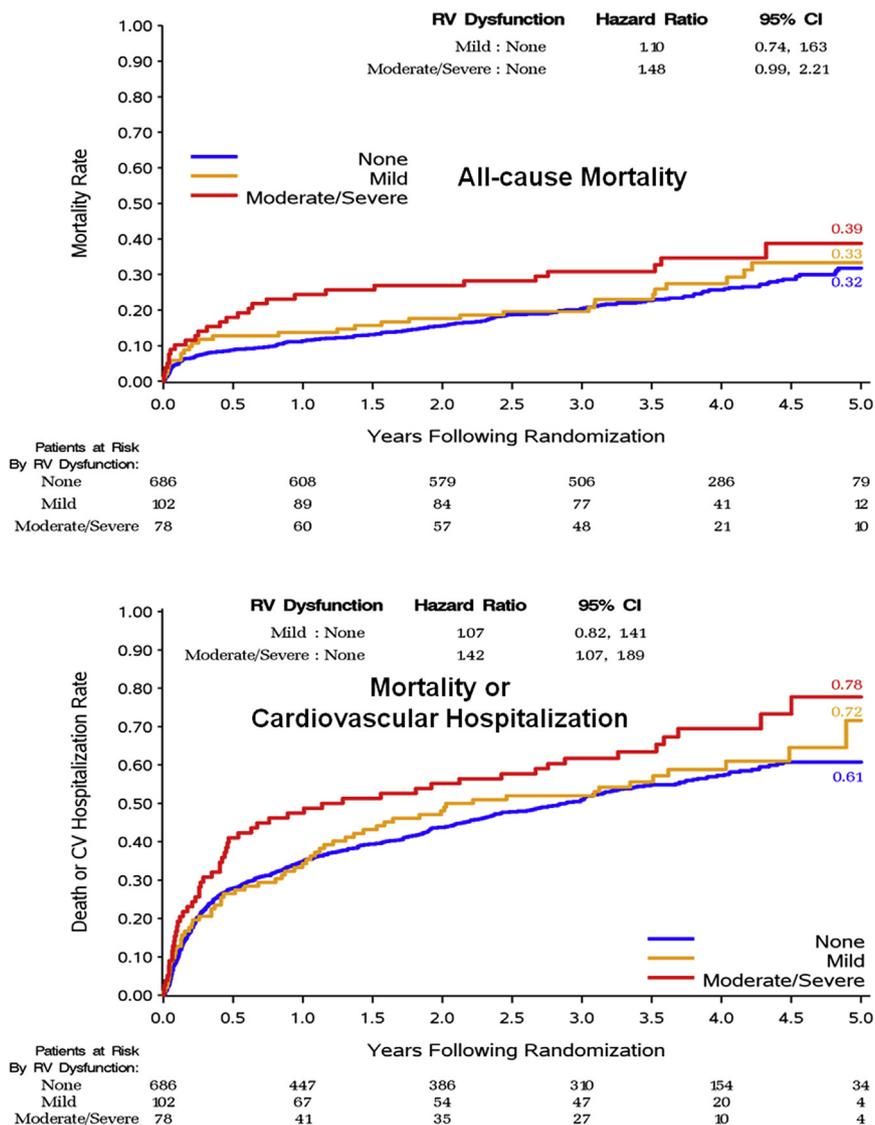


FIGURE 3. Impact of the coexisting RV dysfunction on long-term outcome in the analyzed cohort of STICH patients (n = 866). Kaplan–Meier estimates of event rate by RV dysfunction status. *CI*, Confidence interval; *RV*, right ventricular; *CV*, cardiovascular.

CABG + SVR were not significantly different for the primary (death or CV hospitalization) or the secondary (death) end points (Figure 4, A and B). However, when RV function was moderately or severely reduced, there were significantly higher event rates for CABG + SVR compared with CABG alone for death or CV hospitalization ($P = .028$) and for death alone ($P = .005$) (Figure 4, A and B). There was a statistically significant interaction between RV dysfunction and treatment for the composite end point of death or CV hospitalization ($P = .013$) and for death alone ($P = .001$) because of the markedly higher incidence of clinical outcomes among the patients with moderate/severe RV dysfunction who underwent CABG + SVR. For the death end point, the interaction remained significant even after adjusting for all the other prognostic factors ($P = .022$) (Table 3). There also was a worsening trend for CABG +

SVR with respect to the short-term outcome of surgical death (30-day mortality). Thirty-day mortality among patients with moderately or severely reduced RV function was 5.9% in CABG alone and 13.6% in CABG + SVR, whereas the corresponding 30-day mortality rates among patients with no or mild RV dysfunction were 5.0% for CABG and 4.7% for CABG + SVR.

DISCUSSION

Prevalence of Right Ventricular Dysfunction and Its Association With Left Ventricular Remodeling in Ischemic Cardiomyopathy

This is the largest study to evaluate the prevalence and determinants of RV dysfunction in patients with ischemic cardiomyopathy. In the STICH population, 21% of patients had some degree of RV dysfunction and 9% had moderate

TABLE 3. Prognostic significance of right ventricular dysfunction and the interactive effect of right ventricular dysfunction and treatment: Univariate and multivariable assessments

End points	Main effect of RV dysfunction			Interaction of RV dysfunction and treatment		
	Nonadjusted	Adjusted for LV EF	Adjusted for all prognostic clinical and echocardiography factors*	Nonadjusted	Adjusted for LV EF	Adjusted for all prognostic clinical and echocardiography factors*
Death within 30 d after surgery† (N = 848, events = 45)	0.023	0.164	0.672	0.096	0.152	0.131
All-cause death (N = 866, events = 239)	0.070	0.632	0.711	0.001	0.007	0.022
Death or CV hospitalization (N = 866, events = 506)	0.022	0.281	0.838	0.013	0.041	0.302

Bold indicates $P < .05$. CV, Cardiovascular; EF, ejection fraction; LV, left ventricular; RV, right ventricular. *Prognostic factors identified from previous modeling analyses were used as adjustment variables for the 3 different end points. Atrial flutter/fibrillation, age, mitral regurgitation, creatinine, hemoglobin, end-systolic volume index, and LV EF were included in the adjustment for all 3 end points. In addition to the factors listed, previous myocardial infarction, previous stroke, and New York Heart Association HF class were included for both the death end point and the death or CV hospitalization end point. Canadian Cardiovascular Society angina class was also included in the model for death within 30 days after surgery. Diabetes and hyperlipidemia were also included in the death model. The ability to perform the 6-minute walk test was also included in the death or cardiovascular end point model. †Only patients who actually underwent surgery are included in the analysis of deaths within 30 days after surgery.

or severe dysfunction. Progressively more advanced LV remodeling (larger LV volumes, lower EF, and more severe mitral regurgitation) and worse LV hemodynamic profiles were associated with increasing RV dysfunction. LV systolic and diastolic function parameters, as well as severity of mitral regurgitation, were progressively worse with increasing severity of RV systolic dysfunction.

Mechanisms of Right Ventricular Dysfunction in Ischemic Cardiomyopathy

Several potential underlying mechanisms may explain why progressive RV dysfunction is often accompanied by increasing LV dysfunction.¹⁵ RV dysfunction may reflect primary LV, left atrial, or mitral valve pathology^{16,17} mediated through direct RV compression and the mechanism of RV/LV interdependence or through increased pulmonary pressures and RV afterload due to LV dysfunction.^{18,19} Alternatively, both ventricles might be affected by the same underlying pathologic process (CAD).²⁰ However, the fact that pulmonary artery systolic pressure was found to increase progressively with worsening of RV systolic function suggests that RV dysfunction is related to functional and hemodynamic abnormalities of the LV. This notion is also supported by a recent study of Verhaert and colleagues.²¹ Although many studies have shown that RV dysfunction is a predictor for a poor clinical outcome in patients with HF and reduced LV EF,²² our study is one of the first to demonstrate a relationship between the degree of LV remodeling and the severity of RV dysfunction. The RV is increasingly being recognized as a potential therapeutic target in patients with chronic HF.²³ Although not studied extensively, angiotensin-converting enzyme inhibitors²⁴ and beta-blockers²⁵ seem to improve RV function. Specific therapies targeted indirectly at RV dysfunction in chronic HF have provided

disappointing results.²⁶ Data from Verhaert and colleagues²¹ suggest that RV function may improve when LV abnormalities are treated.

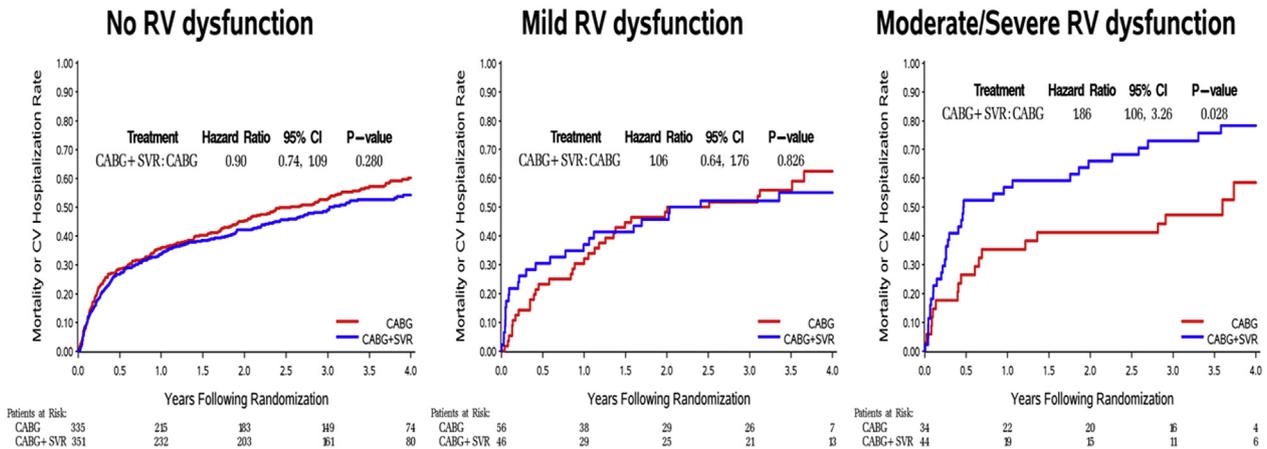
Right Ventricular Dysfunction and Surgical Ventricular Reconstruction

The long-term outcome of the patients who received CABG + SVR in STICH was adversely affected when RV function was at least moderately reduced at baseline. The abrupt reduction in LV size and volume may have increased diastolic stiffness and aggravated diastolic function and filling pressure, especially when LV filling pressure was already markedly elevated at baseline in patients with RV dysfunction. Contrary to the initial belief, we have demonstrated that SVR seems to have a greater benefit in patients with an early stage of ischemic cardiomyopathy and less benefit, or even harm, in patients with a more advanced cardiomyopathy and a larger LV.²⁷ The worse outcome of SVR in the setting of advanced RV dysfunction confirms the earlier report of worse outcome of SVR in patients with a larger LV and more reduced EF associated with RV dysfunction.

Right Ventricular Function Assessment

Many indicators of RV contractility have been proposed (RV EF, RV FAC, tricuspid annular plane systolic excursion [TAPSE], strain, strain rate, RV myocardial performance index, dP/dT max, RV wall motion analysis, tricuspid annular systolic velocity, maximal RV elastance, and Tei index¹⁷), but there is no recognized gold standard imaging modality or parameter for assessment of RV function.²⁸⁻³² Among the numerous quantitative RV parameters studied by Verhaert and colleagues,²¹ only RV systolic strain was predictive of clinical outcome. de Groote and colleagues²² showed radionuclide RV EF but not TAPSE to be the

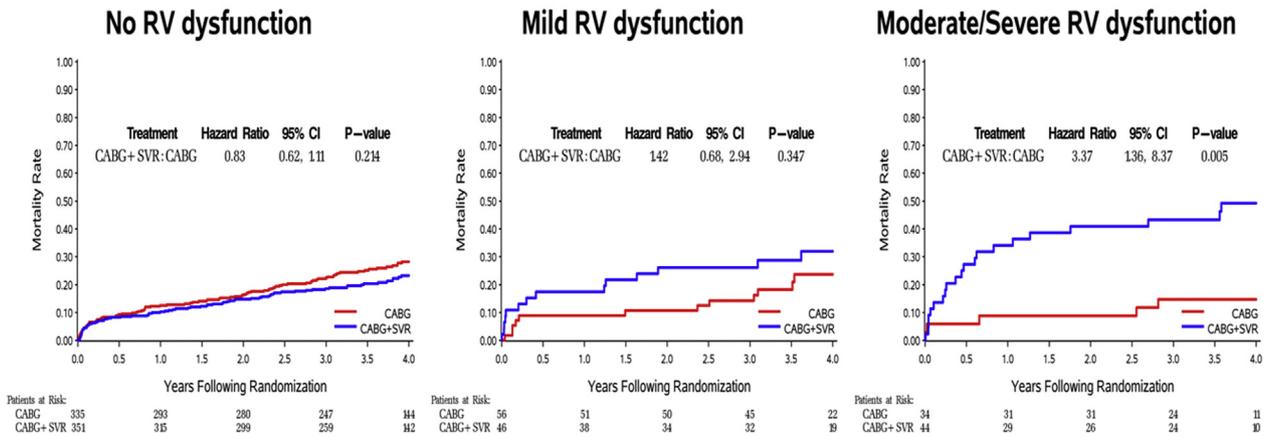
Endpoint=Death or Cardiovascular Hospitalization



Interaction of RV Dysfunction and Treatment (CABG+SVR vs. CABG):
Non-adjusted P-value=0.013
Adjusted P-value=0.302

A

Endpoint=Death



Interaction of RV Dysfunction and Treatment (CABG+SVR vs. CABG):
Non-adjusted P-value=0.001
Adjusted P-value=0.022

B

FIGURE 4. A, Interaction of RV dysfunction and treatment allocation. Kaplan–Meier estimates of event rate by treatment group and LV dysfunction groups. B, Interaction of RV dysfunction and treatment allocation. Kaplan–Meier estimates of event rate by treatment group and LV dysfunction groups. CABG, Coronary artery bypass grafting; CI, confidence interval; CV, cardiovascular; RV, right ventricular; SVR, surgical ventricular reconstruction.

independent predictor of cardiac survival. TAPSE, RV Tei index, RVFAC, and tricuspid systolic annulus velocity were not predictive of long-term outcome³³; however, in the Survival And Ventricular Enlargement trial (SAVE study), RVFAC has been shown to be an independent predictor of mortality and the development of HF in patients with known LV dysfunction.³⁴ Although the optimal method of assessment of RV function is not yet clear,³⁵

the visually and qualitatively assessed RV function used in our study performed well as it related to outcome.

Study Limitations

In our study, moderate to severe RV dysfunction was found in only 9% of the study patients. This may underestimate the true prevalence of the degree of RV dysfunction because patients with significant RV

dysfunction or pulmonary hypertension might have been excluded by study investigators or surgeons because of a perceived high risk for an operation. The evaluation of RV function that we used was a visual assessment. It is difficult to categorize visual assessment, and our classification may not be easily translated to other laboratories. However, differentiation of severe RV dysfunction from normal function is relatively easy by visual assessment. It is more difficult to determine mild and moderate RV dysfunction. However, we believe that our observations are clinically relevant because this visual method is the most practical technique in “real life” practice in almost all echocardiography laboratories. Although the visual assessment of RV function in our study was shown to correlate well with RVFAC, fractional area change was measured after the completion of the trial and once the study results with visual assessment were known. This limitation notwithstanding, the measurements of RVFAC were performed by blinded readers. Also, because a significant proportion of patients could not have RVFAC reliably calculated, it may be that visual assessment, the measure used in this study, is the most widely applicable technique for evaluation of RV function in the general population of patients such as those in the STICH trial.

CONCLUSIONS

In patients with ischemic cardiomyopathy, RV dysfunction is associated with more advanced LV remodeling and hemodynamic abnormalities (larger LV volumes, lower EF, higher filling pressure, and more severe mitral regurgitation). The interaction between RV dysfunction and treatment is significant for mortality after carefully adjusting for other prognostic clinical and echocardiographic factors. When baseline RV function is moderately to severely reduced, the addition of SVR to CABG seems to worsen long-term survival compared with the use of CABG alone.

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