Contribution of Right Ventricular Dysfunction to Heart Failure Mortality: A Meta-Analysis

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Right ventricular systolic dysfunction (RVSD) has been related to prognosis in patients with heart failure (HF) and/or left ventricular systolic dysfunction. However, most of the studies addressing this issue are not large enough, have different inclusion criteria, and use different methods to evaluate RV function to draw definite conclusions. We sought to investigate the association between RVSD and outcomes in patients with left ventricular dysfunction. Eleven studies of 40 (27.5%), with 4732 patients, were included in the meta-analysis. RVSD was present in 2234 patients (47.2%). Four of the studies had admission for HF as an endpoint. We found a significant association between RVSD and overall mortality with significant between-studies heterogeneity and presence of publication bias (funnel plot). A significant association was found between RVSD and admission for HF. RVSD is associated with overall mortality and admission for HF during follow-up. Significant between-studies heterogeneity and publication bias must be taken into account when interpreting this information.

[Rev Cardiovasc Med. *2012;13(2/3):e62-e69 doi: 10.3909/ricm0602*]

KEY WORDS

Right ventricular systolic dysfunction • Left ventricular systolic dysfunction • Heart failure • Outcomes

The proper assessment of right ventricular (RV) systolic function is a difficult task in clinical practice: the geometry of the chamber is complex, longitudinal shortening is a greater contributor to ventricular stroke volume than is short axis shortening, and its performance (very dependent on preload conditions) is linked to the left ventricle (interventricular dependence).[1] In addition, there is no standardized technique to evaluate and quantify RV systolic function, and several methods have been proposed. For these reasons, the right ventricle is frequently overlooked in clinical practice and not routinely evaluated to stratify the risk of patients with heart disease. This is true even when several clinical studies have shown the association of RV systolic dysfunction (RVSD) with clinical outcomes in different clinical scenarios such as heart failure (HF), myocardial infarction,[2] myocarditis,[3] and pulmonary hypertension.[4]

The studies published to date evaluating the prognostic implications of RV function in patients with HF have some limitations: most of them are not large enough to draw definite conclusions; potential bias due to confounding variables are encountered; they used different methods to assess RV function (some of which are not adequately validated); and the cutoff values that define RV dysfunction and outcomes varied between studies. For these reasons we conducted a meta-analysis and systematic review of the literature to explore the prognostic implications of RVSD in patients with HF and/or left ventricular (LV) systolic dysfunction, either of ischemic or nonischemic etiology.

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Methods

Inclusion and Exclusion Criteria

We included studies that analyze the relationship between RV function and outcome in adult patients with chronic LV systolic dysfunction ([LVSD] of ischemic or nonischemic origin) and/or chronic HF. Studies were excluded if (1) overall mortality or admission for HF during follow-up could not be extracted from the data reported or after establishing contact with the corresponding author; (2) a clear cutoff value to define RV dysfunction was not used to divide the whole cohort into groups; (3) only patients with acute heart disease were included (ie, acute myocardial infarction); and (4) duplicate data were provided.

Literature Search

The studies were obtained by searching the MEDLINE and Scopus databases in the month of January 2010 using the following terms: *RV dysfunction* AND *mortality*, and *RV dysfunction* AND *admission HF*. Initial selection of the studies was made according to the title and the abstract, and all those apparently not related with the aim of the meta-analysis were discarded. We excluded studies assessing the relationship between RVSD and outcomes in patients who underwent cardiac transplantation or the implantation of LV assist devices or the influence of RV function in the evaluation of cardiovascular drugs.

Three reviewers read the remaining studies independently and analyzed the literature references they contained. The following data were extracted from each of the original studies: number of patients included, etiology of the heart disease, definition and method used to define RV dysfunction, number of patients with RV dysfunction, mean/median follow-up and deaths, and/or admission for HF during follow-up.

Data Analysis

The two endpoints of the meta-analysis were overall mortality and admission for HF during follow-up. The main measures of association were the odds ratio (OR) and 95% confidence interval (CI). The ORs for the individual studies were combined using the fixed-effects model (Mantel-Haenszel method[5]) or the random-effects model (DerSimonian and Laird test[6]), depending on the results of the heterogeneity analysis. Between-studies heterogeneity was assessed with the χ^2 test and the Galbraith plot.[7] Briefly, the Z statistic (the outcome for each study divided by the square root of its variance) was plotted against the inverse of the standard error of each study in the x-axis. An unweighted regression line constrained through the origin was constructed. The studies farthest from this line (outliers), which have a standard deviation of 1, are those that contribute the most to between-studies heterogeneity.

To assess the presence of publication bias, a funnel plot was constructed, and publication bias was further analyzed with the Galbraith plot (the intercept of the weighted regression line with the y-axis).

An analysis of sensitivity was carried out to assess the influence of each of the original studies on the final result and the effect of the studies that contribute the most to between-studies heterogeneity. Statistical significance for the effects of RV dysfunction on mortality/admission for HF and heterogeneity was established at $P < .05$ and $P < .1$, respectively.

Results

Studies Included

Forty studies were retrieved for analysis, 29 of which were excluded after thorough evaluation: 14 (35.0%) were not related to the aim of the meta-analysis[2,3,8-19]; 8 (20.0%) had a combined endpoint not evaluating total mortality[20-27] (one study[28] was included because the data on total mortality were obtained after establishing contact with the corresponding author); 4 (10.0%) did not have a cutoff value for defining RV dysfunction[29-32]; and 3 (7.5%) provided duplicate data.[33-35] Finally, 11 (26.8%) studies were included, 4 of which had readmission for HF as an endpoint[2,28,36,37] (see the baseline characteristics of the included studies in Table 1).

Qualitative Analysis

The total number of patients recruited was 4732, of which 2234 (47.2%) had RV dysfunction. The baseline characteristics of the studies are shown in Table 1. Seven (63.6%) studies[28,37-42] included patients with either ischemic or nonischemic heart disease, three (27.3%)[2,36,43] included only those with LVSD and/or heart failure of ischemic origin, and one (9.1%) included patients with Chagas cardiomyopathy.[44] The methods used to assess RV function were echocardiography (n = 7),[2,28,36,39-41,44] isotopic ventriculography (n = 3),[37,38,43] or invasive thermodilution techniques (n = 1),[42] with the cutoff values shown in Table 1. In three studies (27.3%),[2,36,44] patients without symptoms of HF could be included; in three other studies (27.3%), patients with normal or relatively preserved LV ejection fraction could be included.[36,41,44]

Quantitative Analysis

Overall Mortality. The results of the combined analysis are shown in the forest plot (Figure 1). We found a significant association between RV dysfunction and overall mortality in the follow-up (OR = 2.98; 95% CI, 2.02-4.39; $P < .001$). We found significant between-studies heterogeneity ($\chi^2 = 41.96$; $P < .001$), primarily based on the results of four publications (see the Galbraith plot, Figure 2).[2,39,41,44] The funnel plot (Figure 3) suggests publication bias, which was also detected in the Galbraith plot, because the weighted regression analysis of the effects of the individual studies (dashed line) intercepts the *y*-axis at 2.65 (95% CI, 0.95-4.32), which is statistically different from zero ($P = .002$).

We repeated the meta-analysis excluding each individual study in turn (Table 2) and the four studies that contributed more to between-studies heterogeneity. All the results indicate a strong association between RV dysfunction and mortality that was higher when the larger studies were excluded.

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Admission for Heart Failure. The overall effect of the four studies (fixed-effects model, Figure 4)[2,28,36,37] that analyze this endpoint was 1.51 (95% CI, 1.27-1.79; $P < .001$). No significant between-studies heterogeneity was found ($\chi^2 = 3.69$; $P = .30$).

Discussion

The results of this study show an association between RVSD and outcomes in patients with LVSD and/or symptomatic HF. However, caution must be exercised when interpreting the data because no definite cause-and-effect relationship can be established, and we detected significant between-studies heterogeneity and a high probability of publication bias.

Possible sources of heterogeneity can be argued even when the same parameter is used to quantify it, the most important being different inclusion criteria, different methods to assess RV function, and different definitions of RVSD. The wide range of prevalence of RVSD found in the meta-analysis (23.6% in the study by Nunes Mdo and colleagues[44] to 77.2% in the study by Anavekar and colleagues[36]) can be explained by the reasons mentioned above. Sensitivity analysis is an approach to test how robust the results obtained are. When we excluded each of the studies in turn and the four studies that contribute more to heterogeneity, we detected changes in the magnitude of the association, which was always statistically significant, confirming a deleterious effect of RV dysfunction. These data reinforce the idea of RV dysfunction as an important prognostic indicator in this group of patients even when between-studies heterogeneity is considered.

Assessment of RV Function

The best method of assessing RV function is still a matter of debate. Due to its widespread availability, echocardiography is used as the first-line imaging modality. Various echocardiography-derived parameters (including three-dimensional echocardiography and strain rate imaging) have been described for right ventricle assessment. Some

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TABLE 1

Baseline Characteristics of the Included Studies

Study	N	Inclusion	Assessment RV Function	Cutoff Value for RV Dysfunction	Patients With RV Dysfunction (%)	Median Follow-up	Etiology
Zornoff LA et al.[2]	416	LVEF ≤ 40% free of HF	FAC (echo)	FAC ≤ 32.2%	19.0	2.6 y	Ischemic and nonischemic
Bistola V et al.[28]	102	LVEF < 35%, NYHA class III/IV	S-wave DTI tricuspid annulus (echo)	S-wave < 7.3	50.9	6 mo	Ischemic and nonischemic
Anavekar NS et al.[36]	522	Signs/symptoms of HF or LVEF ≤ 35% or LVEF ≤ 40% contrast ventriculography	FAC (echo)	FAC ≤ 45%	77.2	24.7 mo	Ischemic
Meyer P et al.[37]	2008	LVEF ≤ 35%, NYHA class III/IV	Radionuclide ventriculography	RVEF < 40%	63.5	24 mo	Ischemic and nonischemic
Di Salvo TG et al.[38]	67	Referred of evaluation of cardiac transplantation; mean LVEF = 22%	Radionuclide ventriculography	RVEF < 35%	64.2	58 wk	Ischemic and nonischemic
Karatasakis GT et al.[39]	40	LVEF < 30%	Echo longitudinal RV shortening	< 1.25	40.0	13.7 mo	Ischemic and nonischemic
Kjaergaard J et al.[40]	817	HF and LVEF ≤ 35%	TAPSE (echo)	< 14 mm	27.8	4.1 y	Ischemic and nonischemic
Dini FL et al.[41]	142	LVEF ≤ 45%, LVEDV > 75 mL/m^2 and moderate/severe mitral regurgitation	TAPSE (echo)	< 16 mm	47.9	20 mo[a]	Ischemic and nonischemic
Ghio S et al.[42]	377	LVEF < 35%	Thermodilution	RVEF < 35%	75.1	17 mo	Ischemic and nonischemic
Polak JF et al.[43]	34	LVEF < 40%	Radionuclide ventriculography	RVEF < 35%	61.8	2 y	Ischemic
Nunes Mdo C et al.[44]	140	LVEDD > 31 m/m^2 and LVEF < 55%	MPI (echo)	> 0.56	23.6	30 mo	Nonischemic (Chagas disease)

[a]Mean follow-up.

DTI, Doppler tissue imaging; Echo, echocardiographic; FAC, fractional area change; HF, heart failure; LVEDD, left ventricular end-diastolic dimension; LVEF, left ventricular ejection fraction; MPI, myocardial performance index; NYHA, New York Heart Association; RV, right ventricular; RVEF, right ventricular ejection fraction; TAPSE, tricuspid annular plane systolic excursion.

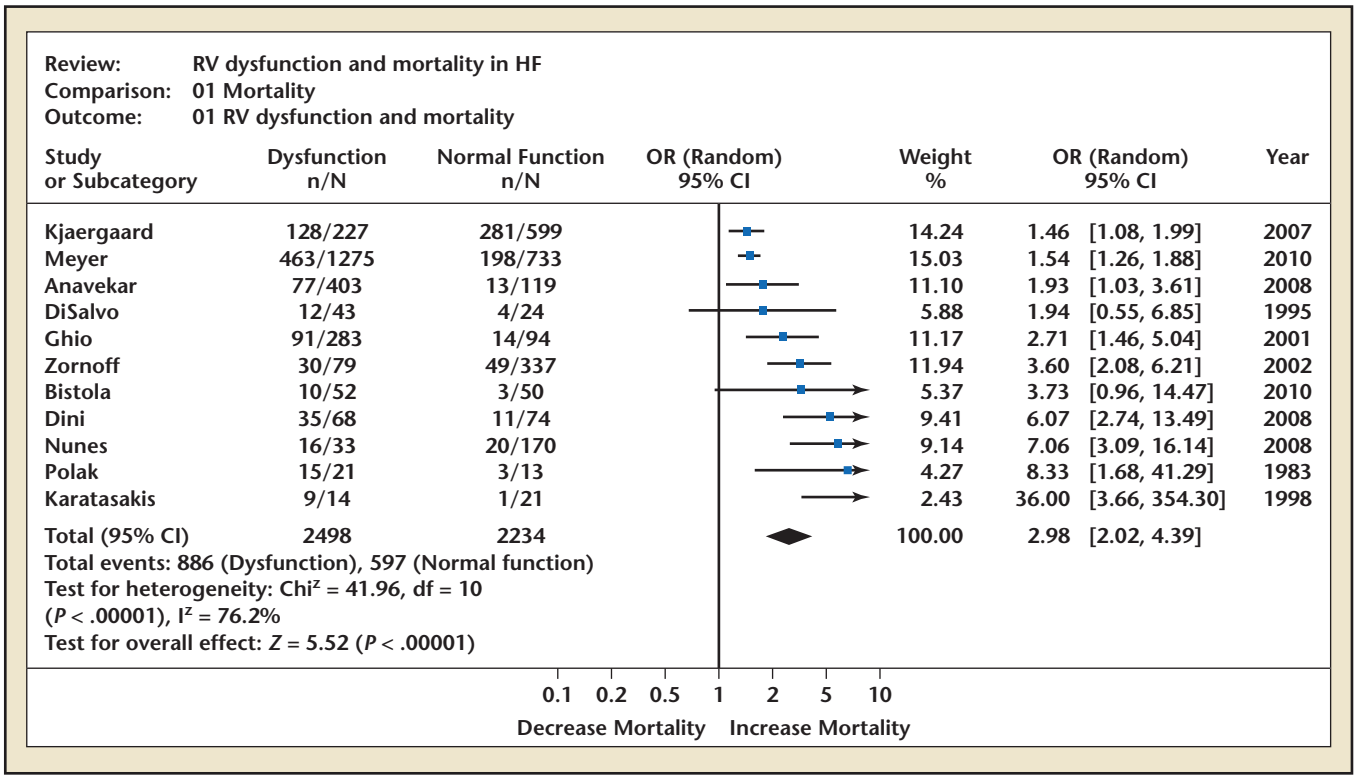

Figure 1. Forest plot: association between RV systolic dysfunction and overall mortality. CI, confidence interval; df, degrees of freedom; HF, heart failure; OR, odds ratio; RV, right ventricular.

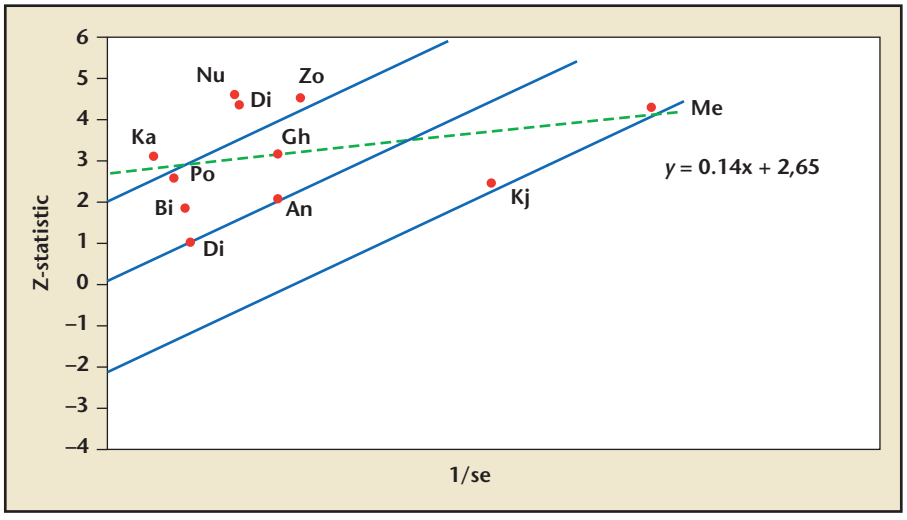

Figure 2. Galbraith plot.

of them are highly correlated with RV ejection fraction.[45,46] Their utility in determining outcome has been analyzed in a recently published study: Damy and associates[27] reported that the peak systolic velocity of the tricuspid annulus (evaluated with tissue Doppler imaging) seems to be superior to its integral, right fractional area change or tricuspid annular plane systolic excursion in the prediction of outcomes for patients with HF. However, it seems clear from the results of the meta-analysis that all methods of quantifying RV function are useful in assessing the risk of this group of patients.

Although cardiac magnetic resonance is considered the most accurate method for assessment of the right ventricle, none of the included studies used this technique. First-pass radionuclide ventriculography was used in two studies,[38,43] which both show an association between RV function and outcomes. Recent advances in radionuclide techniques could contribute to a better understanding of the RV function (systolic and even diastolic) in this clinical scenario.[47] The combination of echocardiography-derived parameters that assess both systolic and diastolic RV performance has been suggested to enhance the prognostic information.[21,22,44] This interesting information should be confirmed in large prospective cohort studies. Funnel and Galbraith plot analyses show a high possibility of publication bias: positive studies (those demonstrating a relationship between RV dysfunction and outcomes), have a higher probability of being published. The analysis of the Galbraith plot suggests that bigger studies show a smaller association.

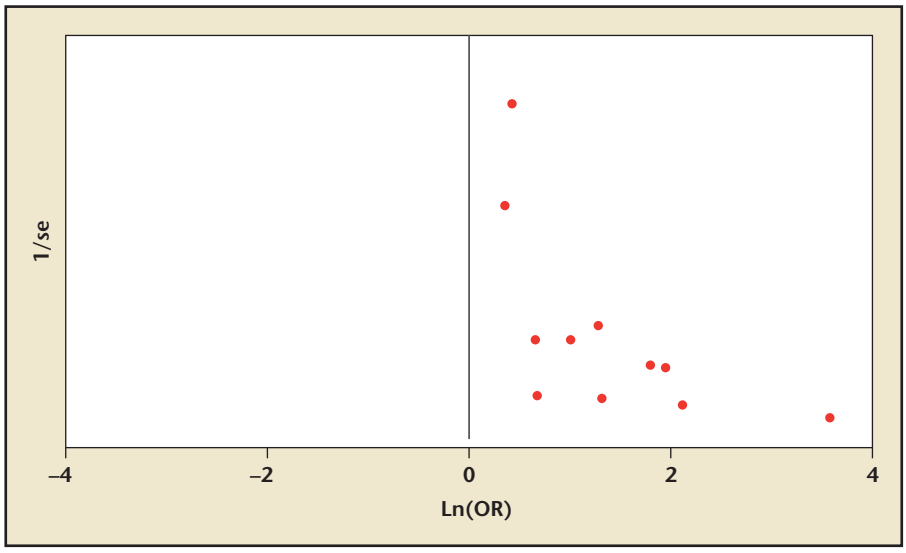

Figure 3. Funnel plot. OR, odds ratio.

TABLE 2

Sensitivity Analysis

Study	Studies	N	OR (95% CI)[a]
Zornoff LA et al.[2]	10	4316	2.90 (1.92-2.37)
Bistola V et al.[28]	10	4630	2.95 (1.97-4.41)
Anavekar NS et al.[36]	10	4215	3.21 (2.08-4.94)
Meyer P et al.[37]	10	2724	3.45 (2.16-5.50)
Di Salvo TG et al.[38]	10	4670	3.09 (2.05-4.65)
Karatasakis GT et al.[39]	10	4697	2.76 (1.91-4.00)
Kjaergaard J et al.[40]	10	3906	3.47 (2.16-5.57)
Dini FL et al.[41]	10	4595	2.71 (1.85-3.97)
Ghio S et al.[42]	10	4360	3.06 (2.00-4.69)
Polak JF et al.[43]	10	4698	2,83 (1.92-4.17)
Nunes Mdo C et al.[44]	10	4529	2.65 (1.83-3.85)
Excluding 4 studies more contributing to heterogeneity (Zornoff LA et al,[2] Karatasakis GT et al,[39] Kjaergaard J et al,[40] and Nunes Mdo C et al.[44])	7	3936	1.66 (1.43-1.93)[b]

[a]Random effects model.
[b]Fixed effects model.
CI, confidence interval; OR, odds ratio.

Cause-and-Effect Relationship

Although a cause-and-effect relationship between RVSD and outcome in the population under study cannot be definitively drawn, some clues extracted from the published studies could be useful to address this issue. First, a temporal sequence between LV systolic function and RV dysfunction, and eventual outcome, can be suspected. The relationship between the functions of both ventricles is not merely the result of the development of passive pulmonary hypertension[48]; complex heart–lung interactions resulting in proliferative pulmonary vascular disease,[49] systolic and diastolic ventricular interdependence, neurohormonal interactions, RV ischemia, and individual susceptibility can be contributory factors.[1,50] Moreover, low RV systolic function may also be a cause of further LV systolic impairment and disease progression.[1] For these reasons, RVSD in this context generally implies more advanced disease; as a consequence, it is biologically plausible

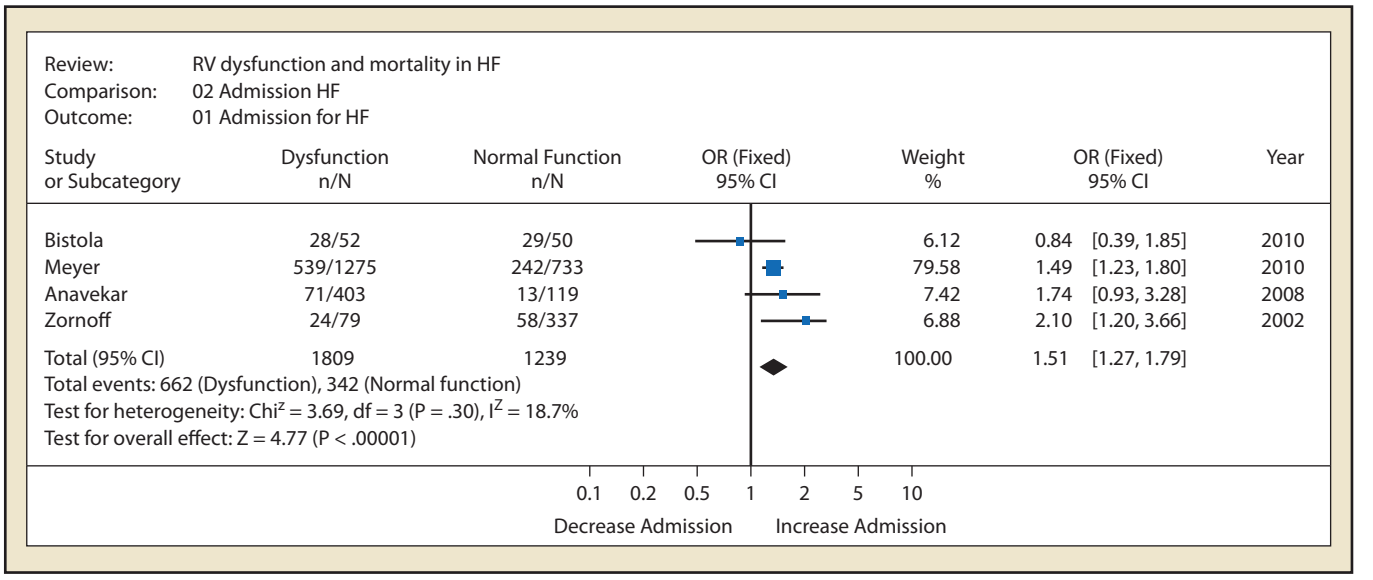

Figure 4. Forest plot: association between RV systolic dysfunction and admission for HF. CI, confidence interval; df, degrees of freedom; HF, heart failure; OR, odds ratio; RV, right ventricular.

to think that this process leads to poor prognosis. The association is strong and consistent: most of the studies[2,28,36-41] (but not all[29,31]) describe in somewhat different clinical scenarios that RV function is a predictor of poor outcome and that the relationship is not weak, even after adjusting for confounding baseline characteristics. Finally, a gradient of RVSD and prognosis has been suggested in some studies[30,37]: the lower the RV function, the poorer the outcome. Taken together, these five points strongly support a causal link between RVSD and outcomes in the group of patients included in this meta-analysis.

RV Function in HF With Preserved Systolic Function

Some of the studies could include patients without LV systolic dysfunction.[2,44] Even in this group of patients, RV dysfunction maintains its prognostic value, suggesting that RVSD is (per se) the factor associated with outcome. The prognostic implications of RV function in patients with HF and preserved LV systolic function cannot be extracted from this meta-analysis, and should be the aim of a well-designed prospective study.

Pulmonary Pressure and RV Function

Arterial pulmonary pressure is a major determinant of RV function, and this is one of the proposed mechanisms to explain RV failure in these patients. Ghio and associates[42] suggested that it is only in instances of pulmonary hypertension that prognosis is related to RV performance. As such, a link between these variables can exist that deserves special investigation.

Conclusions

RVSD is associated with overall mortality and hospital admission for HF in patients with LV systolic dysfunction and/or HF. There is no consensus on how to evaluate RV function and what the appropriate cutoff values to define RVSD are. Significant between-studies heterogeneity and publication bias were detected and should be considered when interpreting the data, although an independent association between RVSD and outcomes can be reasonably suspected. ■

Dr. Ignacio Iglesias-Garriz and Dr. Cristina Olalla-Gómez designed the study, collected data, generated the statistics, and prepared the manuscript. Dr. Carmen Garrote, Dr. María López-Benito, and Dr. Julia Martín participated in data collection and preparation of the manuscript, and Dr. David Alonso and Dr. Miguel A. Rodríguez made a critical evaluation of the data and review of the final manuscript.

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MAIN POINTS

- Right ventricular systolic dysfunction (RVSD) has been related to prognosis in patients with heart failure (HF) and/or left ventricular systolic dysfunction (LVSD). However, there is no standardized technique to evaluate and quantify RVSD.
- There is an association between RVSD and outcomes in patients with LVSD and/or symptomatic HF, although caution must be exercised when interpreting the data because no definite cause-and-effect relationship can be established.
- The best method of assessing RV function is still a matter of debate, although echocardiography is used as the first-line imaging modality due to its widespread availability.