



ORIGINAL RESEARCH

Right Atrial Volume Index in Chronic Systolic Heart Failure and Prognosis

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OBJECTIVES The aim of this study was to determine the relationship between right atrial volume index (RAVI) and right ventricular (RV) systolic and diastolic function, as well as long-term prognosis in patients with chronic systolic heart failure (HF).

BACKGROUND RV dysfunction is associated with poor prognosis in patients with HF, although echocardiographic assessment of RV systolic and diastolic dysfunction is challenging. The ability to visualize the RA allows a quantitative, highly reproducible assessment of the RA volume that can be indexed to body surface area.

METHODS The ADEPT (Assessment of Doppler Echocardiography for Prognosis and Therapy) trial enrolled 192 subjects with chronic systolic HF (left ventricular ejection fraction [LVEF] $\leq 35\%$). The RA volume was calculated by Simpson's method using single-plane RA area and indexed to body surface area (RAVI). RV systolic function was graded as normal, mild, mild-moderate, moderate, moderately severe, or severe dysfunction.

RESULTS In our study cohort, the mean RAVI was 28 ± 15 ml/m², and increased with worsening RV systolic dysfunction, LVEF, and LV diastolic dysfunction (Spearman's $r = 0.61$, $r = 0.26$, and $r = 0.51$, respectively; $p < 0.001$ for all). RAVI correlated modestly with echocardiographic estimates of RV diastolic dysfunction, including tricuspid early/late velocities ratio (Spearman's $r = 0.34$, $p < 0.0001$), hepatic vein systolic/diastolic ratio (Spearman's $r = -0.26$, $p < 0.001$) but not tricuspid early/tricuspid annular early velocities ratio (E/Ea) (Spearman's $r = 0.12$, $p = 0.11$). Increasing tertiles of RAVI were predictive of death, transplant, and/or HF hospitalization (log-rank $p = 0.0002$) and remained an independent predictor of adverse clinical events after adjusting for age, B-type natriuretic peptide, LV ejection fraction, RV systolic dysfunction, and tricuspid E/Ea ratio (hazard ratio: 2.00, 95% confidence interval: 1.15 to 3.58, $p = 0.013$).

CONCLUSIONS In patients with chronic systolic HF, RAVI is a determinant of right-sided systolic dysfunction. This quantitative and reproducible echocardiographic marker provides independent risk prediction of long-term adverse clinical events. (J Am Coll Cardiol Img 2009;2:527–34) © 2009 by the American College of Cardiology Foundation

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Right ventricular (RV) systolic dysfunction is a common abnormality in patients with heart failure (HF) (1,2), pulmonary hypertension (2), pulmonary embolism, and chronic obstructive pulmonary disease (3). Abnormalities in RV systolic function are often associated with poor long-term prognosis (4–6), and therefore, accurate assessment is important in risk prediction. However, precise clinical assessment of abnormalities in RV structure and performance is challenging, primarily because the morphology of the RV is complicated and indices of RV function are often poorly visualized by standard echocardiographic techniques.

The ability to visualize the right atrium (RA) allows a quantitative, highly reproducible assessment of the RA volume that can be indexed to body surface area (7,8). We hypothesized that RA volume can serve as a quantitative marker of RV dysfunction severity. The aim of our study is to determine the relationship between right atrial volume index (RAVI) and echocardiographic indices of left and right side systolic and diastolic performance, as well as long-term prognosis for patients with chronic systolic HF.

METHODS

Study population. We enrolled 192 consecutive ambulatory patients in the ADEPT (Assessment of Doppler Echocardiography for Prognosis and Therapy) study who were referred to the department of cardiovascular medicine at the Cleveland Clinic for evaluation of symptomatic (New York Heart Association [NYHA] functional class II to IV) chronic systolic heart failure

(9), in whom echocardiographic evaluation allowed reliable measurement of RA volume. Eligible patients were 18 to 75 years of age, with left ventricular ejection fraction (LVEF) \leq 35%. Patients were excluded by a history of mitral stenosis or mitral valve surgery, severe mitral regurgitation ($>3+$), or severe aortic stenosis (peak velocity >4 m/s) or aortic regurgitation. The investigators verified clinical data collected from the hospital chart. B-type natriuretic peptide (BNP) was measured by the Christchurch assay, as previously described (9), with values slightly lower than commercial BNP assays. Adverse events (all-cause mortality, cardiac transplantation, or HF hospitalization) were prospec-

tively tracked by scheduled telephone follow-up and validated by chart review. This study was approved by the Cleveland Clinic Institutional Review Board and all subjects provided informed consent.

Transthoracic echocardiography. Comprehensive transthoracic echocardiography was performed using commercially available HDI 5000 (Phillips Medical Systems, N.A., Bothell, Washington) and Acuson Sequoia (Siemens Medical Solutions USA, Inc., Malvern, Pennsylvania) machines. Two-dimensional and color Doppler imaging were performed in standard parasternal and apical views. Left ventricular systolic and diastolic indexes were acquired as previously outlined in the ADEPT trial (9). All images were stored on magneto-optical disc and were measured offline later by an independent investigator who was blinded to the clinical data (9).

All measures of right-side heart structure and performance were averaged over 3 cardiac cycles (5 cycles for atrial fibrillation [AF]). RA area was measured in the apical 4-chamber view at end systole. RAVI was calculated using Simpson's method, in the 4-chamber view, and indexed to body surface area (7,8). The RV was assessed in the apical 4-chamber, parasternal short-axis and subcostal views. RV systolic function was graded on a scale from 0 to 5 based on a semiquantitative visual estimate of normal, mild, mild-moderate, moderate, moderately severe, or severe dysfunction (9). Right ventricular indexes were acquired over 10 consecutive beats using sweep speeds of 50 and 100 cm/s. With pulsed-wave Doppler, we acquired transtricuspid flow using a 1- to 2-mm sample volume placed at the tricuspid leaflet tips in the apical 4-chamber view and hepatic vein flow using a 3- to 4-mm sample volume placed in the hepatic vein 1 cm from the junction of the RA and hepatic vein. Tricuspid annular tissue Doppler imaging (TDI) was acquired with standard pre-sets optimized to eliminate background noise and enhance tissue signals using a 5-mm sample volume placed at the lateral and tricuspid annulus in the 4-chamber view. Measurements from pulsed-wave Doppler included transtricuspid peak E (early) and A (late) velocities, early deceleration time (DT), hepatic vein systolic and diastolic flows, and peak tricuspid annular systolic early (Ea) and late (Aa) velocities. The RV diastolic dysfunction was assessed by echocardiographic measures of tricuspid velocities, including tricuspid E/A ratio, RA area, and hepatic vein systolic/diastolic (S/D) ratio. Because there is no prior classification scheme, RV diastolic dysfunction stage was arbitrarily defined

ABBREVIATIONS AND ACRONYMS

BNP = B-type natriuretic peptide

E/A ratio = tricuspid early/late velocities ratio

E/Ea ratio = tricuspid early/tricuspid annular early velocities ratio

HF = heart failure

HR = hazard ratio

LV = left ventricular

LVEF = left ventricular ejection fraction

RA = right atrium

RAVI = right atrial volume index

ROC = receiver-operator characteristic

RV = right ventricle/ventricular

S/D = hepatic vein systolic/diastolic ratio

TDI = tissue Doppler imaging

on the basis of these echocardiographic measures: stage 0 (normal) was tricuspid E/A ratio 1 to 2 and RA area ≤ 15 cm²; stage I (abnormal relaxation) was tricuspid E/A ratio < 1 ; stage II (pseudonormal) was tricuspid E/A ratio 1 to 2 and RA area > 15 cm²; and stage III (restrictive) was tricuspid E/A ratio ≥ 2 .

Statistical analysis. RA volume and RAVI were treated as non-normally distributed variables and were assessed by nonparametric methods. The Jonckheere-Terpstra test was used to test trends in RAVI across RV systolic dysfunction severity, RV diastolic dysfunction severity, and LV diastolic dysfunction severity. Receiver-operator characteristic (ROC) analysis was used to determine the sensitivity and specificity of RAVI in predicting HF disease severity. Optimal ROC cutoffs were chosen at values maximizing the summation of sensitivity and specificity as provided by JMP 7.0 (SAS Institute, Cary, North Carolina). Spearman's correlation coefficient was used to evaluate significant relationships between clinical and echocardiographic variables and RAVI. For linear regression analysis, a stepwise variable selection procedure was used to select variables significantly related with RAVI with a probability of 0.1 to enter or leave. For outcomes analysis, Cox proportional hazards models included RAVI modeled as a continuous variable along with other known predictors (age, BNP, LVEF, and RV systolic dysfunction, LV diastolic dysfunction, and left atrial volume index). With a total of 192 patients (62 events), this study had a power of 80% to detect a hazard ratio (HR) of 1.43 per standard deviation of RAVI. The power computation was based on a type I error of 0.05. The proportional hazards assumption was verified with log[time] versus log(-log[survival]) plots. The functional forms of the predictors were assessed by checking the martingale residuals. The goodness of fit was assessed by using the chi-square test. Time-dependent ROC analysis was used to determine the sensitivity and specificity of RAVI in predicting adverse clinical events (10). A p value < 0.05 was considered statistically significant. Statistical analyses were performed using JMP 7.0 and SAS 9.1.3 (SAS Institute).

RESULTS

Study population. Baseline clinical and echocardiographic parameters are shown in Table 1. In our study population, the overall mean RA volume was 57 ± 31 ml, with mean RA volume higher in men

Table 1. Baseline Characteristics

Variable	Value
Demographic data	
Mean age, yrs	57 \pm 13
Male, %	72
NYHA functional class III to IV, %	41
Medical history, %	
Ischemic heart failure etiology	41
History of hypertension	51
History of diabetes mellitus	26
History of atrial fibrillation	24
Echocardiographic data	
LVEF, % U	25 \pm 6
LV end-diastolic dimension, cm	6.4 \pm 0.9
LV end-diastolic volume index, ml/m ²	115 \pm 41
LV end-systolic volume index, ml/m ²	87 \pm 35
LA volume index, ml/m ²	42 \pm 17
PA systolic pressure, mm Hg	38 \pm 15
Diastolic stage I/II/III, %	40/26/34
Septal mitral annulus TDI	
Sa wave	4.6 \pm 1.7
Ea wave	4.7 \pm 2.2
Aa wave	6.8 \pm 2.8
Mitral E/Ea ratio	19 \pm 12
RA area, cm ²	20 \pm 7
RA volume index, ml/m ²	28 \pm 15
Tricuspid Doppler flow	
E wave, cm/s	45 \pm 14
A wave, cm/s	39 \pm 12
Deceleration time, ms	206 \pm 60
E/A ratio	1.2 \pm 0.5
Lateral tricuspid annulus TDI	
Sa wave, cm/s	9.9 \pm 3.4
Ea wave, cm/s	8.3 \pm 3.1
Aa wave, cm/s	11.5 \pm 4.9
Tricuspid E/Ea ratio	6.3 \pm 3.8
Hepatic vein peak S/D ratio	1.3 \pm 0.6
RV systolic dysfunction stage $\geq 3+$	26
Mitral regurgitation $\geq 3+$	9.4
Tricuspid regurgitation $\geq 3+$	6.3
Laboratory data	
BNP, Christchurch assay, pg/ml	67 (27-158)
eGFR, ml/min/1.73m ²	69 \pm 24

A = late velocity; Aa = annular late velocity; BNP = B-type natriuretic peptide; E = tricuspid early velocity; Ea = tricuspid annular early velocity; eGFR = estimated glomerular filtration rate; LA = left atrial; LV = left ventricular; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association; PA = pulmonary artery; RA = right atrial; RV = right ventricular; S/D = systolic/diastolic; Sa = annular systolic velocity; TDI = tissue Doppler imaging.

than in women (64 ± 31 ml vs. 40 ± 24 ml, respectively, $p < 0.0001$). When these volumes were indexed to body surface area, the mean and median levels were 28.4 ± 15.3 ml, and mean RAVI was 31 ± 15 ml/m² in men and 21 ± 12 ml/m² in women ($p < 0.0001$). Mean RAVI was 18

Table 2. Univariable and Multivariable Correlation Analysis of Echocardiographic Variables With Indexed Right Atrial Volume (Overall $r = 0.70$, $p < 0.0001$)

Variable	Univariable Analysis		Multivariable Analysis	
	Spearman's r	p Value	Standardized β	p Value
LVEF	-0.26	0.0004	0	—
LV end-diastolic dimension	0.14	0.0546	0	—
LV diastolic dysfunction stage	0.51	<0.0001	0	—
RV systolic dysfunction stage	0.61	<0.0001	0.38	<0.0001
RV diastolic dysfunction stage	0.63	<0.0001	0.42	<0.0001
PA systolic pressure	0.46	<0.0001	0	—
TV Doppler flow				
E wave	0.08	0.2727	0	—
A wave	-0.34	<0.0001	0	—
E/A ratio	0.34	<0.0001	0	—
TV TDI				
Sa wave	-0.37	<0.0001	0	—
Ea wave	-0.07	0.3231	0	—
Aa wave	-0.43	<0.0001	0	—
TV E/Ea ratio	0.12	0.1065	0	—
Hepatic vein				
S wave	-0.29	0.0002	0	—
D wave	0.07	0.4089	0	—
Peak S/D ratio	-0.26	0.0009	0	—

TV = tricuspid valve; other abbreviations as in Table 1.

± 7 ml/m² in patients with normal RV systolic function, and 15 ± 5 ml/m² in patients with normal RV diastolic function. Forty-seven (24%) of 192 patients had AF (35 paroxysmal, 4 permanent, 8 persistent), and patients with AF had higher RAVI (34 ± 17 ml/m² vs. 27 ± 14 ml/m², $p = 0.0012$).

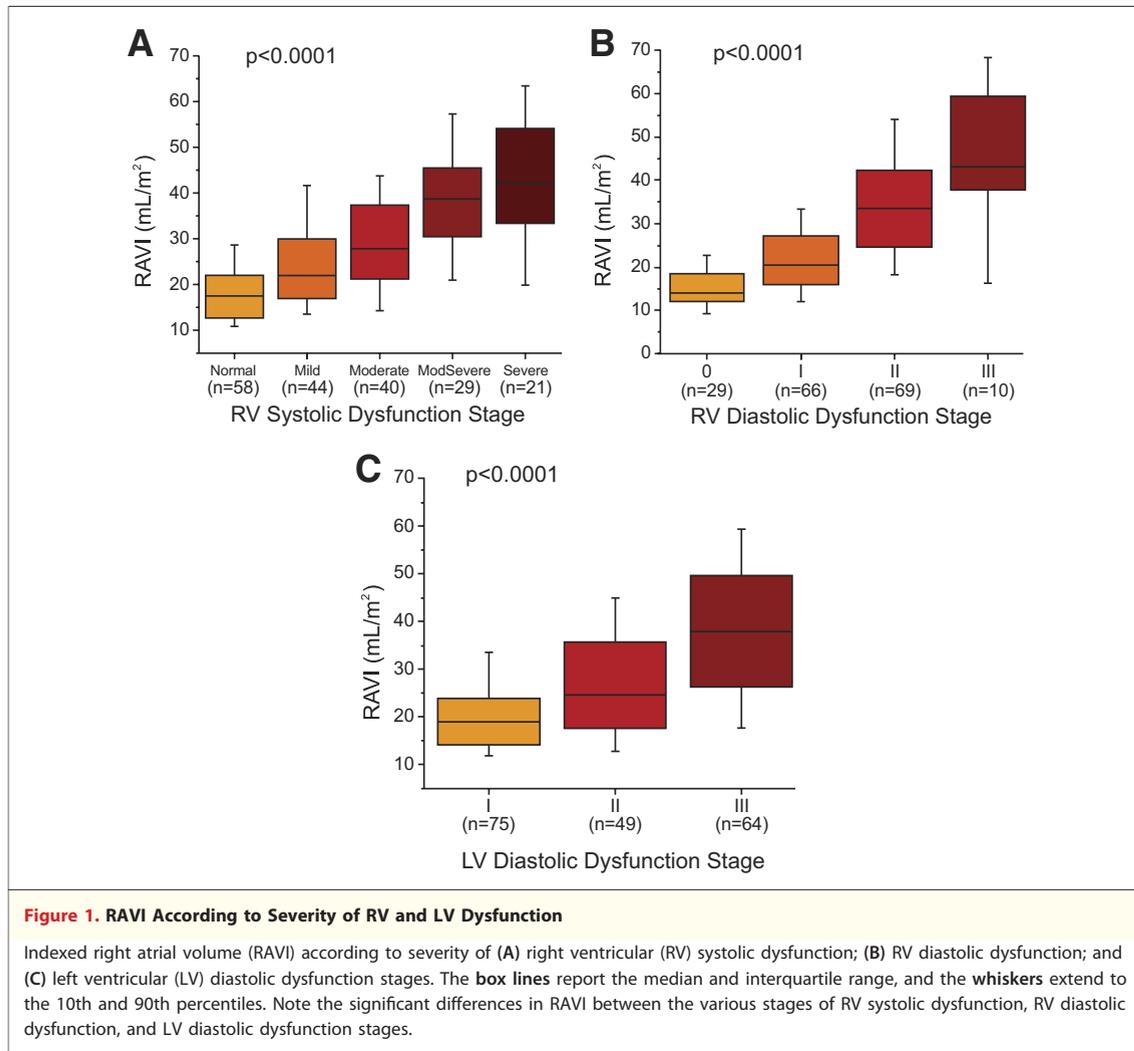
In ROC analysis, $RAVI \geq 41.6$ ml/m² (optimal ROC cutoff) had a 68% sensitivity and a 92% specificity ($p = 0.0004$) for predicting NYHA functional class $\geq III$; $RAVI \geq 30.6$ ml/m² (optimal ROC cutoff) had a 78% sensitivity and a 77% specificity ($p < 0.0001$) for predicting RV systolic dysfunction stage ≥ 3 ; and $RAVI \geq 37.8$ ml/m² (optimal ROC cutoff) had a 80% sensitivity and a 80% specificity ($p = 0.0002$) for predicting RV diastolic dysfunction stage ≥ 3 .

Right ventricular systolic and diastolic function. Right ventricular systolic function was graded as normal in 58 (30%), mild in 44 (23%), moderate in 40 (21%), moderate/severe in 29 (15%), and severe in 21 (11%) subjects. As RV systolic dysfunction worsened, RAVI increased significantly (Table 2, Fig. 1). In multivariable correlation, RV systolic dysfunction stage and RV diastolic dysfunction stage were significantly related to RAVI (Table 2). RAVI also showed significant univariable correlation with pulmonary vein S/D ratio (Spearman's $r = -0.50$,

$p < 0.0001$) and BNP (Spearman's $r = 0.50$, $p < 0.0001$).

Right ventricular diastolic function was defined in 174 subjects, and was graded as stage 0 in 29 (17%) subjects, stage 1 in 66 (38%), stage 2 in 69 (40%), and stage 3 in 10 (6%). RAVI did not correlate with TDI tricuspid E/Ea ratio and modestly correlated with tricuspid E/A ratio ($r = 0.34$, $p < 0.0001$) and hepatic vein S/D ratio ($r = -0.26$, $p < 0.001$) in univariable analysis (Table 2). Significant differences in RAVI were observed between different RV diastolic stages (Fig. 1).

Outcome analysis of RAVI and RA systolic dysfunction. In our study population, there were 34 deaths, 46 deaths and/or transplant, and 62 deaths, transplants, and/or HF hospitalization. In Kaplan-Meier survival analysis, increasing tertiles of RAVI were strongly predictive of death, transplant, or HF hospitalization over a mean follow-up of 36 ± 20 months (log-rank $p = 0.0002$) (Fig. 2). Worsening RV systolic dysfunction grade (≥ 2 vs. < 2) also showed similar poor prognosis (log-rank $p = 0.0017$) (Fig. 2); however, RV diastolic dysfunction stage (≥ 2 vs. < 2) was not associated with survival (log-rank $p = 0.97$). In univariable and multivariable Cox proportional hazards analysis, RAVI modeled as a continuous variable remained an independent predictor of poor outcomes after ad-



justing for age, BNP, LVEF, RV systolic dysfunction stage, and tricuspid E/Ea ratio ($p = 0.005$) (Table 3). Plasma BNP level was also an independent prognostic marker when these 6 variables were combined into the Cox model ($p = 0.009$). In contrast, RV systolic dysfunction grade was a significant predictor in univariable analysis ($p = 0.0002$), but became nonsignificant after adjusting for BNP levels ($p = 0.34$).

Outcome analysis of RAVI and AF. RAVI also predicted adverse clinical events independent of AF in multivariate Cox proportional hazards analysis (Table 3). RAVI remained a significant predictor of adverse clinical events in patients with and without AF (HR: 1.52 [95% confidence interval: 1.01 to 2.18], $p = 0.047$, $n = 47$, and HR: 1.56 [95% confidence interval: 1.20 to 1.99], $p = 0.001$, $n = 145$, respectively). In our population, AF itself was not a predictor of adverse clinical events (HR: 1.34

[95% confidence interval: 0.76 to 2.28], $p = 0.3054$).

DISCUSSION

Until recently, the routine evaluation of RV function on transthoracic echocardiogram has been neglected. In part, this neglect has been due to difficulties in visualization and quantitative assessment of the RV. Additionally, accepted guidelines for the quantification of both systolic and diastolic RV dysfunction remain poorly defined. RV systolic function is now emerging as an important predictor of morbidity and mortality in congestive HF patients (4,11,12). Similarly, RV diastolic dysfunction is also recognized as a common complication in this population (1,2). In our study, RAVI was an independent predictor of mortality and HF hospitalization, after adjusting for age, LVEF, BNP, RV

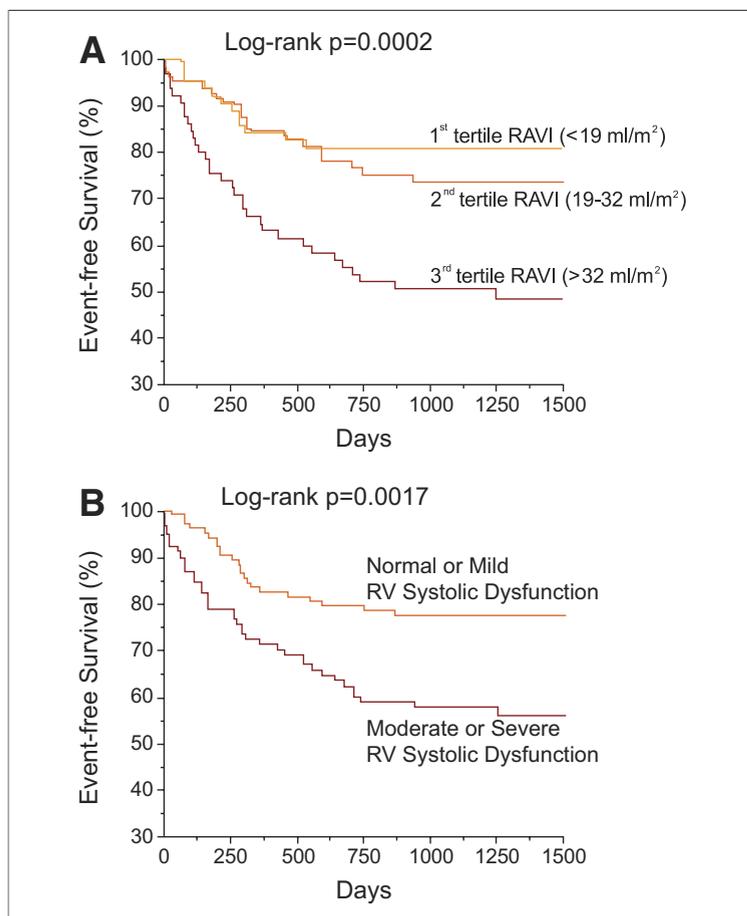


Figure 2. Kaplan-Meier Survival Analysis Across Severity of RAVI and RV Systolic Dysfunction

Kaplan-Meier survival analysis across severity of indexed right atrial volume (RAVI) and right ventricular (RV) systolic dysfunction over a mean follow-up of 36 ± 20 months. (A) Increasing tertiles of RAVI were strongly predictive of death, transplant, or heart failure hospitalization (log-rank $p = 0.0002$); the third tertile of RAVI ($>32 \text{ ml/m}^2$) showed the worst prognosis. (B) Moderate or severe RV systolic dysfunction were strongly predictive of death, transplant, or heart failure hospitalization compared with normal or mild RV systolic dysfunction (log-rank $p = 0.0017$).

systolic dysfunction, and tricuspid E/Ea ratio. Thus, despite limitations in quantifying RV dimensions, echocardiographic assessment of RA size is important in the prognosis of chronic HF.

For the study population, RAVI values are consistent with mean indexed RA volumes calculated in prior studies that utilized a monoplane Simpson's rule (13,14). In a follow-up study, Lambertz et al. (15) calculated a significantly larger mean RAVI of $38 \pm 6 \text{ ml/m}^2$ by employing an alternative RA volume calculation utilizing 2 intersecting echocardiographic views. This technique correlated strongly with RA volumes determined by angiography, and the investigators concluded that monoplane volume determination from the apical 4-chamber view may underestimate RA size, as

only a portion of the true volume is measured. Simpson's method of RA volume calculation omits RA vertical extension and the auricle (15). However, as RA volume was calculated after echocardiograms were performed, adequate subcostal views were not routinely available.

Our study also demonstrated the strong relationship between RAVI and RV systolic dysfunction in both univariable and multivariable analysis. RV systolic dysfunction was noted in 70% of study patients, the majority of whom demonstrated mild or moderate hypokinesis. RV wall hypokinesis is indicated in lower RV ejection fraction and pulmonary perfusion (16,17). Other echocardiographic methods to assess RV systolic dysfunction employ tricuspid annular systolic excursion (18) and TDI tricuspid annular systolic velocity (11). Animal studies suggest that RV pressure overload leading to increasing RA size is more closely related to RV diastolic dysfunction (19). Impaired RV contractility may cause compensatory elevated RAVI for increasing blood volume reservoir, and result in an underperfused state with lower blood volume or oxygen delivery from the pulmonary circulation (20,21). The dilated RA size and depressed RV ejection function may present as reduced exercise tolerance (22).

Interestingly, the RAVI showed only a modest correlation with tricuspid E/A ratio and hepatic vein S/D ratio, but did not correlate with the TDI E/Ea ratio. This suggests that RAVI may be more reflective of the chronicity of RV diastolic function over time (similar to left atrial volume index) rather than a measure of instantaneous diastolic function. This also emphasizes the increased complexity of measuring right-sided diastolic function and the poor correlation of right atrial volume to RV filling pressure due to the additional effect of RA stiffness and contractility (23). Conversely, RAVI was significantly related to LV diastolic dysfunction. In our patient population with chronic LV systolic dysfunction, coexistent LV diastolic dysfunction may exacerbate pulmonary edema and pressures, and transmit further retrograde to manifest as RV overload and subsequent RA enlargement. Prior relationships between RV and LV diastolic measures had been demonstrated in dilated cardiomyopathy patients (2). However, it is undetermined whether left-sided dysfunction precedes or follows right-sided dysfunction. Therefore, the pathophysiology of elevated RAVI in chronic HF is unresolved, and further catheterization studies are needed to deter-

Table 3. Univariable and Multivariable Risk Ratios for Predicting Adverse Clinical Events

Variable	HR (95% CI)	p Value	Goodness of Fit		
			Chi-Square	p Value	AUC (95% CI)*
RAVI†					
Unadjusted	1.56 (1.26–1.91)	<0.0001	15.35	<0.0001	0.69 (0.62–0.77)
Adjusted for age, BNP, LVEF, RVSD	1.81 (1.22–2.64)	0.0033	27.67	<0.0001	0.71 (0.57–0.82)
Adjusted for age, BNP, LVEF, RVSD, tricuspid E/Ea	1.73 (1.18–2.51)	0.0053	29.86	<0.0001	0.70 (0.57–0.83)
Adjusted for age, BNP, LVEF, RVSD, LVDD	1.75 (1.18–2.56)	0.0063	28.84	<0.0001	0.71 (0.57–0.83)
Adjusted for age, BNP, LVEF, RVSD, AF	1.77 (1.20–2.58)	0.0044	32.40	<0.0001	0.71 (0.58–0.84)
Adjusted for age, BNP, LVEF, RVSD, LAVI	1.66 (1.04–2.62)	0.0352	24.27	0.0005	0.68 (0.55–0.81)
RVSD‡					
Unadjusted	1.40 (1.17–1.67)	0.0002	13.89	0.0002	0.68 (0.59–0.74)
Adjusted for age, BNP, LVEF	1.01 (0.76–1.35)	0.9296	19.03	0.0008	0.68 (0.51–0.80)
RVDD‡					
Unadjusted	1.23 (0.90–1.70)	0.1856	1.75	0.1856	0.55 (0.47–0.64)

*Area under the curve (AUC) was based on the prediction at 5 years. The 95% confidence interval (CI) for the area under the ROC curves was obtained by using bootstrap method with a bootstrap size of 200. †Hazard ratios (HRs) per 1 SD increment (1 SD for right atrial volume index [RAVI] = 15.27 ml/m²). ‡Hazard ratios per +1 increment. AF = atrial fibrillation; LAVI = left atrial volume index; LVDD = left ventricular diastolic dysfunction; RVSD = right ventricular systolic dysfunction; other abbreviations as in Table 1.

mine the etiology and effects of elevated RAVI on impaired cardiac function and hemodynamics.

Study limitations. In spite of identifying a strong relationship between RAVI and RV function, there are several limitations to our study. Our study population was relatively small. The RA volumes were calculated using Simpson's method from only 1 view, the apical 4-chamber view. This view places the RA in the far field, diminishing lateral resolution and adversely affecting visualization of the RA endocardium. Whereas the RV was assessed from multiple views, RV systolic function was measured qualitatively, not quantitatively. Invasive hemodynamic data were not available for correlation in any study patients. Last, our study population consisted of patients with significant

LV systolic dysfunction. There was no control group with normal LV function.

CONCLUSIONS

Our findings indicate that, in patients with chronic systolic HF, the RAVI may express the severity of RV systolic dysfunction. This quantitative echocardiographic marker can be used to identify patients with abnormal RV function and a poor prognosis.

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 ■ right ventricular systolic
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 Doppler.