

ORIGINAL RESEARCH

Effectiveness of Rhythm Control of Atrial Fibrillation in Moderate-to-Severe or Severe Secondary Tricuspid Regurgitation



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ABSTRACT

BACKGROUND Secondary tricuspid regurgitation (STR) is heterogeneous and associated with increased morbidity and mortality, but there remains limited evidence on the optimal management strategy. Atrial fibrillation (AF) increases the risk of all types of STR.

OBJECTIVES The authors aimed to study the association of successful rhythm control of AF with decreased severity of moderate-severe/severe STR across clinically relevant subgroups and different etiologies of STR.

METHODS Patients with moderate-severe/severe STR, AF, and follow-up echocardiograms were identified retrospectively. STR types were left-sided valve disease, reduced left ventricular ejection fraction, other causes of pulmonary hypertension, and isolated STR. The primary endpoint was decrease of STR severity to \leq mild. Recurrence of AF was determined. Follow-up was censored at tricuspid valve intervention or last follow-up.

RESULTS Among 1,896 patients (median age 79; 60% women), rhythm control was attempted in 367 (19%) over a median of 21 days. Recurrence of AF occurred in 210 (57%) and was associated with older age, chronic, persistent AF, and lack of antiarrhythmic medication use. Over a median of 1.7 years, tricuspid regurgitation (TR) decreased to \leq mild in 550 patients. Successful control of rhythm without recurrence of AF was strongly associated with decreased TR to \leq mild at the multivariable level (HR: 3.65 [95% CI: 2.24-5.95]; $P < 0.01$). The association remained across different subgroups divided according to TR etiology, age, sex, obesity, burden and chronicity of AF, impaired left ventricular systolic or diastolic function, coexistent significant mitral regurgitation, pulmonary hypertension, and right ventricular remodeling.

CONCLUSIONS Successful rhythm control of AF is associated with improved severity of STR in patients with moderate-severe or severe STR, and an early aggressive attempt should be considered in these patients to decrease risk of recurrence. (JACC Cardiovasc Imaging. 2026;19:673-683) © 2026 by the American College of Cardiology Foundation.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the [Author Center](#).

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**ABBREVIATIONS
AND ACRONYMS**

AF	= atrial fibrillation
AS	= aortic stenosis
ECG	= electrocardiogram
HFpEF	= heart failure with preserved ejection fraction
IVC	= inferior vena cava
LAVI	= left atrial volume index
LV	= left ventricular
LVEF	= left ventricular ejection fraction
MR	= mitral regurgitation
RA	= right atrial
RV	= right ventricular
RVSP	= right ventricular systolic pressure
STR	= secondary tricuspid regurgitation
TTE	= transthoracic echocardiographic
TR	= tricuspid regurgitation

Secondary tricuspid regurgitation (STR) is a heterogeneous lesion arising from various cardiac or pulmonary diseases.¹ Severe STR is associated with worse symptoms, venous congestion, liver and kidney dysfunction, and increased mortality.²⁻⁴ Recent advances in transcatheter interventions demonstrated symptomatic benefit in patients with tricuspid regurgitation (TR),⁵⁻⁷ but optimal timing of intervention remains uncertain. Atrial fibrillation (AF) is common in patients with TR and has been associated with both the development and progression of significant TR.^{2,3,8,9} This was particularly evident in a specific entity of STR, termed "isolated STR," in which overt cardiac disease is absent but AF and heart failure with preserved ejection fraction (HFpEF) are frequent.¹⁰⁻¹³ However, the association of AF and TR expands beyond isolated TR and has been demonstrated across all etiologies of STR including left-sided valve disease, left ventricular (LV)

dysfunction (systolic or diastolic), other causes of pulmonary hypertension, and isolated right ventricular (RV) or right atrial (RA) disease, presenting AF as another potential target for intervention in these patients.¹⁴

Previous studies showing that TR improved in response to rhythm control^{8,15-18} included heterogeneous groups, were frequently small-sized, and without control groups. Therefore, characteristics and types of STR when rhythm control may be beneficial remain unknown. The present study aimed to study the association of rhythm control with decreased severity of moderate-severe or severe STR in patients with AF across clinically relevant subgroups and different etiologies of STR.

METHODS

STUDY POPULATION. The study was approved by the Institutional Review Board, which waived the requirement of informed consent. Only patients who had previously agreed to be included in retrospective research studies were considered. Patients with documented moderate-severe or severe TR (ie, grades III or IV) during a clinically indicated transthoracic echocardiographic (TTE) study 2003 to 2023 at Mayo Clinic sites in the United States (Rochester, Arizona, Florida, and the upper Midwest) were identified retrospectively. Patients were excluded if they had congenital heart disease, previous cardiac implantable electronic devices, previous valve

interventions, previous heart transplants, histories of endocarditis, rheumatic heart disease, carcinoid heart disease, primary tricuspid valve disease, malignancies, liver cirrhosis, end-stage kidney disease on dialysis, or lack of a follow-up echocardiograms at least 30 days from baseline. Subsequently, only patients with known diagnoses of AF, regardless of chronicity, were included, as determined using the International Classification of Diseases (ICD) codes, electrocardiograms (ECGs) or echocardiograms (rhythm recorded during the study by a cardiologist). The baseline (index) examination was the first TTE meeting moderate-severe or severe TR criteria. Therefore, included patients ultimately fulfilled the following 4 inclusion criteria: 1) had moderate-severe/severe secondary TR; 2) had AF; 3) without life-limiting disease; and 4) had follow-up TTE examinations ≥ 30 days after the first diagnoses of moderate-severe/severe TR.

CLINICAL DATA AND DEFINITIONS. Clinical characteristics were extracted from the electronic medical record. Comorbidities were identified using ICD codes. AF was classified as paroxysmal if it terminated spontaneously within 7 days of onset or persistent otherwise. For the purpose of this study, AF and atrial flutter were referred to interchangeably. Rhythm control of AF included catheter ablation, cardioversion, or use of antiarrhythmic medications for ≥ 30 days. Patients were followed using ECGs, TTEs, and Holter monitors for recurrence of AF, and rhythm control without recurrence (which we refer to as "successful rhythm control") was determined. In the case of ablation, recurrence was considered only if it occurred ≥ 90 days after ablation (blinking window).

ECHOCARDIOGRAPHY. All TTEs were performed in routine clinical practice by trained sonographers and reviewed by level III board-certified echocardiologists, according to the guidelines.¹⁹ Severity of TR was determined using an integrative approach based on color flow Doppler, density and shape of the regurgitant jet, proximal isovelocity surface area (effective regurgitant orifice area and regurgitant volume as available), inferior vena cava size, and hepatic vein flow pattern.²⁰ Diastolic dysfunction was defined when ≥ 3 of 4 or ≥ 2 of 3 available diastolic parameters were abnormal (medial mitral e' velocity < 7 cm/s, medial mitral E/e' ratio < 15 during sinus rhythm or < 11 during AF, left atrial volume index (LAVI) > 34 mL/m², and TR velocity > 2.8 m/s).²¹ Right ventricular systolic pressure (RVSP) was estimated using the peak TR jet velocity. Pulmonary hypertension as a categorical variable was

defined as RVSP \geq 50 mm Hg in the absence of RV outflow tract obstruction. RA pressure was estimated based on 2-dimensional (2D) and Doppler assessment of the inferior vena cava (IVC) and hepatic vein flow signals as previously described.²²

Subsets of 25 randomly selected patients from each patients with and without \geq moderate RA enlargement, for a total of 50 patients, were reviewed de novo to confirm RA size assessment and assess agreement using indexed RA volume index (method of disks from the apical 4-chamber view) with reference values according to the most recent guidelines.²³

CLASSIFICATION OF STR ETIOLOGY. All patients had secondary TR by study design. Secondary TR was classified according to the associated cardiac disease, which was identified using a hierarchical order, as described previously.^{3,14,24} First, the presence of organic left-sided valve disease was determined if \geq moderate aortic stenosis (AS)/regurgitation, any mitral stenosis, \geq moderate organic mitral regurgitation (MR), or an aortic/mitral prosthesis were present. Second, the presence of low left ventricular ejection fraction (LVEF) $<$ 50% or ischemic cardiomyopathy associated with \geq moderate ischemic MR was identified. Third, the presence of pulmonary hypertension other than caused by left-sided valve disease or low ejection fraction (EF) was identified. In the last step, in the absence of any of the previous conditions, TR was termed “isolated.” Notably, isolated TR was frequently found to be related to HFpEF in previous studies and is currently frequently referred to as atrial secondary TR.^{10,11}

OUTCOMES. The primary outcome of interest was the decrease of severity of STR from moderate-severe or severe to mild or less. Recurrence of AF was a secondary outcome of interest. All follow-up was censored at the time of tricuspid valve intervention. Patients without the event of interest were censored at their last available TTE at follow-up and before TR intervention when occurred.

STATISTICAL ANALYSIS. Statistical analyses were performed using R programming software, version 4.2.2 (R Foundation for Statistical Computing). A 2-sided value of $P < 0.05$ was considered statistically significant. Continuous variables are reported as median (Q1-Q3) and categorical variables as number (percentage). The analysis of variance (ANOVA) and the Kruskal-Wallis tests were used to compare continuous variables, as appropriate, whereas the chi-square test was used to compare categorical variables. Because of the difficulty in ascertaining true time of recurrence of AF, logistic regression,

rather than Cox regression, was used to identify factors associated with recurrence of AF, while adjusting for time from index examination to rhythm control. The association between rhythm control and the study endpoints of decreased severity of TR was studied using Cox regression, adjusting for clinically relevant variables; as rhythm control occurred after index examination, it was analyzed as a time-dependent covariate.

Because baseline differences existed between patients who did and did not undergo rhythm control, propensity-score matching was additionally performed to compare regression rate of \geq moderate secondary TR to mild or less. The propensity score was calculated using logistic regression (generalized linear method [glm] in the “MatchIt” package in R using greedy nearest-neighbor matching) based on confounders including age, sex, body mass index (BMI), diabetes mellitus, onset of AF within 1 year, paroxysmal (vs persistent AF), low LVEF, moderate/severe RV dysfunction, \geq moderate AS, and RVSP \geq 50 mm Hg and was used to match patients who did or did not undergo rhythm control in a 1:2 ratio. These variables were chosen based on baseline differences. The median matching distance was 0.15 (Q1-Q3: 0.05-0.26). Patients with missing values of any of the matching parameters were excluded from the matching process. In addition, inverse probability weighting was performed as another sensitivity analysis in the overall study population. Propensity scores were estimated using the same confounding factors via a logistic regression model. Subsequently the inverse probability weights were applied in a weight-adjusted Cox model. To minimize the influence of extreme weights and improve model stability, patients with weights $>$ 50 were excluded from this analysis.

RESULTS

BASELINE CHARACTERISTICS. Overall, 1,896 patients with moderate-severe or severe secondary TR and AF were included. Median age was 79 years (Q1-Q3: 72-85 years); 1,141 (60%) were women; and comorbidities were common (Table 1). AF was diagnosed within 1 year before the index examination in 881 (47%) and was paroxysmal in 33% vs persistent in 67%. Low LVEF was present in 561 (30%) and pulmonary hypertension in 1,004 (54%). STR was in the setting of organic left-sided valve disease in 500 (26%), low LVEF or ischemic MR in 445 (24%), other causes of pulmonary hypertension in 490 (26%) and

TABLE 1 Baseline Characteristics in the Overall Cohort and Across Etiologies of TR

	Total (N = 1,896)	Left Valve Disease (n = 500)	Low LVEF/Ischemic MR (n = 445)	Other PH (n = 490)	Isolated (n = 461)	P Value
Age, y	79 (72-85)	82 (77-86)	75 (65-82)	79 (73-84)	79 (71-84)	<0.01
Male	755 (39.8)	170 (34.0)	261 (58.7)	167 (34.1)	157 (34.1)	<0.01
BMI, kg/m ²	27.7 (24.2-32.3)	27.1 (23.8-31.1)	28.2 (24.5-33.2)	28.2 (24.3-33.3)	27.6 (24.3-32.1)	0.25
TR severity						0.12
Moderate-severe	820 (43.2)	202 (40.4)	213 (47.9)	212 (43.3)	193 (41.9)	
Severe	1,076 (56.8)	298 (59.6)	232 (52.1)	278 (56.7)	268 (58.1)	
AF diagnosis within 1 y before baseline	881 (46.5)	208 (41.6)	263 (59.1)	202 (41.2)	208 (45.1)	<0.01
≥ Moderate AR	56 (3.0)	56 (11.2)	—	—	—	—
≥ Moderate AS	254 (13.4)	254 (50.8)	—	—	—	—
≥ Moderate MR	573 (30.3)	279 (55.9)	166 (37.6)	66 (13.5)	62 (13.4)	<0.01
Any MS	51 (2.7)	51 (10.2)	—	—	—	—
Coronary artery disease	799 (42.1)	229 (45.8)	212 (47.6)	194 (39.6)	164 (35.6)	<0.01
Congestive heart failure	1,050 (55.4)	263 (52.6)	309 (69.4)	282 (57.6)	196 (42.5)	<0.01
Chronic lung disease	542 (28.6)	129 (25.8)	124 (27.9)	191 (39.0)	98 (21.3)	<0.01
Diabetes mellitus	626 (33.0)	159 (31.8)	158 (35.5)	176 (35.9)	133 (28.9)	0.07
Hypertension	1,317 (69.5)	353 (70.6)	292 (65.6)	358 (73.1)	314 (68.1)	0.08
Persistent vs paroxysmal AF						<0.01
Paroxysmal	625 (33.3)	139 (28.6)	181 (41.0)	164 (33.6)	141 (30.6)	
Persistent	1,252 (66.7)	347 (71.4)	261 (59.0)	324 (66.4)	320 (69.4)	
EF <50%	561 (29.8)	127 (25.6)	434 (98.2)	—	—	—
PH (RVSP ≥50 mm Hg)	1,004 (53.7)	302 (60.6)	217 (50.2)	485 (100.0)	0 (0.0)	<0.01
Diastolic dysfunction						<0.01
N-Miss	401	114	105	87	95	
1	1,305 (87.3)	352 (91.2)	295 (86.8)	382 (94.8)	276 (75.4)	
Moderate or severe RV enlargement						<0.01
N-Miss	210	44	60	52	54	
1	665 (39.4)	148 (32.5)	160 (41.6)	220 (50.2)	137 (33.7)	
Moderate or severe RV dysfunction						<0.01
N-Miss	326	75	114	83	54	
1	321 (20.4)	75 (17.6)	120 (36.3)	92 (22.6)	34 (8.4)	
Moderate or severe RA enlargement	1,708 (90.1)	453 (90.6)	390 (87.6)	452 (92.3)	414 (89.9)	0.32
Medial e' velocity, cm/sec	7.0 (5.0-8.0)	6.0 (5.0-8.0)	6.0 (4.0-8.0)	7.0 (6.0-8.0)	8.0 (6.0-9.0)	<0.01
Medial E/e' ratio	15.7 (11.4-20.0)	18.3 (13.8-26.0)	15.7 (11.7-20.0)	15.7 (12.0-20.0)	12.9 (10.0-17.1)	<0.01
RVSP, mm Hg	51.0 (41.0-63.0)	54.0 (44.0-65.0)	50.0 (40.0-58.0)	63.0 (56.0-74.0)	41.0 (35.0-45.0)	<0.01
TR velocity, m/sec	3.1 (2.7-3.5)	3.2 (2.8-3.6)	3.0 (2.5-3.3)	3.4 (3.2-3.8)	2.7 (2.4-2.9)	<0.01
LVEDD, mm	46.0 (42.0-52.0)	46.0 (41.0-52.0)	53.0 (47.0-58.0)	45.0 (41.0-49.0)	45.0 (41.0-49.0)	<0.01
LAVI, mL/m ²	50.0 (41.0-64.0)	56.0 (45.0-70.0)	50.0 (42.0-62.0)	48.0 (39.0-65.0)	47.0 (39.0-56.0)	<0.01
LVEF, %	57.0 (45.0-63.5)	58.0 (48.0-64.0)	35.0 (25.0-43.0)	62.0 (58.0-67.0)	60.0 (55.0-64.0)	<0.01
Creatinine, mg/dL	1.1 (0.9-1.4)	1.1 (0.9-1.4)	1.2 (1.0-1.5)	1.1 (0.9-1.4)	1.0 (0.8-1.2)	<0.01
Hemoglobin, mg/dL	12.5 (11.1-13.8)	12.4 (11.0-13.6)	12.8 (11.1-14.1)	12.0 (10.6-13.2)	13.0 (11.5-14.1)	<0.01

Values are median (Q1-Q3) or n (%), unless otherwise indicated.

AF = atrial fibrillation; AR = aortic regurgitation; AS = aortic stenosis; BMI = body mass index; EF = ejection fraction; LAVI = left atrial volume index; LVEDD = left ventricular end diastolic dimension; LVEF = left ventricular ejection fraction; MR = mitral regurgitation; MS = mitral stenosis; PH = pulmonary hypertension; RA = right atrium; RV = right ventricular; RVSP = right ventricular systolic pressure; TR = tricuspid regurgitation.

isolated in 461 (24%). Other baseline characteristics are shown in [Table 1](#).

Patients with organic left-sided valve disease were the oldest. Among these patients, 279 (56%) had ≥ moderate MR; 254 (81%) had ≥ moderate AS; 56 (11%) had ≥ moderate aortic regurgitation (AR); and 51 (10%) had any mitral stenosis. Patients were predominantly women (>60%) in all categories of TR etiologies except for the low LVEF/ischemic MR

group, in which 59% were men. The low LVEF/ischemic MR group had the highest proportion of newly diagnosed AF within 1 year before the index examination as well as paroxysmal AF ([Table 1](#)).

Notably, there was substantial agreement between the reported and the de novo assessed RA size (≥ moderate RA enlargement) in the 50-patient sample with Cohen's kappa of 0.76 (95% CI: 0.58-0.94).

FREQUENCY OF RHYTHM CONTROL. Rhythm control was attempted in 367 (19%) patients over a median of 21 days (Q1-Q3: 1-192 days) from baseline examination. Of these, 178 (49%) received antiarrhythmic medications for at least 30 days; of those, 105 had antiarrhythmic medications along with cardioversion or ablation, 252 (69%) underwent cardioversion attempts, and 51 (14%) underwent ablation attempts. Patients who underwent attempts of rhythm control were younger, more frequently men, more often had a recent (<1 year) diagnosis of AF and paroxysmal (vs persistent) AF, low LVEF and RV dysfunction, and less often had organic left-sided valve disease, pulmonary hypertension, diabetes, and systemic hypertension compared with patients who did not undergo rhythm control (Table 2).

EFFECTIVENESS OF RHYTHM CONTROL. Predictors of recurrence of AF. Overall, recurrence of AF occurred in 210 (57%) patients during follow-up. When adjusted for time to rhythm control, older age (OR: 1.03 [95% CI: 1.02-1.05] per year; $P < 0.001$), more chronic AF (OR: 1.06 [95% CI: 1.00-1.12]; $P = 0.046$), persistent AF (OR: 1.74 [95% CI: 1.12-2.71]; $P = 0.01$), presence of systemic hypertension (OR: 1.62 [95% CI: 1.05-1.20]; $P = 0.03$), and the lack of use of antiarrhythmic medications (OR: 3.45 [95% CI: 2.22-5.26]; $P < 0.001$) were associated with recurrence of AF (Supplemental Tables 1 and 2). Notably, presence of low LVEF or pulmonary hypertension did not seem to be associated with recurrence of AF (Supplemental Tables 1 and 2). Organic left-sided valve disease was numerically more frequent in the group with recurrence of AF, although this did not achieve statistical significance (Supplemental Tables 1 and 2).

Association of rhythm control with decrease in severity of STR. Over median 1.7 years (Q1-Q3: 0.6-3.8 years), TR decreased to mild or less in 550 patients. Achievement of rhythm control without recurrence of AF was strongly associated with decreased TR to mild or less at the univariate level (HR: 6.56 [95% CI: 4.38-9.81]; $P < 0.001$) and when adjusted to age, sex, BMI, type of AF, RVSP, low LVEF, diastolic dysfunction and use of diuretic agents at baseline (HR: 3.65 [95% CI: 2.24-5.95]; $P < 0.01$) (Table 3). Notably, patients with LVEF <50% at baseline who had decreases in TR severity to mild had associated improvement in LVEF and more frequent normalization of LVEF to at least 50% at time of the follow-up TTE compared with patients who did not have decreases in TR severity (LVEF improvement: median +14 [Q1-Q3: +4 to +25] vs +5 [Q1-Q3: -1 to +13]; $P < 0.01$; LVEF

TABLE 2 Baseline Characteristics in Patients Who Underwent or Did Not Undergo Rhythm Control

	Did Not Undergo Rhythm Control (n = 1,529)	Underwent Rhythm Control (n = 367)	P Value
Age, y	80 (74-85)	73 (65-81)	<0.01
Male	592 (38.7)	163 (44.4)	0.045
BMI, kg/m ²	27.5 (24.1-31.9)	28.4 (24.6-33.0)	0.06
Severity of TR			<0.01
Moderate-severe	639 (41.8)	181 (49.3)	
Severe	890 (58.2)	186 (50.7)	
AF diagnosis within 1 y before baseline	654 (42.8)	227 (61.9)	<0.01
Etiology of TR			<0.01
Isolated	365 (23.9)	96 (26.2)	
Left valve disease	436 (28.5)	64 (17.4)	
Low EF/ischemic MR	288 (18.8)	157 (42.8)	
Other PH	440 (28.8)	50 (13.6)	
≥ Moderate AR	43 (2.8)	13 (3.5)	0.46
≥ Moderate AS	234 (15.3)	20 (5.4)	<0.01
≥ Moderate MR	442 (29.0)	131 (35.7)	0.01
Any MS	43 (2.8)	8 (2.2)	0.50
Coronary artery disease	650 (42.5)	149 (40.6)	0.51
Congestive heart failure	833 (54.5)	217 (59.1)	0.11
Chronic lung disease	439 (28.7)	103 (28.1)	0.81
Diabetes	524 (34.3)	102 (27.8)	0.02
Hypertension	1,081 (70.7)	236 (64.3)	0.02
Persistent vs paroxysmal AF			<0.01
Paroxysmal	399 (26.4)	226 (61.9)	
Persistent	1,113 (73.6)	139 (38.1)	
EF <50%	374 (24.6)	187 (51.4)	<0.01
Pulmonary hypertension (RVSP ≥50 mm Hg)	861 (56.9)	143 (40.1)	<0.01
Diastolic dysfunction			0.01
N-Miss	320	81	
1	1,068 (88.3)	237 (82.9)	
Moderate or severe RV enlargement			0.17
N-Miss	180	30	
1	543 (40.3)	122 (36.2)	
Moderate or severe RV dysfunction			0.02
N-Miss	261	65	
1	245 (19.3)	76 (25.2)	
Moderate or severe RA enlargement	1,397 (91.4)	313 (85.3)	<0.01
Medial e' velocity, cm/sec	7.0 (5.0-8.0)	7.0 (5.0-8.0)	0.19
Medial E/e' ratio	15.7 (11.7-21.3)	14.3 (10.0-18.0)	<0.01
RVSP, mm Hg	53.0 (43.0-64.0)	46.0 (39.0-55.0)	<0.01
TR velocity, m/sec	3.1 (2.7-3.5)	2.8 (2.5-3.2)	<0.01
LVEDD, mm	46.0 (41.0-51.0)	48.0 (43.0-55.0)	<0.01
LAVI, mL/m ²	51.0 (43.0-66.0)	47.0 (37.2-57.0)	<0.01
LVEF, %	58.0 (50.0-64.0)	48.0 (31.8-59.0)	<0.01
Creatinine, mg/dL	1.1 (0.9-1.4)	1.1 (0.9-1.4)	0.18
Hemoglobin, mg/dL	12.4 (10.9-13.7)	12.9 (11.4-14.2)	<0.01

Values are median (Q1-Q3) or n (%), unless otherwise indicated.
 AR = aortic regurgitation; AS = aortic stenosis; other abbreviations as in Table 1.

normalization: 44.3% vs 32.6%; $P < 0.01$). Therefore, the association of low LVEF and improved severity of TR is likely in the setting of reversal of the underlying etiology of TR in these patients.

TABLE 3 Multivariable Factors Associated With Decreased TR to Mild or Less

	HR	Lower CI	Upper CI	P Value
Age, per y	0.97	0.96	0.98	<0.01
Male	1.11	0.90	1.36	0.35
BMI, per 1 kg/m ²	1.00	0.99	1.02	0.65
Persistent (vs paroxysmal) AF	0.67	0.54	0.83	<0.01
RVSP, per 1 mm Hg	1.00	0.99	1.00	0.72
Low LVEF <50%	1.78	1.43	2.22	<0.01
Diastolic dysfunction	0.90	0.68	1.21	0.49
Diuretic use	1.17	0.94	1.45	0.16
Rhythm control without recurrence ^a	3.65	2.24	5.95	<0.01

^aAnalyzed as a time-dependent covariate.
Abbreviations as in Table 1.

The association between achievement of rhythm control without recurrence and improvement in TR to mild or less was evidently consistent across subgroups of age (≥ 65 years), sex, BMI (≥ 30 kg/m²), diastolic dysfunction, low LVEF, pulmonary hypertension, TR etiology categories, \geq moderate MR, type of AF, \geq moderate RV dysfunction or enlargement, and chronicity of AF (Figure 1). The benefit seemed to be more pronounced in women and patients with preserved LVEF, although was still clearly apparent in men and patients with reduced LVEF (Figure 1). To investigate this more, we compared baseline characteristics between men and women (Supplemental Table 4). Women seemed to be older with more frequently isolated left-sided valve disease and pulmonary hypertension-related TR and less frequently low LVEF, coronary artery disease, chronic lung disease and RV remodeling. When adjusting for these parameters (age, etiology of TR, coronary artery disease, chronic lung disease, RV function and size), the interaction was weakened (P for interaction = 0.13).

Among patients with low LVEF who underwent rhythm control attempts ($n = 187$), LVEF normalized in 44.0%, compared with 34.0% in patients with low LVEF who did not undergo rhythm control ($P = 0.02$). Furthermore, in a sensitivity analysis excluding patients with left-sided valve disease who underwent interventions, the association of successful rhythm control with decreased TR severity remained in this subgroup of patients (HR: 7.58 [95% CI: 3.09-18.57]).

Notably, when using weight change between baseline and follow-up TTEs as a surrogate of fluid status, there was no significant difference in weight change at time of follow-up TTE between the groups who did or did not undergo rhythm control (median absolute weight change -3.5 kg [Q1-Q3: -9.0 to

-0.4 kg] vs -2.4 kg [Q1-Q3: -8.1 to $+1.0$ kg], respectively; $P = 0.84$) or between the groups who had or did not have regression of TR at follow-up (median absolute weight change -3.0 kg [Q1-Q3: -9.0 to $+0.5$ kg] vs -2.7 kg [Q1-Q3: -8.1 to $+1.0$ kg], respectively; $P = 0.33$).

In a sensitivity analysis in which patients who underwent ablation ($n = 14$) or cardioversion ($n = 137$) attempts before the index date were excluded, rhythm control remained associated with decreased TR to mild or less at follow-up in multivariable analysis (HR: 4.18 [95% CI: 2.54-6.87]; $P < 0.01$). Furthermore, there was no interaction between severity of TR (ie, moderate-severe or severe) and rhythm control in relation to the outcome of decreased TR (P for interaction = 0.46) with successful rhythm control being associated with decreased TR in both groups (moderate-severe, HR: 2.70 [95% CI: 1.43-5.08]; severe TR, HR: 3.88 [95% CI: 1.89-7.99]).

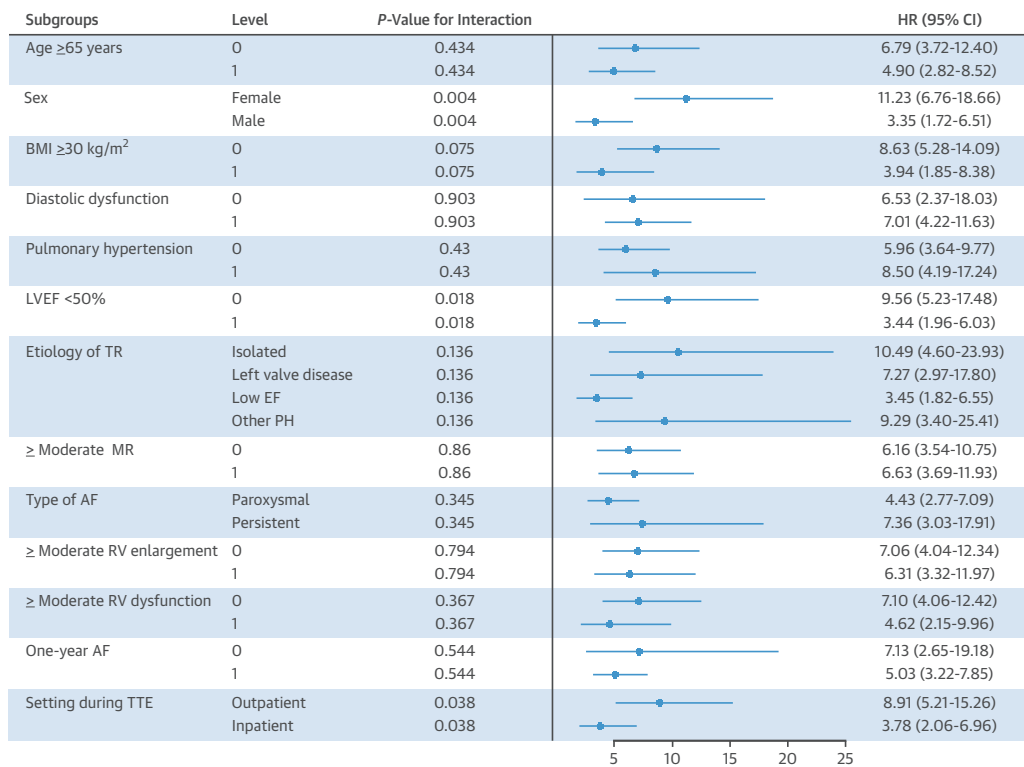
In addition, the propensity score-matched subset of patients with successful rhythm control and no recurrence and patients who did not undergo rhythm control was examined. Baseline characteristics after matching showed no significant baseline differences (Supplemental Table 5). Successful rhythm control as a timed covariate remained associated with regression of \geq moderate TR to mild or less (HR: 3.07 [95% CI: 1.35-6.98]; $P = 0.008$). Similar findings were observed in the inverse probability weighed analysis with successful rhythm control being associated with decreased TR severity (HR: 4.06 [95% CI: 1.77-9.35]; $P < 0.001$).

Baseline characteristics of patients who did or did not undergo rhythm control stratified according to whether TR decreased to mild at follow-up are shown in Supplemental Table 6. Compared with patients who did not undergo rhythm control and did not achieve decreased TR, patients who did not undergo rhythm control and had decrease in TR were younger, more frequently men, had more frequently recent (within 1 year) AF, paroxysmal (vs persistent) AF and low LVEF, lower RVSP, and less prevalence of hypertension.

DISCUSSION

Significant TR confers increased congestion, worsened exertional dyspnea and fatigue, decreased quality of life, increased hospitalizations, and increased mortality risk.^{1,25} However, there remains limited evidence on the optimal management approach of TR. Despite the recent advancements in transcatheter interventions,⁵⁻⁷ timing of

FIGURE 1 Association of Rhythm Control Without Recurrence of AF With Decreased TR Severity to Mild or Less Across Relevant Clinical Subgroups



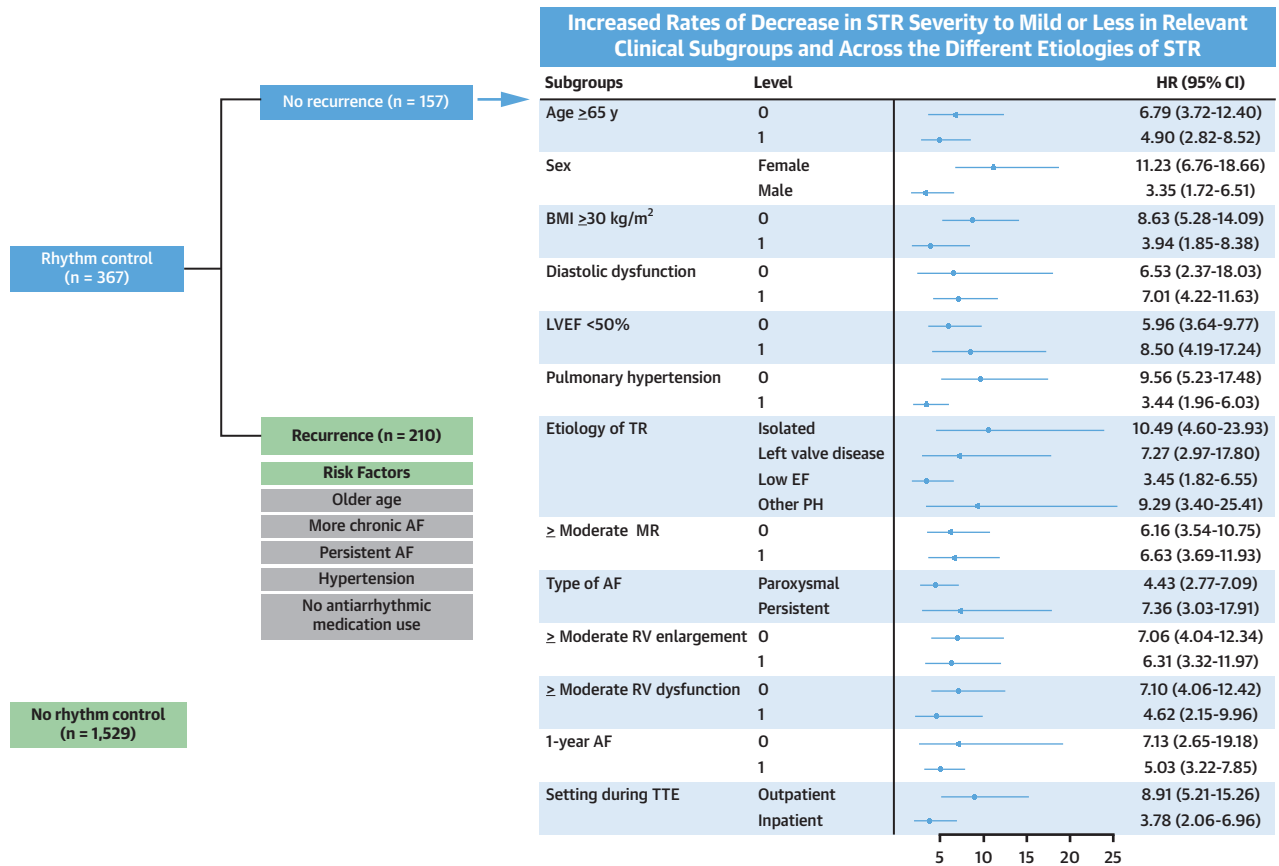
AF = atrial fibrillation; BMI = body mass index; EF = ejection fraction; MR = mitral regurgitation; RV = right ventricle; TR = tricuspid regurgitation.

intervention, and the utility of other management strategies remain unknown, leaving uncertainty in clinical practice in this patient population. The present study (Central Illustration) demonstrates the association of successful rhythm control of AF with decreased severity of TR across clinically relevant subgroups as well as various etiologies of STR in patients with moderate-severe/severe STR and AF. The study also shows that AF recurred in half of the patients who underwent rhythm control interventions, and risk factors for recurrence included older age, systemic hypertension, more chronic, persistent AF, and the lack of use of antiarrhythmic medications. These findings suggest the importance of early aggressive rhythm control in patients with AF and moderate-severe/severe STR and that rhythm control in these patients may be an additional effective

strategy with a potential to avoid TR-targeted valve intervention, although this needs to be confirmed in randomized clinical trials.

The bidirectional relationship between AF and RA remodeling is widely established.^{13,26,27} AF contributes to the development of isolated STR and STR after left-sided valve surgery.²⁸ AF was recently found to increase the risk of incident STR across all inciting etiologies of TR, related to larger RA and higher RVSP compared with sinus rhythm.¹⁴ This highlighted the presence of mixed atrial and ventricular mechanisms in patients with cardiac disease and coexistent AF who develop significant STR, presenting AF as another potential target for intervention in these patients.

Previous studies showing that rhythm control reduces TR severity and reverses right-sided

CENTRAL ILLUSTRATION Effectiveness of Successful Rhythm Control of AF in Moderate-Severe or Severe Secondary TR

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AF = atrial fibrillation; BMI = body mass index; EF = ejection fraction; LVEF = left ventricular ejection fraction; MR = mitral regurgitation; STR = secondary tricuspid regurgitation; RV = right ventricular; TTE = transthoracic echocardiographic; TR = tricuspid regurgitation.

chamber remodeling did not have control groups or distinguish between the distinct categories of STR and frequently included small sample sizes.^{8,15-18} Consequently, the effect of AF treatment across different STR etiologies and clinical characteristics remained unclear, leaving uncertainty about the patient population in which rhythm control should be pursued.^{17,18,29,30} The present study addresses this gap and demonstrates that successful rhythm control was associated with improved severity of STR across various clinical states and echocardiographic diseases, regardless of age, sex, obesity,

burden and chronicity of AF, impaired LV systolic or diastolic function, coexistent significant MR, pulmonary hypertension, and RV remodeling.

Although successful rhythm control was associated with decreased severity of STR in both sexes, the association was stronger in women. Previous studies suggest women are more predisposed to atrial secondary TR and MR, largely driven by atrial remodeling.^{31,32} Proposed mechanisms include sex hormone-mediated inflammatory responses in AF, differences in fibroblast response to annular stress and leaflet adaptation, and structural differences in

the mitral and tricuspid annuli.³³⁻³⁵ Additional analysis in this study suggested that this interaction might be in part related to more frequent low ejection fraction, ischemic heart disease, and LV and RV remodeling in men. Whether there is truly a differential response of TR to successful rhythm control of AF between sexes remains to be evaluated in future studies.

The association of successful rhythm control with decreased severity of STR also persisted across the various inciting etiologies of STR. Notably, a higher proportion of patients with isolated TR or TR in the setting of low LVEF underwent attempts at rhythm control compared with patients with left-sided organic valve disease and other causes of pulmonary hypertension, and patients with \geq moderate AS or MR had numerically higher recurrence rates, which is not surprising. However, when successful and without recurrence of AF, rhythm control was associated with decreased TR in all these patients.

The presence of low LVEF was associated with a decrease in severity of TR at follow-up, and the decrease in TR severity was more frequent in patients who achieved normalization of LVEF compared with those who did not improve their LVEF, suggesting that the inciting etiology of TR (ie, low LVEF) was reversed in the first group. Previous studies have shown that rhythm control of AF in patients with low LVEF and HF, particularly using catheter ablation, decreases rates of all-cause mortality and HF hospitalizations and improves quality of life, LVEF, and markers of congestion;^{36,37} the underlying mechanisms for these improved clinical outcomes are only partially understood. The current study shows that successful rhythm control in patients with low LVEF and moderate-severe or severe STR was associated with improved severity of STR at follow-up and can be a potential contributing mechanism. Conversely, the association of successful rhythm control in decreasing severity of TR seemed to be less strong in patients with low LVEF compared with patients who have normal LVEF, which can be related to availability of other factors contributing to improvement of LVEF such as guideline-directed medical therapy in the first group and scarcity of such factors in the second group, making the association of rhythm control less strong in patients with low LVEF when compared with patients who have preserved LVEF.

Isolated TR was shown to have a strong association with diastolic dysfunction and HFpEF in previous studies.^{10,11} Although the evidence of rhythm control in HFpEF in relation to clinical outcomes is limited to

post hoc subgroup analyses of randomized clinical trials and small trials,^{38,39} our analysis suggests a potential benefit on TR in these patients. Furthermore, successful rhythm control was associated with decreased severity of TR among patients with and without RV enlargement or dysfunction as well as with or without pulmonary hypertension, suggesting benefit across the ventricular and atrial secondary mechanisms of TR.

Early-onset AF, diagnosed within 1 year before moderate-severe or severe TR, was present in about half of our patients, was more likely paroxysmal (as opposed to persistent), and more likely to be targeted with a rhythm control attempt. AF recurred in about half of the patients who underwent rhythm control attempts in this study. This is not surprising, as TR has been associated with increased risk of recurrence of AF after rhythm control.⁴⁰ Recurrence rate of AF appeared to be significantly lower with catheter ablation than cardioversion or antiarrhythmics alone in previous studies,⁴¹ but ablation was performed in only 14% of the current population, likely contributing to the higher observed rate of recurrence in this study. However, in the transcatheter tricuspid valve intervention trials,^{6,7,42} >90% of the patients had AF, and it is uncertain if rhythm control was attempted before consideration of intervention. Even if rhythm control is successful in half of the patients, this would still translate into a significant proportion of patients with a potential to avoid tricuspid valve intervention. Notably, a shorter duration of AF and use of the use of antiarrhythmic medications were associated with lack of recurrence of AF at follow-up. Therefore, early, aggressive rhythm control of AF in these patients may help in decreasing risk of recurrence of AF and in maintaining sinus rhythm. Rhythm control was also shown to decrease the risk of incident atrial secondary TR⁴³ (and MR³²) in previous investigations and may be best performed to prevent TR before it develops.

STUDY LIMITATIONS. The study was retrospective, involving Mayo Clinic sites. Some of the sites (eg, Rochester, Minnesota, USA) are tertiary centers subject to referral bias. Inclusion required follow-up echocardiography, which may select for closer surveillance but aligns with current guideline recommendations for significant TR.⁴⁴ Follow-up timing was variable but addressed using survival analysis. Echocardiograms were interpreted by multiple echocardiologists, and interobserver variability is possible. Nonetheless, echocardiographic grading of TR remained consistent between sites; the

“massive”/“torrential” grades of TR have not been part of the standardized grading of TR in our echocardiography laboratory. In severe TR, RA pressure and RV systolic pressure may be underestimated by echocardiography. The long study period (2003-2023) preceded widespread routine quantification of RA volume, resulting in missing RA volume indices. Consequently, semiquantitative/visual assessment of RA size was used, which can have inaccuracies. However, there was excellent agreement in a 50 random patient sample regarding the assessment of moderate or severe RA enlargement.

Rhythm control was attempted in a small subset, reflecting an era when it was reserved for symptomatic AF despite rate control. Future studies are needed to look at AF burden as a more comprehensive way to evaluate the potential effect of rhythm control and its association with improved TR severity. Although comprehensive analyses were performed, including multivariable adjustment, propensity-score matching, and subgroup analyses, residual confounding may have influenced patient selection for rhythm control. Finally, we included moderate-severe and severe grades of TR in the current study, whereas randomized clinical trials of TR transcatheter interventions focused on least-severe TR grade. Although there was no interaction between severity of TR (moderate-severe vs severe) and rhythm control in association with decreased TR in the current study, future prospective studies and randomized clinical trials are needed to confirm our findings.

CONCLUSIONS

Successful rhythm control of AF in the setting of moderate-severe or severe STR was associated with improved severity of TR, and this association was consistent across different patient and echocardiographic characteristics and inciting etiologies for STR. In patients who underwent rhythm-control attempts, AF recurred in about half, and recurrence

was associated with longer duration of AF, older age, hypertension, and lack of concurrent use of antiarrhythmic medications. The study suggests early, aggressive rhythm control in patients with significant STR and AF may be an effective strategy to improve severity of TR before consideration of valve intervention. Future randomized studies are needed to confirm these findings.

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PERSPECTIVES

COMPETENCY IN MEDICAL KNOWLEDGE: In the present study, we demonstrated that successful rhythm control without recurrence of AF was associated with 3.65-fold increase in chances of decreased severity of TR in patients with moderate-severe or severe secondary TR and AF. Overall, about half of the patients experienced recurrence of AF after rhythm control, and risk factors for recurrence were older age, systemic hypertension, more chronic, persistent AF, and the lack of use of antiarrhythmic medications.

TRANSLATIONAL OUTLOOK: The study presents AF as a potential therapeutic target in patients with severe secondary TR and AF and highlights that earlier and more aggressive rhythm control is warranted to decrease risk of recurrence of AF. Future prospective studies and randomized clinical trials are needed to confirm these findings.

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APPENDIX For supplemental tables, please see the online version of this paper.