

Short-term outcomes of secondary tricuspid regurgitation after left-sided heart valve surgery

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BACKGROUND

Secondary tricuspid regurgitation (TR) is a common manifestation of valvular heart disease, often co-existing with left-sided valvopathies.⁽¹⁾ Historically, a conservative strategy was recommended for the majority of secondary TR as it was believed that most cases would resolve with treatment of the left-sided disease, when followed by decrease in pulmonary arterial systolic pressure.⁽²⁻⁵⁾ Recent evidence suggests that this may indeed be the case with recurrent or progressive TR being uncommon after isolated mitral valve (MV) surgery for degenerative disease.⁽⁶⁾ Furthermore, this is not limited to isolated MV surgery as 20% of patients undergoing combined mitral and aortic valve (AV) surgery will experience spontaneous improvement of TR within 6 months.⁽⁷⁾ However, there is evidence that secondary TR does not always resolve. It can be a progressive disease that may worsen over time and is associated with morbidity, mortality, and poor functional status.^(1,3-5,8-11) In particular, higher grades of TR (moderate or severe) correlate with worse clinical and functional outcomes.⁽¹²⁾ The poorer outcomes associated with worsening TR severity have been demonstrated to be independent of pulmonary artery pressure or right ventricular (RV) dysfunction.⁽¹³⁾

ABSTRACT

Background: Secondary tricuspid regurgitation (TR) is a common finding in patients undergoing surgery for left-sided heart valve disease. The indications for concomitant tricuspid valve (TV) repair have been progressively expanded based on data suggesting adverse sequelae for patients in whom secondary TR is not treated.

Method: This was a prospective observational study of patients undergoing left-sided valve surgery with at least mild TR. Eighty-three patients were enrolled between July 2019 - April 2021. Patients received either conservative management (no TV repair) or concomitant TV repair (TV repair) based upon a guideline-directed, multidisciplinary team approach. Primary outcomes were freedom from recurrent TR, poor functional status, and mortality at 6 months. The secondary outcomes were to identify predictors of recurrent TR and compare no TV repair vs. TV repair outcomes in patients with moderate or severe pre-operative TR.

Results: The mean age was 49 ± 15.5 years and 51.8% (43 of 83) were female. Thirty-seven (44.6%) had rheumatic heart disease. The most common procedures involved the mitral (50.6%) and aortic (28.9%) valves in isolation. Additional procedures were performed in 33 (39.8%) patients, including resection of the left atrial appendage in 21 (63.6%). Pre-operative moderate or severe TR was present in 34 (40.9%) patients, and TV repair was performed in 9 (10.8%) patients who all received rigid ring annuloplasty. At 6 months the 56 patients (67.5%) were free of significant TR, 14 (16.9%) were in a poor functional state and 72 (86.7%) were alive. Suggested predictors of recurrent TR at 6 months were female gender (OR 9.9, p=0.04), rheumatic left-sided valvopathy (OR 14.4, p=0.02), and elevated right ventricular systolic pressure (OR 1.1, p<0.01). An exploratory sub-group analysis did not reveal any primary outcomes differences between no TV repair vs. TV repair at 6 months, despite the latter group demonstrating more high-risk features.

Conclusion: Guideline-directed, multidisciplinary team approach for the management of secondary TR associated with left-sided valve disease produced good overall short-term outcomes that appeared similar whether or not the TV was repaired. Prospective studies with long-term outcomes are required to determine the optimal treatment strategy for secondary TR in patients undergoing left-sided valve surgery.

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Regardless of the severity, untreated pre-operative TR may either fail to improve or progress after left-sided valve surgery in a significant number of patients.^(7,10) Current guidelines reflect this reality, and have adopted a more aggressive management approach toward secondary TR.^(14,15) These guidelines suggest treating both severe and less-than-severe TR, and incorporate symptomatology and tricuspid valve (TV) morphology into the algorithm. The treatment of less-than-severe grades of TR with concurrent TV repair is supported by evidence that suggests improved RV reverse remodeling and reduced heart failure symptoms, without increasing operative risk.⁽³⁾

Despite these factors, only 80% and 40% of severe and less-than-severe cases of secondary TR are repaired according to the Society for Thoracic Surgeons (STS) Adult Cardiac Surgery Database (ACSD).⁽¹⁶⁾ The reason for this may be that the evidence for long term benefit after concomitant TV repair is relatively limited.⁽¹⁷⁾ It remains uncertain whether concomitant TV repair imparts a meaningful improvement to functional class or survival.^(9,18,19) Furthermore, significant TR after TV repair is not uncommon with up to 9% of patients experiencing early moderate or severe TR.⁽²⁰⁾ Given these factors it is not surprising that many surgeons do not concomitantly repair the TV during left-sided valve surgery.⁽²¹⁾ We aimed to determine the short-term outcomes of patients with secondary TR undergoing left-sided valve surgery by prospectively enrolling patients scheduled for surgery.

METHOD

Study design

This was a prospective observational cohort study of consecutive patients undergoing left-sided valve surgery at Tygerberg Hospital in Cape Town, South Africa. Eighty-three patients were enrolled between July 2019 - April 2021. Inclusion criteria included all patients older than 13 years who received repair or replacement of the MV and or the AV or aortic root, with at least mild TR. Exclusion criteria included none or trace TR, primary TV disease (based on clinical features and echocardiography), tricuspid stenosis, presence of pacemaker leads through the tricuspid valve, and redo surgery. Patients who had conservative management of TR formed the no TV repair group, whereas those who had concomitant repair of the TV formed the TV repair group.

Study procedures

Treatment approach

Patients were referred for surgery utilising the weekly heart team meeting. Members of the team included 2 cardiothoracic surgeons, 1 of whom was fellowship trained in structural heart disease, 4 cardiologists with both imaging and structural heart

expertise, and 1 electrophysiologist. Patients who were accepted for left-sided valve surgery had routine assessment of the TV performed. The decision to recommend conservative treatment of the TV vs. concomitant TV repair was based upon a combination of internationally accepted guidelines and expert opinion at our centre. In summary these included severe TR (Class I); or less-than-severe TR with dilated tricuspid annulus (TA) ($\geq 40\text{mm}$ or $\geq 21\text{mm/m}^2$), or previous right heart failure (RHF), or RV dilatation (Class IIa).^(14,15) Other features of TV morphology that were assessed included tenting height (distance between the coaptation point and the annular plane), and tenting area (area contained within the TV leaflets and the annular plane). Once a consensus was reached for each case, a recommendation was made and documented in the clinical notes. If the surgery differed from this initial recommendation the reasons for this were documented in the operative notes. There were no such cases in this study.

Operative approach

The operative approach was median sternotomy with cardiopulmonary bypass (CPB), mild systemic hypothermia and cold blood cardioplegia. For MV replacement part of the contractile apparatus was preserved where feasible. AV replacement was done through a standard aortotomy incision. For valve replacement the sizing and orientation of the prostheses was performed according to manufacturer's guidelines. Braided 2-0 sutures with pledgets were used to secure both the mitral and aortic prostheses. Mitral annuloplasty ring size was determined by the surface area of the anterior leaflet. The implanted left-sided prosthesis included: St. Jude Mechanical / Regent (SJM/SJR) (St. Jude Medical, St. Paul, MN, USA), Carpentier Edwards (CE) Perimount (Edwards Lifesciences, Irvine, CA, USA), and Medtronic CG Future Ring (Medtronic, Minneapolis, MN, USA). These were selected according to the patient's informed preferences. TV repair was performed on the arrested heart, through an oblique right atriotomy and always involved ring annuloplasty with the Medtronic Contour 3D Ring (Medtronic, Minneapolis, MN, USA) using non-pledgeted braided 2-0 sutures. Sizing was performed according to the length of the attachment of the tricuspid septal leaflet. No other TV repair techniques were employed.

Post-operative care and follow-up

Post-operative care occurred in the specialised cardiothoracic surgery unit at Tygerberg Hospital. Follow-up was carried out at the outpatient cardiac surgery clinic 6 months after discharge. This approach was tailored to each patient, and closer follow-up occurred as required. Clinic visits consisted of a thorough clinical assessment, and further tests if indicated. Additional testing including laboratory, chest radiography, electrocardiogram, and echocardiogram were available if required. Poor

functional status was defined as New York Heart Association (NYHA) class III or IV. Features of RHF included raised jugular venous pressure (JVP), liver congestion, ascites, or pedal edema. Adverse events and the cause of death was determined by hospital chart review or information from the physician on duty at the time of the event. Clinical follow-up was complete for 76 of 83 patients (91.6%) and echocardiographic follow-up was complete for 66 of 83 patients (79.5%).

Echocardiographic assessment

All patients underwent standard transthoracic echocardiography pre-operatively and at 6 months after surgery employing the current techniques recommended by the American Society of Echocardiography.⁽²²⁾ Only patients with an indication for transesophageal echocardiography (TEE) underwent TEE. Specific parameters evaluated included chamber dimensions, ventricular function, valvular morphology and function, and pulmonary artery pressure estimation derived from TR Vmax. The RV focused apical 4 chamber views used to measure the TA diameter in diastole, the tenting height and area. Leaflet tethering was considered significant when the tenting height was more than 8mm or the tenting area was more than 16mm².⁽²³⁾ Moderate or severe TR at the 6-month follow-up was considered significant and accordingly termed “recurrent TR”.

Outcomes

The objective of the study was to assess the short-term clinical and echocardiographic outcomes of secondary TR after left-sided valve surgery. Primary outcomes were freedom from recurrent TR, poor functional status, and mortality at 6 months. The secondary objectives were to determine predictors of recurrent TR and a sub-group analysis comparing no TV repair vs. TV repair in patients with moderate or severe TR pre-operatively.

Data collection

Demographic, clinical, and echocardiographic data were collected at 2 time points during the study. The first data collection point was at enrollment and during the course of the index hospitalisation. The second collection point was at the 6-month post-operative clinic visit.

Statistical analysis

Continuous variables are expressed as mean and standard deviation, or median and interquartile range as appropriate. Categorical variables are expressed as counts and percentages. Echocardiography data was analysed using McNemar’s test and the paired t-test where appropriate. Predictors of recurrent TR were assessed by univariate logistic regression analysis using chi-squared analysis or Fisher’s exact test for categorical variables, and Student’s t-test or Mann-Whitney U-test for continuous variables. A p-value=0.05 was considered statistically significant.

However, emphasis was placed on results reporting 95% confidence intervals and clinical significance, over the p-value results. All analysis was conducted using Statistical Package for the Social Sciences, version 28 (SPSS Inc, Chicago, Ill). Data was analysed with the support of the Division of Epidemiology and Biostatistics at the Faculty of Medicine and Health Sciences, University of Stellenbosch.

Ethical considerations

The study was approved by Stellenbosch University Health Research Ethics Committee (HREC Reference Number S18/10/251).

RESULTS

Pre-operative characteristics

Pre-operative characteristics are summarised in Table I. Patients were a mean age of 49.3 ± 15.5 years (range, 17.3 - 79.2 years) and 51.8% (43 of 83) were female. Atrial fibrillation (AF) was present in 23 (27.7%) patients. Most patients were in NYHA functional class II (38.6%) or III (54.2%). Features of RHF were

TABLE I: Pre-operative characteristics.

Variable	Value (n=83)
Age (years), mean (SD)	49.3 (15.5)
Female, n (%)	43 (51.8)
Comorbidities, n (%)	50 (60.2)
Hypertension	34 (41.0)
Diabetes Mellitus	8 (9.6)
Dyslipidemia	9 (10.8)
HIV	9 (10.8)
AF, n (%)	23 (27.7)
NYHA, n (%)	
I	1 (1.2)
II	32 (38.6)
III	45 (54.2)
IV	5 (6.0)
Previous RHF, n (%)	48 (57.8)
Current RHF, n (%)	59 (71.1)
Etiology, n (%)	
Rheumatic	37 (44.6)
Infective	22 (26.5)
Degenerative	19 (22.9)
Ischaemic	3 (3.6)
Congenital	2 (2.4)
EuroScore II, ⁽³⁶⁾ median (IQR)	2.2 (1.5; 3.5)

HIV: Human Immunodeficiency Virus, AF: atrial fibrillation, NYHA: New York Heart Association, RHF: right heart failure.

present in 59 patients (71.1%) at the time of surgery and 48 (57.8%) had been in RHF previously. Thirty-seven patients (44.6%) had rheumatic heart disease, 22 (26.5%) had infective endocarditis, and 19 (22.9%) had degenerative valve disease. The median EuroScore II was 2.2 (IQR: 1.5; 3.5).

Operative data and outcomes

Forty-two (50.6%) left-sided valve procedures were performed that involved the MV in isolation vs. 24 (28.9%) that involved the AV in isolation (Table II). Combined MV / AV procedures accounted for 13 (15.7%) cases, of which 11 were dual-valve replacements and 2 were MV repair with AV replacement. Thirty-three (39.8%) patients received additional procedures,

TABLE II: Operative data and peri-operative outcomes.

Variable	Value (n=83)
Left-Sided Valve Procedure, n (%)	
MV Replacement	25 (30.1)
MV Repair	17 (20.5)
AV Replacement	24 (28.9)
Combined MV / AV	13 (15.7)
Aortic Root Repair or Replacement	4 (4.8)
Additional Procedure, n (%)	33 (39.8)
LAA Resection	21 (63.6)
CABG	9 (27.3)
Other	3 (9.1)
Concomitant TV Repair; n (%)	9 (10.8)
Procedural Time (min)	
CPB, mean ± SD	163.3 ± 53.4
AOC, mean ± SD	121.2 ± 43.1
LOS (days), median (IQR)	18.0 (12.0; 28.0)
Excluding Infective Endocarditis	15.0 (10.0; 19.5)
Morbidities, n (%)	24 (28.9)
POAF	10 (12.0)
Pneumonia or Prolonged Intubation	6 (7.2)
Wound Infection	7 (8.4)
Relook for Bleeding	5 (6.0)
Permanent Pacemaker	1 (1.2)
UTI	3 (3.6)
MACCE	3 (3.6)
Mortality (30-Day), n (%)	
Cardiac	1 (1.3)
Non-Cardiac	0

MV: mitral valve, AV: aortic valve, LAA: left atrial appendage, CABG: coronary artery bypass grafting, TV: tricuspid valve, CPB: cardiopulmonary bypass, AOC: aortic cross clamp, LOS: length of stay, POAF: post-operative atrial fibrillation, UTI: urinary tract infection, MACCE: major adverse cardiac or cerebrovascular event.

and most of these were for resection of the left atrial appendage (LAA) (63.6%) and coronary artery bypass grafting (CABG) (27.3%). TV repair was performed in 9 (10.8%) patients who all received Medtronic 3D contour ring annuloplasty. No other TV repair techniques were employed. CPB time was 163.3 ± 53.4 minutes and the aortic cross clamp time was 121.2 ± 43.1 minutes. The length of hospital stay from surgery to discharge was 18 (IQR 12.0; 28.0) days for the entire group vs. 15 (IQR 10.0; 19.5) days excluding those with infective endocarditis.

Operative outcomes are listed in Table II. One (1.2%) patient required a permanent pacemaker (PPM) for complete heart block following a double valve procedure (MV repair and AV replacement) with no TV repair. Major adverse cardiac and cerebral events (MACCE) occurred in 3 (3.6%) patients who had low cardiac output syndrome due to severe RV failure. There were no cases of cerebrovascular accident or myocardial infarction. There was 1 (1.3%) operative death that occurred due to a complication of AV replacement on post-operative day one.

We defined this in the methods under post-operative care and follow up section: "Clinical follow-up was complete for 76 of 83 patients (91.6%) and echocardiographic follow-up was complete for 66 of 83 patients (79.5%)".

At this point (i.e. operative and preoperative data we had a complete dataset, and there was no loss to follow up yet - therefore n=83).

Clinical outcomes

At 6 months the freedom from recurrent TR, poor functional status, and mortality was 56 (84.8%), 69 (90.8%), and 72 (94.7%) respectively (Table III). The numerator for recurrent TR is 66 (available echo data), and for poor functional status and mortality it was 76 (available clinical follow-up data). At 6 months there were 4 (5.3%) readmissions for heart failure, and 3 of these patients ultimately demised. The remaining readmission was due to left ventricular (LV) failure from rapid ventricular response and inadequate rate control in the setting of chronic AF. At 6 months, 4 (5.3%) patients had died from cardiac causes. There were no non-cardiac deaths. Two patients demised due to RV failure. Both patients had poor pre-operative RV function (TAPSE 13mm and 14mm respectively), with significant pre-operative TR being present in 1 patient. The third mortality at 6 months was due to LV failure from a thrombosed MV prosthesis due to subtherapeutic anticoagulation. The fourth death was classified as an operative or early death within 30 days and was described in the preceding paragraph: "There was 1 (1.3%) operative death that occurred due to a complication of AV replacement on postoperative day one".

Echocardiographic data of the entire cohort

Pre-operative moderate or severe TR was present in 34 (41.0%) patients (Table IV). The proportion of patients with moderate or severe TR decreased significantly over the study

TABLE III: Clinical outcomes of the entire cohort.

Variable	Patients (n=76)
NYHA	
I	51 (67.1)
II	18 (23.7)
III	4 (5.3)
IV	1 (1.3)
Mortality (6-month)	
Cardiac	4 (5.3)
Non-Cardiac	0
Readmission for Heart Failure	4 (5.3)
Freedom from	
Recurrent TR (n=66)	56 (84.8)
Poor Functional Status	69 (90.8)
Mortality	72 (94.7)

NYHA: New York Heart Association, Recurrent TR: moderate or severe tricuspid regurgitation at 6 months, Poor Functional Status: NYHA class III or IV at 6 months, Freedom from Mortality: overall at 6 months.

period (41% vs. 13.6%, OR 0.3, p<0.01). The chamber dimensions, including LV end-systolic diameter (LVIDs 41.9 ± 9.9mm, 95% CI: 39.8 - 44.0 vs. 36.1 ± 8.9mm, 95% CI: 34.1 - 38.1, p<0.01), left atrial (LA) area (32.5 ± 13.0mm², 95% CI: 29.7 - 35.3 vs. 23.7 ± 8.7mm², 95% CI: 21.7 - 25.7, p<0.01), and right atrial (RA) area (20.5 ± 7.6mm², 95% CI: 18.8 - 22.2 vs. 17.6 ± 5.4mm², 95% CI: 16.4 - 18.8, p<0.01) improved significantly between the pre-operative and follow-up studies. The RV systolic function worsened over the study period (TAPSE 18.5 ± 5.3mm, 95% CI: 17.4 - 19.6 vs. 15.5 ± 3.5mm, 95% CI: 14.7 - 16.3, p<0.01) even though the RV systolic pressure (RVSP) improved (53.3 ± 19.1mmHg, 95% CI: 49.2 - 57.4 vs. 32.7 ± 14.7mmHg, 95% CI: 29.3 - 36.1, p<0.01). Measurements of pre-operative TV morphology revealed TA diameter (42.5 ± 7.3mm, 95% CI: 40.9 - 44.1), tenting height (9.1 ± 2.7mm, 95% CI: 8.5 - 9.7), and tenting area (14.6 ± 5.7mm², 95% CI: 13.4 - 15.8).

Predictors of recurrent TR

Logistic regression was restricted to univariate analysis due to the limited study power. Variables that appeared to be significant were female gender (OR 9.9, 95% CI: 1.2 - 84.7, p=0.04), rheumatic left-sided valve (OR 14.4, 95% CI: 1.7 - 123.6, p=0.02), and RVSP (OR 1.1, 95% CI: 1.0 - 1.1, p<0.01) (Table V). AF (OR 3.8, 95% CI: 0.9 - 15.9, p=0.07), RHF (OR

TABLE IV: Echocardiographic data of the entire cohort pre-operatively and at 6 months.

Variable	Pre-operative value (n=83)	6-month value (n=66)	p-value
Left Heart, mean ± SD (95% CI)			
LVEF (%)	48.3 ± 13.4 (45.4 - 51.2)	49.1 ± 11.3 (46.5 - 51.7)	p=0.47
LVIDs (mm)	41.9 ± 9.9 (39.8 - 44.0)	36.1 ± 8.9 (34.1 - 38.1)	p<0.01
LA Area (cm ²)	32.5 ± 13.0 (29.7 - 35.3)	23.7 ± 8.7 (21.7 - 25.7)	p<0.01
Right Heart, n (%) or mean ± SD (95% CI)			
TR			
None	0	28 (43.1)	p<0.01
Mild	49 (59.0)	28 (43.1)	p=0.06
Moderate	24 (28.9)	8 (12.3)	p=0.01
Severe	10 (12.0)	1 (1.5)	p=0.03
RA Area (cm ²)	20.5 ± 7.6 (18.8 - 22.2)	17.6 ± 5.4 (16.4 - 18.8)	p<0.01
TAPSE (mm)	18.5 ± 5.3 (17.4 - 19.6)	15.5 ± 3.5 (14.7 - 16.3)	p<0.01
RVSP (mmHg)	53.3 ± 19.1 (49.2 - 57.4)	32.7 ± 14.7 (29.3 - 36.1)	p<0.01
TA Diameter (mm)	42.5 ± 7.3 (40.9 - 44.1)	-	-
Mild TR (mm)	41.1 ± 6.4 (39.21 - 42.79)	-	-
TV Tenting Height (mm)	9.1 ± 2.7 (8.5 - 9.7)	-	-
TV Tenting Area (mm ²)	14.6 ± 5.7 (13.4 - 15.8)	-	-

LVEF: left ventricle ejection fraction, LVIDs: left ventricle internal dimension in systole, LA: left atrium, TR: tricuspid regurgitation, RA: right atrium, TAPSE: tricuspid annular plane systolic excursion, RVSP: right ventricle systolic pressure, TA: tricuspid annulus, TV: tricuspid valve.

TABLE V: Predictors of recurrent TR.

Variable	Univariable OR (95% CI) (n=83)	p-value
Demographic		
Age	1.0 (0.9 - 1.0)	p=0.55
Female Gender	9.9 (1.2 - 84.7)	p=0.04
Left-Sided Rheumatic Etiology	14.4 (1.7 - 123.6)	p=0.02
AF	3.8 (0.9 - 15.9)	p=0.07
RHF	2.2 (0.3 - 19.2)	p=0.48
NYHA Class (III/IV)	0.4 (0.1 - 1.7)	p=0.22
Operative		
TVA	0.2 (0.1 - 12.4)	p=0.83
Concomitant Procedure	3.6 (0.8 - 16.0)	p=0.09
Echocardiographic		
TA Diameter (mm)	1.1 (1.0 - 1.2)	p=0.35
TV Tenting Height (mm)	1.1 (0.8 - 1.5)	p=0.52
TV Tenting Area (mm ²)	1.0 (0.9 - 1.2)	p=0.87
Moderate or Severe Preoperative TR	3.6 (0.8 - 16.0)	p=0.09
RVSP (mmHg)	1.1 (1.0 - 1.1)	p<0.01
TAPSE (mm)	1.0 (0.8 - 1.1)	p=0.65
LVEF (%)	1.1 (1.0 - 1.1)	p=0.08
LA Area (mm ²)	1.0 (1.0 - 1.1)	p=0.11
RA Area (mm ²)	1.1 (1.0 - 1.2)	p=0.12

AF: atrial fibrillation, TVA: tricuspid valve annuloplasty, RHF: right heart failure, NYHA: New York Heart Association, TA: tricuspid annulus, TV: tricuspid valve, TR: tricuspid regurgitation, RVSP: right ventricle systolic pressure, TAPSE: tricuspid annular plane systolic excursion, LVEF: left ventricle ejection fraction, LA: left atrium, RA: right atrium.

2.2, 95% CI: 0.3 - 19.2, p=0.48), concomitant procedure (OR 3.6, 95% CI: 0.8 - 16.0, p=0.09), and significant pre-operative TR (OR 3.6, 95% CI: 0.8 - 16.0, p=0.09) trended towards an increased risk for recurrent TR at 6 months. We were unable to demonstrate an association of TA diameter, TV tenting height or area, TAPSE, LVEF, or atrial size with recurrent TR at 6 months.

Sub-group analysis of no TV repair vs. TV repair in patients with moderate or severe pre-operative TR

Pre-operative characteristics and echocardiographic data between the groups are summarised in Table VI. There were 25 patients with moderate or severe pre-operative TR that did not receive TV repair, which included 20 (80%) with moderate TR and 5 (20%) with severe TR. The TV repair group had larger pre-operative TA diameter than no TV repair for patients with moderate TR (42.4 ± 5.1mm, 95% CI: 40.4 - 44.4 vs. 48.3 ±

6.9mm, 95% CI: 43.8 - 52.8, p=0.03), although not for severe TR. Pre-operative TV tenting distance (9.3 ± 2.0mm, 95% CI: 8.5 - 10.1 vs. 13.1 ± 2.3mm, 95% CI: 11.6 - 14.6, p<0.01) and TV tenting area (15.1 ± 5.1mm, 95% CI: 13.1 - 17.1 vs. 21.8 ± 4.1mm, 95% CI: 19.1 - 24.5, p=0.01) were larger in TV repair compared with no TV repair. At 6 months patients with TV repair had worse LVEF (52.7 ± 10.7%, 95% CI: 48.0 - 57.4 vs. 41.5 ± 8.7%, 95% CI: 34.5 - 48.5, p=0.03) than those with no TV repair, yet the remaining echocardiographic outcomes between the groups were comparable. Patients in the TV repair group had either no change (n=2), or improvement by 1 (n=3) or 2 grades (n=2) of TR at 6 months. Table VII summarises the operative data and outcomes between the groups. Resection of the LAA occurred more frequently in the TV repair group (28.0% vs. 66.7% p=0.04). Cardiopulmonary bypass and cross clamp times were numerically greater in the TV repair group. Operative morbidity and mortality between the groups were similar, although pneumonia or prolonged ventilation was more frequent in the TV repair group (0% vs. 22.2% p=0.02). The groups had comparable (2.7% vs. 11.1% p=0.20) MACCE. There was no difference in functional status, mortality, readmission, or recurrent TR at 6 months between the no TV repair and TV repair sub-groups.

DISCUSSION

We found good short-term outcomes in a heterogeneous group of patients with secondary TR undergoing left-sided valve surgery employing a guideline-directed, multidisciplinary team approach. The primary outcomes for the entire cohort were characterised by high rates of freedom from recurrent TR (86.2%), poor functional class (90.8%), and mortality (94.7%) at 6 months after surgery.

There are limited and often conflicting data to inform whether these results can be sustained over the medium and long term, especially among patients with no TV repair.⁽²⁴⁾ The natural history and outcomes of secondary TR depend on numerous factors, including etiology of the left-sided valvopathy, degree of pulmonary hypertension, pre-operative TR grade, TA dimension, RV function, and tenting height and tenting area making it difficult to generalise and create a uniform approach.⁽¹⁾ There is conflicting evidence surrounding the notion that progression of TR and deterioration of functional status occurs over time when secondary TR is managed conservatively. At 4.8 years after MV repair for degenerative MV disease, in patients with significant TA dilatation and varying degrees of TR, recurrent TR and worse NYHA functional status was more frequent in those without TV repair than those who had TV repair, yet their mortality rates were similar.⁽²⁵⁾ At 4 years significant recurrent TR was more common in patients who did not undergo TV repair in a cohort of patients undergoing MV replacement

TABLE VI: Pre-operative characteristics and echocardiographic data of no TV repair vs. TV repair in patients with moderate or severe pre-operative TR.

Variable	No TV Repair	TV Repair	p-value
Pre-operative	(n=25)	(n=9)	
Age (years), mean (SD)	47.2 (14.7)	42.9 (14.2)	p=0.45
Female, n (%)	13 (52.0)	5 (55.6)	p=0.86
Comorbidities, n (%)	15 (60.0)	7 (77.8)	p=0.34
AF, n (%)	7 (28.0)	5 (55.6)	p=0.14
NYHA, n (%)			
I	0	0	-
II	8 (32.0)	4 (44.4)	p=0.50
III	16 (64.0)	4 (44.4)	p=0.31
IV	1 (4.0)	1 (11.1)	p=0.44
Previous RHF, n (%)	16 (64.0)	7 (77.8)	p=0.45
With Pre-operative			
Moderate TR	11 (55.0) (n=20)	2 (50.0) (n=4)	p=0.86
Severe TR	5 (100.0) (n=5)	5 (100.0) (n=5)	-
Etiology, n (%)			
Rheumatic	14 (56.0)	9 (100.0)	p=0.03
EuroScore II, ⁽³⁶⁾ median (IQR)	2.8 (2.0)	2.9 (1.4)	p=0.75
Echocardiographic			
Pre-operative	(n=25)	(n=9)	
Six-month	(n=20)	(n=6)	
Left Heart, mean ± SD (95% CI)			
LVEF (%)			
Pre-operative	46.4 ± 14.3 (40.8 - 52.0)	43.2 ± 7.4 (38.4 - 48.0)	p=0.41
Six-month	52.7 ± 10.7 (48.0 - 57.4)	41.5 ± 8.7 (34.5 - 48.5)	p=0.03
LVIDs (mm)			
Pre-operative	41.6 ± 9.7 (37.8 - 45.4)	41.3 ± 8.2 (35.9 - 46.7)	p=0.93
Six-month	34.0 ± 7.5 (30.7 - 37.3)	40.2 ± 2.4 (38.3 - 42.1)	p=0.06
LA Area (cm ²)			
Pre-operative	35.7 ± 15.5 (28.9 - 42.5)	36.5 ± 8.4 (30.7 - 42.3)	p=0.89
Six-month	23.4 ± 6.9 (20.4 - 26.4)	26.5 ± 7.1 (20.8 - 32.2)	p=0.35
Right Heart, n (%) or mean ± SD (95% CI)			
TR (Moderate or Severe)	25 (100.0)	9 (100.0)	-
Pre-operative			
Moderate	20 (80.0)	4 (44.4)	
Severe	5 (20.0)	5 (55.6)	
Six-month	8 (40.0)	1 (16.7)	p=0.29
Moderate	7 (87.5)	1 (100.0)	
Severe	1 (12.5)	0	
RA Area (cm ²)			
Pre-operative	20.6 ± 7.0 (17.9 - 23.3)	28.5 ± 8.3 (22.8 - 34.3)	p=0.01
Six-month	17.7 ± 5.0 (15.5 - 19.9)	20.7 ± 7.2 (14.9 - 26.5)	p=0.24
TA Diameter (mm)	42.2 ± 6.0 (39.9 - 44.6)	49.1 ± 10.2 (42.4 - 55.8)	p=0.08
With Pre-operative			
Moderate TR	42.4 ± 5.1 (40.4 - 44.4)	48.3 ± 6.9 (43.8 - 52.8)	p=0.03
Severe TR	41.6 ± 9.4 (37.9 - 45.3)	49.8 ± 13.0 (41.3 - 58.3)	p=0.14
TV Tenting Height (mm)	9.3 ± 2.0 (8.5 - 10.1)	13.1 ± 2.3 (11.6 - 14.6)	p<0.01
With Pre-operative			
Moderate TR	9.5 ± 2.0 (8.7 - 10.3)	13.3 ± 2.5 (11.7 - 14.9)	p<0.01
Severe TR	8.6 ± 1.8 (7.9 - 9.3)	13.0 ± 2.4 (11.4 - 14.6)	p<0.01
TV Tenting Area (mm ²)	15.1 ± 5.1 (13.1 - 17.1)	21.8 ± 4.1 (19.1 - 24.5)	p=0.01
With Pre-operative			
Moderate TR	15.5 ± 5.1 (13.5 - 17.5)	21.3 ± 3.8 (18.8 - 23.8)	p=0.02
Severe TR	13.4 ± 5.5 (11.2 - 15.6)	22.2 ± 4.7 (19.1 - 25.3)	p=0.01
TAPSE (mm)			
Pre-operative	17.6 ± 5.3 (15.5 - 19.7)	15.6 ± 6.3 (11.2 - 20.0)	p=0.39
Six-month	15.4 ± 3.8 (13.7 - 17.1)	13.8 ± 3.5 (11.0 - 16.6)	p=0.39
RVSP (mmHg)			
Pre-operative	62.4 ± 22.1 (53.7 - 71.1)	57.4 ± 22.8 (42.5 - 72.3)	p=0.57
Six-month	34.5 ± 19.6 (25.9 - 43.1)	38.5 ± 6.1 (33.6 - 43.4)	p=0.63

TV: tricuspid valve, AF: atrial fibrillation, NYHA: New York Heart Association, RHF: right heart failure, TA: tricuspid annulus, TR: tricuspid regurgitation, RVSP: right ventricle systolic pressure, TAPSE: tricuspid annular plane systolic excursion, LVEF: left ventricle ejection fraction, LVIDs: left ventricle internal dimension in systole, LA: left atrium, RA: right atrium.



TABLE VII: Operative data and outcomes of no TV repair vs. TV repair in patients with moderate or severe pre-operative TR.

Variable	No TV Repair	TV Repair	p-value
Operative data	(n=25)	(n=9)	
Left-Sided Valve Procedure, n (%)			
MV Replacement	10 (40.0)	8 (88.9)	p=0.01
MV Repair	9 (36.0)	0	p=0.20
AV Replacement	10 (40.0)	1 (11.1)	p=0.16
Additional Procedure, n (%)			
LAA Resection	7 (28.0)	6 (66.7)	p=0.04
CABG	3 (12.0)	0	p=0.28
Procedural Time (min)			
CPB, mean ± SD (95% CI)	150.4 ± 34.8 (136.8 – 164.0)	200.9 ± 74.5 (152.2 – 249.6)	p=0.08
AOC, mean ± SD (95% CI)	112.2 ± 31.7 (99.8 – 124.6)	141.1 ± 58.3 (103.0 – 179.2)	p=0.07
Operative outcomes	(n=25)	(n=9)	
Morbidities, n (%)			
POAF	5 (20.0)	1 (11.1)	p=0.55
Pneumonia or Prolonged Intubation	0	2 (22.2)	p=0.02
Wound Infection	3 (12.0)	1 (11.1)	p=0.94
Relook for Bleeding	4 (16.0)	0	p=0.20
Permanent Pacemaker	0	0	-
UTI	1 (4.0)	0	p=0.70
MACCE	2 (2.7)	1 (11.1)	p=0.78
Mortality (30-day), n (%)			
Cardiac	0	0	-
Non-Cardiac	0	0	-
Six-month outcomes	(n=23)	(n=7)	
NYHA			
I	15 (65.2)	5 (71.4)	p=0.76
II	6 (26.1)	1 (14.3)	p=0.52
III	2 (8.7)	1 (14.3)	p=0.78
IV	0	0	-
Mortality			
Cardiac	1 (4.3)	1 (14.3)	p=0.36
Non-Cardiac	0	0	-
Readmission for Heart Failure	1 (4.3)	1 (14.3)	P=0.36
Freedom from			
Recurrent TR	15 (75.0) (n=20)	5 (83.3) (n=6)	p=0.67
Poor Functional Status	21 (91.3)	6 (85.7)	p=0.67
Mortality	22 (95.7)	6 (85.7)	p=0.36

TVA: tricuspid valve annuloplasty, MV: mitral valve, AV: aortic valve, LAA: left atrial appendage, CABG: coronary artery bypass grafting, TV: tricuspid valve, CPB: cardiopulmonary bypass, AOC: aortic cross clamp, POAF: post-operative atrial fibrillation, UTI: urinary tract infection, MACCE: major adverse cardiac or cerebrovascular event, NYHA: New York Heart Association, Recurrent TR: moderate or severe tricuspid regurgitation at 6 months, Poor Functional Status: NYHA class III or IV at 6 months, Freedom from mortality: overall at 6 months.

for rheumatic disease, despite both groups having less than moderate TR pre-operatively.⁽²⁶⁾ A third study found that 5 years after MV repair for secondary MR, patients with pre-operative moderate or more TR who did not have TV repair had a significantly higher risk of recurrent TR, poor functional

status, and mortality.⁽²⁷⁾ In contrast, another study showed that at 5.5 years TR progression was unusual in patients undergoing repair of degenerative MV disease without TV repair.⁽²⁸⁾ Our practice was generally characterised by selective treatment of severe TR associated with various left-sided valvopathies, yet

mostly conservative treatment of less-than-severe TR, where other factors like RHF and TV morphology were considered to reach a treatment decision.

The effect that various left-sided valvopathies had on secondary TR outcomes after conservative TV management was summarised by Song, et al.⁽²⁹⁾ who reported that rates of recurrent moderate or severe TR at 8.5 years in 638 patients with pre-operative mild TR, was 8% - 26% for rheumatic MV disease, 5% for degenerative MV disease, and 3% for mixed aortic valve disease. Our study included a significant proportion of patients with rheumatic valve disease (44.6%) associated with mild (66.2%) and moderate or more (33.8%) pre-operative TR who did not undergo TV repair. The long-term outcomes of this cohort are difficult to predict, however it is likely that these patients remain at risk of recurrent TR. Longer follow-up is required to clarify the risk.

Despite these long-term concerns, this study demonstrated that our multidisciplinary team used an individualised, evidence-based approach to achieve good short-term results in this cohort of patients. We performed TV repair for those who had class I indications (i.e. severe TR). In contrast, patients with class II indications for concomitant TV repair (i.e. less-than-severe TR with TA dilatation or previous RHF) were generally treated more conservatively unless compelling indications for surgery existed such as severely enlarged TA or significant TV tethering. This study had a high proportion of patients with mild pre-operative TR (59.0%) who had TA measurement exceeding 40mm (mean=41.1mm). None of these patients received TV repair despite the guidelines suggesting a class II indication for concomitant repair in these circumstances. The 6-month outcomes in this group remained satisfactory in the vast majority of patients. For patients who had moderate TR with TA dilatation (mean=42.4mm), it could be argued that we should have pursued a more aggressive approach as 7 patients had moderate TR at 6 months. Our approach yielded good short-term outcomes and these patients (especially the group with moderate TR) should be followed in the medium and long term to monitor the evolution of TR. A recent trial supported this cautious approach to less-than-severe secondary TR.⁽¹⁹⁾ That trial demonstrated that even though recurrent TR was more common in the no TV repair group at 2 years, the risk of major adverse outcomes, poor functional status, and death were the same in the 2 groups, whereas the PPM rate was almost 6 times higher in the TV repair group. Based on these results, Chikwe and colleagues suggest these seemingly benign medium-term consequences of recurrent TR, together with the significantly increased risk for PPM mean that an aggressive approach to TV

repair is probably not warranted – especially not for high risk patients.⁽³⁰⁾ None of the patients in this study undergoing TV repair required PPM implantation post-operatively.

Reported predictors of recurrent TR after left-sided valve surgery include age, female gender, rheumatic left-sided valvopathy, increased pulmonary artery (PA) pressure or RVSP, AF, RHF, pre-operative TR severity, TV morphology, impaired ventricular function, and increased atrial size.^(5,9,19,21,31,32) This study demonstrated similar findings, with female gender, left-sided rheumatic valve disease, and increased RVSP being significant risk factors for recurrent TR. Although AF, RHF, and pre-operative TR severity did not reach statistical significance, there was a trend to an increased risk of recurrent TR. True differences between the groups were probably underestimated due to the small numbers in our study. Considering that recurrent TR has not been conclusively linked with poorer clinical outcomes in the long term, some authors⁽¹⁷⁾ believe that it may actually be these underlying pre-operative risk factors that are more important for the long-term outcomes of patients with secondary TR. However, they emphasise that 5-year follow-up may not be long enough to assess the true effect of significant TR, which may have a long latent period before significant effects on the heart are observed.

The exploratory analysis between no TV repair and TV repair should be interpreted with caution and is meant to be purely hypothesis generating due to the limited numbers in the groups. No differences in the 6-month outcomes between these sub-groups were detected. The early operative outcomes between the groups were also similar. These seemingly comparable results between the no TV repair and TV repair groups occurred despite the latter demonstrating a higher risk profile including more rheumatic heart valve disease, mitral valve replacement, and LAA resection. There was 1 (1.4%) operative death due to a complication of AV replacement that occurred in the no TV repair group. The observed operative mortality for this diverse group of patients compares favorably with rates of 4% as reported by other authors.^(20,33)

The implantation of 1 (1.4%) PPM was required for complete heart block which occurred following a dual-valve procedure in the no TV repair group. Although this tentative analysis did not demonstrate a higher risk of PPM implantation in the TV repair group, there is conflicting data regarding the risk of PPM after TV repair. Even though a systematic review of mostly observational data failed to demonstrate an association between TV repair and need for early PPM implantation,⁽²⁰⁾ a recent trial revealed that the incidence of pacemaker implantation was significantly higher in the concomitant TV repair group.⁽¹⁹⁾

It is not unexpected that even with low rates of recurrent TR and significantly improved RVSP, RV dysfunction (as measured by diminished TAPSE) was still present at 6 months. There is evidence that irrespective of TV repair status, early RV dysfunction tends to worsen for all patients undergoing surgical repair of MV disease, except in those with pre-operative severe TR and significant RV dysfunction, who often demonstrate transient early improvement in RV function.⁽³⁴⁾ The reasons for early RV dysfunction are uncertain, but may include post-surgical changes from sub-optimal intra-operative myocardial protection of the RV, or could reflect the effect of various loading conditions on the heart (e.g. post-operative changes in pre- and afterload).⁽³⁴⁾ Additionally, we demonstrated that LV dysfunction (measured by LVEF) at 6 months appeared to be more pronounced in the TV repair group. In light of the preceding discussion about RV dysfunction, this is not surprising as it is well established that the function of both ventricles is intimately linked.⁽³⁵⁾ Reassuringly, others have found that medium- to long-term outcomes at 3 - 5 years demonstrated that RV dysfunction resolved, and the improvement of RV function and TAPSE occurred sooner in patients with TV repair vs. no TV repair.^(13,34) In our study, longer follow-up is required to confirm whether or not this subsequent improvement of RV and LV function occurs. It would also be valuable to have additional parameters to assess RV function, including echocardiography based myocardial performance index and RV fractional area change, and cardiovascular magnetic resonance assessments of chamber volume and function.⁽⁴⁾

In conclusion, this study has shown that careful and individualised application of secondary TR guidelines produced good short-term results. Overall, the cohort demonstrated low rates of recurrent TR that were associated with good functional status and low mortality at 6 months. The similar results between the sub-groups are encouraging considering that the TV repair group had more pre-operative and operative risk features than the no TV repair group. It was also reassuring to note that PPM rates were low. Although we have demonstrated good short-term outcomes, longer follow-up is required to assess the long-term outcomes of patients undergoing left-sided valve surgery with associated secondary TR in order to clarify indications for concomitant TV repair.

LIMITATIONS

This study had a number of limitations. It was a single centre study and therefore the results may not be generalisable. Observational studies are also open to treatment allocation bias and hidden confounders. Furthermore, the inclusion of

various types of left-sided heart valve disease may have a confounding effect on TR outcomes. The study duration was relatively short and further follow-up is necessary to determine the long-term effects on clinical and echocardiographic outcomes of secondary TR. The small sample size was underpowered to allow adequate comparisons between no TV repair and TV repair or inferences about predictors of TR. Inclusion of patients with rheumatic heart disease may confound the etiology of TR (histopathological testing was not uniformly performed), and lead to the erroneous inclusion of patients with primary rheumatic TV involvement. To mitigate this TV morphology was carefully interrogated to ensure that only cases of secondary TR were included.

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REFERENCES

- Antunes MJ, Rodríguez-Palomares J, Prendergast B, et al. Management of tricuspid valve regurgitation: Position statement of the European Society of Cardiology Working Groups of Cardiovascular Surgery and Valvular Heart Disease. *Eur J Cardio-thoracic Surg.* 2017;52(6):1022-30. doi:10.1093/EJCTS/EZX279.
- Navia JL, Brozzi NA, Klein AL, et al. Moderate tricuspid regurgitation with left-sided degenerative heart valve disease: To repair or not to repair? *Ann Thorac Surg.* 2012;93(1):59-69. doi:10.1016/j.athoracsur.2011.08.037.
- Arsalan M, Walther T, Smith RL, Grayburn PA. Tricuspid regurgitation diagnosis and treatment. *Eur Heart J.* 2017;38(9):634-8. doi:10.1093/eurheartj/ehv487.
- Mas PT, Rodríguez-Palomares JF, Antunes MJ. Secondary tricuspid valve regurgitation: A forgotten entity. *Heart.* 2015;101(22):1840-8. doi:10.1136/heartjnl-2014-307252.
- Muraru D, Surkova E, Badano LP. Revisit of functional tricuspid regurgitation: Current trends in the diagnosis and management. *Korean Circ J.* 2016; 46(4):443-55. doi:10.4070/kcj.2016.46.4.443.
- David TE, David CM, Manliot C. Tricuspid annulus diameter does not predict the development of tricuspid regurgitation after mitral valve repair for mitral regurgitation due to degenerative diseases [Internet]. Vol. 155, *Journal of Thoracic and Cardiovascular Surgery.* 2018. 2429-2436 p. doi:10.1016/j.jtcvs.2017.12.126.
- Faggion Vinholo T, Mori M, Mahmood SU Bin, et al. Combined valve operations in the aortic and mitral positions with or without added tricuspid valve repair. *Semin Thorac Cardiovasc Surg.* 2020;32(4):665-72. doi:10.1053/j.semtcvs.2020.02.010.
- Kara I, Koksali C, Erkin A, Sacli H, Demirtas M, Percin B, et al. Outcomes of mild to moderate functional tricuspid regurgitation in patients undergoing mitral valve operations: A meta-analysis of 2 488 patients. *Ann Thorac Surg.* 2015;100(6):2398-407. doi:10.1016/j.athoracsur.2015.07.024.
- Rodés-Cabau J, Taramasso M, O'Gara PT. Diagnosis and treatment of tricuspid valve disease: Current and future perspectives. *Lancet.* 2016; 388(10058):2431-42. doi:10.1016/S0140-6736(16)00740-6.
- Pagnesi M, Montalto C, Mangieri A, et al. Tricuspid annuloplasty vs. a conservative approach in patients with functional tricuspid regurgitation undergoing left-sided heart valve surgery: A study-level meta-analysis. *Int J Cardiol.* 2017;240:138-44. doi:10.1016/j.ijcard.2017.05.014.
- Hage A, Hage F, Jones PM, Manian U, Tzemos N, Chu MWA. Evolution of tricuspid regurgitation after repair of degenerative mitral regurgitation. *Ann Thorac Surg.* 2020;109(5):1350-5. doi:10.1016/j.athoracsur.2019.08.025.
- Calafiore AM, Gallina S, Iacò AL, et al. Mitral valve surgery for functional mitral regurgitation: Should moderate-or-more tricuspid regurgitation be treated? A propensity score analysis. *Ann Thorac Surg.* 2009;87(3):698-703. doi:10.1016/j.athoracsur.2008.11.028.
- Chikwe J, Itagaki S, Anyanwu A, Adams DH. Impact of concomitant tricuspid annuloplasty on tricuspid regurgitation, right ventricular function, and pulmonary artery hypertension after repair of mitral valve prolapse. *J Am Coll Cardiol.* 2015;65(18):1931-8. doi:10.1016/j.jacc.2015.01.059.
- Otto CM, Nishimura RA, Bonow RO, et al. 2020 ACC / AHA guideline for the management of patients with valvular heart disease: A report of the American College of Cardiology / American Heart Association Joint Committee on Clinical Practice Guidelines. *J Thorac Cardiovasc Surg.* 2021; 162(2):e183-353. doi:10.1016/j.jtcvs.2021.04.002.
- Vahanian A, Beyersdorf F. 2021 ESC / EACTS guidelines for the management of valvular heart disease. *Eur J Cardio-thoracic Surg.* 2021;60(4):727-800. doi:10.1093/ejcts/ezab389.
- Brescia AA, Ward ST, Watt TMF, et al. Outcomes of guideline-directed concomitant annuloplasty for functional tricuspid regurgitation. *Ann Thorac Surg.* 2020;109(4):1227-32. doi:10.1016/j.athoracsur.2019.07.035.
- Ro SK, Kim JB, Jung SH, Choo SJ, Chung CH, Lee JW. Mild-to-moderate functional tricuspid regurgitation in patients undergoing mitral valve surgery. *J Thorac Cardiovasc Surg.* 2013;146(5):1092-7. doi:10.1016/j.jtcvs.2012.07.100.
- Chan V, Burwash IG, Lam BK, et al. Clinical and echocardiographic impact of functional tricuspid regurgitation repair at the time of mitral valve replacement. *Ann Thorac Surg.* 2009;88(4):1209-15. doi:10.1016/j.athoracsur.2009.06.034.
- Gammie JS, Chu MWA, Falk V, et al. Concomitant tricuspid repair in patients with degenerative mitral regurgitation. *N Engl J Med.* 2022;386(4):327-39. doi:10.1056/nejmoa2115961.
- Veen KM, Etnel JRG, Quanjel TJM, et al. Outcomes after surgery for functional tricuspid regurgitation: A systematic review and meta-analysis. *Eur Hear J - Qual Care Clin Outcomes.* 2020;6(1):10-8. doi:10.1093/ehjqcco/qcz032.
- Zhu TY, Min XP, Zhang HB, Meng X. Pre-operative risk factors for residual tricuspid regurgitation after isolated left-sided valve surgery: A systematic review and meta-analysis. *Cardiol.* 2014;129(4):242-9. doi:10.1159/000367589.
- Zoghbi WA, Adams D, Bonow RO, et al. Recommendations for noninvasive evaluation of native valvular regurgitation: A report from the American Society of Echocardiography developed in collaboration with the Society for Cardiovascular Magnetic Resonance. *J Am Soc Echocardiogr.* 2017;30(4):303-71. doi:10.1016/j.echo.2017.01.007.
- Kim HK, Kim YJ, Park JS, et al. Determinants of the severity of functional tricuspid regurgitation. *Am J Cardiol.* 2006;98(2):236-42. doi:10.1016/j.amjcard.2006.01.082.
- Di Mauro M, Calafiore AM, Penco M, Romano S, Di Giammarco G, Gallina S. Mitral valve repair for dilated cardiomyopathy: Predictive role of right ventricular dysfunction. *Eur Heart J.* 2007;28(20):2510-6. doi:10.1093/eurheartj/ehm375.
- Dreyfus GD, Corbi PJ, Chan KMJ, Bahrami T. Secondary tricuspid regurgitation or dilatation: Which should be the criteria for surgical repair? *Ann Thorac Surg.* 2005;79(1):127-32. doi:10.1016/j.athoracsur.2004.06.057.
- Kim JB, Yoo DG, Kim GS, et al. Mild-to-moderate functional tricuspid regurgitation in patients undergoing valve replacement for rheumatic mitral disease: The influence of tricuspid valve repair on clinical and echocardiographic outcomes. *Heart.* 2012;98(1):24-30. doi:10.1136/heartjnl-2011-300403.
- Calafiore AM, Gallina S, Iacò AL, et al. Mitral valve surgery for functional mitral regurgitation: Should moderate-or-more tricuspid regurgitation be treated? A propensity score analysis. *Ann Thorac Surg.* 2009;87(3):698-703. doi:10.1016/j.athoracsur.2008.11.028.
- Yilmaz O, Suri RM, Dearani JA, et al. Functional tricuspid regurgitation at the time of mitral valve repair for degenerative leaflet prolapse: The case for a selective approach. *J Thorac Cardiovasc Surg.* 2011;142(3):608-13. doi:10.1016/j.jtcvs.2010.10.042.
- Song H, Kim MJ, Chung CH, et al. Factors associated with development of late significant tricuspid regurgitation after successful left-sided valve surgery. *Heart.* 2009;95(11):931-6. doi:10.1136/hrt.2008.152793.
- Chikwe J, Gaudino M. The price of freedom from tricuspid regurgitation. *N Engl J Med.* 2022;386(4):389-90. doi:10.1056/nejme2116776.
- Taramasso M, Gavazzoni M, Pozzoli A, et al. Tricuspid regurgitation: Predicting the need for intervention, procedural success, and recurrence of disease. *JACC Cardiovasc Imaging.* 2019;12(4):605-21. doi:10.1016/j.jcmg.2018.11.034.
- Czapla J, Claus I, Martens T, et al. Midterm comparison between different annuloplasty techniques for functional tricuspid regurgitation. *Ann Thorac Surg.* 2022;114(1):134-41. doi:10.1016/j.athoracsur.2021.07.073.
- Naili MA, Herbst PG, Doubell AF, Janson JJ, Pecoraro AJK. A retrospective audit of mitral valve repair surgery at Tygerberg Hospital. *SA Heart®.* 2018;15(3):182-9. doi:10.24170/15-3-3182.
- Desai RR, Vargas Abello LM, Klein AL, et al. Tricuspid regurgitation and right ventricular function after mitral valve surgery with or without concomitant tricuspid valve procedure. *J Thorac Cardiovasc Surg.* 2013;146(5):1126-1132. e10. doi:10.1016/j.jtcvs.2012.08.061.
- Houston BA, Brittain EL, Tedford RJ. Right ventricular failure. *N Engl J Med.* 2023;388(12):1111-25. doi:10.1056/NEJMra2207410.
- Nashef SAM, Roques F, Sharples LD, et al. To update the European System for Cardiac Operative Risk Evaluation (EuroSCORE) risk model. *Euroscore II. Eur J Cardio-thoracic Surg.* 2012;41(4):734-45. doi:10.1093/ejcts/ezs043.