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Pacemaker implantation after concomitant tricuspid valve repair in patients undergoing minimally invasive mitral valve surgery: Results from the Mini-Mitral International Registry

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ABSTRACT

Objective: Randomized evidence suggests a high risk of pacemaker implantation for patients undergoing mitral valve (MV) surgery with concomitant tricuspid valve repair (cTVR). We investigated the impact of cTVR on outcomes in the Mini-Mitral International Registry.

Methods: From 2015 to 2021, 7513 patients underwent minimally invasive MV with or without cTVR in 17 international centers (MV: n = 5609, cTVR: n = 1113). Propensity matching generated 1110 well-balanced pairs. Multivariable analysis was applied.

Results: Patients with cTVR were older and had more comorbidities. Propensity matching eliminated most differences except for more TR in patients who underwent cTVR (77.2% vs 22.1% MV, P < .001). Mean matched age was 71 years, and 45% were male. European System for Cardiac Operative Risk Evaluation II was still 2.68% (interquartile range [IQR], 0.80-2.63) vs 1.9% (IQR, 1.12-3.9) in matched MV (P < .001). MV replacement (30%) and atrial fibrillation surgery (32%) were similar in both groups. Cardiopulmonary bypass (161 minutes [IQR, 133-203] vs MV: 130 minutes [IQR, 103-166]; P < .001) and crossclamp times (93 minutes [IQR, 66-123] vs MV: 83 minutes [IQR, 64-107]; P < .001) were longer with cTVR. Although in-hospital mortality was similar (cTVR: 3.3% vs MV: 2.2%; P = .5), postoperative pacemaker implantations (9% vs MV: 5.8%; P = .02), low cardiac output syndrome (7.7% vs MV: 4.4%; P = .02), and acute kidney injury (13.8%) vs MV: 10%; P = .01) were more frequent with cTVR. cTVR eliminated relevant TR in most patients (greater-than-moderate TR: 6.8%). Multivariable analysis identified MV replacement, atrial fibrillation, and cTVR as risk factors of postoperative pacemaker implantation.

Conclusions: cTVR in minimally invasive MV surgery is an independent risk factor for pacemaker implantation in this international registry. It is also associated with more bleeding, low output syndrome, and acute kidney injury. It remains unclear whether technical or patient factors (or both) explain these differences. (JTCVS Open 2024;17:64-71)



Pacemaker implantations after mini-mitral surgery ± concomitant tricuspid valve repair.

CENTRAL MESSAGE

Concomitant tricuspid valve repair in mitral valve patients is an independent risk factor for pacemaker implantation. It remains unclear whether technical or patient factors explain these differences.

PERSPECTIVE

Since concomitant tricuspid repair in mitral surgery has the potential to prevent worsening tricuspid regurgitation during follow-up, the finding of increased pacemaker need is important for consenting patients properly and also illustrates the need for more detailed investigations on the underlying causes of postoperative new pacemaker requirements.

See Discussion on page 72.

Abbreviations and Acronyms						
CTSN	= Cardiothoracic Surgical Trials					
	Network					
EuroSCOR	E = European System for Cardiac					
	Operative Risk Evaluation					
IQR	= interquartile range					
MV	= mitral valve					
PS	= propensity score					
TR	= tricuspid regurgitation					
TV	= tricuspid valve					
TVR	= tricuspid valve repair					

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The recent Cardiothoracic Surgical Trials Network (CTSN) trial, assessing the impact of tricuspid valve repair (TVR) at the time of mitral valve (MV) surgery, demonstrated a reduction of severe tricuspid regurgitation (TR) during follow-up by TVR but a significantly increased risk of pacemaker implantation (11.6% absolute difference).¹ This incidence was judged as surprisingly high,^{2,3} and the question arises whether this incidence reflects daily routine.

The Mini-Mitral International Registry reflects a collection of real-world data from 17 expert valve centers across the world, which contains a significant fraction of patients who received MV plus tricuspid valve (TV) surgery and which collected information on postoperative pacemaker implantation as well as other perioperative outcomes.

We assessed the incidence of pacemaker implantation in this patient population in those patients receiving only MV surgery as well as those receiving concomitant tricuspid surgery. We related the findings to other classic outcomes.

METHODS

International Registry and Patient Data

The Mini-Mitral International Registry is an independent registry involving 17 international heart valve centers. The centers combined all patients who received minimally invasive MV operations with or without associated procedures between 2015 and 2021. According to the protocol of the registry, all patients who underwent surgery during the inclusion period had to be provided by the participating centers.⁴ Perioperative characteristics and in-hospital outcomes of 7513 consecutive patients were collected according to Mitral Valve Academic Research Consortium definitions.⁵ Patients with pacemaker implantation before MV surgery, endocarditis, and the need for tricuspid valve (TV) replacement were excluded, resulting in 6722 patients to be included in this analysis. The protocol of the registry, description of the data source, and definition have been published previously.⁴ Ethic boards of all centers approved participation in the registry and this analysis.

Statistical Analysis

Continuous variables were expressed as mean (standard deviation) and categorical variables as percentages. Where continuous variables did not follow a normal distribution (tested using the Kolmogorov–Smirnov test for normality and Q-Q plots), the median and interquartile range (IQR) were reported. Continuous data were compared with the Student *t* test or Mann–Whitney *U* test, as appropriate. Categorical variables were compared by the χ^2 test. Multivariable logistic and linear regression models (using backward stepwise algorithm) were estimated to evaluate the effect of patients' risk profile on outcomes.

To account for potential confounding effects and treatment allocation bias in our analyses, propensity score (PS) matching was performed to generate a study cohort of matched patients treated with cTVR treated and patients treated with MV. PS were estimated using logistic regression modeling with type of intervention (cTVR vs MV) as the dependent variable and relevant covariates as the independent variables. The independent variables were center, sex, age, hypertension, diabetes, smoking, chronic lung disease, renal failure, poor mobility, cerebrovascular disease, peripheral vascular disease, coronary artery disease, New York Heart Association class, preoperative cardiac rhythm, preoperative atrioventricular block, left ventricular ejection fraction, pulmonary hypertension, critical preoperative state, previous cardiac surgery, type of MV surgery, concomitant atrial fibrillation surgery, reintervention, and urgent/emergent status. Patients undergoing cTVR were matched on a one-on-one basis with patients undergoing MV surgery on the basis of PS, by the use of nearest-neighbor matching without replacement, and a caliper of width equal to 0.2 of the standard deviation of the logit of the PS, resulting in equal-size study cohorts. Covariate balance was measured using the standardized differences between the 2 treatment groups, which were calculated as the differences in the means divided by the pooled standard deviation and expressed as a percentage. A standardized difference greater than 10% was considered to represent a meaningful covariate

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TABLE 1. Patient characteristics

	Overall cohort			Propensity-matched cohort				
	cTVR	MV			cTVR	MV		
Characteristics	(n = 1113)	(n = 5609)	SD	<i>P</i> value	(n = 1110)	(n = 1110)	SD	<i>P</i> value
Age, y	72 (64-76)	63 (53-72)	69.4	<.001	72 (64-76)	70 (62-77)	-1.1	.4
Male	496 (44.6)	3405 (60.7)	-32.4	<.001	496 (44.7)	508 (45.8)	-4.9	.5
NYHA class III-IV	713 (64)	2447 (43.6)	42.4	<.001	710 (64)	695 (62.6)	2.2	.6
Arterial hypertension	761 (68.4)	3122 (55.7)	27.7	<.001	758 (68.2)	750 (67.6)	2.7	.7
Diabetes	123 (11)	424 (0.7)	11.1	<.001	123 (11)	124 (11.2)	-0.6	.8
Preoperative atrial fibrillation	695 (62.4)	1465 (26.1)	56.5	<.001	692 (62.3)	669 (60.3)	2.6	.8
Preoperative AV block I II	27 (2.4) 2 (0.2)	194 (3.5) 9 (0.2)	-2.8	.2	27 (2.4) 2 (0.2)	35 (3.2) 1 (0.09)	1.6	.5
Renal impairment (eGFR <85)	851 (76.5)	3047 (54.3)	48.6	<.001	848 (76.4)	832 (75)	0.5	.4
Dialysis	13 (1.2)	48 (0.9)	2.9	.3	13 (1.2)	16 (1.4)	-2.5	.6
Coronary artery disease	189 (17)	717 (12.8)	11.5	.01	188 (16.9)	185 (16.7)	3.3	.4
Chronic lung disease	123 (11)	462 (8.2)	10.3	.02	123 (11)	123 (11)	-	1
Frailty	53 (4.8)	125 (2.2)	11.5	<.001	53 (4.8)	50 (4.5)	0.9	.8
Cerebrovascular disease	21 (1.9)	88 (1.6)	2.2	.47	21 (1.9)	22 (2)	0.7	.8
Peripheral arteriopathy	33 (3)	132 (2.4)	3.6	.23	33 (3)	31 (2.8)	2.1	.5
Previous cardiac surgery	118 (10.6)	341 (6)	14.6	<.001	117 (10.5)	110 (9.9%)	3.8	.6
MV stenosis (moderate to severe)	117 (10.5)	398 (7.1)	12.6	.02	117 (10.5)	124 (11.2)	2.4	.4
MR (moderate to severe)	998 (89.7)	5299 (94.5)	-18	.04	996 (89.7)	1013 (91.3)	-9	.1
MV disease etiology Degenerative Functional Rheumatic Failure previous MV surgery Other	572 (51.3) 336 (30.1) 139 (12.5) 42 (3.8) 24 (2.2)	4261 (76) 627 (11.2) 397 (7.1) 162 (2.9) 162 (2.9)	-26.7	<.001	570 (51.2) 335 (30.2) 139 (12.6) 42 (3.8) 24 (2.2)	607 (54.7) 302 (27.2) 135 (12.2) 34 (3.1) 32 (2.9)	-9.4	.07
Pulmonary hypertension 31-55 mm Hg >55 mm Hg	508 (45.6) 136 (12.2)	1741 (31) 351 (6.3)	38	<.001	507 (45.7) 135 (12.2)	465 (41.9) 149 (13.4)	-0.4	.7
TR Mild Moderate Severe	142 (12.8) 511 (46) 347 (31.2)	1690 (30.1) 865 (15.4) 34 (0.6)	146	<.001	141 (12.7) 511 (46) 346 (31.2)	361 (32.5) 230 (20.7) 15 (1.4)	127	<.001
LVEF >50% 31%-50% 21%-30% ≤20%	797 (71.6) 288 (25.9) 24 (2.2) 4 (0.4)	4748 (84.6) 769 (13.7) 82 (1.5) 10 (0.2)	-30.6	<.001	795 (71.6) 287 (25.9) 24 (2.2) 4 (0.5)	820 (73.9) 249 (22.4) 37 (3.3) 5 (0.5)	1.6	.1
	82 (7.4)	229 (4)	12.6	<.001	81 (7.3)	81 (7.3)	-	1
EuroSCORE II (%)	2.71 (1.42-4.95)	1.15 (0.74-2.21)	39.2	<.001	2.68 (1.41-4.98)	1.9 (1.12-3.9)	15.7	<.001

Data are median (interquartile range) or n (% of total). *cTVR*, Concomitant tricuspid valve repair; *MV*, mitral valve; *SD*, standard deviation; *NYHA*, New York Heart Association; *AV*, atrioventricular; *eGFR*, estimated glomerular filtration rate; *MR*, mitral regurgitation; *TR*, tricuspid regurgitation; *LVEF*, left ventricular ejection fraction; *EuroSCORE*, European System for Cardiac Operative Risk Evaluation.

imbalance. Because of missing baseline data, treatment effect sizes were estimated using the multiple imputation method.⁶ The imputation procedure was performed according to the Rubin's protocol under the assumption that missing data are missing at random. Postoperative outcomes were then

compared between matched groups using standard univariate statistical tests of association. Categorical data were compared using the McNemar test. Continuous data were compared with paired *t* test or Wilcoxon signed rank test, as appropriate. Statistical testing was conducted at the 2-tailed α

TABLE 2. Intraoperative data of the propensity-matched p	patient cohort
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	cTVR	MV	
	(n = 1110)	(n = 1110)	<i>P</i> value
Surgical approach			<.001
Direct vision	431 (38.8)	281 (25.3)	
Video-assisted	397 (35.8)	480 (43.2)	
Totally endoscopic	281 (25.3)	343 (30.9)	
Robotic	2 (0.2)	6 (0.5)	
Surgical access			<.001
Anterolateral	857 (77)	846 (76)	
Transaxillary	97 (8.7)	127 (11.4)	
Periareolar	67 (6)	91 (8.2)	
Partial sternotomy	89 (8)	46 (4.1)	
Conversion to full sternotomy	25 (2.3)	27 (2.4)	.8
Venous cannulation site			<.001
Femoral	477 (43)	709 (63.9)	
Femoral + jugular	528 (47.6)	345 (31)	
Femoral + superior vena cava	94 (8.5)	49 (4.4)	
Other	12 (1)	5 (0.5)	
Myocardial protection*			4
Cardionlegia	1072 (96.6)	1081 (97.4)	
Ventricular fibrillation	27 (2.4)	26 (2 3)	
Beating heart	12 (1)	20(2.3)	
True of MM company	12(1)	2 (0.2)	ſ
Type of MV surgery	772 (70)	757 ((0))	.0
Repair	773 (70)	757 (68)	
Replacement	330 (30)	344 (31)	
Replacement (failed repair)	8 (0.7)	10 (0.9)	
MV repair technique			
Annuloplasty	768 (99.3)	746 (98.5)	.1
Resection	46 (5.9)	95 (12.6)	.01
Sliding	16 (2.1)	17 (2.2)	1
Artificial chords	324 (41.9)	429 (56.7)	.001
Edge-to-edge	8 (1)	18 (2.4)	.06
AF surgery	354 (32)	362 (33)	.8
AF energy source			.2
Cryoablation	280 (25.2)	310 (27.9)	
Radiofrequency ablation	73 (6.6)	51 (4.5)	
Cryo + radiofrequency ablation	1 (0.1)	1 (0.1)	
LAA closure	360 (32.4)	211 (19)	<.001
CPB time, min	161 (133-203)	130 (103-166)	<.001
Aortic crossclamp time, min	93 (66-123)	83 (64-107)	<.001
Repeated aortic crossclamping	25 (2.3)	21 (1.9)	.6

Data are median (interquartile range) or n (% of total). cTVR, Concomitant tricuspid valve repair; MV, mitral valve; AF, atrial fibrillation; LAA, left atrial appendage; CPB, cardiopulmonary bypass. *Myocardial protection during mitral valve surgery.

level of 0.05. Statistical analysis was performed using Statistical Package for Social Sciences, version 29.0 (IBM SPSS Inc).

RESULTS

Table 1 shows the preoperative characteristics of the prepropensity-matched (n = 6722) and the propensity-matched (n = 2220) patients who received MV surgery with or without concomitant TVR. In the

prepropensity-matched patient cohort, 17% of the patients (n = 1113) underwent MV surgery with cTVR and 83% (n = 5609) underwent MV surgery only. Patients were on average 65 years old (IQR, 53-76 years, 58% male), and almost one half of them were in New York Heart Association class III-IV. One third had preoperative atrial fibrillation, 15% had an estimated glomerular filtration rate less than 50 mL/min, and more than 80% had a left

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TABLE 3. In-hospital outcomes of the propensity-matched cohort

	cTVR	MV	
	(n = 1110)	(n = 1110)	P value
In-hospital mortality	37 (3.3)	24 (2.2)	.5
Stroke	23 (2)	13 (1.2)	.3
Intubation time, h	9 (5-18)	7 (5-12)	<.001
Reintubation/tracheostomy	54 (4.9)	49 (4.4)	.5
Bleeding (requiring revision)	102 (9.2)	73 (6.6)	.04
Transfusions (unit)	0 (0-2)	0 (0-2)	<.001
New-onset AF	99 (8.9)	120 (10.8)	.2
Definitive pacemaker implantation	101 (9)	64 (5.8)	.02
Myocardial infarction	16 (1.5)	8 (0.69)	.4
Low cardiac output	86 (7.7)	49 (4.4)	.02
Acute kidney injury Stage 1 Stage 2 Stage 3	108 (9.7) 16 (1.4) 30 (2.7)	61 (5.5) 10 (0.9) 19 (1.7)	.01
Dialysis	36 (3.2)	29 (2.6)	.3
Vascular complications Major Minor	19 (1.7) 7 (0.6)	17 (1.5) 6 (0.5)	.7
Thoracic wound complications	10 (0.9)	18 (1.6)	.2
Redo for early failure	9 (0.8)	14 (1.3)	.1
MR (after MV repair) Mild Moderate Severe	202 (18.2) 5 (0.5) 1 (0.1)	165 (14.9) 15 (1.4) 2 (0.2)	.06
TR Mild Moderate Severe	708 (63.8) 75 (6.8) 9 (0.8)	497 (44.8) 95 (8.6) 6 (0.5)	<.001
ICU stay, h	24.5 (20-96)	24 (20-48)	<.001
Hospital stay, d	10 (7-15)	8 (7-12)	<.001

Data are median (interquartile range) or n (% of total). *cTVR*, Concomitant tricuspid valve repair; *MV*, mitral valve; *AF*, atrial fibrillation; *MR*, mitral regurgitation; *TR*, tricuspid regurgitation; *ICU*, intensive care unit.

ventricular ejection fraction greater than 50%. Among MV pathologies, the degenerative type was most common, followed by functional, rheumatic, and other pathologies in descending frequency. Median European System for Cardiac Operative Risk Evaluation (EuroSCORE) II was 1.3%. Preoperative patient characteristics showed relevant differences, with patients who underwent cTVR being older (72 years [IQR, 64-67 years] vs MV: 63 years [IQR, 53-72 years]; P < .001) and presenting with substantially more risk factors, such as diabetes, atrial fibrillation, kidney disease, and pulmonary hypertension. Propensity matching resulted in 1110 balanced pairs with a median age of 71 years (IQR, 62-77 years) and 45% being male. Here,

preoperative characteristics were similar except for more moderate/severe TR in the patients who underwent cTVR (77.2% vs MV: 22.1%; P < .001). EuroSCORE II was still greater in patients who underwent cTVR (2.68% vs MV: 1.9%; P < .001).

Table 2 shows the intraoperative data of the propensity-matched patient cohort. In contrast to the MV group, patients who underwent cTVR were more often operated under direct vision than video-assisted, endoscopically, or with the robotic approach and mostly through an anterolateral access. Conversion rate to full sternotomy was comparable (cTVR: 2.3 vs MV: 2.4%; P = .8). Bicaval venous cannulation was more common



FIGURE 1. Postoperative permanent pacemaker rate for patients undergoing MV surgery with or without cTVR for the propensity-matched and prepropensity-matched comparison. *cTVR*, Concomitant tricuspid valve repair; *MV*, mitral valve.

in patients who underwent cTVR (57%) with cannulation of the femoral plus jugular vein and less common in direct cannulation of the superior vena cava. In both groups, the vast majority of patients underwent MV surgery on the arrested heart, mostly using single-shot crystalloid cardioplegia (58%). MV repair rate was 70% for all pathologies and 90% for degenerative ones. There were similar rates of concomitant atrial fibrillation surgery (32%) using either cryo- and/or radiofrequency ablation in both matched groups. Cardiopulmonary bypass times (161 minutes vs MV: 130 minutes; P < .001) and aortic crossclamp time (93 minutes vs MV: 83 minutes; P < .001) were longer in the cTVR group.

Table 3 shows the in-hospital outcomes of the propensity-matched cohort. Although in-hospital mortality rates were similar (cTVR: 3.3% vs MV: 2.2%; P = .5), bleeding requiring revision (9.2% vs MV: 6.6%; P = .04), postoperative pacemaker implantations (9% vs MV: 5.8%; P = .02; Figure 1), low cardiac output syndrome (7.7% vs MV: 4.4%; P = .02), and acute kidney injury (13.8% vs MV: 10%; P = .01) were more frequent in the cTVR group. Hospital stay was also longer in this group (10 days vs MV: 8 days; P < .001). Concomitant TVR eliminated relevant TR in the vast majority of patients. Table 3 shows the patients who had any signs of postoperative TR. In 44.8% of patients TR was mild, in 6.8% moderate, and in 0.5% severe. The remaining patients had no TR.

Table 4 shows the multivariable analysis assessing the risk of permanent pacemaker implantation. Multivariable analysis identified MV replacement, atrial fibrillation, and concomitant TVR as independent predictors of postoperative pacemaker implantation.

DISCUSSION

We demonstrate in this large multicenter registry analysis that concomitant TVR in minimally invasive MV surgery is an independent risk factor for pacemaker implantation in the real world. It is also associated with greater risk of bleeding and more low output syndrome and kidney injury, and it is not clear from these data whether technical or patient factors (or both) explain these differences.

The benefits of cTVR in patients undergoing MV surgery have been described previously.¹ Patients with moderate or less-than-moderate TR receiving cTVR during MV surgery had a lower incidence of a composite end point (progression of TR, reoperation for TR, death) than those undergoing MV surgery alone at 2-year follow-up (3.9% vs 10.2%; P = .02). This benefit was mainly driven by lower incidence of TR progression in the cTVR group (0.6% vs MV: 6.1%); relative risk, 0.09; 95% confidence interval, 0.01-0.69), but it was reduced by significantly greater postoperative permanent pacemaker rates (14.1% vs MV: 2.5%; rate ratio, 5.75; 95% confidence interval, 2.27-14.60) bearing the risk for these patients of potential long-term side effects. such as a higher incidence for heart failure.⁷ Thus, the current results of the CTSN trial did not show differences in heart failure rates of patients with and without pacemaker implantation at 2-year follow-up. Therefore, the 5-year follow-up is highly anticipated, as it will provide further insights regarding the clinical impact of pacemaker implantation over time. Balancing the risk of potential long-term pacemaker associated adverse events and the increased morbidity and mortality risk associated with uncorrected TR and reoperation for TR⁸⁻¹¹ it is a matter of individual judgment whether the advantages of concomitant TVR can outweigh the disadvantages.

It is generally accepted that severe TR decreases survival and the degree of TR is related to mortality¹¹ and not addressing mild or moderate TR at time of cardiac surgery led to poorer survival rates in a large retrospective study including more than 20,000 patients.¹² For mild or moderate TR that has not concomitantly been addressed, a progress of TR in approximately 25% of patients has been described, impairing survival and functional outcome.^{2,8,13-15} As it is difficult to predict whether TR improves after left-sided heart valve surgery and reoperation due to the fact severe TR is associated with greater risk for morbidity and mortality,^{16,17} it has been suggested to be addressed during index procedure to prevent future progression.^{2,8,18-20}

However, understanding the risk factors for postoperative pacemaker implantation might be helpful in decisionmaking and surgical planning for cTVR and the sequence of pacemaker implantation. Risk factors, such as age and valvular pathology, and other comorbidities, such as rhythm disturbances, renal impairment, and diabetes, are associated

TABLE	4.	Multivariable	analysis	for	permanent	pacemaker
implanta	tioı	ı (backward stej	pwise logis	tic re	gression)	

Variable	P value	OR	95% Cl
Arterial hypertension	.008	2.797	1.327-5.893
Surgical approach	-	-	-
Venous cannulation site	-	-	-
Concomitant tricuspid repair	.02	1.993	1.128-3.523
Mitral valve replacement	<.001	3.426	2.042-5.746
Preoperative atrial fibrillation	.009	2.566	1.272-5.178
Crystalloid cardioplegia	<.001	0.231	0.113-0.503

OR, Odds ratio; CI, confidence interval.

with greater pacemaker rates.²¹ Complementing the CTSN trial, our registry reflects "real-world practice" in a patient population with a wider range of MV pathologies who received only minimally invasive surgery. Similar to the CTSN trial, we also found greater pacemaker rates in patients who underwent cTVR, although the frequencies differed between the 2 studies. In contrast to the CTSN trial, we found a numerically greater pacemaker rate in patients undergoing isolated MV surgery (5.8%) but a lower one in the patients who underwent cTVR (9.1% vs CTSN trial: 11.1%).¹ For patients undergoing MV surgery only, these greater rates might be explained as they were older, and presented with other MV pathologies requiring more MV replacement as well as greater incidence of atrial fibrillation, thus, these greater pacemaker rates might be explained by comorbidities rather than technical aspects. However, there were no relevant differences in the need for postoperative pacemaker among the centers. For patients undergoing cTVR, other studies showed also greater pacing rates,^{1-3,12} so that surgical–technical aspects cannot be ignored. Beating-heart strategy for TVR might allow detecting surgically induced rhythm disturbances during TVR. In our personal experience, TVR (isolated and concomitant) on the beating heart has resulted in very low heart block and pacemaker rates (unpublished observations). To us, this approach allows for immediate detection of surgically induced rhythm disturbances during TVR and, thus, for procedure adjustment (eg, removal of misplaced annuloplasty sutures). Unfortunately, we are not able to specify this hypothesis, because in the registry the information whether TVR was performed on the arrested or beating heart was only recorded in a small proportion of patients. Nevertheless, future investigations might be able to further elaborate this issue in the context of pacemaker rates and correlating patient prognosis.

Further consideration of TR reflects a unique pathophysiology, as it is often associated with liver dysfunction, which is a critical risk factor in cardiac surgery in particular for patients undergoing isolated TV surgery.²²

In the CTSN trial,¹ no patients had severe TR. In the present patient population, one-third of patients had severe TR. Specifically in these patients, congestion of liver and kidney are possible pathomechanistic scenarios. Liver congestion may contribute to decreased (diastolic) cardiac function and cardiac remodeling,²³ coagulopathy, and immunologic and metabolic impairment.²⁴ In our study, patients who underwent cTVR required more surgical revisions due to a greater incidence of postoperative bleeding, had longer intubation time, and had a greater incidence of low cardiac output and acute kidney injury associated with longer intensive care unit and hospital stay. These factors may all interact with each other and explain the greater incidence of bleeding and heart failure. Since no information on liver function is available from our registry, the aforementioned pathomechanism remains hypothetical.

Limitations

This study has several limitations: It is limited by its retrospective nature and the resulting potential confounders. By propensity matching, we tried to balance these factors as good as possible. Despite these attempts to align the groups, differences remained. Patients in the cTVR group might have undergone surgery at a more severe stage. Greater values for EuroSCORE II might be explained in part by the fact that cTVR is a risk factor itself in the EuroSCORE calculation. However, patients who undergo cTVR carry a greater disease burden by the inherent preoperative degree of TR. This potential hazard can, of course, not be eliminated by propensity matching. As alluded to previously, TR-associated comorbidities such as liver congestion potentially increase perioperative risk, but data from our registry are unable to provide a complete answer. In addition, the registry does not allow for more in-depth analysis of pacemaker implantation because some specific information was not collected (ie, surgical details of TVR or clinical details of pacemaker therapy). For instance, it remains unclear whether surgical, technical, or patient factors contributed more to the postoperative pacemaker rate. Finally, there is one difference to the CTSN trial. In our registry, the majority of patients who received cTVR had moderate-to-severe TR. This difference underscores the difference in comorbidity status, but its contribution to the individual outcome remains unclear.

CONCLUSIONS

Concomitant TVR in minimally invasive MV surgery is an independent risk factor for pacemaker implantation in this international registry. It is also associated with a greater risk of bleeding and more low output syndrome and acute kidney injury. It remains unclear whether technical or patient factors (or both) explain these differences.

Webcast (

You can watch a Webcast of this AATS meeting presentation by going to: https://www.aats.org/resources/pace maker-implantation-after-concomitant-tricuspid-valve-re pair-in-patients-undergoing-minimally-invasive-mitralvalve-surgery-results-from-the-mini-mitral-international-re gistry.



Conflict of Interest Statement

N.B. reported scientific grants: Edwards Lifesciences, Corcym; and speaker's honoraria: Edwards Lifesciences, Medtronic, and Vascular Graft Solutions. J.K. reported speaker honoraria: Edwards, Medtronic, Artivion, and Abbott. All other authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

References

- 1. Gammie JS, Chu MWA, Falk V, Overbey JR, Moskowitz AJ, Gillinov M, et al. Concomitant tricuspid repair in patients with degenerative mitral regurgitation. *N Engl J Med.* 2022;386:327-39.
- 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;65:1931-8.
- David TE, David CM, Tsang W, Lafreniere-Roula M, Manlhiot C. Long-term results of mitral valve repair for regurgitation due to leaflet prolapse. J Am Coll Cardiol. 2019;74:1044-53.
- Berretta P, Kempfert J, Van Praet F, Salvador L, Lamelas J, Nguyen TC, et al. Risk-related clinical outcomes after minimally invasive mitral valve surgery: insights from the Mini-Mitral International Registry. *Eur J Cardiothorac Surg.* 2023;63:ezad090.
- 5. Stone GW, Adams DH, Abraham WT, Kappetein AP, Généreux P, Vranckx P, et al. Clinical trial design principles and endpoint definitions for transcatheter mitral valve repair and replacement: part 2: endpoint definitions: a consensus document from the Mitral Valve Academic Research Consortium. *Eur Heart J*. 2015;36:1878-91.
- Mattei A. Estimating and using propensity score in presence of missing background data: an application to assess the impact of childbearing on wellbeing. *Stat Methods Appl.* 2009;18:257-73.

- Chi MC, Hung KC, Chang SH, Wu VCC, Chou AH, Chan YH, et al. Effect of permanent pacemaker implantation after valve surgery on long-term outcomes. *Circ J*. 2021;85:1027-34.
- Dreyfus GD, Corbi PJ, Chan KM, Bahrami T. Secondary tricuspid regurgitation or dilatation: which should be the criteria for surgical repair? *Ann Thorac Surg.* 2005;79:127-32.
- Bernal JM, Morales D, Revuelta C, Llorca J, Gutierrez-Morlote J, Revuelta JM. Reoperations after tricuspid valve repair. *J Thorac Cardiovasc Surg.* 2005;130: 498-503.
- Singh SK, Tang GH, Maganti MD, Armstrong S, Williams WG, David TE, et al. Midterm outcomes of tricuspid valve repair versus replacement for organic tricuspid disease. *Ann Thorac Surg.* 2006;82:1735-41; discussion 1741.
- 11. Nath J, Foster E, Heidenreich PA. Impact of tricuspid regurgitation on long-term survival. J Am Coll Cardiol. 2004;43:405-9.
- Kelly BJ, Ho Luxford JM, Butler CG, Huang CC, Wilusz K, Ejiofor JI, et al. Severity of tricuspid regurgitation is associated with long-term mortality. J Thorac Cardiovasc Surg. 2018;155:1032-8.e2.
- Benedetto U, Melina G, Angeloni E, Refice S, Roscitano A, Comito C, et al. Prophylactic tricuspid annuloplasty in patients with dilated tricuspid annulus undergoing mitral valve surgery. *J Thorac Cardiovasc Surg.* 2012;143: 632-8.
- 14. Bertrand PB, Koppers G, Verbrugge FH, Mullens W, Vandervoort P, Dion R, et al. Tricuspid annuloplasty concomitant with mitral valve surgery: effects on right ventricular remodeling. *J Thorac Cardiovasc Surg.* 2014;147: 1256-64.
- McCarthy PM, Szlapka M, Kruse J, Kislitsina ON, Thomas JD, Liu M, et al. The relationship of atrial fibrillation and tricuspid annular dilation to late tricuspid regurgitation in patients with degenerative mitral repair. *J Thorac Cardiovasc Surg.* 2021;161:2030-40.e3.
- Jeganathan R, Armstrong S, Al-Alao B, David T. The risk and outcomes of reoperative tricuspid valve surgery. *Ann Thorac Surg.* 2013;95:119-24.
- Farber G, Tkebuchava S, Dawson RS, Kirov H, Diab M, Schlattmann P, et al. Minimally invasive, isolated tricuspid valve redo surgery: a safety and outcome analysis. *Thorac Cardiovasc Surg.* 2018;66:564-71.
- Lee H, Sung K, Kim WS, Lee YT, Park SJ, Carriere KC, et al. Clinical and hemodynamic influences of prophylactic tricuspid annuloplasty in mechanical mitral valve replacement. *J Thorac Cardiovasc Surg.* 2016;151:788-95.
- De Bonis M, Lapenna E, Pozzoli A, Nisi T, Giacomini A, Calabrese M, et al. Mitral valve repair without repair of moderate tricuspid regurgitation. *Ann Thorac Surg.* 2015;100:2206-12.
- Lee JW, Song JM, Park JP, Lee JW, Kang DH, Song JK. Long-term prognosis of isolated significant tricuspid regurgitation. *Circ J.* 2010;74:375-80.
- Ghauri H, Iqbal R, Ahmed S, Ashraf A, Khan MSQ, Malik J, et al. Predictors of permanent pacemaker insertion after mitral valve replacement: a systematic review. *Pacing Clin Electrophysiol*. 2022;45:681-7.
- 22. Färber G, Marx J, Scherag A, Saqer I, Diab M, Sponholz C, et al. Risk stratification for isolated tricuspid valve surgery assisted by model of end-stage liver disease score. J Thorac Cardiovasc Surg. 2023;166:1433-41.e1.
- 23. Poelzl G, Auer J. Cardiohepatic syndrome. Curr Heart Fail Rep. 2015;12:68-78.
- 24. Chacon MM, Schulte TE. Liver dysfunction in cardiac surgery what causes it and is there anything we can do? *J Cardiothorac Vasc Anesth.* 2018;32: 1719-21.

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