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Risk Factors and Outcomes of Permanent Pacemaker Implantation Following Aortic and Mitral Valve Replacements

Aort ve Mitral Kapak Replasmanı Sonrası Kalıcı Kalp Pili İmplantasyonunun Risk Faktörleri ve Sonuçları

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Öz

Bu çalışmada, triküspit anüloplasti (TAP) olan/olmayan mitral kapak ve aort kapak replasmanlarını takiben kalıcı kalp pili implantasyonu (KKPI) için risk faktörlerinin araştırılması amaçlandı. Bu çalışmada Ocak 2014'ten Aralık 2017'ye kadar eşzamanlı TAP olan ve/veya olmayan aort kapak ve mitral kapak replasmanı yapılan hastalar geriye dönük olarak analiz edildi. Çalışmaya toplam 179 ardışık hasta (%48.0 erkek; ortalama yaş 51.7±13.7 yıl) dahil edildi; 165 (%92.17) hasta KKPI olmayan grubda ve 14 (%7.82) hasta KKPI olan gruptaydı. Toplam 179 ardışık hastanın %48.0'i erkek ve ortalama yaş 51.7±13.7 yıldır. İki grup yaş, cinsiyet, vücut kitle indeksi (VKİ), diabetes mellitus (DM), hipertansiyon (HT), ejeksiyon fraksiyonu % (%EF) ve kardiyopulmoner baypas (KPB) süresi açısından farklılık göstermedi (p>0.05). KKPI grubunda daha küçük boyutlarda mekanik veya biyolojik aort kapakları vardı (p<0.05), önemli ölçüde daha yüksek bazal atriyal fibrilasyon (AF) oranı mevcuttu (p<0.05). Geç ölüm oranları KKPI olamayan grupta ve KKPI olan gruplarda sırasıyla %10.9, %35.7 idi (p<0.05). KPI grubunda >2+ postoperatif tricuspid yetersizliği (TY) anlamlı olarak daha yüksekti (p<0.05). Daha dar ve kalsifik aort köklerine bağlı olabilecek KKPI grubunda mekanik veya biyolojik aort kapak boyutları anlamlı olarak daha küçüktü. Başlangıç AF'si olan hastaların yüzdesi KKPI grubunda daha fazlaydı. KKPI uygulanan hastalarda anlamlı derecede yüksek olan >2+ postoperatif TY bir risk faktörü olarak kabul edilemez ancak anlamlı bir sonuçtur.

Anahtar Kelimeler: Aort Kapak Replasmanı, Kalıcı Kalp Pili Implantasyonu, Mitral Kapak Replasmanı

Abstract

This study aimed to search risk factors for permanent pacemaker implantation (PPI) following mitral valve and aortic valve replacements with/without tricuspid annuloplasty (TAP). This study retrospectively analysed patients undergoing aortic valve and mitral valve replacements with/without concomitant TAP from January 2014 to December 2017. A total 179 consecutive patients were included into the study, 165 (92.17%) patients comprised no-PPI group and 14 (7.82%) comprised PPI group. A total 179 consecutive patients were 48.0% men; with the mean age 51.7±13.7 years. The two groups did not differ with respect to age, gender, body mass index (BMI), diabetes mellitus (DM), hypertension (HT), ejection fraction % (EF %) and the duration of cardiopulmonary bypass (CPB) (p>0.05). The PPI group had smaller sizes of mechanical or biological aortic valves (p<0.05), with a significantly higher rate of baseline atrial fibrillation (AF) (p<0.05). The late mortality rates were 10.9%, 35.7% in the no-PPI and the PPI groups, respectively (p<0.05). The PPI group had significantly higher > 2+ postoperative TR (p<0.05). The sizes of mechanical or biological aortic valves were significantly smaller in the PPI group, which may be due to the narrower and calcific aortic roots. The percentage of patients with a baseline AF was greater in the PPI group. The significantly higher incidence of >2+ postoperative TR among patients receiving PPIs can be deemed merely a result, but not a risk factor.

Keywords: Aortic Valve Replacement, Permanent Pacemaker Implantation, Mitral Valve Replacement

Introduction

Temporary conduction diseases frequently emerge following cardiac surgery. But, permanent pace-maker implantation (PPI) is rare, while it considerably increases the duration of mechanical ventilation, the intensive care unit (ICU) and hospital stay, with increased economic burden (1-2).

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As compared with isolated coronary artery bypass grafting (0.8%), the need for PPI following valve surgery (ranging from 3 to 6%) is more often. The rate of PPI was reported to be 5.7% in aortic valve replacement (3, 4). The risk for PPI following heart valve surgery can be due to damage from surgical trauma or ischemic injury to the conduction systems, because the aortic and mitral valves are in close proximity to the sinoatrial node, atrioventricular (AV) node, and bundle of His (5,6).

Recently, cardiac valve replacement has been associated with a long-term risk of PPI following double valve replacement with/without tricuspid valve replacement. Age, gender, emergency surgery, diabetes mellitus, and renal impairment can pose risk factors for PPI (5).

This study aims to search the risk factors for PPI following mitral and aortic valve replacements with/without tricuspid annuloplasty (TAP).

Material and Method

This study retrospectively analyzed patients who had undergone aortic and mitral valve replacements with or without concomitant tricuspid valve repair at the same center from January 2014 through December 2017. A total 179 consecutive patients (48.0% men; with the mean age 51.7±13.7 years) were included into the study; 165 (92.17%) patients comprised PPI group and 14 (7.82%) comprised no-PPI group. This study was approved by the local ethics of cardiac center (number: 2020/14/401). The data on patients' characteristics were collected from the hospital recording systems and archive files. All patients gave their informed written consents. This study was approved by the local ethics of cardiac centre (number: 2020/14/401).

This study determined all patients' demographics characteristics, post-operative complications, mortality and follow up electrocardiographic (ECG) features. Patients who had electively undergone aortic valve and mitral valve replacements with no other concomitant cardiac intervention other than tricuspid valve repair were included. Patients who underwent pre-operative PPI, concomitant surgical ablation and concomitant other cardiac surgeries were excluded.

All surgeries were performed through the median sternotomy by using aortic arterial and bi-caval venous cannulations for cardiopulmonary bypass (CPB). Myocardial protection with mild hypothermia was achieved by intermittent antegrade cardioplegia. In addition, retrograde cardioplegia was left to the discretion of the operation surgeon. While the mechanical prosthetic valve was the most commonly used material as the artificial valve, biological prosthetic valve replacement was performed for elderly or women considering pregnancy. After completing aortic valve and mitral valve replacements in patients undergoing TAP was performed. The technique for TAP was left to the discretion of the operation surgeon (3D Ring-TAP or De-vega-TAP). After the operation, all patients were transferred to the cardiac ICU for the postoperative management.

All data were obtained from hospital recording system and physical examinations were performed in the postoperative period. Patients who had not followed by in our outpatient clinic or had missing data were excluded. During the postoperative period, patients were evaluated based on echocardiographic examination and ECG features. The requirement of PPI was assessed. The two groups were compared according to the post-operative findings and long-term survival rates. In-hospital mortality at one month was defined as early mortality; out-of-hospital mortality was defined as late mortality. We calculated postoperative echocardiographic results at 3 years or those beyond 3 years. The patients were followed for a mean of 3.18 ± 1.56 years, patients (45 %) were followed up more than 3 years.

Statistical analyses were performed by using SPSS (Statistical Package for the Social Sciences) 26.0 software. Parametric continuous data were expressed as mean and standard deviation, while non-parametric continuous data as median and interquartile ranges. Besides the categorical data were defined as frequency and percentage. In the descriptive statistics of the data, mean, standard deviation, median lowest, highest, frequency and ratio values were used. The distribution of variables was with the Kolmogorov Simirnov test. Independent sample t test and Mann-Whitney u test were used in the analysis of quantitative independent data. The Chi-square test was used in the analysis of qualitative independent samples t test and the Fischer test was used when the chi-square test requirements were not met. A p value < 0.05 was considered as statistically significant. Univariate and multivariate logistic regression analysis was performed to calculate the effect level.

Results

The study included a total of 179 patients (48.0% men; the mean age 51.7±13.7 years); 165 (92.17%) patients were included in the no-PPI group, 14 (7.82%) in the PPI group. Valve pathologies and implanted valve types are shown in Table 1.

Table 1. Valve pathologies and implanted valve types

	N	%
Preoperative Diagnosis		
A.S. + M.R.	54	30.2
A.R. + M.R.	51	28.5
A.R. + M.S.	50	27.9
A.S. + M.S.	24	13.4
Mitral Valve Bioprothesis	13	7.3
Mitral Valve Brand of Prosthesis		
Carbomedics	92	51.4
StJude	63	35.2
A.T.S.	11	6.1
Sorin	11	6.1
Medtronic	2	1.1
Aortic Supra-Annular Prosthesis	10	5.6
Aortic Valve Bioprothesis	15	8.4
Aortic Valve Brand of Prosthesis		
Carbomedics	60	33.5
StJude	59	33.0
A.T.S.	37	20.7
Sorin	18	10.1
Medtronic	3	1.7
Perceval (Sutureless)	2	1.1

A.S.: Aortic Stenosis, M.R.: Mitral Regurgitation, A.R.: Aortic Regurtation, M.S.: Mitral Stenosis

The two groups did not differ with respect to age, gender, body mass index (BMI), diabetes mellitus (DM), hypertension (HT), ejection fraction % (EF %) and cardiopulmonary bypass (CPB) time. The sizes of mechanical or biological aortic valve were

significantly smaller in the PPI group (p:0.018). The duration of ventilation, ICU stay and hospital stay did not significantly differ between the two groups. (Table 2).

The PPI group had significantly lower preoperative aortic valve mean gradient than the non-PPI group (p=0.042) (Table 3).

The PPI group had a significantly higher rate of preoperative atrial fibrillation (AF) (p<0.05). The two groups had similar early mortality rates (p=1.000). The no-PPI group had 10.9 % and the PPI group had 35.7% late mortality rates (p=0.021). The PPI group had significantly higher >2+ postoperative TR (p=0.009). The two groups showed no difference with regard to the incidence of PPI given the presence of tricuspid annuloplasty and the type of tricuspid annuloplasty (p=0.836). (Table 4)

In univariate analysis, the comparison of the PPI group with the no-PPI group showed that the preoperative AF, >2+ postoperative TR, the preoperative mean aortic gradient and aortic valve sizes were significantly different (p<0.05). As a result of multivariate analyses, the PPI group had a higher incidence of >2+ postoperative TR than in the PPI group (p<0.05) (Table 5).

Discussion

The rate of postoperative PPI following valve surgery is about 5% (7). This study assessed the risk factors associated with PPI after aortic and mitral valve replacements with or without TAP. The sizes of mechanical or biological aortic valve were significantly smaller in the PPI group, which was consistent with the limited data (8). Additionally, the rate of preoperative AF was significantly higher in the PPI group with significantly higher late mortality rates. Moreover, >2+ postoperative TR was significantly higher in the PPI group, which may be associated with the complications resulting from PPI.

Some studies reported that female gender is a predictive risk factor for PPI (9). However, Ghamdi and colleagues found that there was no difference in PPI with respect to gender. Several studies reported that older age (>75 years) was a risk factor for PPI, however, we did not find any association between the PPI and age (10,11). Moreover, patients >75 years of age and who had a wide QRS were at risk for PPI (11). The presence of arrhythmia and preoperative conduction disturbances, older age, concomitant procedures were linked to enhanced risk for PPI following heart surgery. Additionally, about 30 days after surgery, chronic comorbidities appeared to be linked to enhanced risk for PPI (12). The present study found no difference in older age and the presence of TAP.

Table 2. Comparison of demographic data between the groups

	AV Complete Block (-)		AV Complete Block (+)		P Value
	Mean ±S.D. n- %	Median	Mean ±S.D. n- %	Median	
Age	51.3 ± 13.4	51.0	56.1 ± 16.0	59.0	0,215 [t]
Gender					
Male	82 - 49.7%		4 - 28.6%		0,129 [x2]
Female	83 - 50.3%		10 - 71.4%		
BMI	26.5 ± 4.7	26.0	24.7 ± 4.0	23.0	0,302 [m]
D.M.	16 - 9.7%		0 - 0.0%		0,618 [x2]
H.T.	30 - 18.2		5 - 35.7%		0,112 [x2]
B.S.A.	1.7 ± 0.3	1.8	1.6 ± 0.2	1.6	0,079 [m]
E.F.	55.8 ± 9.7	60.0	59.5 ± 6.4	60.0	0,288 [m]
Mitral Valve size	27.9 ± 2.0	27.0	27.6 ± 2.0	27.0	0,333 [m]
Aortic Valve size	21.5 ± 2.1	21.0	20.4 ± 1.6	20.5	***0,018*** [m]
C.P.B. Time	192.1 ± 61.2	181.5	182.8 ± 63.3	164.0	0,387 [m]
Postoperative M.V. Time	20.6 ± 55.5	13.0	13.2 ± 5.2	12.5	0,817 [m]
Postoperative I.C.U. Length of Stay	4.2 ± 9.2	2.0	4.0 ± 2.0	4.0	0,076 [m]
Postoperative Hospital Length of Stay	14.6 ± 12.1	11.0	17.5 ± 16.0	11.0	0,925 [m]
Follow-Up Duration	3.2 ± 1.5	3.4	2.9 ± 1.8	3.5	0,677 [m]

[m] Mann-Whitney U Test / [t] Independent T-Test / [x2] Chi-Square Test (Fischer). B.M.I.: Body-Mass-Index, D.M.: Diabetes Mellitus, H.T.: Hypertension, B.S.A.: Body Surface Area, E.F.: Ejection Fraction, C.P.B.: Cardio-Pulmonary Bypass, M.V.:Mechanical Ventilation, I.C.U.: Intensive Care Unit

Table 3. Comparison of preoperative and postoperative echocardiographic findings between the groups

	AV Complete Block (-)		AV Complete Block (+)		p Value
	Mean ± S.D.	Median	Mean ± S.D.	Median	
Preoperative Mitral Insufficiency Grade	3.0 ± 0.9	3.0	2.6 ± 1.0	3.0	0,143 [m]
Preoperative Mitral Valve Gradient (Mean)	11.7 ± 6.9	9.5	9.6 ± 2.6	10.0	0,764 [m]
Preoperative Mitral Valve Gradient (Maximum)	21.5 ± 10.3	19.0	17.1 ± 7.8	18.5	0,537 [m]
Preoperative Mitral E.R.O.	32.1 ±10.4	31.0	35.0 ±	35.0	0,788 [t]
Preoperative Mitral Valve Area (cm2)	1.4 ± 0.3	1.4	1.2 ± 0.5	1.1	0,212 [m]
Preoperative Tricuspid Annulus Diameter	4.1 ± 0.8	3.9	4.0 ± 1.0	3.7	0,562 [m]
Preoperative Tricuspid Insufficiency Grade	2.4 ± 0.9	2.0	2.8 ± 0.7	3.0	0,121 [m]
Preoperative Aortic Insufficiency Grade	3.0 ± 0.8	3.0	2.8 ± 1.1	3.0	0,472 [m]
Preoperative Aortic Valve Gradient (Mean)	39.6 ± 16.9	40.0	25.0 ± 11.3	23.5	***0,042*** [t]
Preoperative Aortic Valve Gradient (Maximum)	65.0 ± 27.4	64.0	43.7 ± 17.5	41.5	0,065 [t]
Preoperative Aortic Valve Area (cm2)	0.9 ± 0.3	0.9	1.1 ±	1.1	0,408 [t]
Preoperative LVEDD	5.4 ± 1.0	5.2	5.1 ± 0.6	4.9	0,455 [m]
Preoperative LVESD	3.6 ± 1.0	3.5	3.3 ± 0.5	3.3	0,347 [m]
Preoperative I.V.S.	1.2 ± 0.3	1.1	1.1 ± 0.1	1.1	0,792 [m]
Preoperative PWP	1.1 ± 0.2	1.1	1.1 ± 0.2	1.1	0,879 [m]
Preoperative L.A. Diameter	4.7 ± 0.7	4.6	4.5 ± 0.5	4.4	0,184 [m]
Preoperative PAPs	49.1 ± 16.5	48.0	49.0 ± 13.3	50.0	0,859 [m]
Postoperative Mitral Gradient (Mean)	5.4 ± 2.7	5.0	6.3 ± 5.1	6.0	0,753 [m]
Postoperative Mitral Valve Gradient (Maximum)	11.7 ± 5.7	12.0	13.8 ± 7.7	13.5	0,280 [m]
Postoperative Mitral Valve Area	2.5 ± 0.4	2.5	2.6 ± 0.3	2.5	0,346 [m]
Postoperative Tricuspid Insufficiency Degree	1.3 ± 1.3	1.0	1.5 ± 1.3	2.0	0,532 [m]
Postoperative Aortic Valve Gradient (Mean)	13.2 ± 8.6	13.0	14.8 ± 8.4	13.0	0,740 [m]
Postoperative Aortic Valve Gradient (Maximum)	25.0 ± 14.1	25.0	26.2 ± 13.0	26.5	0,661 [m]
Postoperative LVEDD	5.0 ± 0.6	4.9	4.9 ± 0.8	4.5	0,260 [m]
Postoperative LVESD	3.5 ± 0.7	3.2	3.3 ± 0.9	3.1	0,420 [m]
Postoperative IVS	1.1 ± 0.2	1.1	1.2 ± 0.2	1.1	0,568 [m]
Preoperative P.W.P.	1.0 ± 0.3	1.0	1.1 ± 0.2	1.1	0,280 [m]
Postoperative L.A. Diameter	4.5 ± 0.4	4.5	4.5 ± 0.4	4.5	0,870 [m]
Postoperative E.F.	55.0 ± 11.0	55.0	59.4 ± 4.6	60.0	0,309 [m]

[m] Mann-Whitney U Test / [t] Independent T-Test. ERO: Effective Regurgitant Orifice, LVEDD: Left Ventricular End-Diastolic Diameter, LVESD: Left Ventricular End-Systolic Diameter, IVS: Inter-Ventricular Septum, P.W.P.: Pulmonary Wedge Pressure, L.A.: Left Atrium, PAPs: Systolic Pulmonary Artery Pressure, E.F.:Ejection Fraction.

Table 4. Comparison of main findings between the groups

	AV Complete Block (-)		AV Complete Block (+)		p Value
	n	%	n	%	
Preoperative A.F.	41	24.8 %	7	50.0 %	0,041 [x2]
Preoperative A.F.> Postoperative complate block	0	0.0 %	6	42,9 %	0,000 [x2]
Mitral Valve Bioprothesis	12	7.3 %	1	7.1 %	1,000 [x2]
Aortic Valve- SupraAnnular	9	5.5 %	1	7.1 %	0,567 [x2]
Aortic Valve Bioprothesis	13	7.9 %	2	14.3 %	0i332 [x2]
Postoperative C.V.E.	16	9.7 %	4	28.6 %	0,055 [x2]
Pre.Op. A.F> Postopertive N.S.R.	26	15.8 %	0	0.0 %	0,108 [x2]
Pre.Op. A.F> Postoperative Nodal	4	2.4 %	0	0.0 %	1,000 [x2]
Pre.Op. A.F> Postoperative A.F.	11	6.7 %	1	7.1 %	1,000 [x2]
Pre.op Nodal> Postoperative Nodal	8	4.8 %	0	0.0 %	1,000 [x2]
Pre.op Nodal> Postoperative A.F.	2	1.2 %	0	0.0 %	1,000 [x2]
Aortic P.V.L.	4	2.4 %	0	0.0 %	1,000 [x2]
Mitral P.V.L.	4	2.4 %	1	7.1 %	0,338 [x2]
Aortic and Mitral P.V.L.	2	1.2 %	0	0.0 %	1,000 [x2]
Aortic Valve Thrombosis	1	0.6 %	0	0.0 %	1,000 [x2]
Mitral Valve Thrombosis	2	1.2 %	0	0.0 %	1,000 [x2]
Mitral Valve Vegetation	0	0.0 %	1	7.1 %	0,078 [x2]
Early Mortality	9	5.5 %	0	0.0 %	1,000 [x2]
Late Mortality	18	10.9 %	5	35.7 %	0,021 [x2]
Total Mortality	27	16.4 %	5	35.7 %	0,070 [x2]
Postoperative T.R. above Grade-2	41	24.8 %	8	57.1 %	0,009 [x2]
Postoperative Severe T.R.	18	10.9 %	2	14.3 %	0,659 [x2]
T.A.P. Type					
No Annuloplasty	93	56.4 %	7	50.0 %	
De-Vega annuloplasty	36	21.8 %	4	28.6 %	0,836 [x2]
Ring Ann.	36	21.8 %	3	21.4 %	

[x2] Chi-Square Test (Fischer). A.F.: Atrial Fibrillation Rhythm, De-Vega:Tricuspid DeVega Annuloplasty, C.V.E.: Cerebro-Vascular Event, N.S.R.: Normal Sinus Rhythm, P.V.L.: Para-Valvular Leakage, T.R.: Tricuspid Regurgitation, T.A.P.: Tricuspid Annulo-Plasty

Table 5. Logistic regression analyses

Variable	Crude OR-CI	P value	Adjusted OR-CI	P value
Pre-op AF	3.024 (1.001-9.136)	0.049		
Postoperative elevated TR (>2+)	4.033 (1.321-12.308)	0.014	4.033(1.321-12.308)	0.014
Pre-op Mean aortic gradient	0.928(0.861-1.000)	0.049		

A.F.: Atrial Fibrillation Rhythm, T.R.: Tricuspid Regurgitation

The longer the operations take the more likely the risk for PPI and the more complex becomes the valve surgery. Leyva and colleagues found that the rate of PPI at the follow up at 10 years was higher after AVR (4.22%– 14.4%), MVR (4.38%–15.6%), AVR plus MVR (5.59%–18.3%), and AVR plus MVR plus tricuspid valve replacement (7.89%–25.9%) (5). Moskowitz and colleagues found that the rate of PPI was 6.6% following AVR, 10.5% following MVR, and 13.3% following double valve replacement at one year-follow up (12). The present study showed that the rate of PPI was 7.8% following mitral valve and aortic valve replacements with/without tricuspid annuloplasty at 3 to 5 year-follow ups.

When multiple valve replacements are performed, the duration of surgery takes longer and therefore the risk of bradycardia increases (13). Conduction disorders following heart operation necessitating a PPI result in elevated morbidity rates, longer hospital stay and higher costs (14,5). The current study found that late mortality incidence was higher in the PPI group. Several studies reported that preoperative conduction system diseases represented a risk factor for PPI following cardiac surgery- e.g., bundle branch block and first-degree AV block (14). The present study found that preoperative AF was an increased risk factor for PPI.

The inconsistency in several reports may depend on the variety of surgical procedures, such as valve repair or replacement and concomitant procedures plus valve location, comorbidities and conduction disorders prior to surgeries (15). The guideline by the European Society of Cardiology recommends a seven-day-observation for AV block and a five day-several week observation for sinoatrial dysfunction after cardiac surgery before PPI (15,16). Additionally, the guidelines about bradycardia and cardiac conduction delay by the American College of Cardiology, the American Heart Association, and the Heart Rhythm Society recommend that patients should undergo PPI before discharge, who have a recent postoperative sinus node dysfunction or AV block accompanying persistent symptoms or hemodynamic instability that do not resolve following aortic valve replacement or mitral valve repair or mitral valve replacement (17).

It has been shown that operative trauma may result in PPI (13). Injury to conduction system due to valve surgeries or other operations in close proximity to the AV node makes up a risk factor for conduction disorder associated with AV node (18). The post-operative requirement for PPI is more widely encountered in aortic, mitral and tricuspid valve surgeries (8,19). The present study showed no significant differences in the risk for PPI following aortic and mitral valve replacements with or without concomitant TAP. Jouan et al found that the risk of PPI increased following concomitant TAP on isolated mitral valve surgery group, unlike present study, it may depend on the group of left sided double valve replacement (20). The postoperative requirement for PPI is linked to short-term adverse effects (21). Ghamdi and colleagues reported that prolonged CBP time and cross-clamp time increased the risk for PPI unlike the present study (10). Additionally, some studies found that chronic kidney disease is linked to conduction disturbances and AF due to pathological myocardial remodelling and fibrosis (22,23). The present study found no difference in renal disease between the groups; however, AF was a risk factor for PPI. Elahi et al. showed that smaller valve size (<21 mm) and without stent valves were risk factors for PPI (8). The present study showed that the sizes of mechanical or biological aortic valve were significantly smaller in the PPI group, which may result from damage caused by sutures, pressure from residual calcific material or the placement of the prosthetic valve neighboring conduction systems (24, 25), which may be due to calcific and narrower aortic roots. Combined aortic and mitral valve procedures were linked to an increased risk for PPI than isolated valve procedures (26). Multiple valve replacements, the calcified aortic valve and mitral annular calcification were reported to be linked to a risk for PPI (27).

Francesca N. Delling et al. evaluated 58556 patients and found that the incidence of elevated tricuspid regurgitation was higher after PPI. Significant TR can lead to poor quality of life, which may result in heart failure (28). We also found that the incidence of elevated postoperative TR was significantly higher in the PPI group, which may possibly be associated with the complications following PPI. Elevated TR that occurred following device implantation was caused by injury to the tricuspid valve (laceration/ perforation of leaflets or lead entrapment resulting in scar tissue), or damaged valve coaptation. Hoke et al. analyzed 239 patients and reported the incidence of significant TR (38%), which may result in poor long-term prognosis (29).

Additionally, TR associated with pacemaker implantation could pose significant right-sided heart failure; however, there is insufficient evidence on mortality (30). The present study found that in multivariate analysis, elevated TR 2 after surgery was related to a higher incidence in the PPI group with increased late mortality rates.

In conclusion, the sizes of mechanical or biological aortic valves were significantly smaller in the PPI group, which may be due to the calcific and narrower aortic roots. The percentage of patients with baseline AF was greater in the PPI group. The significantly higher incidence of >2+ postoperative TR among patients receiving PPIs can be deemed merely a result, but not a risk factor.

Ethics Committee Approval: This study was approved by the local ethics of cardiac centre (number: 2020/14/401).

References

1. Emlein G, Huang SKS, Pires LA, Rofino K, Okike ON, Vander Salm TJ. Prolonged bradyarrhythmias after isolated coronary by-pass graft surgery. Am Heart J. 1993;126(1084):90.
2. Chung MK. Cardiac surgery: postoperative arrhythmias. Crit Care Med. 2000;28:136–44.
3. Boughaleb D, Mansourati J, Genet L, Barra J, Mondine P, Blanc JJ. Permanent cardiac stimulation after aortic valve replacement: incidence, predictive factors and long-term prognosis. Arch Mal Coeur Vaiss. 1994; 87:925–30.
4. Lipton IH, Cameron DA, David TE, Mackall J. A Morbidity of permanent pacing following valvular surgery. PACE. 1995;18(1768)
5. Leyva F, Qiu T, McNulty D, Evison F, Marshall H, Gasparini M. Long-term requirement for pace- maker implantation after cardiac valve replace- ment surgery. Heart Rhythm. 2017;14:529–34.
6. Wiggins NB, Chong DT, Houghtaling PL, Hussein AA, Saliba W, Sabik JF. Incidence, indications, risk factors, and survival of patients undergoing cardiac implantable electronic device implantation after open heart surgery. Europace. 2017;19:1335–42.
7. Zakhia Doueihi R, Leloux MF, De Roy L, Krémer R. Permanent cardiac pacing for prolonged second- and third-degree atrioventricular block complicating cardiac valve replacement. Acta Cardiol. 1992; 47:157–66.
8. Elahi M, Usmaan K. The bioprosthesis type and size influence the postoperative incidence of permanent pacemaker implantation in patients undergoing aortic valve surgery. J Interv Card Electrophysiol. 2006;15(2):113–8.
9. Sachweh JS, Vazquez-Jimenez JF, Schondube FA, et al. Twenty years' experience with pediatric pacing: epicardial and transvenous stimulation. Eur J Cardiothorac Surg. 2000;17(4):455-61.
10. Al-Ghamdi B, Mallawi Y, Shafquat A, et al. Predictors of permanent pacemaker implantation after coronary artery bypass grafting and valve surgery in adult patients in current surgical era. Cardiol Res. 2016;7(4):123–9.
11. Turkkan C, Osmanov D, Yildirim E, et al. Bradyarrhythmia development and permanent pacemaker implantation after cardiac surgery. North Clin Istanb. 2018;5(4):288–94.
12. Moskowitz G, Hong KN, Giustino G, et al. Incidence and risk factors for permanent pacemaker implantation following mitral or aortic valve surgery. J Am Coll Cardiol. 2019;74(21):2607–20.
13. Nm VM, Sj H, Jong W, Domburg RT, Pw S, Jaegere PP. Persistent annual permanent pacemaker implantation rate after surgical aortic valve replacement in patients with severe aortic stenosis. Ann Thorac Surg. 2012; 94:1143–9.
14. Schurr UP, Berli J, Berdajs D, et al. Incidence and risk factors for pacemaker implantation following aortic valve replacement. Interact Cardiovasc Thorac Surg. 2010;11(5):556–60.
15. Epstein AE, DiMarco JP, Ellenbogen KA, et al. ACC/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guide- lines (Writing Committee to Revise the ACC/AHA/ NASPE 2002 Guideline Update for Implantation of Cardiac Pacemakers and Antiarrhythmia Devices) developed in collaboration with the American Association for Thoracic Surgery and Society of Thoracic Surgeons. J Am Coll Cardiol 2008;51:1–62.
16. Brignole M, Auricchio A, Baron-Esquivias G et al. 2013 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy: the Task Force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA): The Task Force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA). Eur Heart J. 2013;34(29):2281–329.
17. Writing Committee Members, Kusumoto FM, Schoenfeld MH, Barrett C, et al. 2018 ACC/AHA/HRS guideline on the evaluation and management of patients with bradycardia and cardiac conduction delay: Executive summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines, and the Heart Rhythm Society. Heart Rhythm. 2019;16(9): e227–79.
18. Cardiac Pacemakers -Biological Aspects, Clinical Applications and Possible Complications. Edited by Mart Min, ISBN 978-953-307-639-3, 206 pages, Publisher: InTech, Chapters published September 06, 2011 under CC BY- NC-SA 3.0 license.
19. Raza SS, Li JM, John R, Chen LY, Tholakanahalli VN, Mbai M. Long-term mortality and pacing outcomes of patients with permanent pacemaker implan- tation after cardiac surgery. Pacing Clin Electrophysiol. 2011;34(3):331-8.
20. Jouan J, Mele A, Florens E, et al. Conduction disorders after tricuspid annuloplasty with mitral valve surgery: Implications for earlier tricuspid intervention. J Thorac Cardiovasc Surg. 2016;151(1):99–103.
21. Chi M-C, Hung K-C, Chang S-H, et al. Effect of permanent pacemaker implantation after valve surgery on long-term outcomes. Circ J. 2021;85(7):1027–34.
22. Kestenbaum B, Rudser KD, Shlipak MG, et al. Kidney function, electrocardiographic findings, and cardiovascular events among older adults. Clin J Am Soc Nephrol. 2007;2(3):501–8.
23. Mathew J, Katz R, St John Sutton M, et al. Chronic kidney disease and cardiac remodelling in patients with mild heart failure: results from the Resynchronization Reverses Remodeling in Systolic Left Ventricular Dysfunction (REVERSE) study. Eur J Heart Fail. 2012;14(12):1420-8.
24. Fukuda T, Hawley RL, Edwards JE. Lesions of conduction tissue complicating aortic valvular replacement. Chest. 1976;69(5):605–14.
25. Totaro P, Calamai G, Montesi G, Barzaghi C, Vaccari M. Continuous suture technique and impairment of the atrioventricular conduction after aortic valve replacement. J Card Surg. 2000;15(6):418–22.
26. Gillinov AM, Gelijns AC, Parides MK, et al. Surgical ablation of atrial fibrillation during mitral-valve surgery. N Engl J Med. 2015;372(15):1399–409.
27. Nair CK, Sketch MH, Ahmed I, Thomson W, Ryschon K, Woodruff MP. Calcific valvular aortic stenosis with and without mitral annular calcium. Am J Cardiol. 1987; 60:865–70.
28. Delling FN, Hassan ZK, Piatkowski G, et al. Tricuspid regurgitation and mortality in patients with transvenous

permanent pacemaker leads. Am J Cardiol. 2016;117(6):988–92.
29. Höke U, Auger D, Thijssen J, et al. Significant lead-induced tricuspid regurgitation is associated with poor prognosis at long-term follow-up. Heart. 2014;100(12):960–8.
30. Al-Bawardy R, Krishnaswamy A, Bhargava M, et al. Tricuspid regurgitation in patients with pacemakers and implantable cardiac defibrillators: a comprehensive review: TR in patients with pacemakers and ICDs. Clin Cardiol. 2013;36(5):249–54.