Received: 3 March 2019 | Revised: 29 May 2019 | Accepted: 16 June 2019
DOI: 10.1111/chd.12818

ORIGINAL ARTICLE

Congenital Heart Disease WILEY

Postoperative and short-term atrial tachyarrhythmia burdens after transcatheter vs surgical pulmonary valve replacement among congenital heart disease patients

Subeer K. Wadia MD, MSc[1] | Gentian Lluri MD, PhD[1] | Jamil A. Aboulhosn MD[1] | Hillel Laks MD[2] | Reshma M. Biniwale MD[2] | Glen S. Van Arsdell MD[2] | Daniel S. Levi MD[3] | Morris M. Salem MD[3] | Kevin M. Shannon MD[1,3] | Jeremy P. Moore MD, MSc[1,3]

[1]Division of Cardiology, Ahmanson/UCLA Adult Congenital Heart Disease Center, Los Angeles, California

[2]Division of Cardiothoracic Surgery, UCLA Mattel Children's Hospital, Los Angeles, California

[3]Division of Pediatric Cardiology, Mattel Children's Hospital at UCLA, Los Angeles, California

Correspondence
Jeremy P. Moore, MD, MSc, Division of Cardiology, Ahmanson/UCLA Adult Congenital Heart Disease Center, 100 Medical Plaza, Suite 760, Los Angeles, CA 90095.
Email: JPMoore@mednet.ucla.edu

Abstract

Objective: We examined the atrial tachyarrhythmia (AT) burden among patients with congenital heart disease (CHD) following transcatheter (TC-) or surgical (S-) pulmonary valve replacement (PVR).

Design/Setting: This was a retrospective observational study of patients who underwent PVR from 2010 to 2016 at UCLA Medical Center.

Patients: Patients of all ages who had prior surgical repair for CHD were included. Patients with a history of congenitally corrected transposition of the great arteries, underwent a hybrid PVR procedure, or had permanent atrial fibrillation (AF) without a concomitant ablation were excluded.

Outcome Measures: The primary outcome was a time-to-event analysis of sustained AT. Sustained ATs were defined as focal AT, intra-atrial reentrant tachycardia/atrial flutter, or AF lasting at least 30 seconds or terminating with cardioversion or anti-tachycardia pacing.

Results: Two hundred ninety-seven patients (TC-PVR, n = 168 and S-PVR, n = 129) were included. During a median follow-up of 1.2 years, nine events occurred in TC-PVR group (5%) vs 23 events in S-PVR group (18%). In the propensity adjusted models, the following factors were associated with significant risk of AT after PVR: history of AT, age at valve implantation, severe right atrial enlargement, and S-PVR. In the secondary analysis, TC-PVR was associated with lower adjusted risk of AT events in the postoperative epoch (first 30 days), adjusted IRR 0.31 (0.14-0.97), P = .03, but similar risk in the short-term epoch, adjusted IRR 0.64 (0.14-2.94), P = .57.

Conclusion: There was an increased risk of AT in the first 30 days following S-PVR compared to TC-PVR. Additional factors associated with risk of AT events after PVR were a history of AT, age at valve implantation, and severe right atrial enlargement.

KEYWORDS
atrial arrhythmias, congenital heart disease, pulmonary valve replacement

1 | INTRODUCTION

Advances in early cardiac interventions and surgical techniques have led to improved survival among patients with congenital heart disease (CHD).[1,2] As the number of CHD survivors into adulthood has grown, the development of late sequelae, such atrial tachyarrhythmias (ATs), have become more common.[3-7] Atrial arrhythmias have an estimated incident rate of 50% among adults with complex CHD who survive to age 65, and are associated with a 50% increase in the hazard for mortality compared to those without atrial arrhythmias.[8] In addition, the presence of atrial arrhythmias among CHD patients doubles the risk of stroke or heart failure and triples the risk of cardiac reintervention.[8,9]

Despite the increasing burden of ATs in patients with CHD, the ability to prevent or to alter the natural history remains less certain. For example, among patients with right ventricular outflow tract (RVOT) obstructions or significant pulmonary regurgitation (PR), alleviation of the hemodynamic pathology with surgical pulmonary valve replacement (S-PVR) has only been shown to reduce the risk of recurrent ATs when paired with ablation.[10-12] Without ablation, there is a high risk of supraventricular tachycardia recurrence after S-PVR (up to 66% at 7.5 years in one study[11]). Furthermore, whether S-PVR reduces the risk of developing arrhythmias among patients without baseline atrial arrhythmias has not been well studied.

Over the past decade, transcatheter pulmonary valve replacement (TC-PVR) has also emerged as an approach to treating RVOT obstruction or PR,[13] and has demonstrated comparable hemodynamic and symptomatic outcomes relative to S-PVR.[14-16] To date, no study has quantified the burden of postoperative or short-term ATs following TC-PVR, especially relative to a surgical cohort. In this study, we sought to compare postoperative and short-term atrial arrhythmia burdens among contemporary cohort of patients with CHD who undergo PVR (either TC-PVR or S-PVR). We hypothesized that TC-PVR would be associated with a reduced burden of ATs.

2 | METHODS

2.1 | Study population

This was a single center, retrospective study design with an analysis of a previously collected database of consecutive patients undergoing TC-PVR or S-PVR at UCLA Medical Center between October 2010 through December 2016.[17] The database included patients during this period because TC-PVR became commercially available in the United States in 2010. Data collection was performed in 2017 and subsequent analysis of the data occurred in 2018. Patients of all ages who had prior surgical repair for CHD were included. Patients who had a history of congenitally corrected transposition of the great arteries, underwent a hybrid TC-PVR procedure with surgical plication of the RVOT, and those who had permanent atrial fibrillation (AF) without a concomitant ablation (either radiofrequency ablation [RFA] or surgical MAZE) at time of valve implantation were excluded. The latter was excluded since PVR without ablation was not expected to affect the underlying atrial tachyarrhythmia. Patients with congenitally corrected transposition of the great arteries were not included because these patients were most often diagnosed later in life relative to other complex congenital heart disease patients and had other primary indications for surgery besides PVR, such as systemic atrioventricular valvular regurgitation. For study purposes, the type of valve replacement (TC-PVR vs S-PVR) was defined as the first valve implanted after the onset of the study period.

2.2 | Data collection

After approval from the local Institutional Review Board, the electronic medical record was searched for baseline characteristics, including cardiac and medical history, age at valve implant, symptoms, medications, duration of follow-up, electrocardiographic measurements, 2D echocardiographic findings, invasive hemodynamic data, and indications for valve implantation. Patient charts were reviewed for presence and type of atrial arrhythmias before and after valve implantation, using inpatient telemetry records, electrocardiograms (ECG), cardiopulmonary stress testing, ambulatory ECG monitoring, and cardiac implantable electronic device (CIED) interrogations. The Ahmanson/UCLA Congenital Heart Disease Program policy is for all patients to receive continuous inpatient telemetry until the time of their discharge from the hospital following either surgical or TC-PVR. Routine ECG was assessed at each patient follow up visit. Per institutional protocol, cardiopulmonary exercise stress testing was performed annually and ambulatory ECG monitoring (Holter, event monitors, or ZIO Patch) was performed if the patient had symptoms of palpitations. Percentages of patients who had pre- and post-implant arrhythmia evaluations beyond routine electrocardiograms are provided are provided in the Supporting Information Appendix S1. Baseline "Triedman" clinical arrhythmia scores were calculated to further define the pre-PVR burden of AT.[18] Patient CIEDs were evaluated at a minimum of every 3 months with remote interrogations, as well as annually at clinical visits.

2.3 | Outcomes

The primary outcome was the time to a composite of clinically significant sustained AT. Sustained AT was defined as focal atrial tachycardia, intra-atrial reentrant tachycardia/atrial flutter (IART/AFL), or AF lasting at least 30 seconds in duration or terminating with cardioversion or antitachycardia pacing. Arrhythmia outcomes were considered clinically significant if they led to a change in medications and/or led to an electrophysiologic procedure. After the initial analysis in a time-to-event using the entire study period, secondary analyses were done by subdividing the study period into perioperative (within 30 days after valve implantation) and short-term epochs (events occurring after the first 30 days). For the early epoch, events which occurred in the first 30 days were censored.

TABLE 1 Baseline characteristics

Characteristic	Transcatheter (*n* = 168)	Surgical (*n* = 129)	Significance
Age (years)	21.2 (15.2-30.2)	21.0 (14.1-35.7)	0.91
Sex (% male)	61%	54%	0.21
Follow-up (years)	1.3 (0.2-2.8)	1.0 (0.1–2.9)	0.95
Primary congenital diagnosis (*n*)			
Tetralogy of Fallot	87 (52%)	83 (64%)	0.03
DORV[a]	14 (8%)	3 (2%)	0.03
Pulmonary stenosis	14 (8%)	19 (15%)	0.08
Truncus Arteriosus	14 (8%)	7 (5%)	0.33
Pulmonary atresia/IVS[b]	11 (7%)	5 (4%)	0.31
Aortic disease s/p Ross	14 (8%)	5 (4%)	0.12
Other	14 (8%)	7 (5%)	0.20
Pulmonary atresia	29%	12%	<0.001
≥3 Sternotomies (%)	41%	16%	<0.001
Pre-implant CIED[c] (*n*)	22 (13%)	17 (13%)	0.94
Age of repair (mo.)	12 (5-55)	24 (8-72)	0.06
Prior AT[d]	14%	18%	0.25
Triedman clinical arrhythmia score ≥3 (*n*)	25 (15%)	21 (16%)	0.92
Anticoagulation	6%	9%	0.39
PR interval (ms)	159	158	0.97
Concomitant EP[e] procedure	7%	13%	0.08
Baseline peak TR[f] velocity (m/s)	3.2 (2.9-3.9)	2.7 (2.5-3.6)	0.006
Right atrial enlargement (% mod-sev)	23%	23%	0.47
TR grade (% mod-sev)	18%	18%	0.59
Mean RAP[g] (mm Hg)	9 (7-11.5)	10 (8-13.5)	0.10
Procedure indication			
Regurgitation	30%	61%	<0.001
Stenosis	33%	15%	0.001
Mixed	36%	19%	<0.001
Other	1%	5%	0.02
Right ventricular outflow type			
Conduit	45%	23%	<0.001
Bioprosthesis	36%	3%	<0.001
Native/patch	19%	74%	<0.001
Distribution of implants over study period			
2010-2011	27 (16%)	26 (20%)	0.33
2012-2013	42 (24%)	42 (32%)	0.13
2014-2015	63 (37%)	45 (35%)	0.72
2016	40 (23%)	17 (13%)	0.03

[a]Double-outlet right ventricle.
[b]Intact ventricular septum.
[c]Cardiac implantable electronic device.
[d]Atrial tachyarrhythmia.
[e]Electrophysiologic.
[f]Tricuspid regurgitation.
[g]Right atrial pressure.

2.4 | Statistical analysis

Baseline data are presented as median with interquartile ranges or percentages with number, unless otherwise specified. Differences in the distributions of baseline continuous variables between TC-PVR and S-PVR groups were tested for significance (two-sided $P < .05$) using Mann-Whitney *U* test, while differences in categorical variables were analyzed with Pearson's Chi-square or Fisher's exact tests, as appropriate. Differences in the primary outcome of sustained AT between TC-PVR and S-PVR groups were analyzed in a propensity scored model adjusting for confounders with inverse probability of treatment weighting (IPTW) in a Poisson regression model.[19] In this model, weights are derived from the propensity score and are assigned to individual cases to create a sample in which the distribution of the baseline covariates is independent of treatment assignment. The propensity score's ability to predict grouping was tested with receiver operating characteristic curves. The list of variables used in the propensity score, as well as the propensity score's ability to predict grouping (ie, receiver operating characteristic curve of the propensity score) are provided in the Supporting Information Appendixes S2 and S3, respectively). Importantly, expected differences in duration of post-implant inpatient surveillance between catheter-based and surgically implanted cohorts was included as a variable in the propensity score. Results of statistical model are expressed with adjusted incidence rate ratios (IRR) for the Poisson regression models and are presented with 95% confidence intervals (CI). Kaplan-Meier curves were constructed to display time-to-event figures. Independent predictors in the multivariate model were also tested for multicollinearity (threshold VIF >3 for significance). The analysis was performed on IBM SPSS Statistics 24 (IBM Corporation, Armonk, NY).

3 | RESULTS

Two hundred ninety-seven patients (TC-PVR, $n = 168$ and S-PVR, $n = 129$) were included in this study. Baseline comparison between TC-PVR and S-PVR cohorts is provided in Table 1. Compared to TC-PVR patients, SPVR patients had longer duration of postoperative surveillance and a greater proportion were discharged on anticoagulation, beta blockers, or antiarrhythmic medications (Table 2).

Over a combined follow-up of 516 patient years (median 1.2 years, IQR 0.1-2.9 years), 32 AT events occurred among the 297 patients included undergoing PVR (total burden 11%, Table 3). Of these 32 events, 9 AT events occurred in TC-PVR group (5%) vs 23 AT events in S-PVR group (18%). In the IPTW adjusted Poisson regression model (Table 4), the following factors were associated with significant risk of atrial tachyarrhythmia after PVR: history of atrial arrhythmia, age at valve implantation, severe right atrial enlargement, and S-PVR. S-PVR patients were more likely to undergo a concomitant supraventricular structural intervention (such as atrial septal defect [ASD] closure or atrioventricular valve repair) compared to TC-PVR patients (26% vs 2%, respectively, $P < .001$, description of interventions in Supporting Information Appendix S4). In the secondary analysis, TC-PVR was associated with lower adjusted risk of AT events in the postoperative epoch compared to S-PVR pateints, adjusted IRR 0.31 (0.14-0.97) for TC-PVR vs S-PVR, $P = .03$, but similar adjust risk in the short-term epoch, TC-PVR vs S-PVR adjusted IRR 0.64 (0.14-2.94), $P = .57$ (Figure 1). Distributions of valve implantation over the study period (between 2010 and 2016) was similar between TC-PVR and S-PVR cohorts (Table 1) and no significant era effect difference was noted in AT outcomes (Supporting Information Appendix S5).

TABLE 2 Postoperative comparison of transcatheter and surgical pulmonary valve replacement patients

	TPVR	SPVR	Significance
Post-implant duration of surveillance, days	1 (1-11)	4 (2-44)	<0.001
Initiation of anticoagulation (%)	1%	5%	0.03
Initiation of beta blocker or anti-arrhythmic at discharge (%)	4%	33%	<0.001

The following factors were not associated with atrial arrhythmias in an exploratory analysis ($P > .05$): age at primary repair, indication for PVR (ie, PR vs PS), pulmonary atresia, post-implant duration of inpatient observation, implant location (native outflow vs conduit), number of sternotomies, baseline peak tricuspid regurgitant (TR) velocity, or mean right atrial pressure by catheterization.

4 | DISCUSSION

In this study evaluating the atrial tachyarrhythmia burden following pulmonary valve replacement, there was a decreased risk of postoperative atrial tachyarrhythmias in the first 30 days following TC-PVR when compared to S-PVR, particularly if additional supraventricular structural interventions are performed at time of PVR (such as ASD or atrioventricular valve repair). The mechanism by which these structural interventions lead to an increase in postoperative AT events can only be hypothesized from the present study; however, AT in this context is most likely related to local myocardial inflammation from the procedures themselves. Following confounder adjustment, a history of atrial arrhythmia, age at valve implantation, and severe right atrial enlargement were additional risk factors for atrial tachyarrhythmia after PVR (either TC-PVR or S-PVR approaches).

This study adds to a growing body of evidence highlighting the burden of atrial tachyarrhythmias among patients with CHD and challenging the optimal timing for intervention on dysfunctional RVOTs (either those with chronic regurgitant or obstructive/stenotic lesions). Chronic pulmonic regurgitation or right ventricular outflow tract obstructions lead to increased RV volume/pressures with subsequent tricuspid regurgitation.[20,21] The consequent stress

TABLE 3 Description of atrial tachyarrhythmia episodes

Group	Atrial arrhythmia type	Time since pulmonary valve replacement (days)	Management changes or mode of termination
TC-PVR[a]	Focal Atrial Tachycardia	1	Pace termination
	Focal Atrial Tachycardia	1	Self-limited/BB[d]
	IART/AFL[c]	22	DCCV[e]
	IART/AFL	91	DCCV
	Atrial Fibrillation	128	DCCV
	IART/AFL	146	RFA[f]
	Atrial Fibrillation	167	Rate Control (BB)
	Atrial Fibrillation	204	Amiodarone
	IART/AFL	1022	Amiodarone and BB
S-PVR[b]	Focal Atrial Tachycardia	1	Self-limited/BB
	Atrial Fibrillation	1	Self-limited/BB
	Atrial Fibrillation	2	Amiodarone and BB
	Focal Atrial Tachycardia	2	BB
	Focal Atrial Tachycardia	2	BB
	Atrial Fibrillation	2	Amiodarone
	Focal Atrial Tachycardia	2	Self-limited/BB
	Atrial Fibrillation	2	Amiodarone
	IART/AFL	2	DCCV
	Atrial Fibrillation	3	Amiodarone
	Focal Atrial Tachycardia	3	BB
	Atrial Fibrillation	3	DCCV
	Atrial Fibrillation	18	Amiodarone
	IART/AFL	18	DCCV
	Atrial Fibrillation	26	DCCV
	Focal Atrial Tachycardia	29	BB
	Atrial Fibrillation	95	Rate Control (BB)
	IART/AFL	179	RFA
	IART/AFL	183	RFA
	IART/AFL	372	DCCV
	IART/AFL	657	RFA
	IART/AFL	913	RFA
	Focal Atrial Tachycardia	1022	Flecainide and BB

[a]Transcatheter pulmonary valve replacement.
[b]Surgical pulmonary valve replacement.
[c]Intra-atrial reentrant tachycardia/atrial flutter.
[d]Beta-blocker.
[e]Direct current cardioversion (NOTE: All DCCV patients were placed on anticoagulation and anti-arrhythmic therapy).
[f]Radiofrequency Ablation.

TABLE 4 Adjusted Poisson regression model using inverse probability of treatment weighting with predictors for atrial tachyarrhythmias

Characteristic	Adjusted IRR (95% CI)	*P*-value
Surgical (vs Transcatheter)	3.06 (1.01-9.25)	.04
History of atrial arrhythmia	5.42 (1.94-15.2)	.001
Age at valve implantation (per year)	1.06 (1.04-1.09)	<.001
Severe right atrial enlargement (vs non-severe)	4.30 (1.29-14.3)	.02
Tricuspid regurgitation (per grade)	1.76 (0.93-3.31)	.08

on the right atrium may lead to the development of the arrhythmogenic substrate, which is a recognized contributor to morbidity and mortality in this patient population. In this study, the association between older age and severe right atrial enlargement as independent risk factors for atrial tachyarrhythmias after confounder adjustment is intriguing though not surprising. It remains unknown whether earlier interventions on right ventricular outflow tract lesions before atrial enlargement occurs might reduce the burden of such arrhythmias. Most of the literature on the optimal timing of PVR dates to an era before TC-PVR was an option. With growing adoption and improved operator skills associated with transcatheter techniques, it remains less clear whether the timing for TC-PVR should adhere to guidelines based on these older studies from

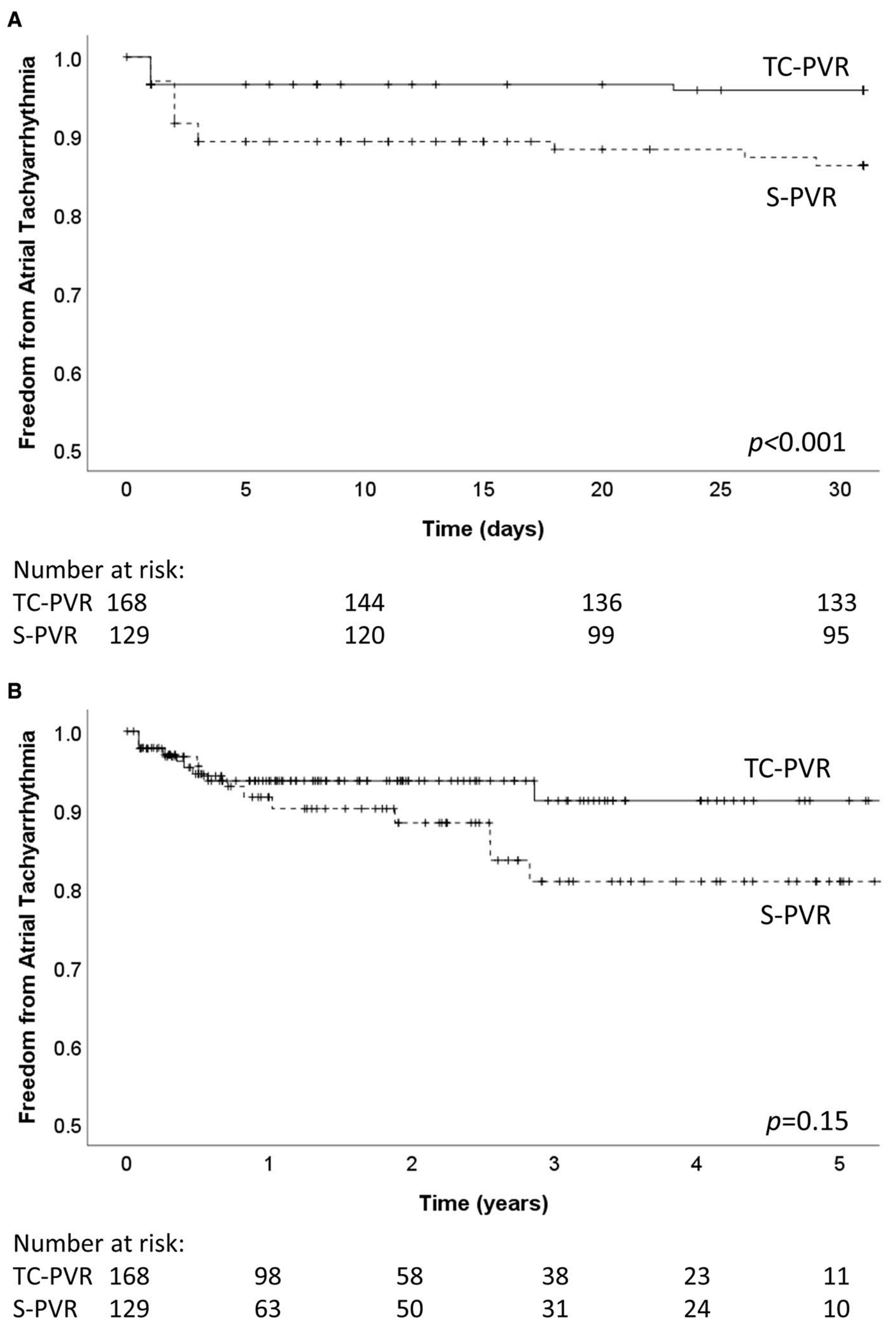

FIGURE 1 Kaplan-Meier curves showing freedom from atrial tachyarrhythmias in (A) the postoperative epoch (within 30 days of valve implantation) and (B) the short-term epoch (after censoring of events in the first 30 days). Solid line: TC-PVR; dashed line: S-PVR; + censored

a S-PVR cohort.[22-26] Future studies should seek to explore atrial sizes and remodeling after transcatheter PVR in greater detail, and whether atrial chamber dimensions might also contribute to guide decision making for optimal timing of intervention to reduce the burden of atrial arrhythmias.

From this study, in combination with previously published data from our institution's cohort, we determined that patients who undergo transcatheter PVR have lower risks of postoperative arrhythmias compared to a surgical approach, with similar short-term risk. This study also identified that treatment of hemodynamically significant regurgitant or stenotic lesion of the RVOT with PVR does not negate the risk of atrial tachyarrhythmias. After censoring events in the first 30 days, the combined freedom from atrial tachyarrhythmias (after TC-PVR or S-PVR) at 5 years was still 78%. Similar to previously published data on a surgical cohort,[27,28] 22% of patients still had an AT event by 5 years after PVR, suggesting that there may be other factors implicated in the development of atrial arrhythmias in this population. Nevertheless, given the associated early benefits from a transcatheter approach, including shorter hospital stay, faster recovery, decreased morbidity/adverse events, reduced 30-day readmission rates, and overall decrease in the arrhythmia burden in the first 30 days after valve implantation,[16,17] TC-PVR should be considered preferentially before S-PVR among eligible CHD patients, ideally at centers experienced to perform such interventions.

4.1 | Study limitations

The following limitations are recognized in this study. This was a retrospective observational study at a single center, advanced tertiary care institution. Lack of randomization with patient selection bias is inherent to the study design. While the use of a propensity scored analysis might attempt to address this bias, it does not eliminate it entirely. The effects of patient selection were evident in the baseline characteristics with differences in primary diagnosis, RVOT types, and the proportion of patients with regurgitant vs stenotic lesions. S-PVR patients were also more likely to undergo concomitant structural interventions (Supporting Information Appendix S4), specifically tricuspid valve annuloplasty, which may impact the postoperative AT burden. Nevertheless, the baseline differences between TC-PVR and S-PVR cohorts in this study are comparable to current practice at other academic institutions.[16] Due to the referral basis for many patients, long-term follow up data was not available in all patients. Additionally, the lack of standardized protocols are inherent to the study design and were especially relevant with regards to echocardiographic assessment of chamber sizes. Right atrial enlargement may have been assessed qualitatively on some studies and therefore is subject to interobserver bias. Finally, the small sample size in the secondary analysis may have contributed to Type II statistical error (failure to reject null hypothesis) due to an underpowered analysis.

5 | CONCLUSIONS

There was a decreased postoperative, but similar short-term, risk of atrial tachyarrhythmia following TC-PVR compared to S-PVR patients. A history of atrial arrhythmia, older age at valve implantation, severe right atrial enlargement, and S-PVR with concomitant structural interventions at time of PVR were positively associated with post-implant AT. Future studies should seek to explore the optimal timing of PVR to reduce the burden of arrhythmias.

ACKNOWLEDGMENT

The authors report no financial contributions to this study. All the authors have contributed significantly to the development of this manuscript. Drs Wadia, Aboulhosn, and Moore are responsible for the data integrity and the interpretation of the overall manuscript content.

CONFLICT OF INTEREST

The authors report no financial contributions or conflicts of interest pertaining to this study.

AUTHOR CONTRIBUTIONS

All of the authors listed have contributed significantly to this manuscript. Drs Wadia and Lluri collected the data and drafted the manuscript. Drs Aboulhosn and Moore conceived and designed the project. Drs Shannon and Van Arsdell revised the manuscript and provided a critical review. Finally, Drs Levi, Salem, Biniwale, and Laks interpreted their respective data, revised the manuscript, and provided final approval for submission.

ORCID

Subeer K. Wadia https://orcid.org/0000-0001-8611-6001

REFERENCES

1. Erikssen G, Liestol K, Seem E, et al. Achievements in congenital heart defect surgery: a prospective, 40-year study of 7038 patients. *Circulation*. 2015;131:337-346.
2. Raissadati A, Nieminen H, Jokinen E, Sairanen H. Progress in late results among pediatric cardiac surgery patients: a population-based 6-decade study with 98% follow-up. *Circulation*. 2015;131:347-353.
3. Gatzoulis MA, Balaji S, Webber SA, et al. Risk factors for arrhythmia and sudden cardiac death late after repair of tetralogy of Fallot: a multicenter study. *Lancet*. 2000;356:975-981.
4. Mandalenakis Z, Rosengren A, Lappas G, Eriksson P, Gilljam T, Hansson PO, Skoglund K, Fedchenko M, Dellborg M. Atrial fibrillation burden in young patients with congenital heart disease. *Circulation*. 2017. https://doi.org/10.1161/CIRCULATIONAHA.117.029590 [epub ahead of print]
5. Khairy P, Aboulhosn J, Gurvitz MZ, et al. Arrhythmia burden in adults with surgically repaired tetralogy of Fallot: a multi-institutional study. *Circulation*. 2010;122:868-875.

6. Lundqvist CB, Potpara TS, Malmborg H. Supraventricular arrhythmias in patients with adult congenital heart disease. *Arrhythm Electrophysiol Rev*. 2017;6:42-49.
7. Labombarda F, Hamilton R, Shohoudi A, et al. Increasing prevalence of atrial fibrillation and permanent atrial arrhythmias in congenital heart disease. *J Am Coll Cardiol*. 2017;70:857-865.
8. Bouchardy J, Therrien J, Pilote L, et al. Atrial arrhythmias in adults with congenital heart disease. *Circulation*. 2009;120:1679-1686.
9. Egbe AC, Connolly HM, McLeod CJ, et al. Thrombotic and embolic complications associated with atrial arrhythmia after Fontan operation: role of prophylactic therapy. *J Am Coll Cardiol*. 2016;68:1312-1319.
10. Therrien J, Siu SC, Harris L, et al. Impact of pulmonary valve replacement on arrhythmia propensity late after repair of tetralogy of Fallot. *Circulation*. 2001;103:2489-2494.
11. Karamlou T, Silber I, Lao R, et al. Outcomes after late reoperation in patients with repaired tetralogy of Fallot: the impact of arrhythmia and arrhythmia surgery. *Ann Thorac Surg*. 2006;81:1786-1793.
12. Discigil B, Dearani JA, Puga FJ, et al. Late pulmonary valve replacement after repair of tetralogy of Fallot. *J Thorac Cardiovasc Surg*. 2001;121:344-351.
13. Chatterjee A, Bajaj NS, McMahon WS, et al. Transcatheter pulmonary valve implantation: a comprehensive systematic review and meta-analyses of observational studies. *J Am Heart Assoc*. 2017;6:e006432.
14. Zablah JE, Misra N, Gruber D, Kholwadwala D, Epstein S. Comparison of patients undergoing surgical versus transcatheter pulmonary valve replacement: criteria for referral and mid-term outcome. *Pediatr Cardiol*. 2017;38:603-607.
15. McRae ME, Coleman B, Atz TW, Kelechi TJ. Patient outcomes after transcatheter and surgical pulmonary valve replacement for pulmonary regurgitation in patients with repaired tetralogy of Fallot: a quasi-meta-analysis. *Eur J Cardiovasc Nurs*. 2017;16:539-553.
16. Steinberg ZL, Jones TK, Verrier E, Stout KK, Krieger EV, Karamlou T. Early outcomes in patients undergoing transcatheter versus surgical pulmonary valve replacement. *Heart*. 2017;103:1455-1460.
17. Wadia SK, Lluri G, Aboulhosn JA, Shivkumar K, Reemtsen BL, Laks H, Biniwale RM, Levi DS, Salem M, Moore JP. Ventricular arrhythmia burden after transcatheter versus surgical pulmonary valve replacement [published online first April 10, 2018]. *Heart*. https://doi.org/10.1136/heartjnl-2017-312769.
18. Triedman JK, Alexander ME, Love BA, et al. Influence of patient factors and ablative technologies on outcomes of radiofrequency ablation of intra-atrial re-entrant tachycardia in patients with congenital heart disease. *J Am Coll Cardiol*. 2002;39:1827-1835.
19. Austin PC. An introduction to propensity score methods for reducing the effects of confounding in observational studies. *Multivariate Behav Res*. 2011;46:399-424.
20. Chaturvedi RR, Redington AN. Pulmonary regurgitation in congenital heart disease. *Heart*. 2007;93:880-889.
21. Bouzas B, Kilner PJ, Gatzoulis MA. Pulmonary regurgitation: not a benign lesion. *Eur Heart J*. 2005;26:433-439.
22. Therrien J, Provost Y, Merchant N, Williams W, Colman J, Webb G. Optimal timing for pulmonary valve replacement in adults after tetralogy of Fallot repair. *Am J Cardiol*. 2005;95:779-782.
23. Borik S, Crean A, Horlick E, et al. Percutaneous pulmonary valve implantation: 5 years of follow-up: does age influence outcomes? *Circ Cardiovasc Interv*. 2015;8:e001745.
24. Aboulhosn J, Levi DS. Percutaneous pulmonary valve implantation: is earlier better? *Circ Cardiovasc Interv*. 2015;8:e002260.
25. Bhagra CJ, Hickey EJ, Van De Bruaene A, Roche SL, Horlick EM, Wald RM. Pulmonary valve procedures late after repair of tetralogy of Fallot: current perspectives and contemporary approaches to management. *Can J Cardiol*. 2017;33:1138-1149.
26. Li WF, Pollard H, Karimi M, et al. Comparison of valvar and right ventricular function following transcatheter and surgical pulmonary valve replacement. *Congenit Heart Dis*. 2018;13:140-146.
27. Cortez D, Barham W, Ruckdeschel E, et al. Noninvasive predictors of perioperative atrial arrhythmias in patients with tetralogy of Fallot undergoing pulmonary valve replacement. *Clin Cardiol*. 2017;40:591-596.
28. Heng EL, Gatzoulis MA, Uebing A, et al. Immediate and midterm cardiac remodeling after surgical pulmonary valve replacement in adults with repaired tetralogy of Fallot: a prospective cardiovascular magnetic resonance and clinical study. *Circulation*. 2017;136:1703-1713.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

How to cite this article: Wadia SK, Lluri G, Aboulhosn JA, et al. Postoperative and short-term atrial tachyarrhythmia burdens after transcatheter vs surgical pulmonary valve replacement among congenital heart disease patients. *Congenital Heart Disease*. 2019;14:838–845. https://doi.org/10.1111/chd.12818