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Outcomes of persistent pulmonary hypertension following transcatheter aortic valve replacement

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Abstract

Objectives—To determine the prevalence and factors associated with persistent pulmonary hypertension (PH) following transcatheter aortic valve replacement (TAVR) and its relationship with long-term mortality.

Methods—Consecutive patients who underwent TAVR from July 2011 through January 2016 were studied. The prevalence of baseline PH (mean pulmonary artery pressure ≥ 25 mm Hg on right heart catheterisation) and the prevalence and the predictors of persistent moderate PH (pulmonary artery systolic pressure (PASP) >45 mm Hg on 1 month post-TAVR transthoracic Doppler echocardiography) were collected. Cox models quantified the effect of persistent PH on subsequent mortality while adjusting for confounders.

Results—Of the 407 TAVR patients, 273 (67%) had PH at baseline. Of these, 102 (25%) had persistent moderate PH. Mortality at 2 years in patients with no baseline PH versus those with PH improvement (follow-up PASP ≤ 45 mm Hg) versus those with persistent moderate PH was 15.4%, 16.6% and 31.3%, respectively ($p=0.049$). After adjusting for Society of Thoracic Surgeons Predicted Risk of Mortality and baseline right ventricular function (using tricuspid annular plane systolic excursion), persistent moderate PH remained associated with all-cause mortality (HR=1.82, 95% CI 1.06 to 3.12, $p=0.03$). Baseline characteristics associated with increased likelihood of persistent moderate PH were moderate tricuspid regurgitation, moderate mitral regurgitation, atrial fibrillation/flutter, early (E) to late (A) ventricular filling velocities (E/A ratio) and left atrial volume index.

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Conclusions—Persistency of even moderate or greater PH at 1 month post-TAVR is common and associated with higher all-cause mortality.

INTRODUCTION

Pulmonary hypertension (PH) is common in patients with severe aortic stenosis (AS) undergoing transcatheter aortic valve replacement (TAVR) and associated with increased mortality post-TAVR.^{1–7} Despite its prognostic role, it remains a missing component of the traditional Society of Thoracic Surgeons risk score assessment. Although invasive right heart catheterisation (RHC) is the gold standard method for PH evaluation, pre-TAVR PH screening is recommended to be performed non-invasively through transthoracic Doppler echocardiography (TTE) with the estimation of pulmonary artery systolic pressure (PASP) using simplified Bernoulli equation.^{8,9}

Studies have suggested that only severe post-TAVR PH is a predictor of mortality on a relatively short follow-up time.^{14,7} However, PH is a heterogeneous entity in which outcomes can be influenced by the presence of right ventricular dysfunction and other comorbidities. Importantly, most studies have used TTE to diagnose PH, and have not performed comprehensive baseline right-sided assessment with adjustment of these parameters into the measured outcome.^{4,7,10} Therefore, we sought to understand the prevalence of persistent moderate or greater PH post-TAVR and its effect on mortality in a contemporary cohort of TAVR patients who underwent comprehensive clinical, echocardiographic and invasive haemodynamic evaluation.

METHODS

Study design

We conducted a retrospective cohort analysis of patients who underwent TAVR at the University of Pittsburgh Medical Center, a large tertiary care centre, between July 2011 and January 2016. Because the purpose was to evaluate the clinical impact of persistent PH post-TAVR, we excluded patients who: (A) had undergone valve-in-valve procedures, (B) lacked baseline RHC data, (C) died prior to post-TAVR 1-month echocardiographic assessment, (D) had uninterpretable tricuspid regurgitation (TR) jet signal to estimate PASP at 1 month post-TAVR, or (E) lacked 1-month follow-up TTE.

Patients underwent comprehensive clinical evaluation by the designated Heart Team and deemed appropriate to undergo TAVR as suggested by the guidelines.^{9,11} Clinical, laboratorial and procedural data were collected. Baseline PH was defined by clinical RHC pre-TAVR as a mean pulmonary artery pressure (mPAP) ≥ 25 mm Hg according to guidelines.¹² Up to 505 patients underwent TAVR during the study period. Persistent moderate PH or greater post-TAVR was defined as PASP > 45 mm Hg on Doppler echocardiography using modified Bernoulli equation. Patients were clinically followed up in our programme at 1, 6 and 12 months post-TAVR, and annually thereafter. Primary outcome was all-cause mortality, which was obtained through querying the social security death index (using the updated Social Security Administration Death Master file, for which our healthcare system is exempt from the 3-year delay period by the Social Security Administration).

Study procedures (echocardiogram, RHC and TAVR procedure)

All included patients undergoing TAVR evaluation underwent comprehensive TTE using commercial systems (Philips Medical Systems, Bothell, WA; Siemens Medical Solution, Malvern, PA; or General Electric, Milwaukee, WI). Both baseline and 1-month follow-up TTE studies were systematically independently reviewed as per guidelines^{13–15} for collection of several imaging parameters. All included patients underwent RHC as part of their clinical care prior to TAVR. Fluid-filled catheters that were connected to pressure transducers were used to measure pressures inside the right atrium, right ventricle (RV), pulmonary artery and pulmonary arterial wedge pressure (PAWP). Lastly, all patients underwent TAVR procedure receiving either balloon-expandable or self-expanding bioprosthesis. Study procedural details are available in online supplementary file 1.

Statistical analysis

Categorical variables are presented as frequency (percentage); continuous variables are presented as mean±SD. For categorical variables, groups are compared using the X² test, except in the case of selected variables with less than five patients in a single cell, where Fisher's exact test was used. For continuous variables, analysis of variance was used to test for differences in the baseline characteristics across all three groups (no baseline PH vs improved PH vs residual PH) while two-sample t-tests were used to test pairwise differences between the two groups that had PH before TAVR (ie, improved PH vs residual PH). Factors associated with residual moderate PH or greater post-TAVR were assessed using univariable and multivariable logistic regression analyses, adjusting for the baseline mPAP. Survival after TAVR is presented using Kaplan-Meier curves; log-rank tests were used to compare unadjusted survival times between groups. Cox proportional hazards models were used to quantify the risk for patients with residual PH versus those in whom PH resolved while adjusting for Society of Thoracic Surgeons Predicted Risk of Mortality (STS-PROM) (as a proxy for overall patient's burden of comorbidities, except for PH) and tricuspid annular plane systolic excursion (TAPSE), an established measure of RV systolic function. The proportional hazards assumption was tested by looking at time-dependent covariates (time*covariate interaction variables) to determine whether the main effects varied over time. Statistical analyses were performed using SAS V9.4 (SAS Institute).

RESULTS

Of the 505 patients identified who underwent TAVR during the study period, 407 met the inclusion criteria. Figure 1 displays the study workflow. Of those, 273 (67%) had baseline PH (mPAP ≥ 25 mm Hg). Patients with baseline PH were further subdivided, based on the 1-month post-TAVR follow-up TTE, into persistent PH group (102 patients or 25%, if PASP ≥ 45 mm Hg) and improved PH group (171 patients or 42%, follow-up PASP < 45 mm Hg).

Baseline clinical characteristics are shown in table 1. Median STS-PROM score was higher in the residual PH versus improved PH group (9.9% vs 8.2%, $p = 0.004$). Preoperative atrial fibrillation was also more common in patients with residual PH than in those with improved PH (62.7% vs 46.2%, $p=0.013$).

Echocardiography

Baseline echocardiographic measurements are shown in table 2. Median follow-up echo was obtained 34 days (IQR 29–36 days) post-TAVR procedure. Baseline moderate to severe mitral regurgitation (MR), moderate to severe TR, higher mean PASP higher early (E) to late (A) ventricular filling velocities (E/A ratio) and larger mean left atrial volume index (LAVI) were seen in patients with residual PH.

Invasive haemodynamics

Baseline RHC data are shown in table 3. Patients with residual PH had higher mPAP (38.9 ± 9.5 vs 34.6 ± 8.4 mm Hg; $p < 0.001$), higher pulmonary vascular resistance (PVR, 3.6 ± 2.3 vs 2.7 ± 2.1 Wood units; $p = 0.004$) and higher transpulmonary gradient (TPG, 15.4 ± 8.6 vs 12.5 ± 8.3 mm Hg; $p = 0.008$). There was no statistically significant difference in diastolic pressure gradient (DPG). In patients with persistent PH, only 13% had a DPG ≥ 7 mm Hg, while 54% had PVR > 3 Wood units and 57% had TPG ≥ 12 mm Hg.

TAVR procedure

Procedural characteristics are shown in online supplementary table 1. The majority of patients underwent transfemoral approach (74.2%), followed by left subclavian approach (13%). There were no statistically significant differences between all three groups for their respective procedural characteristics.

Outcomes

Over a mean follow-up of 20 ± 13 months, there were 82 deaths. The 2-year all-cause mortality in patients with no PH, improved PH and residual PH was 15.4%, 16.6% and 31.3% ($p = 0.049$), respectively (figure 2). There was no statistically significant difference in mortality between patients with no PH at baseline and those with PH at baseline as defined by RHC (see online supplementary figure S1). When patients with PH at baseline were divided based on post-TAVR echo-derived PASP, there was no statistically significant difference in mortality between patients with no PH at baseline and those with improved PH; $p = 0.557$. On the other hand, residual moderate PH as compared with improved PH was associated with an increased risk of all-cause mortality with an unadjusted HR of 1.89 (95% CI 1.12, 3.19, $p = 0.018$). Multivariable analysis showed that persistent PH was independently associated with all-cause mortality (HR=1.82, 95% CI 1.06 to 3.12, $p = 0.03$) even after adjustment for the comprehensive STS-PROM risk score and RV TAPSE.

Pre-TAVR characteristics associated with persistent PH in unadjusted univariate analysis as well as adjusted for baseline mPAP are shown in table 4. After adjusting for mPAP baseline moderate TR, moderate MR, atrial fibrillation or atrial flutter, E/A ratio and LAVI were associated with persistent PH.

We performed a secondary analysis where pre-TAVR echo-derived PASP > 45 mm Hg was used as the entry criteria to diagnose baseline PH rather than invasive mPAP ≥ 25 mm Hg by RHC. Despite discordance between echo and RHC in the quantification of PH severity,¹⁶ survival curves were similar (see online supplementary table). When patient outcomes were analysed based on discordance of pre-TAVR PH diagnosis by echo and RHC, there was no

statistically significant survival difference in patients with baseline echo-derived PASP 45 mm Hg, irrespective of invasively measured mPAP (see online supplementary figure S3). Furthermore, regardless of the PH haemodynamic subtype, all-cause mortality was higher in those with persistent PH versus improved (see online supplementary figures S4 and S5, panels A-C).

DISCUSSION

In this large single-centre study of 407 consecutive patients undergoing TAVR, persistent moderate or greater PH at 1 month post-TAVR is common (25% of overall TAVR cohort or 37.4% of those with baseline PH) and associated with a high 2-year all-cause mortality (31.3%). The presence of residual PH (PASP by TTE >45 mm Hg) was associated with a 1.8-fold increased risk of mortality, even after adjustment for the comprehensive STS-PROM score as well as an index of RV systolic function (TAPSE), suggesting that persistent PH is an independent contributor to mortality in this population. Importantly, post-TAVR PH improvement at 1 month is associated with similar 2-year all-cause mortality as patients with no baseline PH (16.6% vs 15.4%, $p=0.557$). Therefore, not all patients with baseline PH are at risk of poor outcomes post-TAVR. The risk appears to be increased to patients when even moderate PH remains present at 1 month postprocedure. Thus, identification of both baseline and post-TAVR PH is of paramount importance.

Baseline PH has been shown in many studies to be associated with mortality and worse outcomes in patients undergoing TAVR.^{14–17} To our knowledge, only two other studies have evaluated the short-term implications of post-TAVR persistency of PH into outcomes.^{4,7} Testa *et al* reported that post-TAVR patients with persistent severe PH (PASP >60 mm Hg at 1 month post-TAVR) had a 2.4-fold increase at 1-year mortality.⁴ Sinning *et al* demonstrated that patients with persistent severe PH (PASP >60 mm Hg, 90 days post-TAVR) had higher 2-year mortality than patients with improved PH (50% vs 18.6%, $p=0.001$).⁷ The important limitations in both studies included: (1) gold standard RHC was not used to diagnose baseline PH and (2) comprehensive right-sided assessment evaluating RV systolic function was not performed pre-TAVR and/or adjusted into a multivariate model.

RV dysfunction and eventually RV failure are associated with decreased survival in PH.^{17,18} However, in neither of the prior published studies was RV function and/or TR integrated into the multivariate model⁴ and/or measured.⁷ Our findings demonstrated that these worse outcomes persisted even with adjusting for comprehensive STS-PROM score and RV function measured by TAPSE.

RHC prior to TAVR allows accurate PH diagnosis, prognostication and haemodynamic PH classification into isolated precapillary, isolated postcapillary or combined pre-post capillary PH, according to the guidelines.^{12,19} In our study, patients with residual PH had higher baseline mPAP, TPG and PVR, with similar PAWP, signalling a higher prevalence of the combined pre-post capillary PH phenotype. While our cohort is underpowered to detect statistically significant differences based on PH subtype, trends for worse outcomes in patients with persistent PH post-TAVR were consistent in all PH subtypes.

In our study, baseline characteristics associated with increased likelihood of persistent moderate PH were moderate TR, moderate MR, atrial fibrillation/flutter, E/A ratio and LAVI. These findings suggest worse haemodynamic profile with higher burden of concomitant valvular heart disease and worse diastolic function. In a prior study by Testa *et al*, left ventricular ejection fraction <30%, atrial fibrillation and an organic cause of MR were associated with persistent PH.⁴ However, Testa *et al* did not report the prevalence of TR, hence an important limitation of their analysis. Schwartz *et al* have recently shown in a large single-centre study that patients with baseline moderate TR had a higher 6-month PASP (49.4±19 vs 39.8±13 mm Hg) supporting our observations.¹⁸

Overall, our study expands the findings by Testa *et al* and Sinning *et al* and provides crucial proof of the importance of continued PH assessment by showing that even moderate or greater PH at 1 month post-TAVR is strongly associated with worse survival, independent of baseline RV function. Thus, these considerations should be incorporated into management decisions made jointly with the patient and clinical team. With recent advances in the medical therapeutic options for patients with PH, further studies are needed to determine whether baseline and persistent PH in patient with severe AS can be modified by further therapeutic strategies, beyond TAVR.

LIMITATIONS

First, although this represents a large contemporary TAVR cohort from a single centre, this remains an observational study with the limitations that accompany such design, including the exclusion of 75 patients due to missing data (52 patients without baseline RHC data, and 23 patients without follow-up echo at 1 month). However, all included patients underwent a uniform protocolled comprehensive evaluation by the Heart Team prior to TAVR, and all echocardiograms were systematically and independently reviewed. Second, we used RHC to define pre-TAVR PH, but used PASP by TTE to define persistent PH post-TAVR. While RHC remains the most accurate method to diagnose PH, it is not common practice to be performed in the post-TAVR setting. The currently accepted practice of using echocardiography for clinical decisions pre and post-TAVR, and the role of RHC in the care of patients undergoing TAVR need to be further defined particularly for those patients with persistent PH. Lastly, the clinical implications of persistent PH into other important post-TAVR metrics such as quality of life, functional capacity and hospital readmissions need to be equally evaluated in future studies.

CONCLUSION

Longitudinal evaluation of PH severity is necessary in TAVR patients. Persistent moderate or greater PH at 1 month post-TAVR is common and strongly associated with all-cause mortality. PH improvement post-TAVR is associated with an improved survival similar to patients without baseline PH. Whether persistent PH should be a modifiable target for future therapies beyond TAVR requires further investigation.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Key messages**What is already known on this subject?**

Severe pulmonary hypertension (PH) at baseline is associated with worse outcomes after transcatheter aortic valve replacement (TAVR).

What might this study add?

Persistent moderate to severe PH at 1 month is common and is associated with increased mortality, independent of baseline right ventricular function.

How might this impact on clinical practice?

- ▶ Persistent moderate to severe PH at 1 month is common and is associated with increased mortality, independent of baseline right ventricular function.
- ▶ Evaluation of PH at post-TAVR setting is necessary as it might be a target to further improve TAVR outcomes.

Figure 1.

Study population and workflow. mPAP, right heart catheterisation-derived mean pulmonary artery pressure; N, number; PASP, echo-derived pulmonary artery systolic pressure; PH, pulmonary hypertension; RHC, right heart catheterisation; TAVR, transcatheter aortic valve replacement.

Figure 2.
Survival after transcatheter aortic valve replacement (TAVR) in the study population (starting at 30 days post-TAVR).

Table 1

Baseline characteristics

Variables	No baseline PH (n=134)	Improved PH (n=171)	Residual PH (n=102)	p Value *	p Value **
Age	82.9±7.2	82.6±7.8	82.8±7.1	0.859	0.930
Male	66 (49.3%)	94 (55.0%)	50 (49.0%)	0.341	0.510
Prior myocardial infarction	37 (27.6%)	75 (43.9%)	33 (32.4%)	0.060	0.010
Diabetes	48 (35.8%)	70 (40.9%)	38 (37.3%)	0.547	0.638
Dyslipidaemia	102 (76.1%)	136 (79.5%)	77 (75.5%)	0.435	0.676
Pre-TAVR dialysis †	1 (0.7%)	6 (3.5%)	5 (4.9%)	0.751	0.125
Hypertension	117 (87.3%)	155 (90.6%)	98 (96.1%)	0.096	0.067
Atrial fibrillation/flutter	43 (32.1%)	79 (46.2%)	64 (62.7%)	0.005	<0.0001
PVD	49 (36.6%)	58 (33.9%)	40 (39.2%)	0.377	0.672
Prior CABG	40 (29.9%)	58 (33.9%)	27 (26.5%)	0.199	0.420
Prior valve surgeries other than AV	5 (3.7%)	15 (8.8%)	7 (6.9%)	0.575	0.213
Pre-TAVR pacemaker	15 (11.2%)	29 (17.0%)	27 (26.5%)	0.170	0.018
NYHA class 3	94 (70.1%)	136 (79.5%)	74 (72.5%)	0.426	0.691
Chronic lung disease	48 (35.8%)	65 (38.0%)	45 (44.1%)	0.339	0.420
STS-PROM (%)	7.5±3.8	8.2±4.3	9.9±5.2	0.004	<0.001
STS-PROM 8%	52 (38.8%)	79 (46.2%)	60 (58.8%)	0.025	0.006
Beta-blockers	62 (46.3%)	96 (56.1%)	62 (60.8%)	0.620	0.143
ACE-I/ARB	40 (29.9%)	74 (43.3%)	42 (41.2%)	0.734	0.045
Aspirin	110 (82.1%)	142 (83.0%)	83 (81.4%)	0.726	0.938

Categorical variables compared with χ^2 tests unless otherwise specified.

* p Value compares the two groups with baseline PH (improvement in PH vs residual PH);

** p value compares all three groups (no baseline PH vs improvement in PH vs residual PH).

† Variables that used Fisher's exact test.

ACE-I, ACE inhibitor; ARB, angiotensin receptor blocker; AV, aortic valve; CABG, coronary artery bypass grafting; NYHA, New York Heart Association heart failure class; PH, pulmonary hypertension; PVD, peripheral vascular disease; STS-PROM, Society of Thoracic Surgeons Predicted Risk of Mortality score; TAVR, transcatheter aortic valve replacement.

Table 2

Baseline Echocardiographic measurements

Variables	No baseline PH (n=134)	Improved PH (n=171)	Residual PH (n=102)	p Value *	p Value **
LVEF (%)	58.8±10.1	52.9±14.0	51.1±14.7	0.320	<0.0001
LVEF category				0.167	0.0001
LVEF<35 (%)	5 (3.7%)	26 (15.2%)	18 (17.6%)		
LVEF 35–50 (%)	11 (8.2%)	21 (12.3%)	20 (19.6%)		
LVEF 50 (%)	116 (86.6%)	122 (71.3%)	62 (60.8%)		
SVI (mL/m ²)	39.2±9.3	35.4±12.8	33.7±9.3	0.250	0.001
SVI<35 mL/m ²	47 (35.1%)	86 (50.3%)	53 (52.0%)	0.684	0.004
Qualitative RV systolic function [†]				0.103	<0.001
Normal	123 (91.8%)	143 (83.6%)	78 (76.5%)		
Mild dysfunction	2 (1.5%)	9 (5.3%)	9 (8.8%)		
Moderate dysfunction	1 (0.7%)	7 (4.1%)	10 (9.8%)		
Severe dysfunction	0 (0.0%)	1 (0.6%)	0 (0.0%)		
TAPSE (cm)	1.9±0.5	1.8±0.5	1.7±0.5	0.146	0.001
TAPSE<1.7 (cm)	37 (27.6%)	61 (35.7%)	47 (46.1%)	0.138	0.023
PASP (mm Hg)	35.3±11.2	41.7±13.1	58.7±19.3	<0.0001	<0.0001
PASP>45 mm Hg	23 (17.2%)	57 (33.3%)	72 (70.6%)	<0.0001	<0.0001
Moderate to severe MR	8 (6.0%)	20 (11.7%)	21 (20.6%)	0.038	0.003
Moderate to severe TR	7 (5.2%)	18 (10.5%)	36 (35.3%)	<0.0001	<0.0001
AV mean gradient (mm Hg)	49.9±13.8	47.7±16.3	46.7±16.3	0.637	0.253
AV mean gradient <40 (mm Hg)	26 (19.4%)	55 (32.2%)	32 (31.4%)	0.901	0.028
AVA (cm ²)	0.66±0.2	0.64±0.2	0.61±0.2	0.205	0.101
AVAi (cm ² /m ²)	0.37±0.1	0.35±0.1	0.33±0.1	0.335	0.018
LAVI (mL/m ²)	46.7±16.7	47.7±17.0	54.3±15.8	0.004	0.003
Lateral e' (cm/s)	5.4±2.2	5.7±2.3	6.4±2.5	0.119	0.098
Septal e' (cm/s)	4.3±1.6	4.3±1.6	4.5±2.0	0.486	0.726
Lateral E/e'	20.8±12.1	23.2±10.7	22.9±10.4	0.901	0.465

Variables	No baseline PH (n=134)	Improved PH (n=171)	Residual PH (n=102)	p Value *	p Value **
Septal E/e '	25.8±12.4	29.6±12.1	32.6±14.2	0.252	0.050
E/A ratio	0.9±0.5	1.3±0.7	1.8±1.0	0.002	<0.0001

Categorical variables compared with X² tests unless otherwise specified.

* p Value compares the two groups with baseline PH (improvement in PH vs residual PH);

** p value compares all three groups (no baseline PH vs improvement in PH vs residual PH).

[#] Variables that used Fisher's exact test.

AV, aortic valve; AVA, aortic valve area; AVAi, aortic valve area index; E/A ratio, early (E) to late (A) ventricular filling velocities; LAVI, left atrial volume index; LVEF, left ventricular ejection fraction; MR, mitral regurgitation; PASP, pulmonary artery systolic pressure; PH, pulmonary hypertension; RV, right ventricle; SVI, stroke volume index; TAPSE, tricuspid annular plane systolic excursion; TR, tricuspid regurgitation.

Table 3

Baseline right heart catheterization measurements

Variables	No baseline PH (n=134)	Improved PH (n=171)	Residual PH (n=102)	p Value *	p Value **
RAP (mm Hg)	5.3±3.3	10.2±4.9	11.5±5.3	0.048	<0.0001
RVSP (mm Hg)	35.2±8.4	54.1±12.5	64.0±16.1	<0.0001	<0.0001
PASP (mm Hg)	33.7±7.3	54.6±13.2	63.5±16.2	<0.0001	<0.0001
mPAP (mm Hg)	19.6±3.6	34.6±8.4	38.9±9.5	0.0001	<0.0001
PAWP (mm Hg)	11.7±5.0	22.1±9.0	23.6±7.1	0.161	<0.0001
PAWP>15 mm Hg	19 (14.2%)	141 (82.5%)	88 (86.3%)	0.544	<0.0001
Aortic SBP (mm Hg)	136±31.2	144±26.8	140±28.6	0.362	0.111
Thermal CO (L/min)	4.5±1.4	4.7±1.2	4.3±1.2	0.017	0.070
Thermal index (L/min/m ²)	2.5±0.6	2.5±0.6	2.4±0.7	0.068	0.159
Fick CO (L/min)	5.2±1.4	4.9±1.6	4.7±1.2	0.365	0.096
Fick index (L/min/m ²)	2.9±0.7	2.7±0.9	2.6±0.6	0.877	0.012
AV mean gradient (mm Hg)	42.6±13.9	47.0±15.7	42.4±12.9	0.055	0.065
AV mean gradient <40	39 (29.1%)	33 (19.3%)	29 (28.4%)	0.080	0.026
AVA (cm ²)	0.72±0.22	0.70±0.22	0.77±0.25	0.055	0.142
AVAi (cm ² /m ²)	0.40±0.10	0.37±0.11	0.43±0.13	0.009	0.024
PVR (Wood units, using Fick CO)	1.7±0.9	2.7±2.1	3.6±2.3	0.004	<0.0001
PVR>3 Wood units	7 (5.2%)	59 (34.5%)	49 (48.0%)	0.044	<0.0001
PVR (Wood units, using thermal CO)	1.9±1.7	2.9±1.7	3.8±2.4	0.001	<0.0001
PVR>3 Wood units	18 (13.4%)	55 (32.2%)	54 (52.9%)	0.001	<0.0001
DPG (mm Hg)	1.3±1.9	1.73±3.0	2.5±4.8	0.100	0.014
DPG>7 mm Hg	4 (3.0%)	13 (7.6%)	13 (12.7%)	0.161	0.017
TPG (mm Hg)	7.9±5.5	12.5±8.3	15.4±8.6	0.008	<0.0001
TPG>12 mm Hg	12 (9.0%)	73 (42.7%)	58 (56.9%)	0.030	<0.0001
PH subclassification				0.224	<0.0001
None	128 (95.5%)	0 (0.0%)	0 (0.0%)		
Precapillary PH	0 (0.0%)	26 (15.2%)	13 (12.7%)		

Variables	No baseline PH (n=134)	Improved PH (n=171)	Residual PH (n=102)	p Value *	p Value **
Isolated postcapillary PH	0 (0.0%)	95 (55.6%)	50 (49.0%)		
Combined pre/post capillary PH	0 (0.0%)	46 (26.9%)	38 (37.3%)		

Categorical variables compared with χ^2 tests unless otherwise specified.

* p Value compares the two groups with baseline PH (improvement in PH vs residual PH);

** p value compares all three groups (no baseline PH vs improvement in PH vs residual PH).

AV, aortic valve; AVA, aortic valve area; AVAI, aortic valve area index; CO, cardiac output; DPG, diastolic pressure gradient; mPAP, mean pulmonary artery pressure; PASP, pulmonary artery systolic pressure; PAWP, pulmonary artery wedge pressure; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; RAP, right atrial pressure; RVSP, right ventricular systolic pressure; SBP, systolic blood pressure; TPG, transpulmonary gradient.

Table 4

Relationship between preoperative factors and residual PH

	Unadjusted		Adjusted for pre-TAVR mPAP			
	OR	95% CI	p Value	OR	95% CI	p Value
Age	1.003	0.97 to 1.04	0.859	1.01	0.97 to 1.05	0.452
LVEF (vs 50%)						
LVEF<35 (%)	1.36	0.69 to 2.67	0.369	1.20	0.59 to 2.42	0.609
LVEF 35–50 (%)	1.87	0.94 to 3.72	0.072	1.86	0.91 to 3.75	0.086
Moderate to severe MR *	2.02	1.02 to 3.95	0.041	2.11	1.05 to 4.21	0.035
Moderate to severe TR *	4.72	2.49 to 8.95	<0.0001	4.69	2.41 to 9.08	<0.0001
Chronic lung disease	1.28	0.77 to 2.10	0.339	1.31	0.78 to 2.19	0.296
AV mean gradient <40 mm Hg	0.97	0.56 to 1.64	0.901	0.94	0.54 to 1.62	0.823
TAPSE<1.7 cm	1.47	0.88 to 2.44	0.139	1.23	0.72 to 2.09	0.445
SVI<35 mL/m ²	1.11	0.66 to 1.86	0.684	0.98	0.57 to 1.66	0.932
PVR>3Wood units	1.70	1.01 to 2.84	0.044	1.00	0.55 to 1.82	0.996
DPG>7 mm Hg	1.78	0.78 to 4.00	0.166	1.24	0.52 to 2.96	0.626
LAVI (per 10 mL/m ²)	1.27	1.07 to 1.49	0.004	1.25	1.02 to 1.48	0.008
Atrial fibrillation or atrial flutter	2.07	1.23 to 3.47	0.006	1.94	1.14 to 3.29	0.013
E/A ratio	2.17	1.26 to 3.75	0.006	2.14	1.24 to 3.70	0.006

* Pre-TAVR.

AV, aortic valve; DPG, diastolic pressure gradient; LAVI, left atrial volume index; E/A ratio, E/A ratio, early (E) to late (A) ventricular filling velocities; LVEF, left ventricular ejection fraction; mPAP, mean pulmonary artery pressure; MR, mitral regurgitation; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; SVI, stroke volume index; TAPSE, tricuspid annular plane systolic excursion; TAVR, transcatheter aortic valve replacement; TR, tricuspid regurgitation.