



Prospective Observational Study on the Accuracy of Predictors of Permanent Pacemaker Secondary to High-Grade Atrioventricular Conduction Block After TAVI (CONDUCT-TAVI)

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BACKGROUND: The incidence of permanent pacemaker implantation (PPMI) due to high-grade atrioventricular block after transcatheter aortic valve implantation (TAVI) is 10% to 15% at 1-year, and current prediction algorithms remain unreliable.

METHODS: CONDUCT-TAVI (Prospective Observational Study on the Accuracy of Predictors of Permanent Pacemaker Secondary to High-Grade Atrioventricular Conduction Block After TAVI) is a prospective observational study of 200 patients undergoing TAVI across 2 centers. Baseline demographic, anatomic, and procedural characteristics were recorded, followed by targeted electrophysiology studies and continuous rhythm monitoring using implantable loop recorders for 1 year. The primary outcome was PPMI secondary to high-grade atrioventricular block, and secondary outcomes included early (≤ 48 hours) and late (> 48 hours) PPMI, new-onset persistent left bundle branch block, and new-onset atrial fibrillation. Predictors were assessed using multivariable logistic regression.

RESULTS: PPMI due to high-grade atrioventricular block occurred in 21.0% of patients (early PPMI: 13.5%, late PPMI: 7.5%). Key predictors included preexisting right bundle branch block (adjusted odds ratio, 5.45 [95% CI, 1.67–17.84]; $P=0.005$), Δ HV [His-Ventricle] interval > 10 ms (adjusted odds ratio, 3.62 [95% CI, 1.23–10.67]; $P=0.020$), and pre-TAVI rapid atrial pacing-induced atrioventricular Wenckebach (adjusted odds ratio, 3.70 [95% CI, 1.37–9.98]; $P=0.010$). The CONDUCT-TAVI score combined these variables with high predictive accuracy (area under the curve=0.794) and negative predictive value (98%). New-onset persistent left bundle branch block (> 24 hours) was observed in 19.1%, and new-onset atrial fibrillation in 21.7% at 1 year.

CONCLUSIONS: The incidence of conduction abnormalities remains high after TAVI, and after factoring in anatomic, procedural, and electrophysiological factors, a baseline right bundle branch block and electrophysiology study-derived measures of atrioventricular conduction were the most significant predictors of PPMI. The CONDUCT-TAVI score incorporates these findings to help implanters stratify low-risk patients and tailor follow-up care.

GRAPHIC ABSTRACT: A [graphic abstract](#) is available for this article.

Key Words: atrial fibrillation ■ atrioventricular block ■ electrophysiology ■ humans ■ incidence

Postprocedural high-grade atrioventricular conduction block (HGAVB) is a common complication of transcatheter aortic valve implantation (TAVI),

primarily due to the proximity of the membranous septum to the aortic valve complex. This leaves the Bundle of His and left bundle branch vulnerable to injury, with new

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WHAT IS KNOWN

- Despite advancements in valve engineering and implantation techniques, high-grade atrioventricular block requiring permanent pacemaker implantation is still prevalent after transcatheter aortic valve implantation, believed to occur in up to $\approx 15\%$ of all cases.
- There is a lack of a reliable and reproducible prediction algorithm, despite various anatomic, procedural, and electrophysiological risk factors.

WHAT THE STUDY ADDS

- Continuous rhythm monitoring using an implantable loop recorder over 1 year revealed that high-grade atrioventricular block requiring a permanent pacemaker occurs in 21.0% of patients, with over a third (7.5%) developing it after 48 hours.
- A significant number of patients (21.5%) were diagnosed with new-onset atrial fibrillation, predominantly in a paroxysmal form with varying burdens.
- The CONDUCT-TAVI score, which includes electrophysiologically derived parameters such as preexisting right bundle branch block, changes in His-Ventricle interval, and pretranscatheter aortic valve implantation rapid atrial pacing-derived atrioventricular Wenckebach, showed high predictive accuracy and a negative predictive value of 98% for new pacemaker implantation at 1 year, thereby assisting implanters in patient stratification for rapid discharge.

Nonstandard Abbreviations and Acronyms

AVW	atrioventricular Wenckebach
CHB	complete heart block
CT	computed tomography
HGAVB	high grade atrioventricular block
LBBB	left bundle branch block
NOAF	new onset atrial fibrillation
OR	odds ratio
PPMI	permanent pacemaker implantation
RAP	rapid atrial pacing
RBBB	right bundle branch block
TAVI	transcatheter aortic valve implantation

permanent pacemaker implantation (PPMI) occurring in 10% to 15%, and persistent left bundle branch block (LBBB) in 20% to 25%.^{1,2} Both conditions are linked to adverse patient outcomes at long-term follow-up.^{3,4}

Several anatomic factors may contribute to post-TAVI conduction disease, including severe calcification, a short membranous septum, and a tapering left ventricular outflow tract. Procedural risk factors include self-expanding valves, deep implantation, and oversizing ($>20\%$).

Electrophysiological predictors include preexisting right bundle branch block (RBBB), postprocedural PR interval prolongation, or the emergence of any new bundle branch block. Recently, periprocedural electrophysiology studies have helped in evaluating His bundle conduction by measuring the HV interval⁵ and implementing rapid atrial pacing to assess the atrioventricular Wenckebach (AVW) cycle length.⁶ Despite numerous risk factors, a reliable predictive algorithm for PPMI post-TAVI is still lacking.

HGAVB typically occurs within 48 hours post-TAVI but occurs late (>48 hours) in 2% to 14%.^{7,8} Late HGAVB often arises after discharge and may contribute to unexplained sudden cardiac death, although its underlying mechanisms and risk factors require further investigation. In addition, new-onset atrial fibrillation (NOAF) after TAVI can increase mortality, rehospitalization, stroke rates, bleeding, and PPMI,⁹ with an incidence estimated at 10% at 1 year.⁹ As the trend shifts toward early discharge after TAVI, accurately identifying and stratifying arrhythmias is essential for improving and streamlining follow-up care.

METHODS

CONDUCT-TAVI (CONDUCT-TAVI: Prospective Observational Study on the Accuracy of Predictors of Permanent Pacemaker Secondary to High-Grade Atrioventricular Conduction Block After TAVI) (REGISTRATION: URL: <https://www.australianclinicaltrials.gov.au/>; Unique identifier: ACTRN12621001700820) is an Australian prospective observational study, and the rationale and design have been previously described.¹⁰ The study was approved by the local ethics committee, all participants provided informed consent, and amendments to the protocol were submitted to the committee before implementation (notably, a reduction in follow-up length from 2 years to 1). The data that support the findings of this study are available from the corresponding author upon reasonable request.

Study Population

Consecutive elective transfemoral TAVI were screened (2021–2023) during the institutional Structural Heart Team meeting and were excluded if they had a prior pacemaker, defibrillator, or prior aortic valve surgery. All study participants provided written informed consent.

Preprocedural Imaging

Before the planned TAVI, all patients underwent routine transthoracic echocardiography, and ECG-gated multidetector computed tomography (CT-TAVI)

CT-TAVI images were reviewed independently offline using specialized software (3Mensio imaging software; Pie Medical Imaging, the Netherlands). During peak ventricular systole, valve morphology (bicuspid versus tricuspid), annular and left ventricular outflow tract dimensions, and coronary cusp calcium volumes at the device landing zone were recorded. The device landing zone was defined as a 15 mm segment encompassing 10 mm above and 5 mm below the annular plane (Figure S1). Separately, the infraannular membranous septum length was obtained using a previously validated technique.¹¹ The

infraannular membranous septum length was measured by a single observer and averaged over 3 separate measurements.

TAVI Procedure Details

TAVI procedures were conducted at 2 centers. All cases were transfemoral, under conscious sedation, with ultrasound-guided vascular access. The balloon-expandable Sapien (S3/S3 Ultra, Edwards Lifesciences, Irvine) and the self-expandable Evolut (Evolut R/Pro/Pro Plus/FX; Medtronic, Dublin, Ireland) were used at the discretion of the implanter. The Cusp Overlap technique¹² for the self-expandable platform and the High Deployment Technique¹³ for the balloon-expandable platform were used. Implantation depth was measured fluoroscopically from the nadir of the noncoronary cusp in a co-planar (3-cusp or 2-cusp) view, as described in prior studies.^{11,13}

Electrophysiology Study

Targeted electrophysiology study was conducted using a dedicated multipolar catheter (BIOTRONIK, Berlin, Germany) inserted via the right femoral vein and externally connected to a mobile electrophysiology system (Figures S2 and S3). The His signal was measured by positioning the catheter at the septal aspect of the tricuspid annulus and defined as the intervening signal between the atrial and ventricular signals. The atrial-His interval was measured from the onset of the atrial signal to the earliest His signal on the His bundle recording. The HV interval was defined as the earliest His signal to the earliest ventricular signal on the surface ECG. Measurements were required to be reproducible (± 2 ms) over at least 3 cardiac cycles.

Right atrial pacing (RAP) was performed using the method described by Krishnaswamy et al,⁶ which involved pacing at heart rates from 70 to 120 bpm in 10 bpm increments until AVW block (Mobitz I) was observed (defined as progressive PR interval prolongation followed by a nonconducted paced atrial beat and a shorter return PR interval). If the patient's atrial rate was above 70 bpm, pacing was commenced at the next increment. Patients with atrial fibrillation (AF) or complete heart block (CHB) did not undergo RAP.

Continuous Rhythm Monitoring

Patients without a permanent pacing requirement underwent loop recorder implantation before discharge (BIOMONITOR III, BIOTRONIK, Berlin, Germany). Loop recorders were programmed to detect bradycardia below 40 bpm, pauses over 3 seconds, tachycardia above 180 bpm, and irregular heart rates. CardioMessenger Home Monitoring (BIOTRONIK, Berlin, Germany) was reviewed daily by the study team. All arrhythmias were co-reported by a cardiac technologist and electrophysiologist. NOAF was defined as at least 30 seconds of irregular R-R activity, without detectable organized atrial activity. Clinically relevant arrhythmias were referred to the treating cardiologist for management (eg, see Figures S4 and S5).

Follow-Up and End Points

All patients were followed up for 1 year. In exceptional circumstances, remote follow-up was conducted via telephone. Table S1 provides details on the CONDUCT-TAVI patient schedule. The primary study end point was the binary occurrence of new PPMI due to HGAVB within 1 year of TAVI. The decision to

implant a pacemaker was left to the treating team, and the results of the electrophysiology study or volumetric calcium analysis were not used to dictate management. Secondary end points included time-to-event (PPMI due to HGAVB, early (≤ 48 hours) and late (>48 hours to 1 year) PPMI, as well as new-onset persistent LBBB (≥ 24 hours), and NOAF.

Sample Size and Statistical Analysis

The sample size was determined using the Wilson interval method to calculate a 2-sided 95% CI with a specified width of 0.15. The estimated 1-sample sensitivity was 0.98, while the prevalence of the primary outcome was projected at 0.15. Based on these parameters, a required sample size of 194 was calculated. Considering a projected drop-out rate of 5%, a total enrollment sample size of 205 was proposed. These calculations were conducted with the assistance of a biostatistician using Power Analysis and Sample Size Software, version 20.0.3.

Continuous variables were presented as mean $\pm$ SD if data were normally distributed, or median with interquartile range if data was not normally distributed, while categorical variables were reported as percentages. Normality was assessed using the Shapiro-Wilk test. We used Fisher exact tests for categorical variables, and the Mann-Whitney *U* test (Wilcoxon rank-sum; nonnormal distribution) or Student *t* test (normal distribution) for continuous variables. Logistic regression assessed the relationship between variables and the primary end point. Variables with $P < 0.10$ in univariate analyses were included in multivariable models to reduce the risk of type II error, which may lead to a failure to detect a true association. Collinearity was assessed using correlation matrices, variable inflation factors, and clinical judgment, and was managed by selecting a variable from collinear groups based on a combination of the statistical strength of association, likelihood ratio, and clinical relevance or applicability. Similarly, a Kaplan-Meier and Cox regression secondary analysis was also performed using time-to-event (new PPMI secondary to HGAVB). Odds ratios (ORs), hazard ratios, and 95% CIs were calculated. Significance was set at $P < 0.05$. Analyses were performed using SPSS, version 29.0 (IBM, Armonk, NY).

RESULTS

Study Population and Clinical Characteristics

From October 2021 until December 2023, 354 consecutive elective transfemoral patients underwent TAVI at the 2 centers recruiting for this study. Of these, 143 patients were excluded, and 11 patients withdrew, with 200 patients included in the study (Figure 1). The baseline characteristics and preprocedural echocardiography parameters were similar across PPMI and non-PPMI groups and are described in Table 1.

CT-TAVI and Procedural Characteristics

Table 1 also highlights the CT and procedural characteristics of the 2 cohorts. The no PPMI group had a longer membranous septum length compared with PPMI group (3.1 versus 2.0 mm; $P = 0.003$). No differences were seen in total or differential calcium volume, or annular ellipticity.

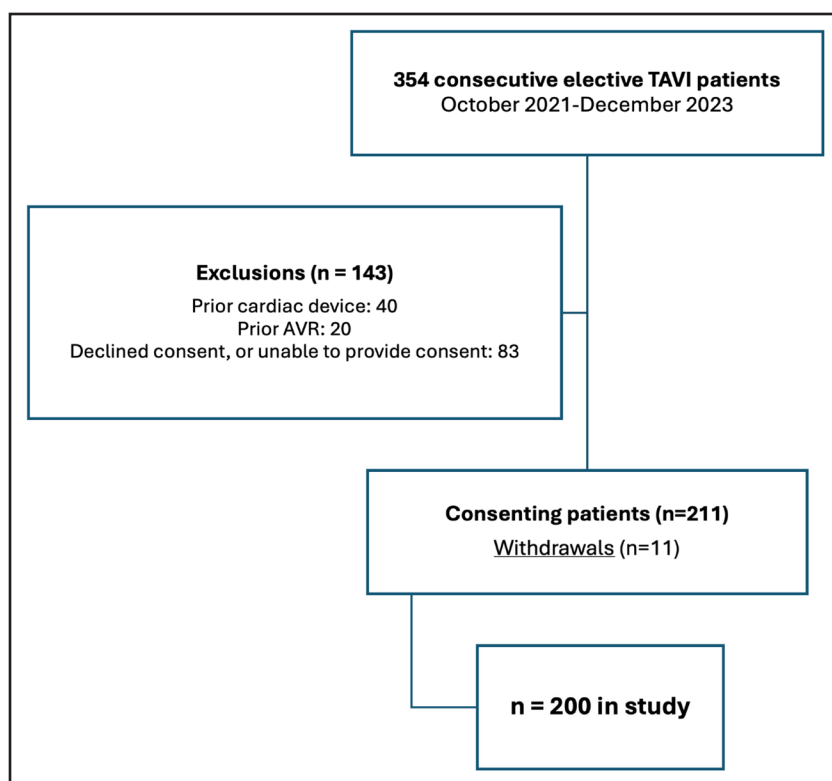


Figure 1. CONDUCT-TAVI study pathway. AVR indicates aortic valve replacement; and TAVI, transcatheter aortic valve implantation.

In the PPMI group, there was a greater proportion of self-expanding valves used (78.6 versus 57.0%, $P=0.01$; Table 1), while annular (19.7 versus 14.8%; $P<0.001$) and left ventricular outflow tract oversizing percentage (19.3 versus 14.5%; $P=0.007$) was higher. The absolute implant depth was not different, however the calculated implant depth subtracted by the membranous septum length (infraannular membranous septum length; mm) was lower in the PPMI group (-1.84 versus -0.45 mm; $P=0.004$). Transient CHB was more common in the PPMI group (54.8 versus 10.8%; $P<0.001$). There was no difference in length of stay across the groups.

ECG and Electrophysiology Study Characteristics

On baseline pre-TAVI ECG (Table 2), the PPMI cohort had longer QRS length (108.0 versus 88.0 ms; $P<0.001$), and higher proportion baseline RBBB (40.5% versus 8.8%; $P<0.001$). On immediate post-TAVI ECG (0 hour), the PPMI cohort had longer QRS duration (152.0 ms versus 132.0 ms; $P<0.001$), and greater proportion of both LBBB (52.9% versus 44.0%; $P=0.005$) and RBBB (37.0% versus 10.3%; $P=0.035$). At 4 hours post-TAVI, the PR interval (207.8 ms versus 190.2 ms; $P=0.016$) and QRS intervals (152.0 ms versus 112.0 ms; $P<0.001$) were longer in the PPMI group, with a higher proportion of RBBB (39.1% versus 9.0%; $P=0.007$). The QRS interval remained longer at 24 hours in the PPMI group

(150.0 ms versus 101.0 ms; $P<0.001$), although no other differences were observed.

All patients underwent AH and HV interval measurement, while 80.5% of the cohort underwent RAP to induce AVW (Table 2). AVW was more commonly induced in the PPMI cohort pre- (56.3% versus 21.9%; $P<0.001$), and post-TAVI (64.7% versus 24.2%; $P<0.001$). No difference was observed in AH and HV intervals, however the Δ HV interval was longer in PPMI group (14.0 ms versus 6.0 ms; $P=0.005$).

Primary Outcome and Other Clinical Outcomes

A total of 42 (21.0%) patients reached the primary outcome of new PPMI due to HGAVB (Table 3). Of these, 27 (13.5% of total cohort) were inserted ≤ 48 hours (early PPMI) while 15 (7.5% of cohort) required PPMI >48 hours (late PPMI). 6 (3.0%) patients received PPMI due to reasons unrelated to AV block (sick-sinus syndrome, or tachy-brady syndrome). Median time to PPMI was 1.5 (± 16.0) days, and the median time in the late PPMI cohort was 37 (± 87.0) days (Table 4). Freedom from PPMI secondary to HGAVB is illustrated in Figure 2, while the indications and long-term pacing requirements are reported in Table S2.

The incidence of new-onset LBBB immediately after TAVI was 43.6%, and 19.1% at 24 hours. New-onset LBBB at ≥ 30 days was 20.7%. NOAF was detected in 30 of the 138 (21.7%) patients without known history of AF (Table 3) with median time to diagnosis being

Table 1. Baseline Demographics, Medications, Bloods, Imaging, and Procedural Results

	No PPMI (n=158)	PPMI (n=42)	P value
Demographics			
Age, y; median (IQR)	83.0 (10.0)	81.0 (12.0)	0.823
Male, n (%)	98 (62.0)	32 (67.2)	0.070
BMI, kg/m ² ; median (IQR)	27.4 (7.5)	28.4 (7.2)	0.085
STS score, %; median (IQR)	3.0 (2.9)	1.6 (2.5)	0.428
Hypertension, n (%)	127 (80.4)	36 (84.5)	0.508
Hyperlipidemia, n (%)	64 (40.5)	26 (61.9)	0.860
Diabetes, n (%)	40 (25.3)	15 (35.7)	0.181
Coronary artery disease, n (%)	58 (36.7)	12 (28.6)	0.367
Prior percutaneous coronary intervention, n (%)	41 (25.9)	12 (28.6)	0.844
Prior coronary artery bypass grafting, n (%)	17 (10.8)	4 (9.5)	0.999
Peripheral arterial disease, n (%)	9 (5.7)	3 (7.1)	0.719
Prior atrial fibrillation, n (%)	48 (30.4)	14 (33.3)	0.711
Prior stroke, n (%)	15 (9.5)	2 (4.8)	0.534
Chronic obstructive pulmonary disease, n (%)	16 (10.1)	5 (11.9)	0.778
Chronic kidney disease, n (%)	43 (27.2)	13 (31.0)	0.700
Medications			
β-Blocker, n (%)	60(38.0)	13 (30.1)	0.473
Calcium-channel blocker, n (%) (nondihydropyridine)	9 (5.7)	3 (7.1)	0.718
Digoxin, n (%)	9 (5.7)	2 (4.8)	0.99
Other antiarrhythmic, n (%)	9 (5.7)	3 (7.1)	0.99
Anticoagulation, n (%)	32 (20.3)	9 (21.4)	0.833
Aspirin, n (%)	72 (45.6)	16 (38.1)	0.451
Bloods			
Hemoglobin, g/L; mean (SD)	126.8±16.5	128.1±18.6	0.343
Creatinine, μmol/L; median (IQR)	89.5 (36.5)	90.0 (47.5)	0.119
eGFR, mL/min per 1.73 m ² ; median (IQR)	62.0 (31.0)	67.0 (30.5)	0.323
Echocardiography			
Mean gradient, mm Hg; median (IQR)	42.0 (17.3)	43.0 (14.0)	0.654
Aortic valve area, cm ² ; median (IQR)	0.8 (0.3)	0.8 (0.3)	0.362
LVEF, %; median (IQR)	60.0 (5.0)	60.0 (7.5)	0.192
CT-TAVI			
Bicuspid	11 (7.0)	3 (9.4)	0.679
MSL, mm; mean (SD)	3.1±2.3	2.0±2.2	0.003
Annulus area, mm ² ; median (IQR)	466.1 (114.1)	517.1 (145.1)	0.099
Annulus perimeter, mm; mean (SD)	77.9±8.4	80.0±7.5	0.069
Ellipticity index, mean (SD)	0.2±0.06	0.2±0.06	0.466

(Continued)

Table 1. Continued

	No PPMI (n=158)	PPMI (n=42)	P value
Aortic take-off angle (degrees), median (IQR)	50.0 (11.0)	50.0 (13.0)	0.275
DLZ calcium, mm³, median (IQR)			
Total	705.0 (222.0)	762.5 (730.0)	0.862
NCC	279.0 (300.0)	402.5 (388.0)	0.224
RCC	185.0 (255.0)	203.0 (218.0)	0.602
LCC	188.0 (222.0)	174.5 (201.0)	0.285
LVOT calcium, mm³; median (IQR)	9.0 (58.0)	15.0 (34.0)	0.309
Procedural			
Valve type, n (%)			0.011*
Balloon-expandable	68 (43.0)	9 (21.4)	
Self-expandable	90 (57.0)	33 (78.6)	
Valve model, n (%)			0.063*
S3	21 (13.3)	1 (2.4)	
S3 Ultra	47 (29.7)	8 (19.0)	
Evolut R	73 (46.2)	28 (66.7)	
Evolut Pro	8 (5.1)	1 (2.4)	
Evolut Pro Plus	4 (2.5)	3 (7.1)	
Evolut FX	5 (3.2)	1 (2.4)	
Valve size, mm; n (%)			0.002*
20	3 (1.9)	1 (2.4)	
23	23 (14.6)	1 (2.4)	
26	57 (36.1)	8 (19.1)	
29	48 (30.4)	14 (33.3)	
34	27 (17.1)	18 (42.9)	
Annular oversizing (%), mean, SD	14.8±9.2	19.7±8.1	<0.001
LVOT oversizing (%), mean, SD	14.5±12.1	19.3±10.6	0.007*
TAVI guidewire, n (%)			0.005*
Safari	77 (48.7)	10 (23.8)	
Lunderquist	81 (51.3)	32 (76.2)	
Predilatation, n (%)	27 (17.1)	10 (23.8)	0.267
Postdilatation, n (%)	6 (3.8)	1 (2.4)	0.657
Implant depth, mm; mean (SD)	3.54±1.95	3.77±2.14	0.267
MSL-ID, mm; mean, SD	−0.45±2.88	−1.84±3.1	0.005*
Transient CHB in TAVI, n (%)	17 (10.8)	23 (54.8)	<0.001*
Length of stay, d; median (IQR)	3.0 (1.0)	3.0(4.0)	0.400

*Indicates statistical significance.

BMI indicates body mass index; CHB, complete heart block; CT, computed tomography; DLZ, device landing zone; eGFR, estimated glomerular filtration rate; IQR, interquartile range; LCC, left coronary cusp; LVEF, left ventricular ejection fraction; LVOT, left ventricular outflow tract; MSL, membranous septum length; MSL-ID, membranous septum length-implant depth; PPMI, permanent pacemaker implantation; NCC, noncoronary cusp; RCC, right coronary cusp; STS, Society of Thoracic Surgeons, and TAVI, transcatheter aortic valve implantation.

20.5 (±68.0) days. As explored in Table 4, among participants with NOAF, 28 individuals (93.3%) were initiated on anticoagulation therapy. The status of AF varied, with 4 (13.3%) remaining in persistent AF at

Table 2. Pre- and Post-TAVI Electrophysiology Characteristics

	No PPMI (n=158)	PPMI (n=42)	P value
12-lead ECG			
Baseline ECG			
Heart rate, ms; mean	71.6±12.7	69.6±12.3	0.184
Rhythm, n (%)			0.305
Sinus rhythm	89 (56.3)	25 (59.5)	
First-degree AVB	42 (26.6)	7 (16.7)	
High-degree AVB	0 (0.0)	0 (0.0)	
AF or flutter	27 (16.1)	10 (23.8)	
PR, ms; median (IQR)	180.0 (47.0)	180.0 (45.5)	0.870
QRS, ms; median (IQR)	88.0 (24.0)	108.0 (63.0)	<0.001*
LBBB, n (%)	9 (5.7)	3 (7.1)	0.718
RBBB, n (%)	14 (8.8)	17 (40.5)	<0.001*
Immediately post-TAVI			
Heart rate, ms; median (IQR)	78.0(19.5)	75.0(16.5)	<0.001*
Rhythm, n (%)			<0.001*
Sinus rhythm	61 (38.6)	6 (14.3)	
First-degree AVB	59 (37.3)	10 (23.8)	
High-degree AVB	5 (3.1)	19 (45.2)	
AF or flutter	30 (19.0)	5 (11.9)	
Other	3 (1.9)	2 (4.8)	
PR, ms; median (IQR)	196.0 (52.0)	208.0 (34.0)	0.228
QRS, ms; median (IQR)	132.0 (42.0)	152.0 (26.0)	<0.001*
LBBB, n (%)	82 (52.9)	12 (44.0)	0.005*
RBBB, n (%)	16 (10.3)	10 (37.0)	0.035*
4-hour post-TAVI			
Heart rate, ms; median (IQR)	74.0 (18.0)	68.0 (21.0)	0.024*
Rhythm, n (%)			<0.001
Sinus rhythm	72 (45.9)	11 (26.2)	
First-degree AVB	53 (33.8)	9 (21.4)	
High-degree AVB	2 (1.3)	15 (35.7)	
AF or flutter	29 (18.5)	5 (11.9)	
PR, ms; mean, SD	190.2±32.7	207.8±40.5	0.016*
QRS, ms; median (IQR)	112.0 (45.0)	152.0 (34.0)	<0.001*
LBBB, n (%)	53 (34.2)	11 (39.3)	0.237
RBBB, n (%)	14 (9.0)	11 (39.1)	0.007*
Other	0 (0.0)	0 (0.0)	
24-hour post-TAVI			
Heart rate (ms), median (IQR)	70.0(16.5)	68.0(25.0)	0.033
Rhythm			<0.001*
Sinus rhythm	83 (52.5)	9 (21.4)	
First-degree AVB	46 (29.1)	8 (19.0)	
High-degree AVB	1 (0.6)	20 (47.7)	
AF or flutter	28 (17.7)	5 (11.9)	

(Continued)

Table 2. Continued

	No PPMI (n=158)	PPMI (n=42)	P value
PR, ms; median (IQR)	186.0 (43.0)	194.0 (81.0)	0.171
QRS, ms; median (IQR)	101.0 (44.0)	150.0 (54.0)	<0.001*
LBBB, n (%)	38 (24.2)	10 (38.5)	0.576
RBBB, n (%)	15 (9.5)	9 (21.4)	0.057
Electrophysiology study			
Pre-TAVI			
AH, ms; median (IQR)	104.0 (45.0)	108.5 (53.5)	0.345
HV, ms; median (IQR)	56.0 (13.5)	59.0 (17.5)	0.407
RAP performed, n (%)	129 (81.6)	32 (76.2)	0.511
Inducible AVW, n (%)	28 (21.9)	18 (56.3)	<0.001*
Post-TAVI			
AH, ms; median (IQR)	108.0 (59.5)	109.5 (59.5)	0.364
HV, ms; median (IQR)	66.0 (16.0)	77.0 (34.0)	0.075
RAP performed, n (%)	120 (75.9)	17 (41.5)	<0.001*
Inducible AVW, n (%)	29 (24.2)	11 (64.7)	0.001*
ΔAH, ms; median (IQR)	4.0 (18.0)	6.0 (36.3)	0.281
ΔHV, ms; median (IQR)	6.0 (12.0)	14.0 (19.5)	0.005*

*Indicates statistical significance.

AF indicates atrial fibrillation; AH, Atrial-His interval; AVB, atrioventricular block; AVW, atrioventricular Wenckebach; HV, His-Ventricle interval; IQR, inter-quartile range; LBBB, left bundle branch block; PPMI, permanent pacemaker implantation; RAP, rapid atrial pacing; RBBB, right bundle branch block; and TAVI, transcatheter aortic valve implantation. Significance defined as $p < 0.05$ (in boldface).

the 1-year follow-up, while the remainder (86.7%) remained in paroxysmal AF. NOAF was detected peri-procedurally (day 0) in 3 (10.0%) individuals, while 7 (23.3%) individuals were diagnosed within the first week (days 1–7). The remaining 20 (66.7%) individuals were diagnosed after the first week, continuing until the 1-year mark.

Other clinical outcomes are also represented in Table 3. A total of 15 (7.5%) patients died at 1 year, of which 5 (2.5%) were due to a cardiovascular cause. The rate of cardiovascular rehospitalization was 17.0%, and major vascular complication or bleeding was seen in 4.0%. There were 13 (6.5%) strokes (or TIA), and 3 (1.5%) cases infective endocarditis.

Logistic Regression

Table 5 lists the univariable and multivariable logistic regression of prespecified predictors of PPMI. Collinearity was managed by selecting a single variable based on statistical and clinical relevance (for further details on management of collinearity (Table S3). Preexisting RBBB (adjusted OR, 5.45 [95% CI, 1.67–17.84]; $P=0.005$), $\Delta HV > 10$ (or CHB; adjusted OR, 3.62 [95% CI, 1.23–10.67]; $P=0.020$) and pre-TAVI RAP induced AVW (adjusted OR, 3.70 [95% CI, 1.37–9.98]; $P=0.010$) were independently associated with permanent pacemaker implantation at 1 year.

Table 3. Primary Outcome and Clinical Outcomes

Clinical outcomes	Overall (n=200)
PPMI	48 (24.0)
PPMI secondary to HGAVB, n (%)	42 (21.0)
PPMI secondary to early HGAVB (<48 h)	27 (13.5)
PPMI secondary to late HGAVB (>48 h – 1 y)	15 (7.5)
PPMI secondary to tachy-brady syndrome or sick-sinus syndrome	6 (3.0)
Time to pacemaker, d; median	1.5 (±16.0)
Time to delayed (>48 h) pacemaker, d; median	37.0 (±87.0)
New onset LBBB	
New LBBB immediately post-TAVI, n (%)	82/188 (43.6)
New LBBB at 4 h, n (%)	52/188 (27.7)
New LBBB at 24 h (persistent), n (%)	36/188 (19.1)
New LBBB at 1 mo (permanent), n (%)	39/188 (20.7)
Tachyarrhythmia (1 y)	
NOAF, n (%)	30/138 (21.7)
Time to NOAF, d; median	20.5 (68.0)
Ventricular tachycardia or fibrillation, n (%)	1 (0.5)
Clinical outcomes (1 y)	
All-cause mortality, n (%)	15 (7.5)
Cardiovascular mortality, n (%)	5 (2.5)
Any prehospitalization, n (%)	60 (30.0)
Cardiovascular prehospitalization, n (%)	34 (17.0)
Major vascular complication or bleeding, n (%)	8 (4.0)
Stroke or TIA, n (%)	13 (6.5)
Endocarditis, n (%)	3 (1.5)

HGAVB indicates high-grade atrioventricular block; LBBB, left bundle branch block; NOAF, new onset atrial fibrillation; PPMI, permanent pacemaker implantation; TAVI, transcatheter aortic valve implantation; and TIA, transient ischaemic attack.

The regression coefficients (Table 5) of these independently significant variables were used for risk score development which resulted in an area under the curve of 0.802 (95% CI, 0.731–0.874; Figure S5). Subsequently, a simplified version of the model: 1 point for preexisting RBBB, 1 point for $\Delta HV > 10$ (or CHB), and 1 point for Pre-TAVI RAP induced AVW, was found to perform similarly (area under the curve, 0.794 [95% CI, 0.722–0.865]; Figure 3) and was used to form the CONDUCT-TAVI score (Figure 3). The score distribution is presented in Figure 3, and a score of 0 (38.0% of the cohort) carried a sensitivity of 95%, and negative predictive value of 98% for PPMI at 1 year. The score performed similarly across both valve types used ($P=0.307$; Figure 4).

On Cox regression analyses (secondary analyses; Table S4), preexisting RBBB (adjusted OR, 4.35 [95% CI, 1.23–14.12]; $P=0.014$) and pre-TAVI RAP induced AVW (adjusted OR, 4.51 [95% CI, 1.57–12.96]; $P=0.005$) remained independently significant on multivariable assessment. Other secondary outcomes included early and late PPMI, as well as persistent LBBB (Table S5). Preexisting RBBB remained the strongest predictor

regardless of PPMI timing. RAP induced AVW was also associated with both early and late PPMI. Membranous septum length, ΔPR interval, periprocedural CHB, self-expanding valves and oversizing percentage were associated with early but not late PPMI. Post-procedure HV prolongation was associated with only late PPMI. Otherwise, implantation depth and ΔPR interval were associated with persistent LBBB.

DISCUSSION

CONDUCT-TAVI is the first prospective study to combine demographic, anatomic, electrophysiological and procedural predictors of PPMI, coupled with continuous rhythm monitoring follow-up to 1 year. Key findings included: (1) the incidence of new PPMI resulting from HGAVB was 21.0% at 1-year, with 13.5% occurring within the first 48 hours and 7.5% after 48 hours; (2) new persistent LBBB was observed in 19.1%, and NOAF in 21.7% at 1 year post-TAVI; (3) the independent predictors of PPMI identified were preexisting RBBB, $\Delta HV > 10$ ms (or CHB), and pre-TAVI RAP induced AVW block, which formed the CONDUCT-TAVI score that had negative predictive value of 98%.

PPMI Secondary to High-Grade Atrioventricular Block

The incidence of PPMI at 1 year after TAVI has varied, with the PARTNER (balloon-expandable) and Evolut (self-expanding) low-risk cohorts reporting rates of 7.5% and 19.4%, respectively.^{14,15} Recent nonindustry sponsored mixed-valve studies, DEDICATE¹ and UK-TAVI,² reported rates of 11.8% and 14.2%, respectively. We observed a higher incidence (21.0%) which may partly reflect enhanced HGAVB detection via implanted loop recorders. We noted 7 (3.5%) patients to have transient late asymptomatic HGAVB (Table S2) treated with PPMI, in accordance with current European¹⁶ and American¹⁷ guidelines which advocate for permanent pacing (Class 1) in permanent or paroxysmal second-degree Mobitz Type II or complete atrioventricular block (HGAVB), regardless of symptom status.

Conduction system recovery after TAVI is variable and challenging to predict. A 2021 meta-analysis reported over 50% of patients were no longer pacing dependant at 1 year,¹⁸ aligning with our observation that 31% of patients had <10% ventricular pacing at extended follow-up (Table S2). Similarly, while immediate new LBBB occurred in 43.6%, it persisted at 24 hours in only 19.1%. While these findings caution against premature pacemaker implantation, HGAVB often presents transiently and therefore a low pacing percentage may not necessarily reflect a healed conduction system.

Importantly, not all post-TAVI bradyarrhythmia are caused via valve-induced injury. Historically, severe

Table 4. Patients With NOAF (n=30) and Description of Characteristics, Including Time to NOAF, AF Status at Completion of Follow-Up, Average Daily AF Burden (%), and Total AF Episodes Across Period of Follow-Up (Derived From Implantable Loop Recorder Data), and Whether Anticoagulation was Commenced Due to the Diagnosis of NOAF

Patient	Time from TAVI to NOAF (days)	AF status at completed follow-up	AF burden (average % of day)	Total AF episodes (to completed follow-up)	Anticoagulation commenced
1	14	Paroxysmal	<0.1	3	Yes
2	13	Paroxysmal	13.7	304	Yes
3	4	Paroxysmal	<0.1	3	Yes
4	24	Paroxysmal	0.1	47	Yes
5	1	Paroxysmal	0.3	2244	Yes
6	0	Paroxysmal	2.3	86	Yes
7	93	Paroxysmal	<0.1	4	Yes
8	4	Paroxysmal	1.3	19	Yes
9	80	Paroxysmal	1.0	43	Yes
10	0	Paroxysmal	15.4	11	Yes
11	154	Paroxysmal	<0.1	1	No
12	20	Paroxysmal	<0.1	3	Yes
13	25	Paroxysmal	21.1	1968	Yes
14	6	Paroxysmal	<0.1	35	Yes
15	253	Paroxysmal	<0.1	3	Yes
16	22	Persistent	100	NA	Yes
17	7	Paroxysmal	38.7	87	Yes
18	33	Paroxysmal	1.0	256	Yes
19	223	Paroxysmal	<0.1	8	Yes
20	15	Paroxysmal	<0.1	4	Yes
21	21	Paroxysmal	33.4	693	Yes
22	3	Persistent	100	NA	Yes
23	57	Persistent	100	NA	Yes
24	294	Paroxysmal	<0.1	3	Yes
25	14	Paroxysmal	1.0	33	Yes
26	7	Paroxysmal	6.1	615	Yes
27	267	Paroxysmal	2.5	98	Yes
28	0	Persistent	100	NA	Yes
29	93	Paroxysmal	0.6	5	No
30	42	Paroxysmal	6.9	133	Yes

AF indicates atrial fibrillation; NA, not applicable; NOAF, new onset atrial fibrillation; and TAVI, transcatheter aortic valve implantation.

calcific aortic stenosis has been shown to independently prolong AV conduction, secondary to calcium infiltration or fibrosis.^{19,20} Urena et al²¹ reported HGAVB occurred in 5.5% of patients monitored the day before TAVI. In our single-arm post-TAVI design, the proportion of patients with preexisting conduction disease is difficult to estimate.

RBBB is a well-known risk factor, since mechanical valve-induced injury most commonly affects the Bundle of His and left bundle branch. We documented 15.5% of our cohort had a baseline RBBB, higher than in most contemporary studies, which may have also influenced our high incidence of PPMI. Results from the Society of Thoracic Surgeons/ American College of Cardiology registry recorded a baseline RBBB rate of 10.3%,²² while

the PARTNER cohorts of balloon-expandable valves²³ and the 2023 OPTIMIZE PRO²⁴ study of self-expanding valves had rates of 8.1% and 6.9%, respectively. In baseline RBBB, PPMI has been reported in roughly 50% of cases regardless of valve type,²⁵ and we noted a similar rate. Prophylactic PPMI before TAVI in patients with baseline RBBB may shorten length of stay, decrease procedural duration, and lower cardiovascular readmission.^{26,27} With prospective validation, this may represent a safe approach for incorporating patients with baseline RBBB into expedited discharge protocols.

Incorporating RAP to induce AVW at rates between 70 and 120 bpm to specifically assess AV nodal conduction post-TAVI was first proposed by Krishnaswamy et al.⁶ Beyond validating their results, we demonstrate

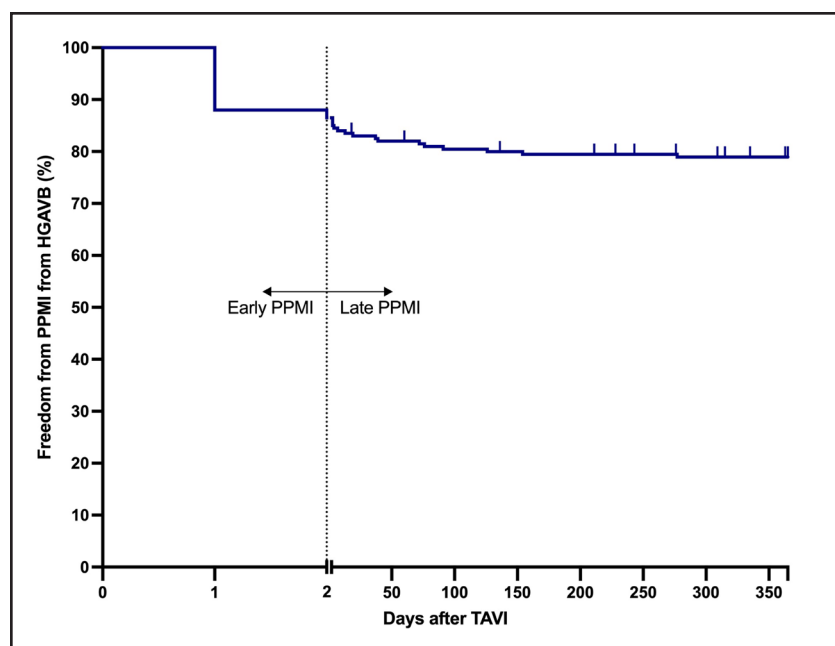


Figure 2. Freedom from permanent pacemaker implantation (PPMI) from high-grade atrioventricular block (HGAVB) at 1 year after transcatheter aortic valve implantation (TAVI).

it can be performed before TAVI and serve as an independent predictor of PPMI, even after adjusting for other variables. A drawback of RAP is its futility in CHB or AF. On the other hand, the HV interval can be assessed in AF and is also less prone to vagal and medication influence. As a sensitive marker of infra-Hisian conduction, Rivard et al¹⁵ published that $\Delta HV > 13$ ms increased PPMI risk. We observed statistical significance at a lower threshold ($\Delta H > 10$ ms) and combined it with periprocedural CHB where it was assumed the HV interval was prolonged to the point of being absent.

The CONDUCT-TAVI Score combines the 3 independent factors (baseline RBBB, $\Delta HV > 10$ ms (or CHB), or pre-TAVI RAP induced AVW) and demonstrated strong predictive accuracy (area under the curve, 0.794) with a linear correlation to PPMI risk (Figure 3). Notably, a score of 0, observed in 38% of the cohort, had a negative predictive value of 98%, which may aid implanters in identifying low-risk patients suitable for rapid discharge. Given that this score necessitates the measurement of the HV interval and rapid atrial pacing, it may initially seem limited to centers equipped to incorporate electrophysiology studies into their TAVI procedures. However, our study demonstrates that mobile electrophysiology systems provide an accessible and cost-effective alternative.

Self-expanding valves historically have had comparatively higher PPMI incidence, attributed to greater oversizing, radial force, and typically a deeper implant. However, redistribution of radial force along with the introduction of the cusp-overlap deployment technique²⁴ have reportedly reduced PPMI rates. Cusp-overlap isolates the noncoronary cusp during deployment, thereby elongating the left ventricular

outflow tract to achieve a precise high implant, which may explain why self-expanding valves did not independently predict PPMI on our multivariable analyses. Similarly, factors such as oversizing, membranous septum length, and implantation depth, which are believed to amplify the interaction between the prosthesis and conduction system, did not remain significant predictors on multivariable analyses.

There was no association found between calcium volume and distribution with PPMI, aligning with a recent meta-analysis,²⁸ which may be due to conflicting mechanisms of His Bundle injury: via direct infiltration or asymmetrical valve expansion, driven by opposing calcium distribution. Similarly, we did not find any association between annular ellipticity and conduction, which has also been proposed previously.²⁹

The comprehensive evaluation of late PPMI post-TAVI constitutes a notable strength of this study, particularly given heterogeneous prior results.⁸ Late PPMI occurred in 7.5%, with half occurring in the first month, followed by a tapering yet persistent risk up to 1 year (Figure 2). The recently published D-PACE³⁰ scoring system for delayed PPMI identified self-expanding valves, implantation depth, baseline RBBB, new bundle branch block, and PR prolongation as independent predictors in a primarily retrospective cohort. However, it did not include anatomic and electrophysiology study-derived parameters, which we have shown to be significant contributors.³⁰ Our subanalyses (Table S3) suggest that early PPMI was driven more by procedural factors (valve type, membranous septum, and oversizing), whereas late PPMI correlated with markers of impaired baseline infra-Hisian conduction, which may serve as directions for future research.

Table 5. Logistic Regression of Prespecified Variables to Predict New PPMI After TAVI

	Univariable, OR (95% CI)	P value	Multivariable, OR (95% CI)	P value
Age, y	1.01 (0.96–1.07)	0.624		
Female	0.51 (0.23–1.11)	0.091*	1.29 (0.44–3.80)	0.648
BMI	1.05 (0.98–1.12)	0.145		
STS score	0.94 (0.80–1.11)	0.475		
MSL, mm	0.80 (0.67–0.94)	0.008*		
Annular ellipticity	0.70 (0.01–214.3)	0.901		
Total DLZ calcium volume	1.00 (1.00–1.01)	0.327		
NCC LVOT calcium volume	1.01 (1.0–1.01)	0.379		
Pre-TAVI PR	1.00 (0.99–1.01)	0.574		
ΔPR (pre/post-TAVI)	1.03 (1.01–1.05)	0.013*		
RBBB	6.99 (3.07–15.96)	<0.001*	5.45 (1.67–17.84) β: 1.696*	0.005*
LBBB	1.27 (0.33–4.92)	0.726		
Preexisting AF	1.15 (0.56–2.37)	0.713		
Pre-TAVI HV interval	1.02 (0.99–1.05)	0.241		
Post-TAVI HV interval	1.04 (1.01–1.08)	0.015*		
ΔHV >10 (or CHB)	5.11 (2.29–11.40)	<0.001*	3.62 (1.23–10.67) β: 1.308*	0.020*
AVW Pre	4.59 (2.03–10.37)	<0.001*	3.70 (1.37–9.98) β: 1.287*	0.010*
AVW Post	5.75 (1.96–16.92)	0.001*		
Self-expanding valve	2.77 (1.24–6.18)	0.013*	2.05 (0.40–10.66)	0.392
Annular oversizing %	1.07 (1.03–1.12)	0.002*	1.03 (0.95–1.13)	0.481
LVOT oversizing %	1.04 (1.01–1.07)	0.025*		
Implant depth	1.06 (0.89–1.27)	0.508		
Implant depth-MSL	0.85 (0.75–0.97)	0.013*	0.85 (0.71–1.01)	0.070
Pre- or postdilatation	1.28 (0.57–2.88)	0.551		
New persistent LBBB (24 h)	0.71 (0.28–1.84)	0.482		

*Indicates statistical significance.

AF indicates atrial fibrillation; AVW, atrioventricular Wenckebach; β, beta coefficient; BMI, body mass index; CHB, complete heart block; DLZ, device landing zone; HV, his-ventricle interval; LBBB, left bundle branch block; LVOT, left ventricular outflow tract; MSL, membranous septum length; NCC, noncoronary cusp; OR, odds ratio; PPMI, permanent pacemaker implantation; RBBB, right bundle branch block; STS, Society of Thoracic Surgeons Risk Score; and TAVI, transcatheter aortic valve implantation.

New-Onset Atrial Fibrillation

Without continuous rhythm monitoring, major contemporary randomized trials have reported NOAF incidence between 7.0% and 9.8% at 1 year,^{14,15} while a recently published study used Holter monitoring to report an incidence of 7.0% at 14 days post-TAVI.³¹ This is the first study to use loop recorder implantations to document the incidence of NOAF at 1 year after TAVI is 21.7%. The specific role of TAVI in triggering AF remains uncertain, as both age and aortic stenosis related myocardial fibrosis, diastolic impairment and atrial enlargement predispose to AF. Prior work in patients with heart failure reported subclinical device-detected AF in 23% at 4 years.³² We observed 46.5% patients either had a previous history of AF or developed NOAF within 1-year post-TAVI. Whether our findings merely indicate a heightened incidence of

device-detected AF within the high-risk aortic stenosis population, or whether electrical and anatomic remodeling post-TAVI directly contributes, could be a direction of future work.

However, the management implications of device-detected NOAF remain unclear. The LOOP study³³ which randomized patients with cryptogenic stroke to loop recorder implantations, reported increased AF detection and anticoagulation, but without a reduction in stroke risk.³³ Contrastingly, a 2015 study monitored patients for 24 hours before their TAVI, detecting NOAF in 6.4%, which was associated with a 7-fold increase in 30-day stroke risk. Among the 13 patients in our cohort with ischemic stroke or TIA, 5 had known AF, while 3 were identified with NOAF. The thromboembolic risk may differ in TAVI populations, and prior studies conducted in stroke cohorts may not be generalizable. Currently, major

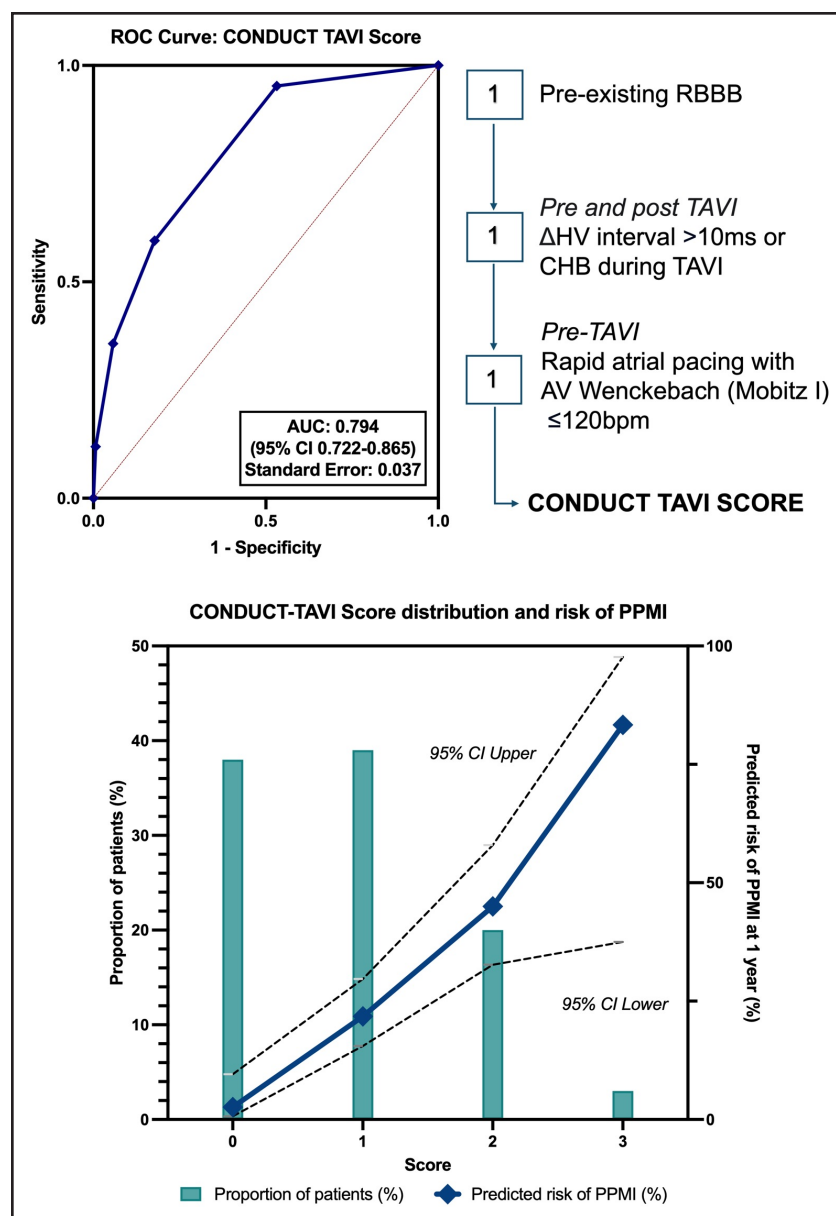


Figure 3. Receiver operator characteristic (ROC) curve CONDUCT-TAVI score (top); CONDUCT-TAVI score distribution across cohort (bar graph, left axis), and respective predicted risk of permanent pacemaker implantation (PPMI) at 1 year (line graph, right axis) with 95% CIs (dotted line).

AUC indicates area under the curve; CHB, complete heart block; HV, his-ventricle; and RBBB, right bundle branch block.

European and American guidelines continue to recommend anticoagulation for patients with a high thromboembolic risk and device-detected subclinical AF episodes lasting longer than 5 minutes.

Limitations

The nonblinded observational design of the study may have introduced potential bias. The primary outcome (PPMI due to HGAVB) was determined at the discretion of the implanter or treating cardiologist, rather than by the study protocol. While the 2 most common valve systems were used, the results should not be generalized to other commercially available valve platforms. Furthermore, implantation depth was estimated based on fluoroscopic projections, and variability in 2-cusp and 3-cusp

views may have introduced minor measurement inconsistencies. Finally, as an observational cohort design, the study used a sensitivity-based power calculation rather than being specifically designed for predictive modelling. Consequently, the CONDUCT-TAVI score, which was developed and evaluated within this population, would benefit from external validation to enhance the model's credibility.

Conclusions

This prospective study of 200 patients undergoing elective TAVI, combined with 1 year of continuous rhythm monitoring, suggests that the true incidence of HGAVB, new persistent LBBB and NOAF may be higher than previously recognized. Furthermore, after accounting for

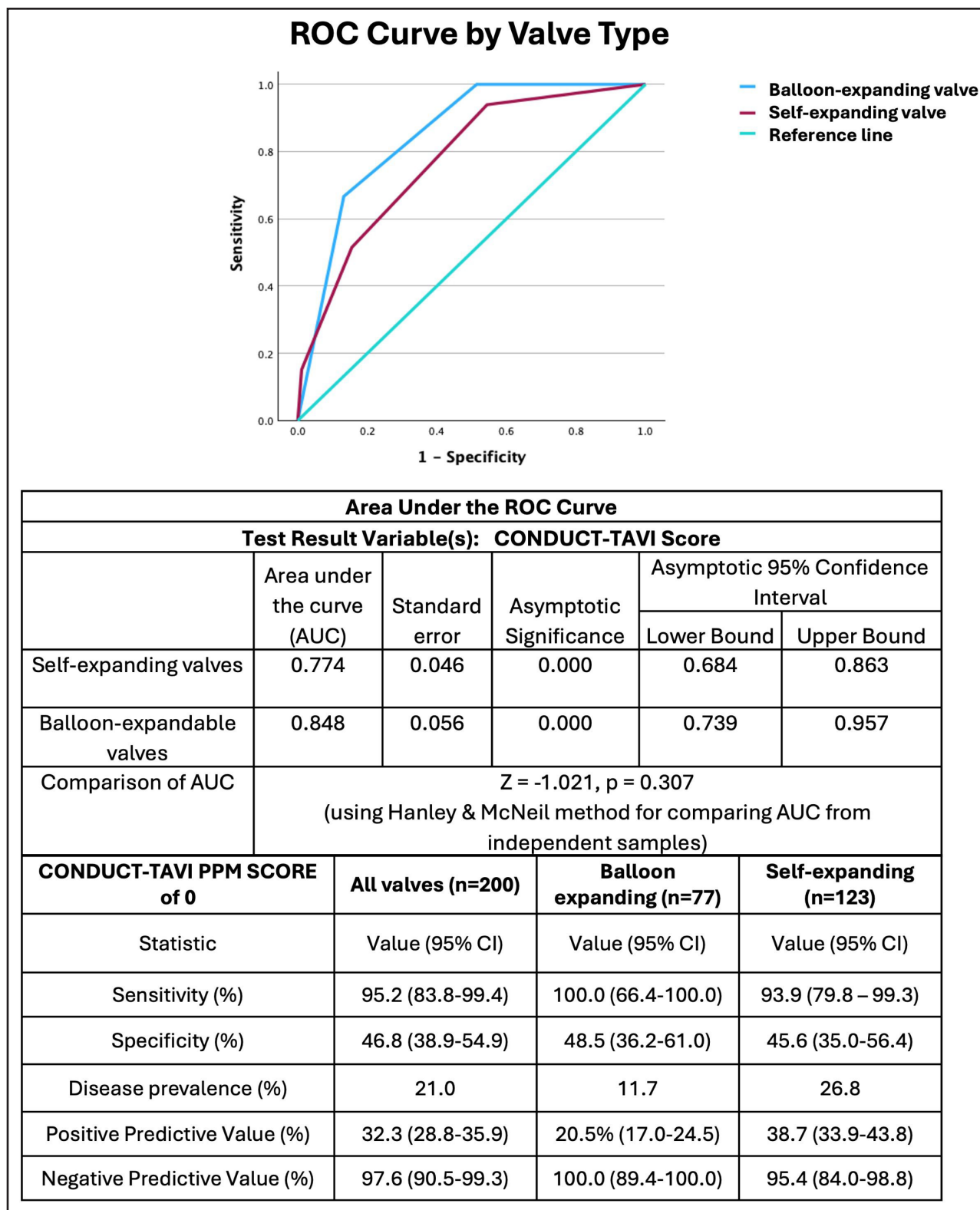


Figure 4. Performance of the CONDUCT-TAVI score when arranged by valve type.

AUC indicates area under the curve; CI, confidence interval; PPM, permanent pacemaker; and ROC, received operator characteristic.

demographic, anatomic, electrophysiological and procedural factors, the key independent predictors of PPMI risk were found to be preexisting RBBB and impaired AV nodal and infra-Hisian conduction. These were used to develop

the CONDUCT-TAVI Score, which demonstrated a negative predictive value of 98% within this cohort. Incorporating these findings into clinical practice can enable implanters to more effectively stratify patients for early discharge.

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Disclosures

None.

Supplemental Material

Supplementary Methods

Tables S1–S6

Figures S1–S5

REFERENCES

- Blankenberg S, Seiffert M, Vonthein R, Baumgartner H, Bleiziffer S, Borger MA, Choi YH, Clemmensen P, Cremer J, Czerny M, et al; DEDICATE-DZHK6 Trial Investigators. Transcatheter or surgical treatment of aortic-valve stenosis. *N Engl J Med*. 2024;390:1572–1583. doi: 10.1056/NEJMoa2400685
- Investigators TUTT. Effect of transcatheter aortic valve implantation vs surgical aortic valve replacement on all-cause mortality in patients with aortic stenosis: a randomized clinical trial. *JAMA*. 2022;327:1875–1887. doi: 10.1001/jama.2022.5776
- Zito A, Princi G, Lombardi M, D'Amario D, Vergallo R, Aurigemma C, Romagnoli E, Pelargonio G, Bruno P, Trani C, et al. Long-term clinical impact of permanent pacemaker implantation in patients undergoing transcatheter aortic valve implantation: a systematic review and meta-analysis. *Europace*. 2022;24:1127–1136. doi: 10.1093/europace/euac008
- Rao K, Ahmed M, Brieger D, Baer A, Hansen P, Bhindi R. The prognostic relevance of a new bundle branch block after transcatheter aortic valve implantation: a systematic review and meta-analysis. *Struct Heart*. 2024;9:100392. doi: 10.1016/j.shj.2024.100392
- Rivard L, Schram G, Asgar A, Khairy P, Andrade JG, Bonan R, Dubuc M, Guerra PG, Ibrahim R, Macle L, et al. Electrocardiographic and electrophysiological predictors of atrioventricular block after transcatheter aortic valve replacement. *Heart Rhythm*. 2015;12:321–329. doi: 10.1016/j.hrthm.2014.10.023
- Krishnaswamy A, Sammour Y, Mangieri A, Kadri A, Karrthik A, Banerjee K, Kaur M, Giannini F, Pagliaro B, Ancona M, et al. The utility of rapid atrial pacing immediately post-TAVR to predict the need for pacemaker implantation. *JACC Cardiovasc Interv*. 2020;13:1046–1054. doi: 10.1016/j.jcin.2020.01.215
- Reiter C, Lambert T, Kellermair J, Blessberger H, Fellner A, Nahler A, Grund M, Steinwender C. Delayed total atrioventricular block after transcatheter aortic valve replacement assessed by implantable loop recorders. *JACC Cardiovasc Interv*. 2021;14:2723–2732. doi: 10.1016/j.jcin.2021.09.003
- Rao K, Chan B, Baer A, Hansen P, Bhindi R. A systematic review of delayed high-grade atrioventricular block after transcatheter aortic valve implantation. *CJC Open*. 2024;6:86–95. doi: 10.1016/j.cjco.2023.10.003
- Ryan T, Grindal A, Jinah R, Um KJ, Vadakken ME, Pandey A, Jaffer IH, Healey JS, Belley-Coté EF, McIntyre WF. New-onset atrial fibrillation after transcatheter aortic valve replacement. *JACC Cardiovasc Interv*. 2022;15:603–613. doi: 10.1016/j.jcin.2022.01.018
- Rao K, Bhatia K, Chan B, Cowan M, Saad N, Baer A, Sriharan H, Bromhead I, Whalley D, Allahwala UK, et al. Prospective observational study on the accuracy of predictors of high-grade atrioventricular conduction block after transcatheter aortic valve implantation (CONDUCT-TAVI): study protocol, background and significance. *BMJ Open*. 2023;13:e070219. doi: 10.1136/bmjopen-2022-070219
- Jilalawi H, Zhao Z, Du R, Staniloae C, Saric M, Neuburger PJ, Querijero M, Vainrib A, Hisamoto K, Ibrahim H, et al. Minimizing permanent pacemaker following repositionable self-expanding transcatheter aortic valve replacement. *JACC Cardiovasc Interv*. 2019;12:1796–1807. doi: 10.1016/j.jcin.2019.05.056
- Tang GHL, Zaid S, Michev I, Ahmad H, Kaple R, Undemir C, Cohen M, Lansman SL. “Cusp-overlap” view simplifies fluoroscopy-guided implantation of self-expanding valve in transcatheter aortic valve replacement. *JACC Cardiovasc Interv*. 2018;11:1663–1665. doi: 10.1016/j.jcin.2018.03.018
- Sammour Y, Banerjee K, Kumar A, Lak H, Chawla S, Incognito C, Patel J, Kaur M, Abdelfattah O, Svensson LG, et al. Systematic approach to high implantation of SAPIEN-3 valve achieves a lower rate of conduction abnormalities including pacemaker implantation. *Circ Cardiovasc Interv*. 2021;14:e009407. doi: 10.1161/CIRCINTERVENTIONS.120.009407
- Mack MJ, Leon MB, Thourani VH, Makkar R, Kodali SK, Russo M, Kapadia SR, Malaisrie SC, Cohen DJ, Pibarot P, et al; PARTNER 3 Investigators. Transcatheter aortic-valve replacement with a balloon-expandable valve in low-risk patients. *N Engl J Med*. 2019;380:1695–1705. doi: 10.1056/NEJMoa1814052
- Popma JJ, Deeb GM, Yakubov SJ, Mumtaz M, Gada H, O'Hair D, Bajwa T, Heiser JC, Merhi W, Kleiman NS, et al; Evolut Low Risk Trial Investigators. Transcatheter aortic-valve replacement with a self-expanding valve in low-risk patients. *N Engl J Med*. 2019;380:1706–1715. doi: 10.1056/NEJMoa1816885
- Glikson M, Nielsen JC, Kronborg MB, Michowitz Y, Auricchio A, Barbash IM, Barrabés JA, Boriani G, Braunschweig F, Brignole M, et al; ESC Scientific Document Group. 2021 ESC guidelines on cardiac pacing and cardiac resynchronization therapy: developed by the Task Force on Cardiac Pacing and Cardiac Resynchronization Therapy of the European Society of Cardiology (ESC) with the special contribution of the European Heart Rhythm Association (EHRA). *Eur Heart J*. 2021;42:3427–3520. doi: 10.1093/eurheartj/ehab364
- Kusumoto FM, Schoenfeld MH, Barrett C, Edgerton JR, Ellenbogen KA, Gold MR, Goldschlager NF, Hamilton RM, Joglar JA, Kim RJ, 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. *Circulation*. 2019;140:e333–e381. doi: 10.1161/CIR.0000000000000627
- Ravaux JM, Di Mauro M, Vernooij K, Van't Hof AW, Veenstra L, Kats S, Maessen JG, Lorusso R. One-year pacing dependency after pacemaker implantation in patients undergoing transcatheter aortic valve implantation: systematic review and meta-analysis. *JTCVS Open*. 2021;6:41–55.e15. doi: 10.1016/j.jxjon.2021.02.002
- Yater WM. Heart block due to calcareous lesions of the bundle of His. *Ann Intern Med*. 1935;8:777–789. doi: 10.7326/0003-4819-8-7-777
- Dry TJ, Willis FA. Calcareous disease of the aortic valve: a study of two hundred twenty-eight cases. *Am Heart J*. 1939;17:138–157. doi: 10.1016/s0002-8703(39)90486-2
- Urena M, Hayek S, Cheema AN, Serra V, Amat-Santos IJ, Nombela-Franco L, Ribeiro HB, Allende R, Paradis JM, Dumont E, et al. Arrhythmia burden in elderly patients with severe aortic stenosis as determined by continuous electrocardiographic recording: toward a better understanding of arrhythmic events after transcatheter aortic valve replacement. *Circulation*. 2015;131:469–477. doi: 10.1161/CIRCULATIONAHA.114.011929
- Auffret V, Webb JG, Eltchaninoff H, Muñoz-García AJ, Himbert D, Tamburino C, Nombela-Franco L, Nietlispach F, Moris C, Ruel M, et al. Clinical impact of baseline right bundle branch block in patients undergoing transcatheter aortic valve replacement. *JACC Cardiovasc Interv*. 2017;10:1564–1574. doi: 10.1016/j.jcin.2017.05.030
- Chen S, Dizon Jose M, Hahn Rebecca T, Pibarot P, George I, Zhao Y, Blanke P, Kapadia S, Babaliaros V, Szeto Wilson Y, et al. Predictors and 5-year clinical outcomes of pacemaker after TAVR. *JACC Cardiovasc Interv*. 2024;17:1325–1336. doi: 10.1016/j.jcin.2024.03.034
- Grubb Kendra J, Gada H, Mittal S, Nazif T, Rodés-Cabau J, Fraser Douglas GW, Lin L, Rovin Joshua D, Khalil R, Sultan I, et al. Clinical impact of standardized TAVR technique and care pathway. *JACC Cardiovasc Interv*. 2023;16:558–570. doi: 10.1016/j.jcin.2023.01.016

25. Weferling M, Liebetrau C, Renker M, Fischer-Rasokat U, Choi YH, Hamm CW, Kim WK. Right bundle branch block is not associated with worse short- and mid-term outcome after transcatheter aortic valve implantation. *PLoS One*. 2021;16:e0253332. doi: 10.1371/journal.pone.0253332
26. Fukutomi M, Hokken T, Wong I, Bieliauskas G, Daemen J, de Jaegere P, Van Mieghem N, Søndergaard L, De Backer O. Prophylactic permanent pacemaker strategy in patients with right bundle branch block undergoing transcatheter aortic valve replacement. *Catheter Cardiovasc Interv*. 2021;98:E1017–E1025. doi: 10.1002/ccd.29914
27. Zorman M, Bamford P, Coronelli M, Barnes C, Saunderson C, Gamble J, Dawkins S, Kharbanda RK, Newton J, Banning AP, et al. Prophylactic permanent pacemaker implantation for baseline right bundle branch block in patients undergoing transcatheter aortic valve replacement: clinical efficacy, safety, and long-term pacing requirement. *Struct Heart*. 2024;8:100326. doi: 10.1016/j.shj.2024.100326
28. Litkouhi PN, Rao K, Baer A, Hansen PS, Bhindi R. Aortic valve and left ventricular outflow tract calcium distribution and conduction outcomes after transcatheter aortic valve replacement: a systematic review and meta-analysis. *Struct Heart*. 2024;9:100389. doi: 10.1016/j.shj.2024.100389
29. Bianchini F, Bianchini E, Romagnoli E, Aurigemma C, Zito A, Busco M, Nesta M, Bruno P, Laezza D, Giambusso N, et al. Anatomical annulus predictors of new permanent pacemaker implantation risk after balloon-expandable transcatheter aortic valve implantation. *Am J Cardiol*. 2024;224:26–35. doi: 10.1016/j.amjcard.2024.05.034
30. Bendandi F, Taglieri N, Ciurlanti L, Mazzapicchi A, Foroni M, Lombardi L, Palermo F, Filice F, Ghetti G, Bruno AG, et al. Development and validation of the D-PACE scoring system to predict delayed high-grade conduction disturbances after transcatheter aortic valve implantation. *EuroIntervention*. 2025;21:e119–e129. doi: 10.4244/EIJ-D-24-00850
31. Nuche J, Soliman F, Chavarria J, Okoh AK, Alvarado Mora H, Nault I, Natarajan MK, Russo M, Philippon F, Rodés-Cabau J. New-onset atrial fibrillation detected by ambulatory ECG monitoring after transcatheter aortic valve implantation. *EuroIntervention*. 2024;20:591–601. doi: 10.4244/EIJ-D-23-01014
32. Shahzeb Khan M, Kahwash R, Zile MR, Sarkar S, Van Dorn B, Gerritse B, Butler J. Incidence of atrial fibrillation detected by an insertable cardiac monitor in a large real-world cohort of heart failure patients with reduced and preserved ejection fraction. *Eur Heart J*. 2024;45:ehae666.494. doi: 10.1093/eurheartj/ehae666.494
33. Svendsen JH, Diederichsen SZ, Højberg S, Krieger DW, Graff C, Kronborg C, Olesen MS, Nielsen JB, Holst AG, Brandes A, et al. Implantable loop recorder detection of atrial fibrillation to prevent stroke (The LOOP study): a randomised controlled trial. *Lancet*. 2021;398:1507–1516. doi: 10.1016/S0140-6736(21)01698-6