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Mechanisms Underlying Alterations in Cardiac Conduction After Transcatheter Aortic Valve Replacement

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IMPORTANCE Transcatheter aortic valve replacement (TAVR) to treat aortic stenosis is complicated by heart block requiring permanent pacemaker implantation in at least 10% of cases.

OBJECTIVES To better understand mechanisms underlying heart block complicating TAVR and improve prediction of intraprocedural and delayed heart block.

DESIGN, SETTING, AND PARTICIPANTS This cohort study was conducted at a single academic medical center in Boston, Massachusetts, from May 2021 to January 2024 among all patients undergoing TAVR, except those with preexisting pacemakers. A total of 409 consecutive patients undergoing TAVR were prospectively studied. An electrophysiologic study was performed at the beginning and end of the TAVR procedure. An electrophysiologist monitored the electrocardiogram (ECG) and His bundle recording continuously during the procedure. Patients were followed up for 1 year. Occurrence of high-grade atrioventricular (AV) block was related to ECG and electrophysiological, anatomic, and procedural variables. Data analysis was performed from March 2023 to May 2025.

EXPOSURES An electrophysiologist monitored the ECG and intracardiac electrograms continuously during the valve implant; patients with preexisting right bundle-branch block (RBBB) or periprocedural conduction abnormalities were discharged with an ECG monitor.

MAIN OUTCOME AND MEASURES The primary outcome was Mobitz type II or complete heart block.

RESULTS A total of 409 consecutive patients were enrolled, among whom median (IQR) age was 78.5 (73.1-83.5) years and 182 patients (44.5%) were female. Forty patients (9.7%) developed heart block requiring permanent pacemakers: block developed during the TAVR procedure in 15 patients and after TAVR in 25. Block was persistent in all patients developing block during the TAVR but paroxysmal in 20 of 25 patients with post-TAVR block. Block localized to the AV node during TAVR in 6 cases (all resolved) and in 3 patients (7.5%) with delayed block. In the remaining 9 patients that developed intraprocedural block and 22 patients developing postprocedural block, the block was infranodal. Preexisting RBBB was the only ECG or electrophysiological predictor for intraprocedural block, but preexisting RBBB did not predict postprocedural block. The best predictors of delayed heart block were His-ventricular interval of 80 milliseconds or longer at the end of the implant procedure, PR interval longer than 300 milliseconds, and AV Wenckebach cycle length of 500 milliseconds or longer post-TAVR.

CONCLUSIONS AND RELEVANCE In this cohort study, the characteristics and mechanisms causing AV block during TAVR differed from delayed block. Both AV nodal and infranodal block contributed to heart block accompanying TAVR procedures.

Editor's Note page 34

Multimedia

Supplemental content

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Therapy of aortic stenosis has changed radically since the advent of transcatheter aortic valve replacement (TAVR) as an alternative to surgery. While TAVR is highly effective and safe, it is complicated by heart block in 10% or more of cases. This risk has remained fairly constant in spite of increasing case volume.[1-3]

While pacing is indicated in patients with persistent heart block, management of patients with prolongation of the PR or His-ventricular (HV) intervals, transient heart block, or new bundle-branch block during the procedure is unsettled. Published studies have evaluated the contribution of electrocardiographic (ECG) and intracardiac atrioventricular (AV) conduction measures, as well as anatomic and procedural variables. However, reported studies are limited by relatively small size and inconsistent end points. Most observers believe AV block is due to impingement on the His bundle.[4] However, the contribution of the AV node to AV block remains unclear.

The purposes of this study were to better delineate the mechanisms and prognosis attending conduction abnormalities in patients undergoing TAVR procedures. We sought to reevaluate the prognostic utility of intracardiac measurement of AV conduction during TAVR procedures.

Key Points

Question What are the site(s) of heart block in patients undergoing transcatheter aortic valve replacement (TAVR), and what are markers of risk for heart block?

Findings In this cohort study of 409 patients undergoing TAVR, His bundle studies identified block in both the atrioventricular (AV) node and His bundle and were predictive of delayed block. These observations suggest mechanisms underlying intraprocedural block differ from those causing postprocedural block.

Meaning Differing prediction models are necessary to identify patients at risk for heart block occurring during vs following TAVR implants.

Methods

We prospectively studied consecutive patients undergoing TAVR at Beth Israel Deaconess Medical Center in Boston, Massachusetts. Patients with preexisting pacemakers or implantable cardioverter-defibrillators were excluded. One patient was excluded when their TAVR procedure was aborted after coronary angiography at the beginning of the procedure revealed the need for surgical revascularization. A second patient was excluded when the ECG and intracardiac recordings performed at the beginning of the TAVR revealed previously unrecognized 2:1 AV conduction block. Two additional patients refused participation, and 3 could not be enrolled because of language barriers. The study was approved by the Beth Israel Deaconess Medical Center Institutional Review Board, and written informed consent was obtained from all patients. Race and ethnicity were defined by study participants and were assessed to determine if they affected outcomes.

Patients underwent TAVR with either a balloon-expandable valve (SAPIEN 3 Ultra or RESILIA valves [Edwards LifeSciences]) or a self-expandable valve (Evolut PRO+ valve [Medtronic]) after review by the heart team.

A 12-lead ECG was recorded before and after the TAVR procedure, then daily until discharge. An electrophysiologic study (EPS) was performed immediately before and after the valve replacement.[5] Intracardiac electrical recording and stimulation were performed using a portable system with 10 bipolar channels (EP-Tracer [Schwarzer Cardiotek]). Intracardiac bipolar signals were filtered at 40 to 500 Hz. ECG leads I, II, III and V1 and intracardiac electrograms were monitored by the electrophysiologist. Various quadripolar and hexapolar catheters were used in the first 26 cases. We then used 6F steerable octapolar or decapolar catheters (Abbott Inquiry [Abbott Medical]). The catheter was inserted and manipulated by the structural heart team cardiologists or cardiac surgeons as previously described.[5] The catheter was inserted through a femoral vein and initially advanced to the lateral right atrium. We performed incremental atrial pacing to AV Wenckebach, or a minimal paced cycle length of 350 milliseconds. The catheter was then advanced to the tricuspid valve annulus to record the most proximal His bundle electrogram (HBE) possible. Stable, reproducible atrio-His (AH) and HV intervals were recorded. The catheter was then advanced to the mid-right ventricular septum. Retrograde (VA) conduction was assessed at paced cycle length of 600 milliseconds. This catheter was used to perform rapid pacing during valve deployment. We attempted to record ventricular electrograms with the distal poles and a continuous His bundle recording with the proximal poles, but this was not always possible (stability of the catheter for right ventricular [RV] pacing was prioritized over the His recording). The electrophysiologist monitored the ECG, catheter position, and intracardiac electrograms continuously throughout the procedure. After valve deployment, EPS was repeated: VA conduction, recording HBE, and determination of AV Wenckebach cycle length.

Patients that developed persistent high-grade heart block during the procedure had either a temporary pacing catheter placed via the internal jugular vein or a permanent pacemaker placed following the TAVR procedure, per standard institutional protocol. EPS observations did not influence the determination to implant permanent pacemakers.

Patients were monitored on telemetry after the TAVR. If no new conduction disturbance occurred during the TAVR procedure, patients were discharged 1 day after implantation. Patients with new conduction disturbances were observed at least 1 additional day, until AV conduction was stable. Patients with right bundle-branch block (RBBB) or new conduction disturbances were discharged with a mobile telemetry monitor for 2 weeks (Zio AT [iRhythm] or 5-in-1 Mini Patch Monitor [First-Call Medical]). Initial follow-up was at 1 month, with final follow-up at 1 year.

Patients who developed persistent complete heart block (CHB), or Mobitz II AV block, after the procedure had permanent pacemakers implanted. For patients not in persistent CHB, the pacemakers were programmed initially to a rate below their intrinsic sinus rate (usually 60 beats per

minute) with long AV intervals to minimize unnecessary ventricular pacing.

The primary end point was the development of new high-grade AV conduction block, defined as either CHB, or Mobitz II second-degree AV block.

Implant Depth Assessment

Angiography after valve deployment was performed to confirm implant depth, except in patients with advanced kidney failure. Using a Picture Archiving and Communication System (PACS), a straight line was traced at the bottom of the left coronary cusp and noncoronary cusp to define the annular plane. The implant depth was determined by measuring the perpendicular distance from the annulus to the intraventricular valve end under the left coronary cusp and noncoronary cusp (eFigure 1 in Supplement 1).[6]

Computed Tomography Measurements

3mensio software (Pie Medical Imaging) was used to obtain all standard TAVR measurements and membranous septum length, except for 4 patients who had a noncontrast computed tomography (CT) scan only due to advanced kidney failure. Annular and left ventricular outflow tract (LVOT) calcium burden were quantified in the following grades: grade 0, indicating no calcification; grade 1, small, nonprotruding calcifications; grade 2, protruding (>1 mm) or extensive (>50% of cusp sector) calcifications; and grade 3, protruding (>1 mm) and extensive (>50% of cusp sector) calcifications.

Definitions

We used standard criteria for recognition of RBBB and Straus Criteria for left BBB (LBBB).[7,8]

Statistical Analysis

Categorical variables were described as number and percentage of the total and compared with the χ^2 test. Continuous variables were described as median and interquartile range and compared with the Wilcoxon rank sum test. STATA version 17 (StataCorp) was used for all analyses. Logistic regression was performed to associate clinical and electrophysiologic parameters with the outcomes of intraprocedural and late AV block separately. Sensitivity and specificity were calculated based on clinically meaningful cut points. Due to the small number of intraprocedural and late AV blocks, multivariable modeling was not performed. *P* value tests were 2-tailed, and $P < .05$ was considered statistically significant.

Results

We enrolled 409 consecutive patients undergoing TAVR, among whom median (IQR) age was 78.5 (73.1-83.5) years and 182 patients (44.5%) were female. A balloon-expandable valve was used in 368 cases (90.0%) and a self-expanding valve in 41 cases (10.0%). Thirty-four of the procedures (8.3%) were valve-in-valve (VIV) (10 with self-expanding valves).

Patient characteristics are typical of reported TAVR populations (**Table 1**). Few patients had a history of syncope.

Table 1. Baseline Patient Characteristics

Variable	All patients (N = 409), No. (%)
Age, median (IQR), y	78.5 (73.1-83.5)
Sex	
Female	182 (44.5)
Male	227 (55.5)
Race and ethnicity[a]	
Asian	3 (0.7)
Black	7 (1.7)
Hispanic	6 (1.5)
White	390 (95.4)
Other[d]	3 (0.7)
Body mass index, median (IQR)[b]	28.2 (24.4-33.0)
Diabetes	137 (33.5)
Hypertension	362 (88.5)
Hyperlipidemia	344 (84.1)
Atrial fibrillation[c]	107 (26.2)
CKD (eGFR <60 mL/min/1.73 m^2)	118 (28.9)
Receiving hemodialysis	7 (5.9)
CAD	226 (55.4)
Prior PCI	77 (18.9)
Prior cardiac surgery	69 (16.9)
Prior CABG	40 (9.8)
Prior AVR/r	34 (8.3)
Prior MVR/r	3 (0.7)
LVEF, median (IQR), %	60.0 (57.0-65.0)
AVA, median (IQR), cm^2	0.78 (0.62-0.90)
Mean gradient, median (IQR), mm Hg	42 (36-54)
Peak AoV velocity, median (IQR), m/s	4.2 (3.9-4.7)
Bicuspid valve	62 (15.2)
TAVR-in-SAVR (valve-in-valve)	34 (8.3)
Length of membranous septum, median (IQR), mm	3.7 (2.3-5.3)
LVOT calcium burden	
None	250 (61.3)
Mild	112 (27.5)
Moderate	35 (8.6)
Severe	11 (2.7)
Annulus calcium burden	
None	226 (55.5)
Mild	143 (35.1)
Moderate	32 (7.9)
Severe	6 (1.5)

Abbreviations: AVA, aortic valve area; AoV, aortic valve; AVR/r, aortic value replacement or repair; CABG, coronary artery bypass graft; CAD, coronary artery disease; CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; LVEF, left ventricular ejection fraction; LVOT, left ventricular outflow tract; MVR/r, prior mitral valve replacement or repair; PCI, percutaneous coronary intervention; SAVR, surgical aortic valve replacement; TAVR, transcatheter aortic valve replacement.

[a] Defined by study participants.

[b] Calculated as weight in kilograms divided by height in meters squared.

[c] Atrial fibrillation refers to rhythm in operating room prior to TAVR.

[d] Includes those identifying as Native Hawaiian or Other Pacific Islander or those who chose not to disclose.

Table 2. Atrioventricular (AV) Conduction Changes During the Transcatheter Aortic Valve Replacement (TAVR) Procedure

Type of conduction disturbance	No.[a] Intraprocedural block	Postprocedural block
PR prolongation	4	0
Second-degree block (AV Wenckebach)	3	0
Second-degree block (Mobitz II)	5	3
Complete heart block	55	22

[a] Number of patients developing conduction abnormalities during or after the TAVR procedure. No patient developed >1 conduction disturbance.

Patients had severe aortic stenosis; median (IQR) peak aortic valve velocity was 4.2 (3.9-4.7) meters per second. Thirty-four patients had undergone prior surgical aortic valve replacement. The median (range) length of hospitalization after TAVR was 1 (1-10) days. Longer hospitalizations were due primarily to persistent heart failure. Atrial fibrillation was present in 10% of patients before the procedure. The baseline PR interval was prolonged (>200 milliseconds) in 24% of patients. RBBB or LBBB were present in 13% and 5%, respectively.

AV Conduction Disturbances

During the TAVR procedure 63 patients (15.4%) developed AV block (**Table 2**): AV Wenckebach in 3 patients, Mobitz II second-degree AV block in 5, and CHB in 55. Four additional patients developed markedly prolonged PR intervals without block. AV block was present at the end of the procedure in 19 patients: AV Wenckebach in 2, Mobitz II block in 3, and CHB in 14. The median duration of transient AV block was 2 minutes, with a mean (range) duration of 17 minutes (5 seconds-150 minutes). Pacemakers were placed at the end of the procedure in 24 patients: 20 temporary and 4 permanent. Heart block that developed during the valve implant persisted in 11 cases in which a temporary pacemaker was placed. Thus, a total of 15 patients (3.7%) received permanent pacemakers due to persistent heart block developing during the TAVR procedure.

High-grade block developed postprocedure in 25 patients (6.3%) (Table 2). In 4 patients, the heart block occurred prior to hospital discharge, while heart block occurred after discharge in 21 patients. Postprocedure heart block occurred a median (range) of 4 days (1-10 days) after the procedure. Delayed AV block occurred in only 3 of 48 patients that had transient AV block during the TAVR procedure. In contrast to patients developing intraprocedural block, 19 of 25 patients (76.0%) that developed complete or Mobitz II block after the procedure had self-limited episodes of paroxysmal block lasting, on average, 11.5 seconds (median: 6 seconds), or more prolonged periods of episodic block lasting 56 minutes to 11 hours (comparing percentage of persistent AV block developing during [100%] or after [20%] TAVR: $P < .001$) (eFigure 2 in Supplement 1). Three patients experienced syncope; the remaining patients reported no symptoms or only transient lightheadedness.

Site of AV Conduction Block

Most patients (58 of 63) with AV block during the TAVR procedure developed intra- or infra-His block. However, we documented changes in AV nodal conduction in 5 patients during the procedure: marked increase in the PR/AH interval, 2:1 AV node block, AV node Wenckebach, and 1 case with CHB, which resolved after 11 minutes without recurrence (no pacemaker was implanted). In 3 patients, we documented complete AV node block, confirmed by EPS, following the procedure (1 of these patients also developed AV nodal block during TAVR). The high-grade or CHB occurred in these cases 1, 3, and 5 days post-TAVR (eFigure 3 in Supplement 1). Thus, of the 40 patients with AV block leading to a pacemaker, 3 (7.5%) were localized to the AV node.

Variables Associated With Conduction Block During TAVR

Several factors correlated with risk of intraprocedural heart block (**Table 3**). Almost one-fifth of patients with baseline RBBB developed persistent AV block requiring permanent pacemakers. No other baseline ECG parameter predicted intraprocedural block. New LBBB developing during the procedure was associated with increased risk for intraprocedural block, although only 2 of 170 patients exhibiting new LBBB during the procedure developed persistent heart block. New-onset RBBB was observed in 5 patients (1.2%). In 3 cases, the block appeared due to catheter manipulation, and in 2 of these 3 cases, the RBBB resolved by the first postoperative day. The third patient developed persistent CHB during the procedure. No baseline electrophysiologic parameter was associated with intraprocedural heart block. In contrast to prior reports, patients with a longer membranous septum had higher risk of AV block (Table 3). Additionally, greater amounts of calcium in the LVOT and aortic annulus were associated with increased AV block risk, as were larger prosthetic valves and oversizing of balloon-expandable valves.

Variables Associated With Delayed Conduction Block

Predictors of delayed AV block differed from factors associated with intraprocedural AV block (**Table 4**). Increasing age was associated with delayed AV block. Preexisting RBBB was not associated with risk for delayed AV block. One patient with new-onset RBBB developed CHB 2 days postprocedure. New LBBB at any time during or after TAVR was not associated with delayed AV block.

Marked prolongation of the HV interval at the end of the TAVR was significantly associated with delayed AV block, as were longer PR intervals and longer Wenckebach-paced cycle length at baseline and at the end of the procedure.(Table 4) Neither transient AV block during the procedure nor the duration of intraprocedural block correlated with risk for delayed AV block.

One patient undergoing VIV developed delayed AV block. Valve size did not influence occurrence of delayed AV block. The presence of coronary disease was associated with delayed but not intraprocedural block. Although depth of implant was not related to intraprocedural heart block, patients developing delayed AV block had greater noncoronary

Table 3. Factors Associated With Intraprocedural Atrioventricular Block (AVB)

Variable	No. (%)			*P* value
	All patients (N = 409)	No persistent AVB (n = 394)	Persistent AVB (n = 15)	
Age, median (IQR), y	78.5 (73.1-83.5)	78.4 (73.1-83.5)	80.0 (71.2-86.6)	.53
Sex				
Female	182 (44.5)	174 (44.2)	8 (53.3)	.48
Male	227 (55.5)	220 (55.8)	7 (46.7)	
Bicuspid valve	62 (15.2)	62 (15.7)	0	.10
Valve-in-valve	34 (8.3)	34 (8.6)	0	.23
LBBB pre-TAVR	21 (5.1)	21 (5.3)	0	.36
LBBB peri-TAVR[a]	170 (41.6)	168 (42.6)	2 (13.3)	.02
RBBB pre-TAVR[a]	52 (12.7)	43 (10.9)	9 (60.0)	<.001
New RBBB post-TAVR	5 (1.2)	4 (1.0)	1 (6.7)	.05
Pre-HV, median (IQR), ms	59.0 (53.0-65.0)	59.0 (53.0-65.0)	62.0 (54.0-68.0)	.48
Pre-AH, median (IQR), ms	92.0 (75.0-115.0)	93.0 (76.0-115.0)	76.0 (70.0-86.0)	.07
Baseline PR, median (IQR), ms	177.0 (158.0-200.0)	178.0 (158.0-200.0)	164.0 (153.0-196.0)	.37
Pre-TAVR AVWB CL, median (IQR), ms	420.0 (370.0-480.0)	420.0 (370.0-490.0)	430.0 (360.0-450.0)	.51
Balloon-expandable vs self-expanding valve	368 (90.0)	356 (90.4)	12 (80.0)	.19
Pre-valve dilatation	72 (17.8)	69 (17.7)	3 (20.0)	.82
Post-valve dilatation	78 (19.3)	75 (19.3)	3 (20.0)	.94
Depth of implant, median (IQR)				
NCC	3.6 (2.7-4.4)	3.6 (2.6-4.4)	4.0 (3.3-4.9)	.13
LCC	3.0 (1.5-4.0)	3.0 (1.5-3.9)	4.0 (2.3-4.4)	.06
Length of membranous septum, median (IQR), mm[a]	3.7 (2.3-5.3)	3.7 (2.3-5.2)	5.3 (3.5-6.0)	.02
Annulus area, median (IQR), mm^2	461.3 (402.7-530.8)	460.8 (402.7-532.3)	464.5 (391.9-513.6)	.83
Annulus perimeter, median (IQR), mm	77.4 (72.4-83.3)	77.4 (72.4-83.3)	77.2 (71.3-81.5)	.84
LVOT area, median (IQR)	453.9 (393.7-528.1)	455.2 (393.8-531.1)	435.2 (370.6-484.3)	.38
LVOT calcium score, median (IQR)	1730.3 (670.0-5707.4)	1624.5 (650.6-5660.7)	3958.5 (1477.5-7760.8)	.08
Annulus calcium burden[a]				<.001
None[a]	226 (55.5)	222 (56.6)	4 (26.7)	.02
Mild	143 (35.1)	137 (34.9)	6 (40.0)	.69
Moderate	32 (7.9)	29 (7.4)	3 (20.0)	.08
Severe[a]	6 (1.5)	4 (1.0)	2 (13.3)	<.001
LVOT calcium burden[a]				<.001
None[a]	250 (61.3)	246 (62.6)	4 (26.7)	.005
Mild	112 (27.5)	108 (27.5)	4 (26.7)	.94
Moderate[a]	35 (8.6)	28 (7.1)	7 (46.7)	<.001
Severe	11 (2.7)	11 (2.8)	0	.51
Oversized balloon-expandable valve[a]	143 (35.0)	133 (33.8)	10 (66.7)	.009

Abbreviations: AH, atrio-His interval; AVWB CL, atrioventricular Wenckebach cycle length (in milliseconds); HV, His-ventricular interval; LBBB, left bundle-branch block; LCC, left coronary cusp; LVOT, left ventricular outflow tract; NCC, noncoronary cusp; PR, PR interval; RBBB, right bundle-branch block; TAVR, transcatheter aortic valve replacement.

[a] Indicates significance.

cusp implant depth. In contrast to intraprocedural block, patients with delayed block had shorter lengths of the membranous septum (Table 4).

Optimal ECG and electrophysiological parameters to predict delayed AV block are summarized in **Table 5**. Only marked prolongation of the HV (≥80 milliseconds) and PR longer than 300 milliseconds after implant significantly correlated with risk for delayed heart block. Intraprocedural changes in AH, HV, or Wenckebach cycle length were not associated with delayed block. We analyzed the PR interval and AV Wenckebach cycle length at the end of the implant procedure as continuous variables for all patients, as well as

Table 4. Factors Associated With Postprocedural Atrioventricular (AV) Block[a]

Variable	No. (%)			*P* value
	All patients (N = 394)	No AV block (n = 369)	AV block + pacemaker (n = 25)	
Age, median (IQR), y[a]	78.4 (73.1-83.5)	78.0 (73.1-83.4)	81.7 (74.3-86.7)	.048
LBBB				
Pre-TAVR	21 (5.3)	20 (5.4)	1 (4.0)	.76
Peri-TAVR	168 (42.6)	155 (42.0)	13 (52.0)	.33
Post-TAVR	124 (31.5)	117 (31.7)	7 (28.0)	.70
At discharge	60 (15.2)	53 (14.4)	7 (28.0)	.07
Postdischarge	8 (2.0)	8 (2.2)	0	.46
New LBBB anytime predischarge	179 (45.4)	165 (44.7)	14 (56.0)	.27
RBBB pre-TAVR	43 (10.9)	41 (11.1)	2 (8.0)	.63
New RBBB post-TAVR	4 (1.0)	3 (0.8)	1 (4.0)	.12
Pre-HV, median (IQR), ms	59 (53-65)	59 (53-65)	63 (55-70)	.10
Post-HV, median (IQR), ms[a]	65 (58-76)	64 (58-75)	70 (60-93)	.02
Post-HV, ms				
≥70	146 (39.2)	133 (38.1)	13 (56.5)	.08
≥80[a]	67 (18.0)	58 (16.6)	9 (39.1)	.007
≥90[a]	36 (9.7)	29 (8.3)	7 (30.4)	<.001
70-89	110 (29.6)	104 (29.8)	6 (26.1)	.71
Marked peri-TAVR AV delay[a]	2 (0.5)	1 (0.3)	1 (4.0)	.01
Peri-TAVR AVWB	2 (0.5)	2 (0.5)	0	.71
Peri-TAVR Mobitz II	5 (1.3)	5 (1.4)	0	.56
Peri-TAVR CHB	40 (10.2)	38 (10.3)	2 (8.0)	.71
Peri-TAVR CHB or Mobitz II	45 (11.4)	43 (11.7)	2 (8.0)	.58
Transient AV block	38 (9.6)	36 (9.8)	2 (8.0)	.77
Time in AV block if transient, median (IQR), min	2.0 (0.5-6.0)	1.8 (0.5-6.0)	2.0 (0.8-8.0)	.82
Baseline QRS, median (IQR), ms	98 (90-116)	98 (88-116)	100 (94-108)	.48
Pre-AH, median (IQR), ms	93 (76-115)	92 (76-114)	109 (76-133)	.10
Post-AH, median (IQR), ms	95 (76-118)	94 (74-117)	118 (81-150)	.07
Baseline PR, median (IQR), ms[a]	178 (158-200.0)	176 (158-200)	200 (172-240)	.007
Post-TAVR PR, median (IQR), ms[a]	200 (177-230)	200 (177-226)	230 (200-278)	.004
Pre-TAVR PR >200 ms[a]	85 (24.4)	75 (22.9)	10 (47.6)	.01
Pre-TAVR PR >300 ms[a]	3 (0.9)	2 (0.6)	1 (4.8)	.046
Post-TAVR PR >200 ms	165 (49.3)	152 (48.4)	13 (61.9)	.23
Post-TAVR PR >300 ms[a]	15 (4.5)	12 (3.8)	3 (14.3)	.03
Post-TAVR PR >400 ms[a]	3 (0.9)	0	3 (14.3)	<.001
Pre-TAVR AVWB CL, median (IQR), ms[a]	420 (370-490)	420 (370-480)	470 (400.0-550)	.02
Post-TAVR AVWB CL, median (IQR), ms[a]	440 (380-510)	430 (380-510)	495 (395.0-640)	.045
Change in AVWB CL, median (IQR), ms	10.0 (0.0-30.0)	10.0 (0.0-30.0)	20.0 (−5.0 to 55.0)	.20
Valve-in-valve	34 (8.6)	33 (8.9)	1 (4.0)	.39
Bicuspid valve	62 (15.7)	57 (15.4)	5 (20.0)	.55
CAD[a]	215 (54.7)	196 (53.3)	19 (76.0)	.03
Depth of implant, median (IQR)				
NCC[a]	3.6 (2.6-4.4)	3.6 (2.6-4.3)	4.4 (3.3-4.7)	.03
LCC	3.0 (1.5-3.9)	3.0 (1.5-3.9)	3.0 (2.4-4.6)	.19
Length of membranous septum, median (IQR), mm[a]	3.7 (2.3-5.2)	3.7 (2.3-5.3)	2.8 (1.9-3.6)	.04
LVOT calcium score, median (IQR)	1624.5 (650.6-5660.7)	1620.0 (648.4-5697.7)	2144.2 (861.2-5410.4)	.74
Annulus calcium burden				.19
None	222 (56.6)	211 (57.5)	11 (44.0)	.19
Mild	137 (34.9)	125 (34.1)	12 (48.0)	.16
Moderate	29 (7.4)	28.0 (7.6)	1.0 (4.0)	.50
Severe	4 (1.0)	3.0 (0.8)	1.0 (4.0)	.13

(continued)

Table 4. Factors Associated With Postprocedural Atrioventricular (AV) Block[a] (continued)

Variable	No. (%) All patients (N = 394)	No AV block (n = 369)	AV block + pacemaker (n = 25)	*P* value
LVOT calcium burden				.62
None	246 (62.6)	230 (62.5)	16 (64.0)	.88
Mild	108 (27.5)	102 (27.7)	6 (24.0)	.69
Moderate	28. (7.1)	25 (6.8)	3 (12.0)	.33
Severe	1 (2.8)	11 (3.0)	0	.38
Oversized balloon-expandable valve	133 (33.8)	125 (33.9)	8 (32.0)	.85

Abbreviations: AH, atrio-His interval; AVWB CL, atrioventricular Wenckebach cycle length (in milliseconds); CAD, coronary artery disease; CHB, complete heart block; HV, His-ventricular interval; LBBB, left bundle-branch block; LCC, left coronary cusp; LVOT, left ventricular outflow tract; NCC, noncoronary cusp; PR, PR interval; RBBB, right bundle-branch block; TAVR, transcatheter aortic valve replacement.

[a] Values in this Table exclude the 15 patients that developed persistent intraprocedural AV block and received permanent pacemakers. Values for electrocardiogram and intracardiac variables labeled "post-" refer to values measured at the end of the TAVR procedure.

[b] Indicates significance.

Table 5. Predictive Utility of Electrocardiogram (ECG) and Electrophysiologic (EP) Measures for Delayed Atrioventricular (AV) Block[a]

Measure	OR (95% CI)	*P* value	Sensitivity, %	Specificity, %	PPV	NPV
Post-HV ≥80 ms	3.28 (1.36-7.95)	.008	39.13	83.62	0.14	0.95
Post-TAVR PR >300 ms	4.19 (1.09-16.21)	.04	14.29	96.18	0.20	0.94
Post-AVWB ≥500 ms	2.43 (0.97-6.03)	.06	50.00	70.81	0.10	0.95

Abbreviations: AVWB, longest-paced atrial cycle length at which AV Wenckebach block occurred; NPV, negative predictive value; OR, odds ratio; PPV, positive predictive value.

[a] Sensitivity, specificity, and positive and negative predictive values for ECG and EP parameters. "Post-" values refer to measurements made at the conclusion of the TAVR procedure.

those with new LBBB (eFigures 4-11 in Supplement 1). This analysis did not appreciably improve our ability to identify patients at highest risk.

Effect of VIV Cases on Results

We analyzed associations with AV block after excluding the 34 patients that underwent VIV TAVR (eTables 1 and 2 in Supplement 1). This resulted in 2 changes. Excluding patients who underwent VIV TAVR, implant depth measured at the left coronary cusp was significantly greater in patients developing intraprocedural block (eTable 1 in Supplement 1). Analysis of postprocedural block excluding patients who underwent VIV TAVR found no significant association with depth of implant (eTable 2 in Supplement 1).

Mortality and Follow-Up

One periprocedural death occurred due to left ventricular perforation by the transcatheter valve guidewire, with pericardial tamponade and subsequent hemodynamic decompensation. One death attributed to progressive heart failure occurred 19 days after TAVR. There were 8 later deaths within the first year, none of which were sudden.

One patient developed CHB 8 months after TAVR. They had no periprocedural conduction abnormality. They were in persistent atrial fibrillation with chronically slow ventricular rates and presented with fatigue 8 months after the procedure. We judged this related to progressive intrinsic conduction disease, not the TAVR.

Consistent with the observation that 80% of patients with AV block developing after the procedure had paroxysmal block, while AV block was persistent in those developing intraprocedural block, we observed differences in pacing dependence at 1 month and 1 year. Patients that developed block during TAVR exhibited a median of 97.4% ventricular pacing at 1 month and median of 93.5% ventricular pacing at 1 year. In contrast, patients that developed block after TAVR demonstrated significantly less ventricular pacing at both 1-month follow-up (median: 5.8%) and 1-year follow-up (median: 9.2%) (eFigure 12 in Supplement 1). There was no significant difference in the programmed lower rate or AV delays for these 2 patient groups.

Discussion

Several novel observations in this study may increase our understanding of mechanisms causing heart block complicating TAVR and improve utilization of pacemakers in these patients. It is generally assumed that AV block is due to injury to the His bundle.[9] We have demonstrated that AV node as well as His bundle block may occur. We show that most cases of intraprocedural AV delay and block resolve and that this not a risk factor for delayed heart block. Preexisting RBBB was clearly the most significant risk factor for intraprocedural block, but it was not associated with delayed AV block. New-onset RBBB was very uncommon (5 patients [1.2%]). Two of these 5 patients developed CHB. Thus, as reported recently, new-onset RBBB is uncommon but appears to carry high risk for progression to CHB.[10] New LBBB was not associated with delayed heart block. In this series of predominantly balloon-expandable valves, anatomic and procedural variables made relatively minor contributions to risk for delayed heart block. We did not

observe any significant difference in outcome related to the type of valve used. However, since only 10% (n = 41) self-expanding valves were used, we cannot exclude an effect of valve type. Finally, baseline electrophysiologic measurements did not predict risk for delayed heart block, but measurements of AV Wenckebach and HV interval at the end of the procedure were associated with delayed block.

The explanation why block may occur within the AV node is likely anatomic variation in location of the specialized conducting system. An anatomic, fluoroscopic, and electrophysiology study found the His bundle located within the triangle of Koch in more than one-third of cases. In 48% of cases, the largest His deflection was found to be on the ventricular aspect of the septal leaflet of the tricuspid valve, meaning that the distal portions of the AV node are very close to the membranous septum and noncoronary cusp. The compact AV node was located at the apex of the triangle of Koch in 46% of hearts, thus potentially vulnerable to aortic prosthesis placement.[11]

Our observations suggest that the mechanisms underlying intraprocedural block differ from those causing delayed heart block: preexisting RBBB was associated with intraprocedural but not delayed block; prolonged PR interval and AV Wenckebach cycle length pre-TAVR predicted delayed but not intraprocedural block. Length of the membranous septum was directly related to intraprocedural heart block, whereas delayed block was inversely associated with membranous septal length.[12,13] Aortic annular calcium and LV outflow tract calcium burden were associated with intraprocedural block but not delayed block. Finally, the nature of block differed between the 2 types; block during the procedure was persistent, whereas most delayed blocks were paroxysmal and relatively brief. As a result, models to predict delayed block will have to incorporate different variables than those identifying patients at risk for intraprocedural block.

Prior studies of patients undergoing TAVR have identified a variety of ECG and electrophysiologic abnormalities associated with delayed AV block (eTable 3 in Supplement 1). Isolated PR prolongation before and/or after the procedure has been associated with risk for AV block in some studies, but not others.[14,15] We found no predictive value of the baseline PR interval for intraprocedural block, but baseline PR intervals longer than 200 milliseconds and end-of-procedure PR intervals longer than 300 milliseconds were associated with significantly increased risk for delayed block.

Baseline RBBB is generally acknowledged as a risk for heart block, but most studies do not compare risk for intraprocedural vs delayed block.[15,16] As expected, we observed persistent intraprocedural heart block in 20% of patients with preexisting RBBB.[17] However, RBBB did not predict delayed block, as reported once previously in a small study of 6 patients.[18] This is an important consideration for postprocedure monitoring and use of temporary pacing.

New-onset LBBB has been associated with increased risk for delayed heart block or pacemaker implantation.[16,19-21] Using our end point of Mobitz II or CHB, we found no association between new LBBB and delayed CHB. The 2020 American College of Cardiology (ACC) Expert Consensus Decision Pathway document proposes that new LBBB or increase in PR/QRS duration of 20 milliseconds or longer in patients without preexisting RBBB warrants continued transvenous pacing for at least 24 hours.[4] We did not follow this suggestion and observed no deleterious effects on outcomes.

The reported utility of electrophysiologic studies in patients undergoing TAVR has been variable (eTable 3 in Supplement 1). Some studies report no utility.[18,22-25] Other studies found electrophysiologic studies provided useful prognostic information.[9,26-32] One study used atrial pacing to assess AV Wenckebach cycle length only at the end of the TAVR procedure.[33] These investigators used an arbitrary cutoff of Wenckebach cycle of 500 milliseconds or longer to determine need for a pacemaker. Unfortunately, the end points in that study are not defined clearly, nor was use of pacemakers consistent. Most studies have been small (<200 patients). Furthermore, most have used variable criteria to implant pacemakers, such as absolute HV interval (<55, <60, or <70 milliseconds) or percentage increase in HV (25%), rather than actual spontaneous CHB (eTable 3 in Supplement 1).[26,28-30] The 2021 European Society of Cardiology Guidelines suggest it is reasonable to consider pacing in patients with HV of 70 milliseconds or longer in patients with new LBBB and QRS duration longer than 150 milliseconds or PR longer than 240 milliseconds.[34] If we had followed the proposed criteria for implanting pacemakers proposed by these publications, we would have implanted multiple pacemakers unnecessarily (eg, using criteria of an HV of 70-79 milliseconds at procedure end) in more than 50 patients.

Limitations

This is a single-center study. Although this is the largest prospective study we are aware of utilizing continuous ECG and electrophysiologic monitoring, the number of end points is relatively low. Most valve implants in this series involved a balloon-expandable valve; observations may not apply equally to self-expanding valves

Another limitation common to most published studies of conduction abnormalities after TAVR is interpretation of the amount of pacing after discharge. Percentage pacing is virtually meaningless if pacemaker programming is not specified and controlled. The amount of ventricular pacing may be affected by the lower programmed rate, as well as programmed AV delays. We programmed pacers to the lowest clinically appropriate pacing rate (usually 60 beats per minute) and the longest appropriate AV delays in order to minimize ventricular pacing dependence.

Conclusions

In conclusion, according to the results of this cohort study, multiple anatomic, procedural, electrocardiographic, and electrophysiologic factors influence the development of heart block after TAVR. It is clear that electrophysiologists can guide interventional cardiologists to place catheters to record His bundle electrograms with minimal prolongation of the TAVR procedure. Electrophysiologic measurements at the end of TAVR procedures may guide permanent pacemaker implan-

tation beyond those provided by the ECG alone.[5] ECG measurements combined with His bundle studies can provide physiologic data to predict delayed heart block. While these measurements have high negative predictive value, the positive predictive values remain relatively low. Our observations suggest that mechanisms for intraprocedural block differ from delayed block. Therefore, they require different prediction models. Finally, we have documented that both AV nodal block as well as infranodal block may cause heart block following TAVR.

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REFERENCES

1. Carroll JDMM, Mack MJ, Vemulapalli S, et al; STS-ACC TVT Registry of Transcatheter Aortic Valve Replacement. STS-ACC TVT registry of transcatheter aortic valve replacement. *J Am Coll Cardiol*. 2020;76(21):2492-2516. doi:10.1016/j.jacc.2020.09.595

2. Valle JA, Li Z, Kosinski AS, et al. Dissemination of transcatheter aortic valve replacement in the United States. *J Am Coll Cardiol*. 2021;78(8):794-806. doi:10.1016/j.jacc.2021.06.028

3. Harvey JE, Puri R, Grubb KJ, et al. Decreasing pacemaker implantation rates with Evolut supra-annular transcatheter aortic valves in a large real-world registry. *Cardiovasc Revasc Med*. 2024;69:1-9. doi:10.1016/j.carrev.2024.05.024

4. Lilly SM, Deshmukh AJ, Epstein AE, et al. 2020 ACC expert consensus decision pathway on management of conduction disturbances in patients undergoing transcatheter aortic valve replacement: a report of the American College of Cardiology Solution Set Oversight Committee. *J Am Coll Cardiol*. 2020;76(20):2391-2411. doi:10.1016/j.jacc.2020.08.050

5. Poulin MF, Ultimo B, Omar W, et al. Routine electrophysiology study during transcatheter aortic valve replacement: a safety and feasibility pilot study. *JACC Cardiovasc Interv*. 2023;16(20):2584-2586. doi:10.1016/j.jcin.2023.07.017

6. Piayda K, Hellhammer K, Veulemans V, et al. Navigating the "optimal implantation depth" with a self-expandable TAVR device in daily clinical practice. *JACC Cardiovasc Interv*. 2020;13(6):679-688. doi:10.1016/j.jcin.2019.07.048

7. Strauss DG, Selvester RH, Wagner GS. Defining left bundle branch block in the era of cardiac resynchronization therapy. *Am J Cardiol*. 2011;107(6):927-934. doi:10.1016/j.amjcard.2010.11.010

8. Surawicz B, Childers R, Deal BJ, et al; American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; American College of Cardiology Foundation; Heart Rhythm Society; Endorsed by the International Society for Computerized Electrocardiology. AHA/ACCF/HRS recommendations for the standardization and interpretation of the electrocardiogram: part III: intraventricular conduction disturbances: a scientific statement from the American Heart Association Electrocardiography and Arrhythmias Committee, Council on Clinical Cardiology; the American College of Cardiology Foundation; and the Heart Rhythm Society. *J Am Coll Cardiol*. 2009;53(11):976-981. doi:10.1016/j.jacc.2008.12.013

9. Reiter C, Lambert T, Kellermair J, et al. Intraprocedural dynamics of cardiac conduction during transcatheter aortic valve implantation: assessment by simultaneous electrophysiological testing. *Heart Rhythm*. 2021;18(3):419-425. doi:10.1016/j.hrthm.2020.10.018

10. Michowitz Y, Yagel O, Shrem M, et al. New-onset RBBB After transcatheter aortic valve replacement: incidence and risk factors for permanent pacemaker implantation. *JACC Clin Electrophysiol*. Published online August 7, 2025. doi:10.1016/j.jacep.2025.06.013

11. Cabrera JÁ, Anderson RH, Macías Y, et al. Variable arrangement of the atrioventricular conduction axis within the triangle of Koch: implications for permanent His bundle pacing. *JACC Clin Electrophysiol*. 2020;6(4):362-377. doi:10.1016/j.jacep.2019.12.004

12. Boonyakiatwattana W, Maneesai A, Chaithiraphan V, et al. Preprocedural and procedural variables that predict new-onset conduction disturbances after transcatheter aortic valve replacement. *BMC Cardiovasc Disord*. 2022;22(1):135-146. doi:10.1186/s12872-022-02576-y

13. Hamdan A, Guetta V, Klempfner R, et al. Inverse relationship between membranous septal length and the risk of atrioventricular block in patients undergoing transcatheter aortic valve implantation. *JACC Cardiovasc Interv*. 2015;8(9):1218-1228. doi:10.1016/j.jcin.2015.05.010

14. Perel N, Tovia-Brodie O, Schnur A, et al. Post-transcatheter aortic valve implantation isolated PR prolongation: incidence and clinical significance. *Europace*. 2023;26(1):euae011. doi:10.1093/europace/euae011

15. Mangieri A, Lanzillo G, Bertoldi L, et al. Predictors of advanced conduction disturbances requiring a late (≥48 h) permanent pacemaker following transcatheter aortic valve replacement. *JACC Cardiovasc Interv*. 2018;11(15):1519-1526. doi:10.1016/j.jcin.2018.06.014

16. Zahid S, Khan MZ, Ullah W, et al. In-hospital outcomes of TAVR patients with a bundle branch block: insights from the National Inpatient Sample 2011-2018. *Catheter Cardiovasc Interv*. 2022;100(3):424-436. doi:10.1002/ccd.30341

17. Jørgensen TH, De Backer O, Gerds TA, Bieliauskas G, Svendsen JH, Søndergaard L. Immediate post-procedural 12-lead electrocardiography as predictor of late conduction defects after transcatheter aortic valve replacement. *JACC Cardiovasc Interv*. 2018;11(15):1509-1518. doi:10.1016/j.jcin.2018.04.011

18. Pavlicek V, Mahfoud F, Bubel K, et al. Prediction of conduction disturbances in patients undergoing transcatheter aortic valve replacement. *Clin Res Cardiol*. 2023;112(5):677-690. doi:10.1007/s00392-023-02160-0

19. Urena M, Webb JG, Cheema A, et al. Impact of new-onset persistent left bundle branch block on late clinical outcomes in patients undergoing transcatheter aortic valve implantation with a balloon-expandable valve. *JACC Cardiovasc Interv*. 2014;7(2):128-136. doi:10.1016/j.jcin.2013.08.015

20. Isogai T, Dykun I, Agrawal A, et al. Early resolution of new-onset left bundle branch block after transcatheter aortic valve implantation with the SAPIEN 3 valve. *Am J Cardiol*. 2022;168:117-127. doi:10.1016/j.amjcard.2021.12.032

21. Akin I, Kische S, Schneider H, et al. Surface and intracardiac ECG for discriminating conduction disorders after CoreValve implantation. *Clin Res Cardiol*. 2012;101(5):357-364. doi:10.1007/s00392-011-0400-6

22. López-Aguilera J, Segura Saint-Gerons JM, Mazuelos Bellido F, et al. Atrioventricular conduction changes after CoreValve transcatheter aortic valve implantation. *Rev Esp Cardiol (Engl Ed)*. 2016;69(1):28-36. doi:10.1016/j.rec.2015.02.026

23. Badenco N, Chong-Nguyen C, Maupain C, et al. Respective role of surface electrocardiogram and His bundle recordings to assess the risk of atrioventricular block after transcatheter aortic valve replacement. *Int J Cardiol*. 2017;236:216-220. doi:10.1016/j.ijcard.2017.02.029

24. Rubín JM, Avanzas P, del Valle R, et al. Atrioventricular conduction disturbance characterization in transcatheter aortic valve implantation with the CoreValve prosthesis. *Circ Cardiovasc Interv*. 2011;4(3):280-286. doi:10.1161/CIRCINTERVENTIONS.111.961649

25. Serban T, Knecht S, du Lavallaz JDF, et al. Ventricular pacing burden in patients with left bundle branch block after transcatheter aortic valve replacement therapy. *J Cardiovasc Electrophysiol*. 2023;34(6):1464-1468. doi:10.1111/jce.15920

26. Rivard L, Schram G, Asgar A, et al. Electrocardiographic and electrophysiological predictors of atrioventricular block after transcatheter aortic valve replacement. *Heart Rhythm*. 2015;12(2):321-329. doi:10.1016/j.hrthm.2014.10.023

27. Tovia-Brodie O, Ben-Haim Y, Joffe E, et al. The value of electrophysiologic study in decision-making regarding the need for pacemaker implantation after TAVI. *J Interv Card Electrophysiol*. 2017;48(2):121-130. doi:10.1007/s10840-016-0218-2

28. Badertscher P, Knecht S, Spies F, et al. Value of periprocedural electrophysiology testing during transcatheter aortic valve replacement for risk stratification of patients with new-onset left bundle-branch block. *J Am Heart Assoc*. 2022;11(15):e026239. doi:10.1161/JAHA.122.026239

29. Knecht S, Schaer B, Reichlin T, et al. Electrophysiology testing to stratify patients with left bundle branch block after transcatheter aortic valve implantation. *J Am Heart Assoc*. 2020;9(5):e014446. doi:10.1161/JAHA.119.014446

30. Ferreira T, Da Costa A, Cerisier A, et al. Predictors of high-degree conduction disturbances and pacemaker implantation after transcatheter aortic valve replacement: prognostic role of the electrophysiological study. *Pacing Clin Electrophysiol*. 2021;44(5):843-855. doi:10.1111/pace.14225

31. Raad M, Greenberg J, Altawil M, et al. The transcatheter aortic valve replacement-conduction study: the value of the His-ventricular interval in risk stratification for post-TAVR atrioventricular-block. *Struct Heart*. 2024;8(5):100296. doi:10.1016/j.shj.2024.100296

32. Massoullié G, Ploux S, Souteyrand G, et al. Incidence and management of atrioventricular conduction disorders in new-onset left bundle branch block after TAVI: a prospective multicenter study. *Heart Rhythm*. 2023;20(5):699-706. doi:10.1016/j.hrthm.2023.01.013

33. Krishnaswamy A, Sammour Y, Mangieri A, et al. The utility of rapid atrial pacing immediately post-TAVR to predict the need for pacemaker implantation. *JACC Cardiovasc Interv*. 2020;13(9):1046-1054. doi:10.1016/j.jcin.2020.01.215

34. Glikson M, Nielsen JC, Kronborg MB, et al; ESC Scientific Document Group. 2021 ESC guidelines on cardiac pacing and cardiac resynchronization therapy. *Eur Heart J*. 2021;42(35):3427-3520. doi:10.1093/eurheartj/ehab364

Editor's Note

Mechanistic Insights Into Post-TAVR Atrioventricular Block

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Heart block is one of the more frequent adverse events of aortic valve replacement therapy, in both surgical aortic valve replacement and transcatheter aortic valve replacement (TAVR). Data from the Society of Thoracic Surgeons/American College of Cardiology Transcatheter Valve Therapy Registry report a 30-day permanent pacemaker (PM) implantation rate of 11.3%, with extensive site-level variation (0%-36%).[1] Current guidance from the 2020 American College of Cardiology Expert Consensus Decision Pathway recommends PM implantation for symptomatic bradycardia or complete heart block (CHB) and monitoring with consideration of electrophysiology study (EPS) for new or progressive conduction disturbances.[2] Yet more than 2 decades after the introduction of TAVR, uncertainty remains regarding which conduction changes truly warrant permanent pacing, and different strategies have been proposed for conduction disturbance management.[3]

Related article page 25

In this issue of *JAMA Cardiology*, Waks et al[4] provide important mechanistic insight from 409 patients who underwent EPS at the beginning and end of TAVR. Conduction abnormalities were common: 13% developed intraprocedural CHB, but only 3% had persistent block by the end of the case. Transient atrioventricular (AV) block (AVB) typically resolved within minutes and was not associated with delayed high-grade block. Overall, 3.7% required PM implantation for intraprocedural block, and 6.3% developed delayed heart block necessitating a PM; in most, this was after discharge. Unlike the persistent AVB in intraprocedural cases, most delayed episodes were brief, self-limited, and asymptomatic, although 3 patients presented with syncope.

Several key observations are worthy of focus. First, the site of intraprocedural block was localized to the AV node in 8% of patients, and of the 40 patients ultimately receiving a PM, 7.5% had AV nodal block and not infrahisian block. Second, most intraprocedural AV block resolves and does not predict delayed CHB, suggesting that transient conduction injury need not trigger reflexive PM implantation or prolonged temporary pacing. Although no electrophysiological parameter was associated with intraprocedural block, a longer membranous septum, greater amounts of calcium, larger prosthetic valves, and oversizing were. Third, baseline right bundle-branch block increases risk of intraprocedural but not delayed heart block. Fourth, new left bundle-branch block, while frequent, was not associated with delayed heart block, offering reassurance that extended monitoring may be unnecessary in such cases. Last, delayed heart block correlated instead with specific electrophysiological parameters at the end of TAVR (HV interval ≥80 milliseconds, PR interval >300 milliseconds, longer AV Wenckebach), not with transient intraprocedural block.

Concordant with differences in presentation between intraprocedural and delayed heart block, at 1 year, patients with persistent intraprocedural AVB were far more pacing dependent than those with delayed block (94% vs 9%), emphasizing distinct mechanisms and prognoses. These findings challenge the instinctive caution that has characterized post-TAVR management of AVB. The data support consideration of a more selective, physiology-guided strategy, which may