Outcomes of Acute Conduction Abnormalities Following Transcatheter Aortic Valve Implantation With a Balloon Expandable Valve and Predictors of Delayed Conduction System Abnormalities in Follow-up

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Transcatheter aortic valve implantation (TAVI) is an acceptable treatment for severe aortic stenosis in high or intermediate risk patients. Conduction abnormalities are a known complication of TAVI. Most abnormalities occur perioperatively but can develop later. The predictors of delayed conduction abnormalities are unknown. Patients who underwent TAVI at our institution were reviewed. Patients with a pre-existing pacemaker were excluded. Baseline, in-hospital, and 30-day follow-up ECGs were reviewed. Patient and procedural characteristics were analyzed to look for predictors of acute and delayed abnormalities. Ninety-eight patients were included. All valves implanted were balloon expandable, most commonly SAPIEN S3 (78%). Thirty-seven (37.7%) patients developed abnormalities before discharge. Of these patients, 20 (57.1%) had complete resolution at 30-day follow-up. No patients with new conduction abnormalities during hospitalization had additional abnormalities at 30-day follow-up. Five (5.1%) patients developed new conduction abnormalities following discharge. Overall, 22 (22.4%) patients had conduction abnormalities at 30-day follow-up which were not present at baseline. Predilatation ($p = 0.003$), higher ratios of balloon ($p = 0.03$) or valve ($p = 0.05$) size to left ventricular outflow tract, and previous myocardial infarction ($p = 0.034$) were predictive of acute conduction abnormalities. Baseline right bundle branch block ($p = 0.002$), longer baseline ($p < 0.001$) and discharge ($p = 0.004$) QRS duration, moderate, or severe aortic insufficiency ($p = 0.002$) and atrial fibrillation ($p = 0.031$) were predictors of new conduction abnormalities after discharge. In conclusion, most new in-hospital conduction abnormalities resolve by 30-day follow-up. In-hospital conduction abnormalities are related to technical aspects of TAVI while delayed conduction abnormalities are related to baseline conduction system disease. **(Am J Cardiol 2019;123:1845−1852)**

Transcatheter aortic valve implantation (TAVI) has become an accepted treatment option for patients with severe aortic stenosis who are at elevated surgical risk.[1,2] The development of conduction system disease is a known complication following TAVI.[3−10] The development of left bundle branch block (LBBB) and need for permanent pacemaker implantation (PPI) have both been demonstrated to increase the risk of mortality and heart failure following TAVI.[11−14] Patients who develop conduction abnormalities at the time of TAVI without indications for PPI pose a unique clinical challenge without specific recommendations in the guidelines.[15,16] Most conduction defects observed occur in the perioperative period however new conduction abnormalities can occur following discharge.[17] Predictors of conduction abnormalities following hospital discharge and whether new conduction abnormalities following TAVI are predictive of progressive His-Purkinje disease remains unclear. The purpose of this study was 2-fold; to determine the outcomes of new conduction abnormalities that develop during index hospitalization for TAVI and to determine the predictors of conduction abnormalities that develop following hospital discharge.

Methods

The study was approved by the George Washington University Investigational Review Board. All patients who underwent TAVI at our institution from May 2015 to March 2018 were evaluated for this study. Patients who had a pre-existing permanent pacemaker or did not have a 30-day follow-up available were excluded from the investigation.

All patients who underwent TAVI at our institution had undergone evaluation by our multidisciplinary structural heart team to determine the appropriateness of TAVI. As part of our protocol, all patients underwent transthoracic echocardiograms and cardiac computed tomography or

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transesophageal echocardiograms for aortic annulus sizing. Prosthesis size was chosen by the implanting physician using the manufacturer's sizing recommendations. Valve oversizing was defined as an implanted valve area that was greater than the measured aortic annular area on cardiac computed tomography. All patients had an ECG before TAVI. Postprocedurally patients were admitted to the Cardiac Care Unit and were placed on continuous telemetry monitoring and had serial ECGs performed. All patients had 30-day follow-up arranged at our institution or with their referring physician. Those patients who had follow-up outside of our institution had records of their visit relayed to our structural heart program coordinator.

Baseline patient characteristics, echocardiographic and cardiac computed tomography measurements, procedural specifics, and electrocardiograms were collected retrospectively. All baseline, in-hospital, and 30-day follow-up ECGs were reviewed. Conduction abnormalities were defined using the 2009 AHA/ACCF/HRS expert consensus for the standardization and interpretation of the electrocardiogram.[18] For the purpose of this study, conduction disease before discharge was defined as the highest degree of His-Purkinje dysfunction observed during the index hospitalization. Worsening conduction abnormalities were defined as the presence of a new conduction abnormality not seen on ECG in the comparative time period. Resolution of conduction abnormalities was defined as the absence of a conduction abnormality that developed before hospital discharge on the 30-day follow-up ECG. Available pacemaker interrogation reports at the 30-day follow-up were analyzed to look for the amount of right ventricular pacing.

Separate analyses were performed between patients who had stable ventricular conduction and those who had new conduction abnormalities across the different time periods. Patient characteristics and procedural specifications were analyzed to look for predictors of new conduction abnormalities. Continuous variables were expressed as mean ± SD and were compared using the Student *t* test. Discrete variables were compared using the chi-square test. All tests were conducted using an $\alpha = 0.05$ as the probability for a Type I error.

Results

A total of 146 patients underwent TAVI at our institution during the investigative time period. Seven (4.8%) patients died before their 30-day follow-up visit. Follow-up data were unavailable for 5 (3.4%) patients. Thirty-six (24.7%) patients had a pre-existing pacemaker. Ultimately, 98 (67.1%) patients were included in the analysis (Figure 1).

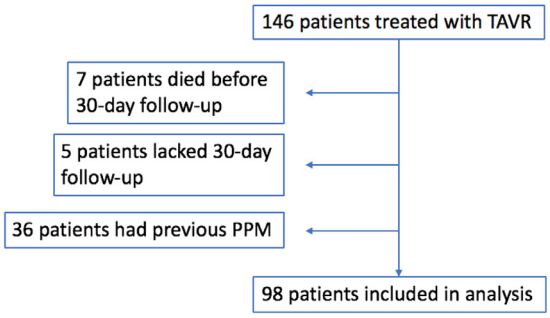

Figure 1. Flow chart of patients from dataset included in analysis.

Table 1 contains the baseline demographic and clinical characteristics of our patient population. Overall, the mean (±SD) age of the study group was 79.6 ± 9.1 years. A total of 27 (27.6%) patients had baseline ventricular conduction abnormalities. The most common baseline conduction abnormality seen was left anterior fascicular block (LAFB; n = 14, 14.3%) followed by right bundle branch block (RBBB; n = 13, 13.3%), and LBBB (n = 3, 3.1%). Baseline bifascicular block was observed in a total of 5 (5.1%) patients. All valves implanted were balloon expandable, with the majority being the Edwards Lifesciences SAPIEN S3 (n = 72, 73.5%). One (1.0%) patient required multiple valves during TAVI. Predilatation before valve deployment took place in 67 (68.4%) patients.

Thirty-seven (37.8%) patients developed new conduction abnormalities before hospital discharge. Eight (8.2%) patients developed complete heart block (CHB) immediately following valve deployment, and a total of 14 (14.3%) patients developed CHB during the index hospitalization. Seven (7.1%) patients underwent PPI before discharge. The timing of the development and resolution of conduction abnormalities in patients through the course of the study is depicted in Figures 2 and 3. Patients who had new conduction abnormalities before hospital discharge were more likely to have had predilatation before valve deployment ($p = 0.003$), higher ratios of predilatation balloon size ($p = 0.030$) or valve size ($p = 0.05$) to left ventricular outflow tract diameter, and history of myocardial infarction ($p = 0.034$). In contrast, these patients were less likely to have a history of atrial fibrillation ($p = 0.046$) or a prior aortic valve replacement ($p = 0.033$). Baseline His-Purkinje status was similar between patients who developed new conduction abnormalities before hospital discharge compared with those who did not. Table 1 displays the patient and procedural characteristics predictive of new conduction abnormalities before hospital discharge.

Twenty (54.1%) of the 37 patients who developed conduction abnormalities before discharge had complete normalization to their baseline electrical conduction by 30-day follow-up. Of the 14 patients who had developed CHB during hospitalization, 7 had no CHB and did not require PPI by 30-day follow-up. Of the 16 patients who developed LBBB during hospitalization, 10 had no LBBB at 30-day follow-up. Baseline LAFB ($p = 0.041$) was predictive of persistence of new conduction abnormalities. Shorter QRS duration at discharge ($p = 0.02$) and smaller aortic valve areas ($p = 0.05$) were predictive of resolution of new conduction abnormalities by 30-day follow-up. Table 2 displays the patient and procedural characteristics predictive of resolution of new conduction abnormalities by 30-day follow-up.

Five (5.1%) patients developed new conduction abnormalities by 30-day follow-up that were not observed before discharge. Four (4.1%) of these patients required PPI for CHB and 1 (1.0%) patient developed a LBBB. None of the patients who developed new conduction abnormalities after discharge had developed conduction abnormalities before hospital discharge. Baseline RBBB ($p = 0.002$), longer

Table 1
Comparison of baseline characteristics predictive of new conduction abnormalities before discharge versus those with stable ECG findings

Variable	Total (n = 98)	New abnormality (n = 37)	No change (n = 61)	p value
Baseline Characteristics				
Age (years)	79.6 ± 9.1	78.8 ± 8.1	80.0 ± 9.6	0.512
Women	32 (33%)	15 (41%)	17 (28%)	0.195
White	75 (77%)	30 (81%)	45 (74%)	0.512
Black	17 (17%)	6 (16%)	11 (18%)	
Other	6 (6%)	1 (3%)	5 (8%)	
Myocardial infarction	23 (24%)	13 (35%)	10 (16%)	0.034
Percutaneous coronary intervention	28 (29%)	13 (35%)	15 (25%)	0.262
Coronary bypass	27 (28%)	11 (30%)	16 (26%)	0.707
Other prior cardiac surgery	20 (20%)	5 (14%)	15 (25%)	0.187
Prior aortic valve replacement	7 (7%)	0	7 (11%)	0.033
Stroke	15 (15%)	4 (11%)	11 (18%)	0.336
Peripheral artery disease	25 (26%)	9 (24%)	16 (26%)	0.834
Smoker	8 (8%)	2 (5%)	6 (10%)	0.437
Hypertension	85 (87%)	30 (81%)	55 (90%)	0.199
Diabetes	34 (35%)	13 (35%)	21 (34%)	0.943
End-stage renal disease	6 (6%)	2 (5%)	4 (7%)	0.818
Chronic obstructive pulmonary disease	21 (21%)	7 (19%)	14 (23%)	0.902
STS Score	5.0 ± 2.8	3.8 ± 3.5	4.4 ± 2.8	0.393
Hostile chest	20 (20%)	7 (19%)	13 (21%)	0.776
Left ventricular ejection fraction (%)	55.7 ± 12.6	56 ± 14	55 ± 12	0.811
Heart failure 2 weeks prior	74 (76%)	27 (73%)	47 (77%)	0.649
NYHA class				
I	1 (1%)	0	1 (2%)	0.643
II	46 (47%)	20 (54%)	26 (43%)	
III	42 (43%)	14 (38%)	28 (46%)	
IV	9 (9%)	3 (8%)	6 (10%)	
Atrial fibrillation	21 (21%)	4 (11%)	17 (28%)	0.046
"Porcelain" aorta	6 (6%)	3 (8%)	3 (5%)	0.523
Moderate or severe aortic insufficiency	13 (13%)	3 (8%)	10 (16%)	0.241
Aortic annular calcification	50 (51%)	21 (57%)	29 (48%)	0.376
Mitral valve disease	77 (79%)	26 (70%)	51 (84%)	0.119
Left ventricular dimension-diastole (cm)	4.6 ± 0.9	4.2 ± 1.4	3.0 ± 1.8	0.500
Interventricular septal thickness-diastole (cm)	1.2 ± 0.3	0.9 ± 0.7	1.1 ± 0.5	0.157
Left ventricular outflow tract diameter (cm)	2.0 ± 0.2	2.0 ± 0.2	2.1 ± 0.2	0.227
Aortic annular minimum axis (cm)	2.2 ± 0.3	2.1 ± 0.5	1.9 ± 0.8	0.171
Aortic annular area (cm^2)	4.7 ± 1.0	4.7 ± 1.3	4.6 ± 1.1	0.569
Mean gradient (mmHg)	45 ± 14	43 ± 14	45 ± 14	0.502
Aortic valve area (cm^2)	0.7 ± 0.2	0.7 ± 0.3	0.7 ± 0.5	0.676
Electrocardiographic data				
PR interval (ms)	182 ± 32	187 ± 39	178 ± 27	0.257
QRS duration (ms)	103 ± 26	105 ± 25	103 ± 27	0.701
RBBB	13 (13%)	6 (16%)	7 (11%)	0.502
LAFB	14 (14%)	8 (22%)	6 (10%)	0.106
LPFB	2 (2%)	1 (3%)	1 (2%)	0.718
LBBB	3 (3%)	0	3 (5%)	0.171
Procedural specifications				
Valve type				
Edwards SAPIEN S3	72 (74%)	25 (68%)	47 (77%)	0.293
Edwards XT	20 (20%)	8 (22%)	12 (20%)	
Edwards Ascendra	6 (6%)	4 (11%)	2 (3%)	
Valve size (mm)				
29	13 (13%)	7 (19%)	6 (10%)	0.071
26	49 (50%)	15 (41%)	34 (56%)	
23	31 (32%)	15 (41%)	16 (26%)	
20	5 (5%)	0	5 (8%)	
Access				
Transfemoral	91 (93%)	33 (89%)	58 (95%)	0.335
Transapical	6 (6%)	4 (11%)	2 (3%)	
Transaortic	1 (1%)	0	1 (2%)	
Predilatation	67 (68%)	32 (86%)	35 (57%)	0.003

(*continued*)

Table 1 (***Continued***)

Variable	Total (n = 98)	New abnormality (n = 37)	No change (n = 61)	p value
Perivalvular leak	32 (33%)	15 (41%)	17 (28%)	0.247
Multiple valves deployed	1 (1%)	1 (3%)	0	0.197
Balloon size (mm)	22 ± 3	22 ± 3	22 ± 3	0.736
Balloon size:Left ventricular outflow tract diameter	11.2 ± 1.8	11 ± 4	8 ± 5	0.030
Balloon size:Aortic annular area	4.8 ± 0.9	4.8 ± 0.9	4.8 ± 0.9	0.754
Valve size:Left ventricular outflow tract diameter	12.4 ± 1.3	12.8 ± 1.4	12.2 ± 1.2	0.050
Valve size:Aortic annular area	5.5 ± 0.7	5.3 ± 1.2	5.6 ± 0.8	0.202
Valve oversizing	14 (16%)	6 (17%)	8 (15%)	0.110

All variables are expressed as mean ± SD or n (%).

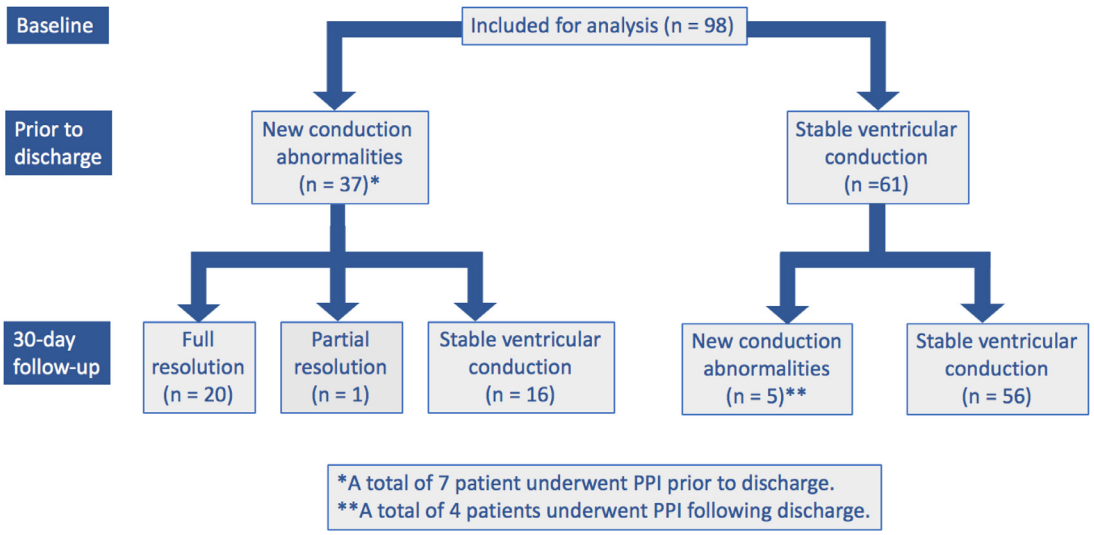

Figure 2. Flow chart of the development and resolution of conduction abnormalities before hospital discharge and at 30-day follow-up.

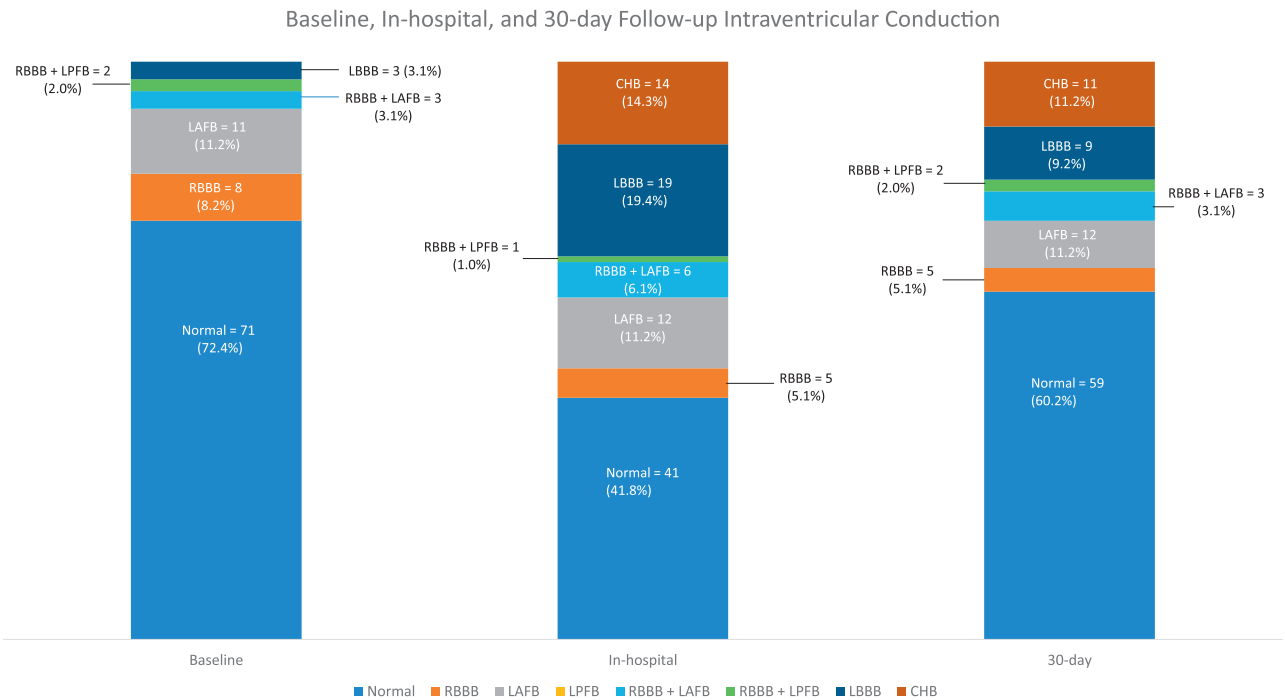

Figure 3. Incidence of specific conduction abnormalities at baseline, by hospital discharge, and at 30-day follow-up.

Table 2
Comparison of characteristics predictive of resolution of new conduction abnormalities following hospital discharge

Variable	Recovery (n = 21)	Persistent (n = 16)	p value
Baseline Characteristics			
Age (years)	80.1 ± 8.0	77.1 ± 8.2	0.263
Women	8 (38%)	7 (44%)	0.729
White	16 (76%)	14 (88%)	0.204
Black	5 (24%)	1 (6%)	
Other	0	1 (6%)	
Myocardial infarction	5 (24%)	8 (50%)	0.098
Percutaneous coronary intervention	5 (24%)	8 (50%)	0.098
Coronary bypass	6 (29%)	5 (31%)	0.860
Other prior cardiac surgery	3 (14%)	2 (13%)	0.875
Prior aortic valve replacement	0	0	1
Stroke	1 (5%)	3 (19%)	0.175
Peripheral artery disease	5 (24%)	4 (25%)	0.933
Smoker	0	2 (13%)	0.096
Hypertension	18 (86%)	12 (75%)	0.410
Diabetes	5 (24%)	8 (50%)	0.098
End-stage renal disease	1 (5%)	1 (6%)	0.843
Chronic obstructive pulmonary disease	3 (15%)	4 (25%)	0.428
STS Score	3.9 ± 3.8	3.7 ± 3.3	0.895
Hostile chest	3 (14%)	4 (25%)	0.410
Left ventricular ejection fraction (%)	56 ± 15	56 ± 14	0.922
Heart failure 2 weeks prior	15 (71%)	12 (75%)	0.809
NYHA class			
I	0	0	0.665
II	10 (48%)	10 (63%)	
III	9 (43%)	5 (31%)	
IV	2 (10%)	1 (6%)	
Atrial Fibrillation	1 (5%)	3 (19%)	0.175
"Porcelain" aorta	2 (10%)	1 (6%)	0.718
Moderate or severe aortic insufficiency	2 (10%)	1 (6%)	0.718
Aortic Annular Calcification	10 (48%)	11 (69%)	0.199
Mitral valve disease	16 (76%)	10 (63%)	0.367
Left ventricular dimension-diastole (cm)	4.3 ± 1.3	4.1 ± 1.5	0.684
Interventricular septal thickness-diastole (cm)	0.9 ± 0.8	1.0 ± 0.5	0.846
Left ventricular outflow tract diameter(cm)	4.6 ± 1.5	4.9 ± 1.1	0.520
Mean gradient (mmHg)	2.0 ± 0.2	2.0 ± 0.2	0.454
Aortic valve area (cm^2)	2.2 ± 0.3	2.0 ± 0.6	0.223
Aortic annular minimum axis (cm)	45 ± 16	41 ± 10	0.291
Aortic annular area (cm^2)	0.6 ± 0.3	0.8 ± 0.2	0.050
Electrocardiographic data			
PR interval (ms)	194 ± 43	178 ± 31	0.241
QRS duration (ms)	100 ± 21	111 ± 29	0.189
RBBB	2 (10%)	4 (25%)	0.206
LAFB	2 (10%)	6 (38%)	0.041
LPFB	0	1 (6%)	0.246
LBBB	0	0	
Delta QRS > 38 ms	5 (38%)	7 (70%)	0.133
QRS at discharge (ms)	129 ± 18	146 ± 14	0.020
Procedural specifications			
Valve type			
Edwards SAPIEN S3	15 (71%)	10 (63%)	0.395
Edwards XT	5 (24%)	3 (19%)	
Edwards Ascendra	1 (5%)	3 (19%)	
Valve size (mm)			
29	2 (10%)	5 (31%)	0.134
26	11 (52%)	4 (25%)	
23	8 (38%)	7 (44%)	
20	0	0	
Access			
Transfemoral	20 (95%)	13 (81%)	0.175
Transapical	1 (5%)	3 (19%)	
Transaortic	0	0	
Predilatation	18 (86%)	14 (88%)	0.875
Perivalvular leak	7 (19%)	8 (22%)	0.687
Multiple valves deployed	0	1 (6%)	0.246
Balloon size (mm)	22.6 ± 3.0	22.3 ± 3.3	0.813
Balloon size:Left ventricular outflow tract diameter	10.4 ± 4.0	10.8 ± 3.4	0.799
Balloon size:Aortic annular area	4.9 ± 0.9	4.9 ± 0.9	0.942
Valve size:Left ventricular outflow tract diameter	12.5 ± 1.0	13.1 ± 1.7	0.236
Valve size:Aortic annular area	5.1 ± 1.4	5.4 ± 0.7	0.445
Valve oversizing	4 (20%)	2 (13%)	0.605

All variables are expressed as mean ± SD or n (%).

baseline QRS duration ($p < 0.001$), longer QRS at discharge ($p = 0.004$), baseline moderate or severe aortic insufficiency ($p = 0.002$), and history of atrial fibrillation ($p = 0.031$) were predictive of new conduction abnormalities following discharge.

Twenty-two (22.4%) patients had new conduction abnormalities by 30-day follow-up which were not present at baseline. Eleven (11.2%) patients underwent PPI for CHB by 30-day follow-up (7 before discharge and 4 following discharge). After CHB, the most commonly observed new conduction abnormality was LBBB (n = 7, 7.1%) followed by bifascicular block (n = 3, 3.1%), and LAFB (n = 1, 1.0%). There were no new isolated RBBB or LPFB observed. Table 3 displays the patient and procedural characteristics predictive of new conduction abnormalities at 30-day follow-up compared with baseline. Baseline RBBB ($p = 0.004$), baseline LAFB ($p = 0.008$), longer baseline QRS duration ($p = 0.02$), an increase in QRS duration of > 38 ms at discharge ($p < 0.001$), and longer QRS duration at hospital discharge ($p < 0.001$) were predictive of new conduction abnormalities at 30-day follow-up compared with baseline.

Pacemaker interrogation reports were available for 8 of the 11 patients who underwent PPI. Six (75%) of the patients were right ventricular pacing > 95% of the time. The remaining 2 patients were right ventricular pacing 45% and 1% at their 30-day follow-up.

Table 3
Comparison of characteristics predictive of new conduction abnormalities including periprocedural + those developing by 30-day follow-up compared with baseline

Variable	New abnormality (n = 22)	No change (n = 76)	p value
Baseline Characteristics			
Age (years)	78 ± 8.1	80 ± 9.3	0.443
Women	8 (36%)	24 (32%)	0.673
White	20 (91%)	55 (72%)	0.169
Black	1 (5%)	16 (21%)	
Other	1 (5%)	5 (7%)	
Myocardial infarction	9 (41%)	14 (18%)	0.028
Percutaneous coronary intervention	9 (41%)	19 (25%)	0.146
Coronary bypass	8 (36%)	19 (25%)	0.293
Other prior cardiac surgery	3 (14%)	17 (22%)	0.371
Prior aortic valve replacement	1 (5%)	6 (8%)	0.591
Stroke	4 (18%)	11 (14%)	0.671
Peripheral artery disease	6 (27%)	19 (25%)	0.830
Smoker	3 (14%)	5 (7%)	0.287
Hypertension	17 (77%)	68 (89%)	0.137
Diabetes	8 (36%)	26 (34%)	0.852
End-stage renal disease	1 (5%)	5 (7%)	0.726
Chronic obstructive pulmonary disease	5 (23%)	16 (21%)	0.815
STS Score	3.9 ± 3.0	4.3 ± 3.1	0.608
Hostile chest	6 (27%)	14 (18%)	0.364
Left ventricular ejection fraction (%)	54 ± 15	56 ± 12	0.506
Heart failure 2 weeks prior	17 (77%)	57 (75%)	0.827
NYHA class			
I	0	1 (1%)	0.821
II	12 (55%)	34 (45%)	
III	8 (36%)	34 (45%)	
IV	2 (9%)	7 (9%)	
Atrial fibrillation	6 (27%)	15 (20%)	0.448
"Porcelain" aorta	1 (5%)	5 (7%)	0.726
Moderate or severe aortic insufficiency	4 (18%)	9 (12%)	0.440
Aortic annular calcification	15 (68%)	35 (46%)	0.068
Mitral valve disease	15 (68%)	62 (82%)	0.178
Left ventricular dimension-diastole (cm)	4.2 ± 1.7	4.0 ± 1.6	0.735
Interventricular septal thickness-diastole (cm)	1.0 ± 0.5	1.1 ± 0.6	0.465
Left ventricular outflow tract diameter (cm)	42 ± 15	45 ± 14	0.301
Aortic annular minimum axis (cm)	0.8 ± 0.2	0.6 ± 0.2	0.005
Aortic annular area (cm^2)	2.0 ± 0.2	2.1 ± 0.2	0.049
Mean gradient (mmHg)	2.0 ± 0.7	2.0 ± 0.7	0.694
Aortic valve area (cm2)	4.8 ± 1.0	4.6 ± 1.2	0.543
Electrocardiographic data			
PR interval (ms)	176 ± 28	183 ± 33	0.379
QRS duration (ms)	118 ± 34	99 ± 22	0.02
RBBB	7 (32%)	6 (8%)	0.004
LAFB	7 (32%)	7 (9%)	0.008
LPFB	1 (5%)	1 (1%)	0.345
LBBB	0	3 (4%)	0.344
Delta QRS >38 ms	7 (47%)	5 (7%)	<0.001
QRS at discharge (ms)	147 ± 25	107 ± 24	<0.001
Procedural specifications			
Valve type			
Edwards SAPIEN S3	16 (73%)	56 (74%)	0.196
Edwards XT	3 (14%)	17 (22%)	
Edwards Ascendra	3 (14%)	3 (4%)	
Valve size (mm)			
29	5 (23%)	8 (11%)	0.159
26	8 (36%)	41 (54%)	
23	9 (41%)	22 (29%)	
20	0	5 (7%)	
Access			
Transfemoral	19 (86%)	72 (93%)	0.349
Transapical	3 (14%)	3 (4%)	
Transaortic	0	1 (1%)	
Predilatation	17 (77%)	50 (66%)	0.308
Perivalvular leak	9 (41%)	23 (30%)	0.491
Multiple valves deployed	1 (5%)	0	0.062
Balloon size (mm)	22 ± 3.2	22 ± 3.2	0.537
Balloon size:Left ventricular outflow tract diameter	11 ± 3.1	8.9 ± 4.8	0.147
Balloon size:Aortic annular area	4.9 ± 1.0	4.8 ± 0.8	0.632
Valve size:Left ventricular outflow tract diameter	13.1 ± 1.6	12.3 ± 1.1	0.018
Valve size:Aortic annular area	5.5 ± 0.8	5.4 ± 1.0	0.656
Valve oversizing	3 (14%)	11 (16%)	0.855

All variables are expressed as mean ± SD or n (%).

Discussion

Based on our single-center retrospective study, the development of new conduction abnormalities following TAVI remains a common occurrence. Overall, the rates of PPI and new LBBB at 30-day follow-up in our study were similar to previous investigations.[3-10] The majority of new conduction abnormalities occurred in the perioperative period which is similar to what has been previously demonstrated.[17] Fortunately, new conduction abnormalities in the perioperative period failed to progress and the majority resolved by 30-day follow-up. This suggests that patients who develop new conduction abnormalities in the perioperative period are at no higher risk of developing progressive His-Purkinje dysfunction than patients who had stable conduction following TAVI. Characteristics predictive of new perioperative conduction abnormalities were primarily related to technical specifics of TAVI rather than baseline His-Purkinje disease. Predilatation and valve or balloon oversizing have been implicated as predictors of new conduction abnormalities in the past.[5,7,10]

Five patients developed new abnormalities by 30-day follow-up that were not present at discharge. In contrast to TAVI specifics predictive of in-hospital conduction defects, baseline His-Purkinje disease was predictive of new abnormalities following hospital discharge. Our study found

RBBB at baseline and prolonged baseline or discharge QRS durations to be predictive of the development of subacute conduction abnormalities. Additionally, baseline LAFB was predictive of persistence of new in-hospital conduction abnormalities at 30-day follow-up. Previous investigations have identified RBBB, LAFB, and prolonged QRS duration as predictors of new conduction abnormalities following TAVI.[5,7,19] In our study, the presence of at least moderate aortic insufficiency before TAVI was also predictive of subacute changes. This finding may be related to the degree of calcification of the aortic annulus. Although annular calcification itself was not predictive, there is no grading of severity of calcification. The patients with aortic insufficiency may have more extensive calcification than others, putting them at higher risk for conduction system injury.

The discrepancy between the predictors of conduction abnormalities in the periods of our investigation suggests different mechanisms of injury leading to His-Purkinje dysfunction. According to our investigation, acute conduction abnormalities appear to be more related to direct mechanical injury as a result of technical aspects of valve deployment such as prosthesis or balloon oversizing and predilatation. The majority of our patients who developed acute abnormalities had recovery of normal function the injury that occurs immediately following TAVI suggesting the acute injury is often transient. Moreno, et al demonstrated hemorrhage and edema involving the His bundle during post-mortem analysis of a patient who developed CHB following TAVI.[20] The acute injury leading to conduction disease is likely a result of this mechanical injury leading to hemorrhage and edema, and the degree to which this is present is likely what determines if the injury will be persistent or transient. In contrast, subacute abnormalities appear more related to baseline conduction system disease. Following the initial injury, healing and fibrosis begin. Patients with preexisting conduction system disease presumably have a higher degree of fibrosis in the area of the His-Purkinje fibers. As the healing process begins after the initial injury takes place, a smaller amount of fibrosis may be sufficient to lead to clinically significant changes.

RBBB and its relation to conduction abnormalities following TAVI deserve special consideration. Investigations have found baseline RBBB as the most significant electrocardiographic predictor of PPI after TAVI.[5,19,21] Additionally, studies examining the incidence of conduction abnormalities following TAVI have found the right bundle to be injured less frequently than other His-Purkinje fascicles.[5,6,8] Similar to the previously mentioned investigations, our analysis found RBBB to be the most significant baseline electrocardiographic predictor of subacute conduction abnormalities and that no patients developed a new isolated RBBB. This finding suggests that injury to the left-sided fascicles is more common which is likely a result of the proximity of the left fascicles to the aortic annulus.[22] Although loss of conduction through the right bundle does occur in CHB or bifascicular block following TAVI, it is unlikely to occur unless the injury is sufficient to damage it in addition to the left-sided fascicles. In patients with a preexisting RBBB, a smaller injury could lead to loss of function of the left bundle, and as a result, a complete atrioventricular block will develop. Patients with baseline RBBB warrant special attention and particularly careful planning with regards to valve sizing and deployment. In patients with baseline RBBB it is difficult to know the appropriate period of time to monitor for the development of CHB from our study. Information regarding the specific time to development of CHB was not available. If a high index of suspicion for developing CHB exists, delayed discharge or cardiac monitoring upon discharge should be a considered. Toggweiler et al proposed a minimum of 48 hours of monitoring should new conduction abnormalities develop following implantation, we propose a similar approach to patients with pre-existing RBBB.[23]

This study has several limitations. The retrospective nature of our study could introduce biases and unmeasured confounders, including selection bias. The sample size of our study is small, which is not powered to identify factors that have a small effect on our outcomes. Multivariable analysis was not able to be used in this study because of the sample size. Our patients are from a single-center, which makes it difficult to generalize our results to the overall population.

Disclosures

The authors have no conflicts of interest to disclose.

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