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CLINICAL RESEARCH

The R-wave amplitude in V1 on baseline electrocardiogram correlates with the occurrence of high-degree atrioventricular block following left bundle branch block after transcatheter aortic valve replacement

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Aims Several procedural and electrocardiogram (ECG) parameters have been associated with the occurrence of high-degree atrioventricular block (AVB) requiring permanent pacemaker implantation (PPI) after transcatheter aortic valve replacement (TAVR). We hereunder sought to assess if the baseline R-wave amplitude in V1 ECG lead of patients with normal QRS duration undergoing TAVR is associated with a higher patient's risk for developing high-degree AVB following left bundle branch block (LBBB).

Methods and results In this retrospective single-centre study in 720 consecutive patients who underwent TAVR, 141 (19.6%) patients with normal QRS duration developed a new LBBB after TAVR. The 24 (17%) patients who underwent PPI for reasons other than high-degree AVB were excluded from further analysis. In the remaining 117 study patients, 14 (12%) developed high-degree AVB requiring PPI (Group 1) while the remaining 103 (88%) patients did not (Group 2). There were no significant differences in baseline demographic or procedural characteristics nor in PR interval, QRS duration, and QRS axis between these two groups. The incidence of left anterior hemiblock was higher in Group 1 (3 of 14, 21.4%) than that in Group 2 (9 of 103, 8.7%), but the difference was not statistically significant ($P = 0.156$). The R-wave amplitude in V1 was smaller in Group 1 than that in Group 2 (0.029 ± 0.04 mV vs. 0.11 ± 0.14 mV, $P = 0.0316$). In the receiver-operating characteristics analysis, the cut-off for R-wave amplitude pre-TAVR was 0.03 mV, area under the curve = 0.7219 ($P = 0.0002$).

Conclusion The R-wave amplitude in lead V1 during baseline ECG in patients with normal QRS duration may predict the occurrence of high-degree AVB following new LBBB after TAVR.

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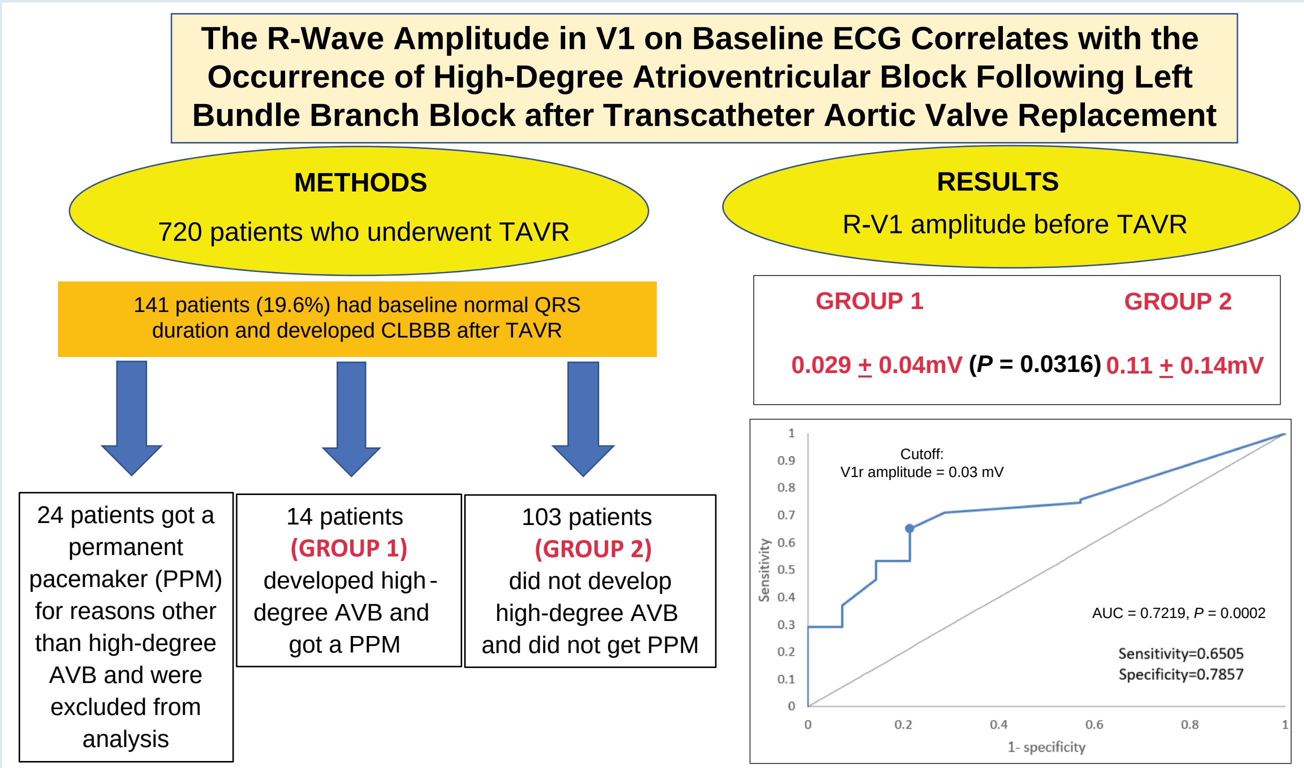
Graphical Abstract



Keywords Transcatheter aortic valve replacement • Left bundle branch block • Atrioventricular block • Permanent pacemaker implantation

What's new?

- Transcatheter aortic valve replacement (TAVR) is associated with a non-negligible incidence of left bundle branch block (LBBB) deteriorating into high-degree atrioventricular block (AVB) requiring permanent pacemaker implantation (PPI).
- Several procedural and electrocardiogram (ECG) risk factors have been reported in patients with new-onset LBBB and high-degree AVB after TAVR.
- We hereunder report that the R-wave amplitude in lead V1 during baseline ECG in patients with normal QRS duration may predict the occurrence of high-degree AVB requiring PPI following new LBBB after TAVR.
- If the results of our study are validated by others, they may have important clinical implications for the management of patients undergoing TAVR.

Introduction

Transcatheter aortic valve replacement (TAVR) has revolutionized the treatment of severe aortic stenosis and is increasingly performed in catheterization laboratories.

New-onset complete left bundle branch block (LBBB) is the most common acquired conduction abnormality after TAVR,[1–3] and its deterioration to high-degree atrioventricular block (AVB) requiring permanent pacemaker implantation (PPI) remains a significant complication.[1,3,4]

Several procedural risk factors have been associated with new-onset LBBB after TAVR.[1] In addition, first-degree AVB, left anterior hemiblock, and right bundle branch block (RBBB) have been found to be electrocardiogram (ECG) predictors for PPI.[1]

To our best knowledge, no study has assessed the amplitude of R-wave in V1 in patients undergoing TAVR who have no baseline apparent conduction disturbance. While a physiologic R wave >0.1 mV (1 mm) in V1 would suggest intact left-to-right ventricular septal activation and normal conduction over the LBB, a small R wave in V1 might indicate left septal fibrosis and/or a minimal degree of LBBB. Whether such small R wave in V1 before TAVR would represent an aggravating factor for the future occurrence of high-degree AVB requiring PPI in patients displaying LBBB after TAVR is unknown.

In the present study, we analysed the R-wave amplitude in V1 during baseline ECG in patients with normal QRS duration who developed LBBB post-TAVR and assessed its possible relationship with the occurrence of AVB requiring PPI.

Methods

Population and clinical data collection

This is a retrospective single-centre study involving patients who underwent TAVR between 12/2008 and 4/2021 in our tertiary medical centre at Hadassah Medical Center, Jerusalem. The study was approved by the Institutional Research Ethics Board at our institution.

All patients who developed a new LBBB after TAVR procedure were screened. Only those who had normal baseline QRS duration (<110 ms)[5] before TAVR were analysed. Patients who exhibited RBBB after TAVR were excluded.

Demographic and clinical data were collected from the hospital's electronic medical file system including hospitalization summary and TAVR report. Procedure variables included valve type and size, as well as the use of balloon dilatation technique.

Electrocardiogram collection and measurements

Surface 12-lead ECG tracings were recorded at least once daily before and after TAVR during the hospital stay. The first post-TAVR ECG showing new LBBB was selected for measurements.

Electrocardiograms were recorded at a sweep speed of 25 mm/s and a calibration of 10 mm/mV and digitally stored in the MUSE ECG database (GE Healthcare, Chicago, IL, USA). The following data were obtained from ECG software calculations: baseline rhythm, PR interval, QRS width, QRS axis, and QTc interval.

The amplitude of R wave in V1 lead was measured after significant magnification of the scanned pre- and post-TAVR ECGs. These measurements were independently performed 'on-line' by the two first co-authors (O.Y. and B.B.) (simultaneous measurements from scanned data). In case of discordance, consensual agreement was searched. If this could not be achieved, the senior investigator (B.B.) adjudicated the conflicting value.

Intra- and inter-investigator (O.Y. and B.B.) agreements were also tested using Bland–Altman figures. For all measurements tested, high agreements were found (mostly, 95% of the observations were found to be within the Bland–Altman 95% confidence interval). In addition, Pearson's correlations were calculated between pre -and post-measurements for inter- and intra-comparisons. For all calculations, the correlations were above 97% and mostly at 99%.

Table 1 Reasons for permanent pacemaker implantation unrelated to high-degree atrioventricular block

Reasons	Number of patients
LBBB with QRS and PR interval prolongation	8
Alternating LBBB and RBBB	2
Wide LBBB	4
Prolonged HV interval during electrophysiologic study	5
Severe sick sinus syndrome	5

LBBB = left bundle branch block; RBBB = right bundle branch block.

Definitions

Complete LBBB was defined according to standard classification[5] including QRS width of ≥120 ms.

The patients who underwent permanent pacemaker implantation (PPI) after TAVR were classified into two groups: (i) patients in whom high-degree AVB (≥ 2:1 AVB) developed within the first days following TAVR and (ii) patients who did not exhibit high-degree AVB after TAVR but had other indications for PPI (*Table 1*). The second group of patients was excluded from further analysis.

Statistical analysis

Continuous data are presented as mean ± standard deviation (SD). Categorical variables are shown as frequencies and percentages. Differences in baseline characteristics were compared using a paired *t*-test for continuous variables or Fisher's exact test for categorical variables. The differences in ECG parameters within the groups were calculated based on a paired *t*-test or the McNemar test for continuous and

Table 2 Baseline demographic and procedural characteristics

	Group 1 $n = 14$	Group 2 $n = 103$	Total $n = 117$	*P* value[a]
Gender, *n* (%)				
Female	9 (64.2)	66 (64.1)	75 (64.1)	0.9879
Male	5 (35.8)	37 (35.9)	42 (35.9)	
Age (years)				
Mean ± SD	84.1 ± 5.7	80.7 ± 6.1	81.1 ± 6.1	0.0512
Days discharge after TAVI[a]				
Mean ± SD	7.1 ± 3.2	5.9 ± 3.5	6.1 ± 3.45	0.2267
Balloon pre-dilatation, *n* (%)[a]				
No	6 (42.9)	55 (53.3)	61 (52.1)	0.4588
Yes	8 (57.1)	48 (46.7)	56 (47.9)	
Balloon post-dilatation, *n* (%)[a]				
No	14 (100)	90 (87.1)	104 (88.8)	0.361
Yes	0 (0)	13 (12.9)	13 (11.2)	
Valve company, *n* (%)[a]				
Edwards	4 (28.6)	37 (35.9)	41 (35.1)	0.7678
Medtronic	10 (71.4)	66 (64.1)	76 (64.9)	
Valve size (mm), *n* (%)[a]				
23	2 (14.3)	16 (5.5)	18 (15.3)	
26	5 (35.7)	38 (36.9)	43 (36.8)	0.4921
29, 34	7 (50)	49 (47.6)	56 (47.9)	

[a]P value between Group 1 and Group 2 was calculated based on a *t*-test or Fisher's exact test for continuous and categorical, respectively.

Table 3 Baseline and post-TAVR ECG parameters

		Group 1	Group 2	Total	P Value[a]		
		n = 14	n = 103	n = 117	Group 1	Group 2	Between groups
Pre-TAVR rhythm	Atrial fibrillation	1 (7.1)	8 (7.8)	9 (7.7)			0.9999
n (%)	Sinus rhythm	13 (92.9)	95 (92.2)	108 (92.3)			
PR (ms)	Baseline	168.2 ± 43.0	176.3 ± 34.6	177.5 ± 35.9			0.4940
	After TAVR	198.6 ± 32.8	186.1 ± 35.4	189.8 ± 39.5			0.2899
	Change	30.4 ± 30.1	9.8 ± 26.5	12.3 ± 29.8	0.011	0.0007	0.0234
QRS (ms)	Baseline	89.8 ± 11.2	93.4 ± 9.6	93.0 ± 9.8			0.1683
	After TAVR	142.8 ± 14.3	150.1 ± 37.0	149.2 ± 35.0			0.4661
	Change	53.2 ± 12.1	56.7 ± 37.5	56.3 ± 35.4	<0.0001	<0.0001	0.7333
	QRS type, n (%)						
	Notch	11 (78.6)	78 (75.7)	89 (76.1)			0.9999
	Slurred	3 (21.4)	25 (24.37)	28 (23.9)			
QTc (ms)	Baseline	446.5 ± 29.6	432.7 ± 31.2	434.3 ± 31.2			0.1207
	After TAVR	509.6 ± 41.8	489.4 ± 50.5	491.8 ± 49.7			0.1546
	Change	63.1 ± 30.7	56.7 ± 43.8	57.4 ± 42.4	<0.0001	<0.0001	0.5992
QRS axis (deg)	Baseline	0.93 ± 33.63	9.34 ± 33.11	8.33 ± 33.14			0.3752
	After TAVR	−17.5 ± 24.25	−14 ± 34.94	−14.4 ± 33.78			0.7177
	Change	−18.4 ± 36.34	−23.3 ± 45.42	−22.7 ± 44.33	0.0802	<0.0001	0.6997
R-V1 amplitude (mV)	Baseline	0.03 ± 0.04	0.11 ± 0.15	0.1 ± 0.14			0.0316
	After TAVR	0.01 ± 0.03	0.04 ± 0.1	0.04 ± 0.1			0.2197
	Change	−0.02 ± 0.04	−0.07 ± 0.12	−0.06 ± 0.12	0.0907	<0.0001	0.1240

ms = milliseconds; deg = degrees; mV = millivolts.

[a]The P-value within arms was calculated based on a paired t-test or McNemar test for continuous and categorical, respectively. The P-value between arms was calculated based on a t-test or Fisher's exact test for continuous and categorical, respectively.

categorical, respectively. The differences between groups were calculated based on a *t*-test or Fisher's exact test for continuous and categorical, respectively.

Receiver-operating characteristics (ROC) analysis was performed to calculate a cutoff of R-wave amplitude in V1 to assess those patients who developed high-degree AVB requiring PPI. For all analyses, we used the R software version 4.2.1.1. A $P < 0.05$ was considered statistically significant.

Results

Study group

From a total of 720 consecutive patients who underwent TAVR between 2008 and 2021 in our medical centre, 141 (19.5%) patients who had a baseline QRS complex of normal duration (<110 ms) developed a new LBBB post-TAVR. After excluding the 24 (17%) patients who underwent PPI for reasons other than high-degree AVB (*Table 1*), the remaining 117 patients comprised the study group. Of these 117 patients, 14 (12%) developed high-degree AVB requiring PPI (Group 1) while the remaining 103 (88%) patients did not (Group 2).

Baseline demographic and procedural characteristics

Most of the study patients (64.1%) were females and the mean age of the cohort was 81.1 ± 6.1 years (*Table 2*). The length of stay in the hospital was 6 ± 3.4 days. About half of the study patients underwent balloon pre-dilatation (46%), while approximately two-thirds of patients were implanted with a Medtronic valve (64.6%).

There were no significant differences in baseline demographic or procedural characteristics between patients who developed high-degree AVB and those who did not (*Table 2*).

Pre- and post-transcatheter aortic valve replacement electrocardiogram parameters

Of the 117 study patients, 108 (92.3%) displayed sinus rhythm at baseline and after TAVR while the remaining 9 patients had atrial fibrillation (*Table 3*). There was no significant difference in the baseline heart rhythm between Group 1 and Group 2 patients.

There were no significant differences in baseline ECG parameters (PR interval, QRS duration, QRS axis, and QTc interval) between Group 1 and Group 2 patients. The incidence of left anterior hemiblock was higher in Group 1 (3 of 14, 21.4%) than that in Group 2 (9 of 103, 8.7%) but the difference was not statistically significant ($P = 0.156$).

As expected, upon development of LBBB after TAVR, the PR, QRS, and QTc values were similarly and significantly increased in both groups. However, only the change of the PR interval increase was statistically different between the groups.

The QRS axis was similar in both groups before and after TAVR with significant similar negative change in the QRS axis.

The baseline R-wave amplitude in V1 in the study group was 0.1 ± 0.14 mV. It was significantly smaller in Group 1 (0.03 ± 0.04 mV) than that in Group 2 (0.11 ± 0.15 mV) ($P = 0.0316$). The change in the R-wave amplitude before and after TAVR was statistically significantly different only in Group 1. However, during LBBB after TAVR, the V1 R-wave amplitude was not different between Groups 1 and 2.

Based on the ROC analysis, the cutoff of the R-wave amplitude in ECG lead V1 was 0.03 mV, with an area under the curve of 0.7219 ($P = 0.0002$), sensitivity of 65.05%, and specificity of 78.57% (*Figure 1*).

Three representative case of study patients are displayed in *Figures 2–4*. *Figure 1* shows the case of a patient who had a very small R-V1 amplitude before TAVR (0.01 mV), who developed LBBB and subsequently complete AVB after the procedure. *Figures 3* and *4* show the cases of two patients who developed LBBB after TAVR without subsequent high-degree AVB. In these patients, baseline R-V1 amplitudes were 0.17 mV and 0.22 mV, respectively.

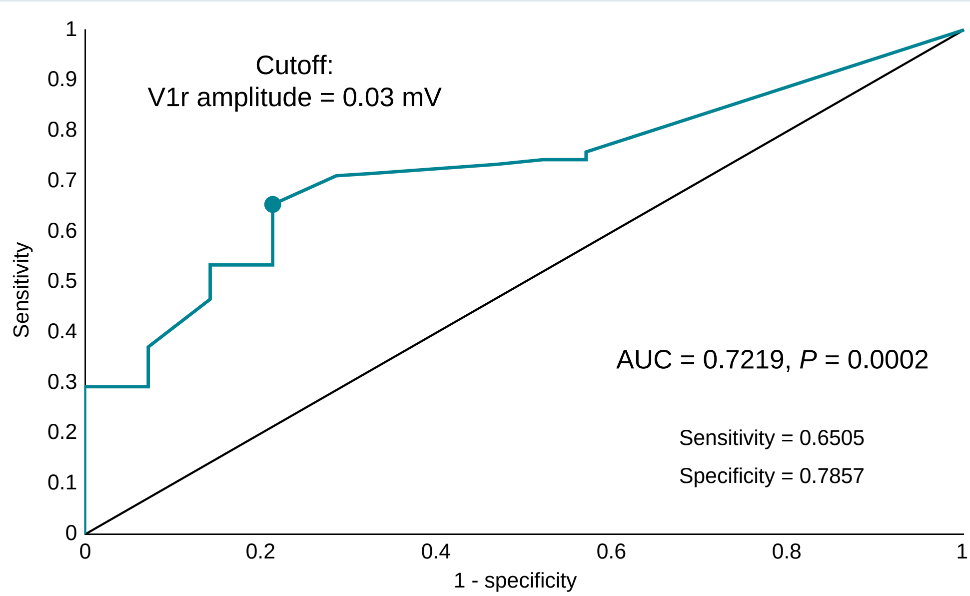


Figure 1 Receiver-operating characteristic curve of R-wave amplitude in electrocardiogram (ECG) lead V1 for predicting the occurrence of CAVB requiring PPI in patients exhibiting LBBB after TAVR. Abbreviations: AUC = area under the curve; CAVB = complete atrioventricular block; LBBB = left bundle branch block; PPI = permanent pacemaker implantation; TAVR = transcatheter aortic valve replacement.

Discussion

The results of our study show two main findings: (i) the baseline R-wave amplitude in V1 in TAVR patients who developed a new LBBB correlated with the development of high-degree AVB requiring PPI, and (ii) upon development of LBBB, the difference in the R-wave amplitude in V1 was not significantly different in patients who developed high-degree AVB requiring PPI or not.

The initial part of the QRS complex represents the progression of activation in the interventricular septum. During normal activation, the interventricular septum is predominantly activated from the left toward the right side of the septum, producing a positive (R wave) deflection in V1, usually of 1–3 mm (0.1–0.3 mV) amplitude. In the presence of conduction disturbance in the LBB, the left septal vector loses its predominance, resulting in smaller R-wave amplitude in V1 (< 0.1 mV) and sometimes in a QS pattern. This is in agreement with the results of our study showing that the R amplitude in V1 was markedly reduced in all patients who developed LBBB after TAVR.

It is well known that the presence of a pre-existing RBBB before TAVR is associated with a substantial increased risk of high-degree AVB after TAVR.[1] Less well known is that complete LBBB before TAVR was also found to be associated with a higher risk of post-procedural PPI requirement. In a study of 3404 TAVR recipients in whom 11.7% patients had LBBB at baseline, Fisher *et al.* showed that pre-existing LBBB was associated with a significantly increased risk of PPI by 30 days (21.1% vs. 14.8%; $P = 0.006$).[7]

In our study, by definition, all the patients had a QRS duration <110 ms; therefore, none of the Group 1 patients who developed high-degree AVB did fulfil the criteria of even incomplete LBBB (QRS 110–119 ms) as defined by Surawicz *et al.*[5] However, the small R-wave amplitude in V1 may suggest some conduction delay in the LBB system. Interestingly, a QRS pattern similar to that observed in our Group 1 patients has been sometimes attributed to intrahisian disease with split left-sided His[8,9] consistent with the theory of functional longitudinal dissociation of the His bundle fibers.[10,11]

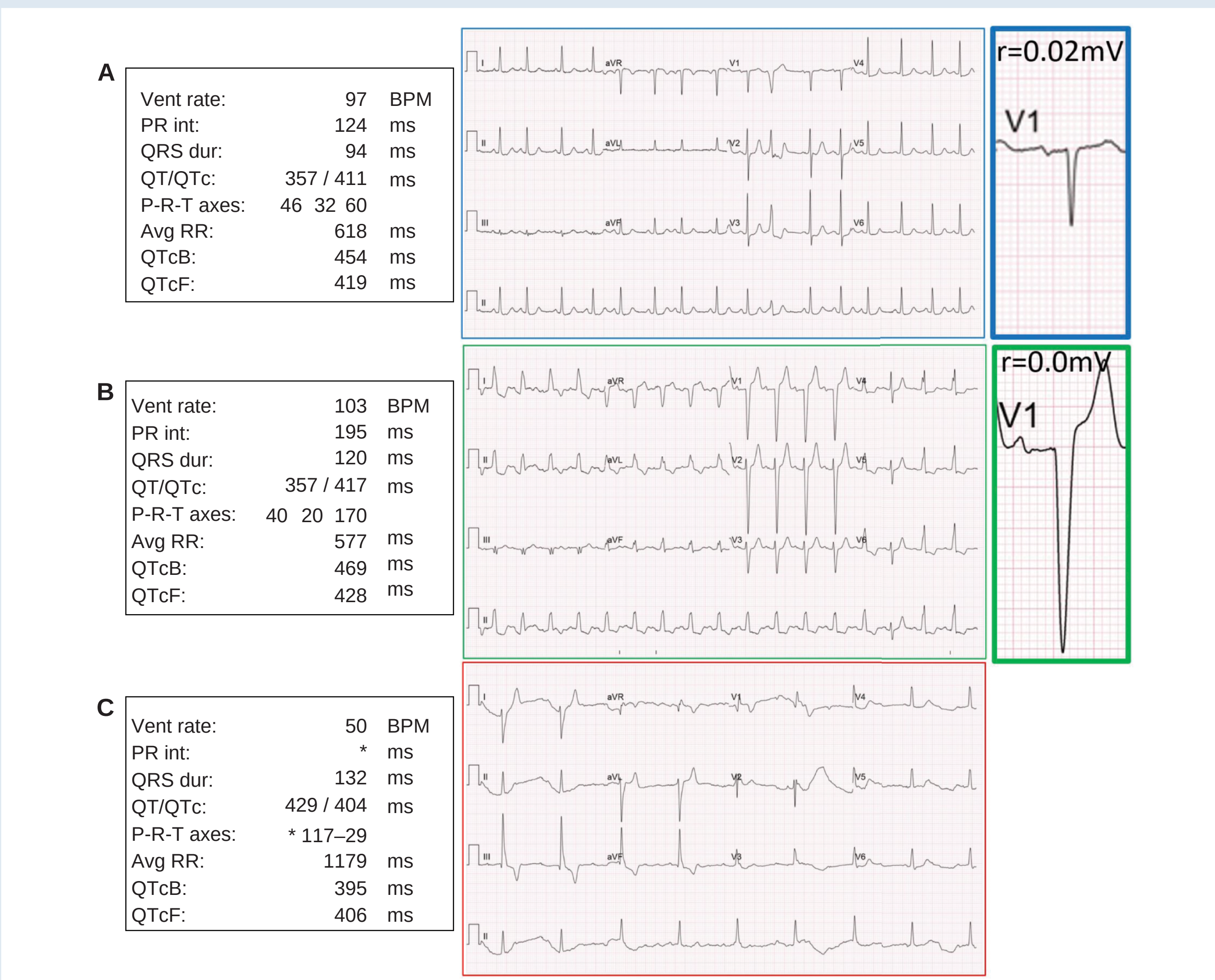


Figure 2 A 89-year-old woman with severe aortic stenosis and a significant obstruction of the left anterior descending artery requiring stent implantation. (*A*) Baseline electrocardiogram (ECG) before TAVR. The QRS is narrow (duration 94 ms), and the PR interval is normal (124 ms). R-wave in V1 is small (0.02 mV). (*B*) Twenty-four hours after TAVR, there is a LBBB (QRS duration 120 ms) associated with a prolongation of PR interval at 195 ms. R-wave in V1 is 0.0 mV. (*C*) Forty-four hours after TAVR, CAVB is documented with a ventricular escape rhythm of 50/min having a duration of 132 ms and a morphology of RBBB and right axis deviation suggesting an origin close to the left anterior fascicle. The patient received a permanent pacemaker.

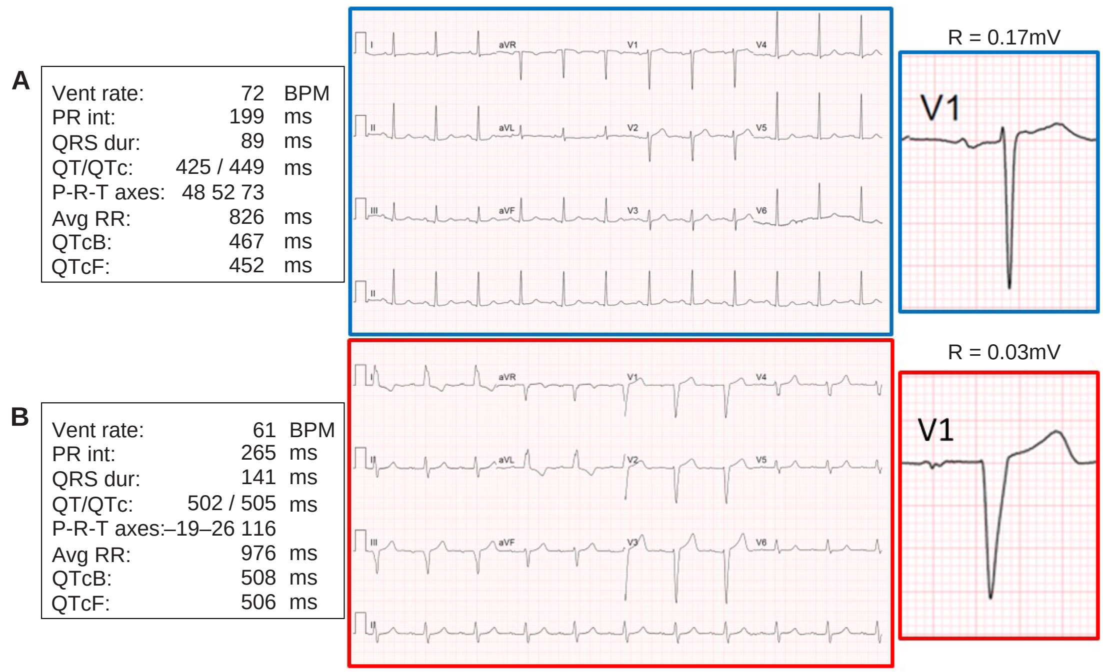


Figure 3 A 73-year-old woman with severe aortic stenosis. (*A*) Baseline ECG before TAVR showing narrow QRS duration (89 ms) with R-V1 amplitude of 0.17 mV. (*B*) After TAVI, LBBB occurred with a resulting decrease of R-V1 to 0.03 mV. This patient did not develop a high-degree AV block despite the marked increase of PR by 66 ms during LBBB. Note a partial interatrial block (*P* duration >120 ms after TAVR).[6]

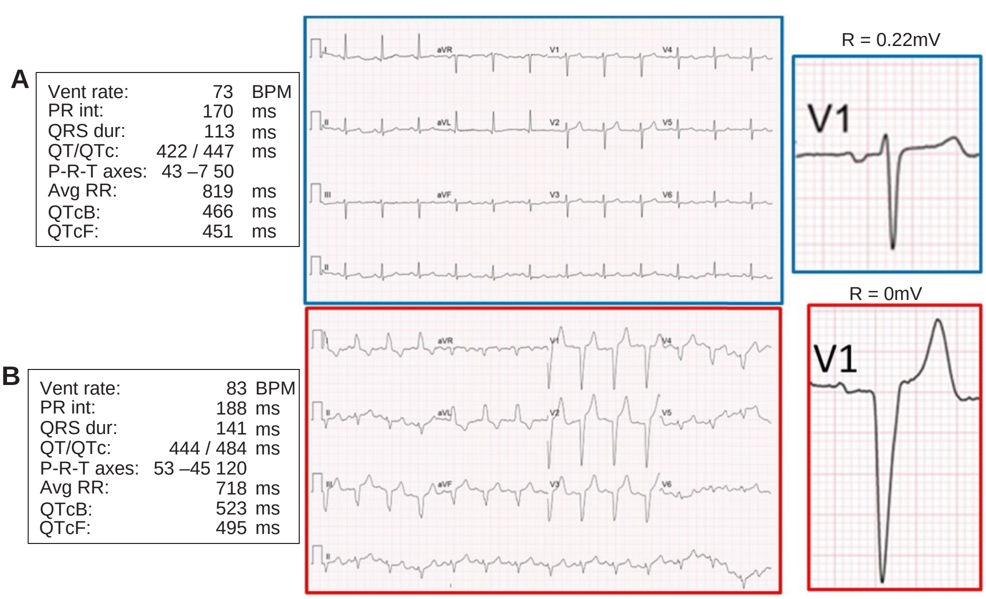


Figure 4 A 76-year-old woman with severe aortic stenosis. (*A*) Baseline electrocardiogram (ECG) before TAVR showing R-V1 amplitude of 0.22 mV; (*B*) After TAVR, LBBB is present with a marked decrease of R-V1 amplitude (0 mV). This patient did not develop high-degree AVB.

Since it is assumed that TAVR may damage the LBB and the RBB at their proximal parts because of their close anatomic relationship with the membranous septum at the junction of the non and right coronary cusps, one may speculate that this prior conduction disturbance (presumably in the His bundle) played an aggravating role in the occurrence of high-degree infranodal AVB after TAVR.

We did not find any prior report in the literature showing findings similar to those of our present paper. One reason could be that what we assume to represent a mild conduction delay in the LBB was not recognized and that the tracing was considered as normal.

In patients with LBBB undergoing right heart catheterization, complete AVB or only RBBB may develop following catheter-induced

RBB trauma. Padanilam *et al.*[12] found that in LBBB patients who develop catheter trauma to RBB during right heart catheterization, an initial R wave of ≥ 1 mm in lead V1 presuming intact left-to-right ventricular septal activation identifies LBBB patients at low risk of complete AVB. In our study, there was no significant difference in the amplitude of R wave in V1 during LBBB after TAVR between the groups who subsequently developed high-degree AVB requiring PPI or not. Whether the anatomical site responsible for the RBBB (right septum after catheter-induced trauma vs. left septum after TAVR) plays a role in the difference observed in the two settings is unknown.

Study limitations

First, our study was retrospective in nature. Second, the group of patients with high-degree AVB requiring PPI was relatively small that prevented to build an accurate multivariable model. Third, in patients with documented AVB, an electrophysiologic study was not performed to precisely determine the site of AVB. However, although the AV node may be damaged after TAVR,[13] it is assumed that high-degree AVB is commonly located in the infranodal conduction system in these patients.[14] Fourth, measurements of the R-wave in V1 in standard 12-lead ECG are not an easy task because of the usual small amplitude of the R wave in this derivation. Therefore, we performed these measurements on scanned ECGs using maximal amplification, taking into consideration the line thickness of the trace in our measurements. Fifth, correct positioning of the precordial electrodes (especially of V1 and V2 electrodes at the 4th intercostal space) also has a critical importance since recording higher precordial position may be associated with smaller V1 amplitude.[15]

Clinical implications

If the results of our study are validated by others, they may have important clinical implications for the management of patients undergoing TAVR, such as requiring a more careful patient follow-up including strict ECG monitoring and prolonged stay in the cardiology department.

Conclusions

The R-wave amplitude in lead V1 during baseline ECG in patients with normal QRS duration may predict the occurrence of high-degree AVB following new LBBB after TAVR.

Author's contribution

O.Y. and B.B. have full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

Funding

None declared.

Conflict of interest: None declared.

Data availability

The data that support the findings of this study are available from the corresponding author upon reasonable request.

References

1. Rodés-Cabau J, Ellenbogen KA, Krahn AD, Latib A, Mack M, Mittal S *et al.* Management of conduction disturbances associated with transcatheter aortic valve replacement: JACC scientific expert panel. *J Am Coll Cardiol* 2019;**74**:1086–106.
2. Badertscher P, Knecht S, Zeljković I, Sticherling C, de Asmundis C, Conte G *et al.* Management of conduction disorders after transcatheter aortic valve implantation: results of the EHRA survey. *Europace* 2022;**24**:1179–85.
3. Glikson M, Nielsen JC, Kronborg MB, Michowitz Y, Auricchio A, Barbash IM *et al.* 2021 ESC guidelines on cardiac pacing and cardiac resynchronization therapy. *Europace* 2022; **24**:71–164.
4. Ravaux JM, Di Mauro M, Vernooy K, Kats S, Mariani S, Ronco D *et al.* Permanent pacemaker implantation following transcatheter aortic valve implantation using self-expandable, balloon-expandable, or mechanically expandable devices: a network meta-analysis. *Europace* 2021;**23**:1998–2009.
5. Surawicz B, Childers R, Deal BJ, Gettes LS. 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: endorsed by the international society for computerized electrocardiology. *Circulation* 2009;**119**:e235–40.
6. Vicent L, Fernández-Cordón C, Nombela-Franco L, Escobar-Robledo LA, Ayesta A, Ariza Solé A *et al.* Baseline ECG and prognosis after transcatheter aortic valve implantation: the role of interatrial block. *J Am Heart Assoc* 2020;**9**:e017624.
7. Fischer Q, Himbert D, Webb JG, Eltchaninoff H, Muñoz-García AJ, Tamburino C *et al.* Impact of preexisting left bundle branch block in transcatheter aortic valve replacement recipient. *Circ Cardiovasc Interv* 2018;**11**:e006927.
8. Upadhyay GA, Cherian T, Shatz DY, Beaser AD, Aziz Z, Ozcan C *et al.* Intracardiac delineation of septal conduction in left bundle-branch block patterns. *Circulation* 2019; **139**:1876–88.
9. Upadhyay GA, Tung R. Intermittent left bundle branch block: what is the mechanism? *J Cardiovasc Electrophysiol* 2019;**30**:1380–3.
10. Narula OS. Longitudinal dissociation in the His bundle: bundle branch block due to asynchronous conduction within the His bundle in man. *Circulation* 1977;**56**: 996–1006.
11. El-Sherif N, Amay-Y-Leon F, Schonfield C, Scherlag BJ, Rosen K, Lazzara R *et al.* Normalization of bundle branch block patterns by distal His bundle pacing: clinical and experimental evidence of longitudinal dissociation in the pathologic his bundle. *Circulation* 1978;**57**:473–83.
12. Padanilam BJ, Morris KE, Olson JA, Rippy JS, Walsh MN, Subramanian N *et al.* The surface electrocardiogram predicts risk of heart block during right heart catheterization in patients with preexisting left bundle branch block: implications for the definition of complete left bundle branch block. *J Cardiovasc Electrophysiol* 2010;**21**: 781–5.
13. Tovia-Brodie O, Ben-Haim Y, Joffe E, Finkelstein A, Glick A, Rosso R *et al.* The value of electrophysiologic study in decision-making regarding the need for pacemaker implantation after TAVI. *J Interv Card Electrophysiol* 2017;**48**:121–30.
14. Reiter C, Lambert T, Kellermair J, Blessberger H, Fellner A, Strasser B *et al.* Intraprocedural dynamics of cardiac conduction during transcatheter aortic valve implantation: assessment by simultaneous electrophysiological testing. *Heart Rhythm* 2021;**18**:419–25.
15. Zema MJ, Luminais SK, Chiaramida S, Goldman M, Kligfield P. Electrocardiographic poor R wave progression III. The normal variant. *J Electrocardiol* 1980;**13**: 135–42.