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CASE REPORT

CLINICAL CASE

Seventy Seconds of Asystole

Delayed Atrioventricular Block Post-TAVR From His-Bundle Injury and Phase 4 Block

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ABSTRACT

BACKGROUND Conduction system disturbance is a leading complication of transcatheter aortic valve replacement (TAVR). Conventional predictors of post-TAVR pacing include preexisting right bundle branch block, new left bundle branch block, and annular calcification.

CASE SUMMARY A 71-year-old woman with severe aortic stenosis and left bundle branch block (PR: 160 ms, QRS: 138 ms) underwent TAVR. The QRS paradoxically narrowed postprocedure (104 ms), and inpatient telemetry showed no high-grade atrioventricular block before discharge. Five days later, an outpatient real-time monitor captured 71 seconds of asystole due to complete heart block. She presented with syncope and underwent pacemaker implantation.

DISCUSSION The paradoxical QRS narrowing reflects His-bundle injury producing a proximal conduction delay in the His bundle, with recovery of conduction in the left bundle branch indicative of gap phenomenon. The recovery of conduction in the left bundle also highlights the dynamic nature of a diseased left bundle that can facilitate prolonged, pause-dependent complete heart block without a stable escape rhythm.

TAKE-HOME MESSAGES Delayed complete heart block after TAVR is rare, life-threatening, and may occur in the absence of conventional predictors. Real-time ambulatory monitoring enables timely diagnosis. (JACC Case Rep. 2026;■:110318) © 2026 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 71-year-old woman with severe, symptomatic aortic stenosis (AS) and multivessel coronary artery disease was referred for transcatheter aortic valve replacement (TAVR). She reported NYHA functional class II exertional dyspnea in the prior 2 weeks. Her baseline 12-lead electrocardiogram (ECG) showed sinus rhythm with left bundle branch block (LBBB), a PR interval of 160 ms, and a QRS duration of 138 ms (**Figure 1A**).

On examination, she was euvolemic, with a normal blood pressure and a regular pulse. Cardiac auscul-

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ABBREVIATIONS AND ACRONYMS

AS = aortic stenosis

AV = atrioventricular

CHB = complete heart block

ECG = electrocardiogram

LBBB = left bundle branch block

TAVR = transcatheter aortic valve replacement

tation revealed a grade 3/6 systolic murmur at the right upper sternal border.

PAST MEDICAL HISTORY

The patient's medical history included coronary artery disease treated with percutaneous coronary intervention of the mid left anterior descending artery. Staged revascularization of the left circumflex and right posterior descending arteries was planned after TAVR. She also had type 2 diabetes mellitus and peripheral vascular disease. She had undergone an ablation of a left lateral accessory pathway for orthodromic atrioventricular re-entrant tachycardia, without recurrence.

DIFFERENTIAL DIAGNOSIS

When 71 seconds of paroxysmal complete heart block (CHB) was detected, the leading diagnosis was delayed high-grade atrioventricular (AV) block from TAVR-related His-Purkinje injury. Neurocardiogenic syncope and vagal-mediated CHB were inconsistent with the stability of the preceding P-P intervals and clinical history. An ischemic precipitant was unlikely given a troponin I level of 0.027 ng/mL on presentation to the emergency department. Cardiac amyloidosis could be considered given the small, hypertrophied left ventricle (LV) and severe AS without severe annular calcification. However, the baseline ECG showed normal QRS voltages, making this diagnosis less likely.

INVESTIGATIONS

Preprocedural transthoracic echocardiography showed a small LV (internal diameter: 3.5 cm) with preserved ejection fraction (71%). The aortic valve area was calculated as 1.33 cm^2 with a mean gradient of 21 mm Hg, a dimensionless index of 0.32, and a normal stroke volume index (37.2 mL/m^2). Because of the small LV cavity and discordance of AS severity measurements with her symptoms, an invasive valve study was performed, which confirmed low-gradient severe AS: aortic valve area 0.81 cm^2 and mean gradient 35 mm Hg, with a normal cardiac index. Gated cardiac computed tomography showed an aortic annulus area of 412 mm^2 and a valve calcium score of 1,074 Agatston units (below the sex-specific threshold of 1,200 for women).[1] The preprocedural assessment and the absence of conventional conduction-risk predictors are summarized in Table 1. Five days after TAVR, a real-time ambulatory monitor recorded 71 seconds of ventricular asystole.

TAKE-HOME MESSAGES

- After TAVR, paradoxical narrowing of a baseline left bundle branch block can be concerning rather than reassuring, as it can reflect His-bundle injury. This injury can result in a proximal conduction delay in the His bundle, with recovery of conduction in the left bundle branch indicative of gap phenomenon. The recovery of conduction in the left bundle also highlights the dynamic nature of a diseased left bundle that may produce prolonged pause-dependent CHB without a stable escape rhythm even when conventional predictors of post-TAVR CHB are absent.
- Real-time outpatient rhythm monitoring is essential for the timely detection and treatment of delayed, potentially life-threatening atrioventricular block after TAVR.

Regular sinus P waves at a P-P interval of 710 ms were followed by a premature P wave (coupling interval 569 ms). Every subsequent sinus P wave then failed to conduct throughout the 71-second window, producing sustained complete AV block without an escape rhythm (Figure 2).

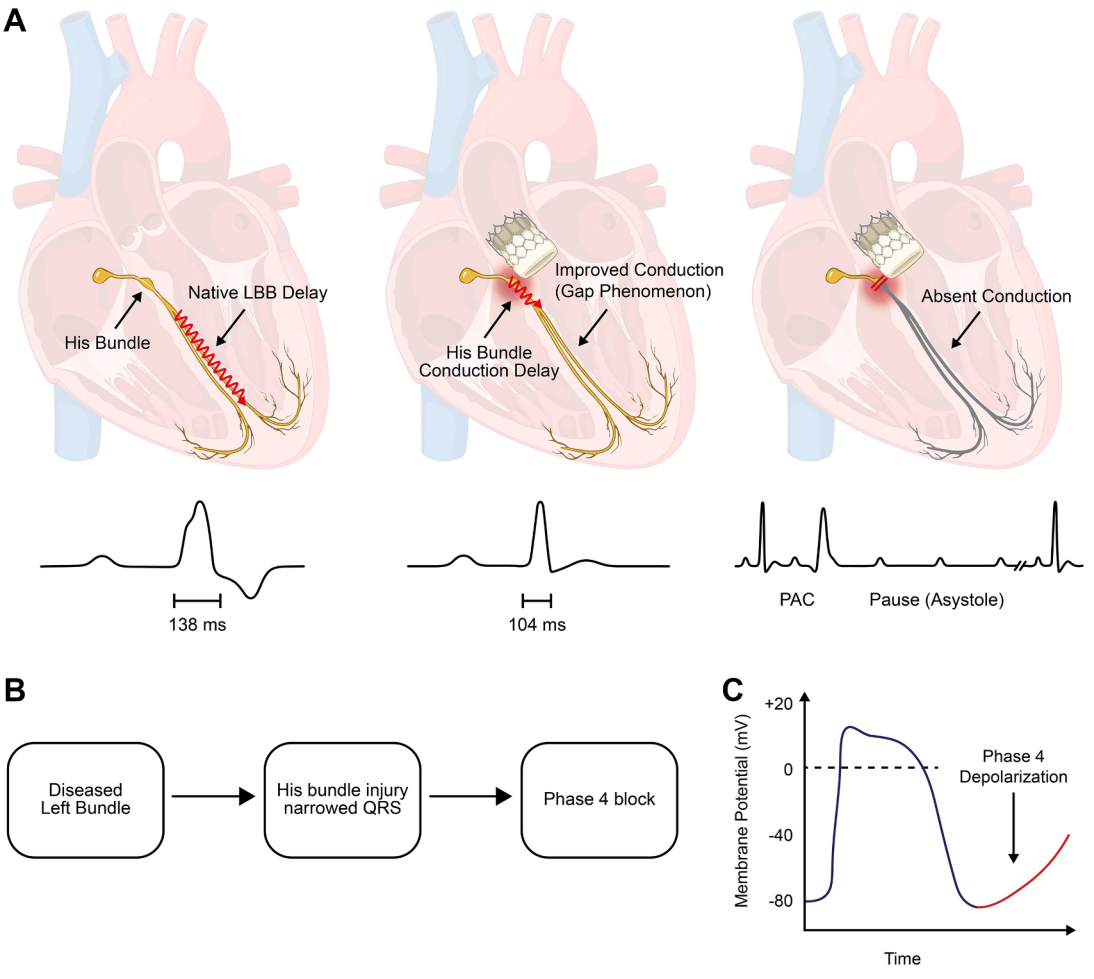
MANAGEMENT

The patient underwent TAVR with a 23-mm Edwards SAPIEN 3 Ultra RESILIA balloon-expandable bioprosthesis (Figure 3). During valve deployment, transient QRS widening to 140 ms with a left bundle branch morphology was noted, but this resolved by the end of the case. The postprocedural ECG showed narrowing of the QRS to 104 ms, with the LBBB resolving into a nonspecific intraventricular conduction delay and a normal PR interval of 148 ms (Figure 1B). She remained free of high-grade AV block on inpatient telemetry and was discharged on postoperative day 1 with a real-time ambulatory monitor. On postoperative day 5, the monitoring service issued an automatic alert for asystole. By then, she had already presented to the emergency department with syncope. In the emergency department, she had multiple episodes of paroxysmal CHB, and a temporary pacing wire was placed.

OUTCOME AND FOLLOW-UP

Despite a 71-second pause and recurrent syncope, the patient arrived alert with a normal neurologic examination and full return of consciousness between

VISUAL SUMMARY **Proposed Mechanism: His-Bundle Injury and the Gap Phenomenon**



(A) Pre-TAVR: Conduction delay in a diseased left bundle produces left bundle branch block (wide QRS, 138 ms). (B) Immediate post-TAVR: His-bundle injury slows proximal conduction, allowing both bundles to recover and conduct, resynchronizing ventricular activation and paradoxically narrowing the QRS to 104 ms (gap phenomenon)—a marker of His-bundle injury, not recovery. (C) Phase 4 complete heart block: After a premature atrial contraction and pause, diastolic (phase 4) depolarization raises the resting membrane potential above the excitable threshold, abolishing infra-His conduction and leaving an unstable escape rhythm. LBB = left bundle branch; PAC = premature atrial contraction; TAVR = transcatheter aortic valve replacement.

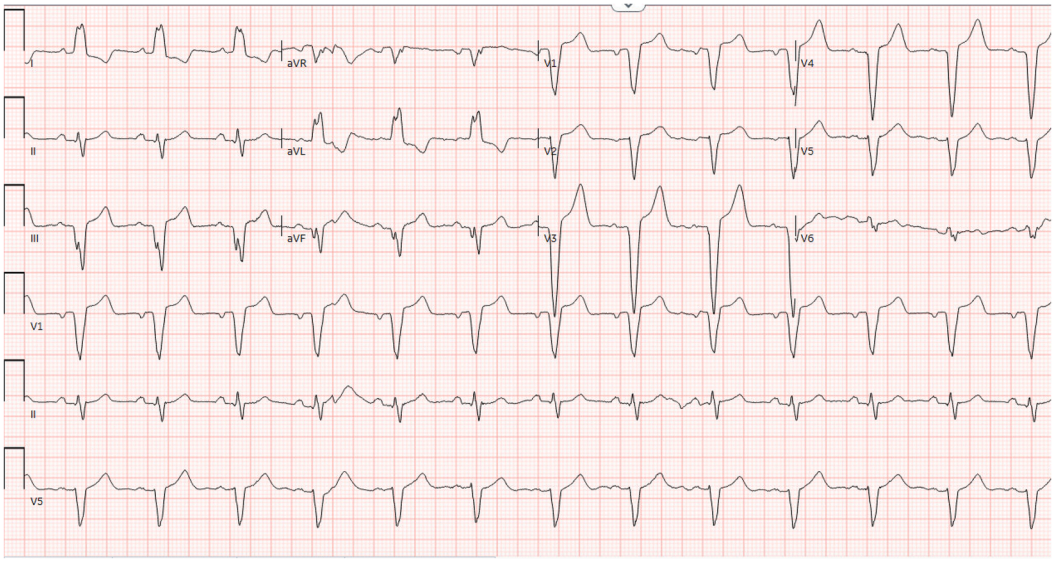
episodes of CHB. She underwent uncomplicated dual-chamber pacemaker implantation and was discharged in stable condition with no further syncope.

Device interrogation was performed 1 month after pacemaker implantation. Because her implantation was performed elsewhere, we reviewed her settings when she presented for elective percutaneous coronary intervention. The ventricular pacing burden was 76.3%, but her pacemaker settings included a sensed AV delay of 110 ms. When ventricular pacing was inhibited, the underlying rhythm was sinus at approximately 77 beats/min, with intact AV conduction and a normal AV interval of 130 to 140 ms. We reprogrammed her device to a physiologic sensed AV delay of 220 ms. This high pacing requirement, combined with her normal AV interval when she does conduct, could indicate ongoing paroxysmal CHB, although this is difficult to ascertain with her initial programmed AV delay of 110 ms. Importantly, pacing burden can be low in paroxysmal AV block, as intermittent episodes of CHB can be life-threatening yet only require short periods of ventricular pacing.

FIGURE 1 12-Lead Electrocardiograms Before and After TAVR

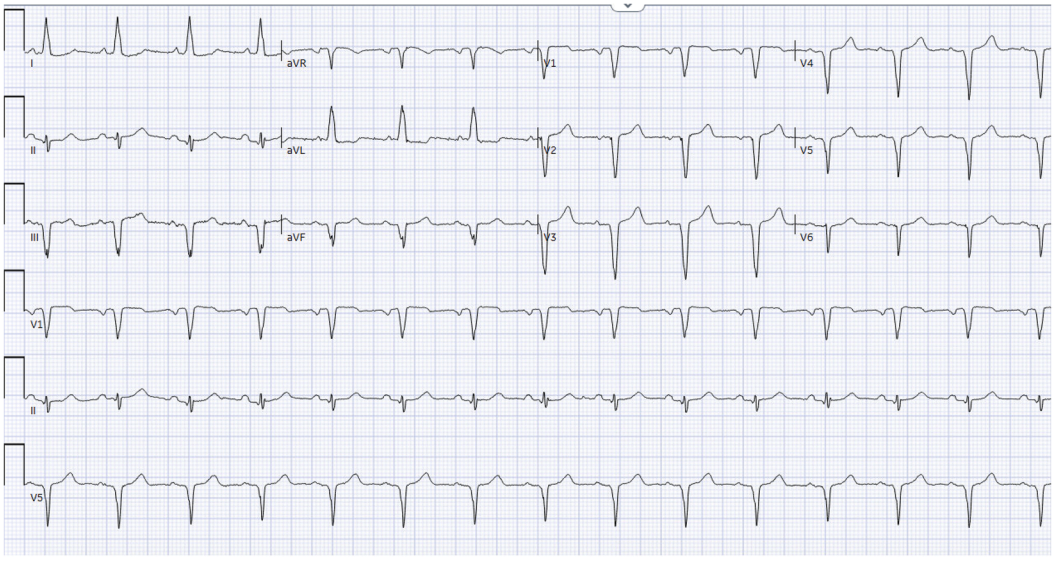
A **Before TAVR**

Sinus rhythm with LBBB (QRS 138 ms, PR 160 ms)



B **After TAVR (prior to discharge)**

Sinus rhythm with IVCD (QRS 104 ms, PR 148 ms)



(A) Baseline: sinus rhythm, PR 160 ms, LBBB with QRS 138 ms. (B) Post-TAVR: paradoxical QRS narrowing to 104 ms with resolution of LBBB into a nonspecific intraventricular conduction delay and poor R-wave progression (V_1-V_4); normal PR interval of 148 ms.
IVCD = intraventricular conduction delay; LBBB = left bundle branch block; TAVR = transcatheter aortic valve replacement.

TABLE 1 Preprocedural and Procedural Assessment, Highlighting the Absence of Conventional Conduction-Risk Predictors

Domain	Finding	Conduction-Risk Implication
Baseline ECG	• Sinus rhythm • LBBB (no RBBB) • PR 160 ms; QRS 138 ms	Only LBBB present (weaker predictor than RBBB)
Post-TAVR ECG	QRS narrowed to 104 ms (LBBB → nonspecific IVCD); PR 148 ms	Concerning, not reassuring—QRS narrowing reflects His-bundle injury facilitating left bundle repolarization (gap phenomenon)
Echocardiography	Small LV (3.5 cm), EF 71%, concentric LVH; AVA 1.33 cm^2 → 0.81 cm^2 (invasive); mean gradient 21→35 mm Hg	No high-risk conduction feature
Invasive hemodynamics	AVA 0.81 cm^2, mean gradient 35 mm Hg, LVEDP 18 mm Hg, normal CI	Confirmed severe low-gradient AS
Coronary angiography	Two-vessel disease; patent prior LAD stent	
Gated cardiac CT	Annulus 412 mm^2 (22.9 mm); valve calcium score 1,074 (subsevere); RCA ostial height 11 mm	No heavy annular calcification
Procedure	23-mm Edwards SAPIEN 3 Ultra RESILIA; modest oversizing; transient QRS 140 ms, resolved	Balloon-expandable; no high-grade block
Inpatient telemetry	No evidence of AV block; discharged postoperative day 1	Benign

AS = aortic stenosis; AV = atrioventricular; AVA = aortic valve area; CI = cardiac index; CT = computed tomography; ECG = electrocardiogram; EF = ejection fraction; IVCD = intraventricular conduction delay; LAD = left anterior descending artery; LBBB = left bundle branch block; LV = left ventricle; LVEDP = left ventricular end-diastolic pressure; LVH = left ventricular hypertrophy; QRS = QRS duration; RBBB = right bundle branch block; RCA = right coronary artery; TAVR = transcatheter aortic valve replacement.

Therefore, a low pacing burden should not be taken as evidence against clinically significant conduction system disease.

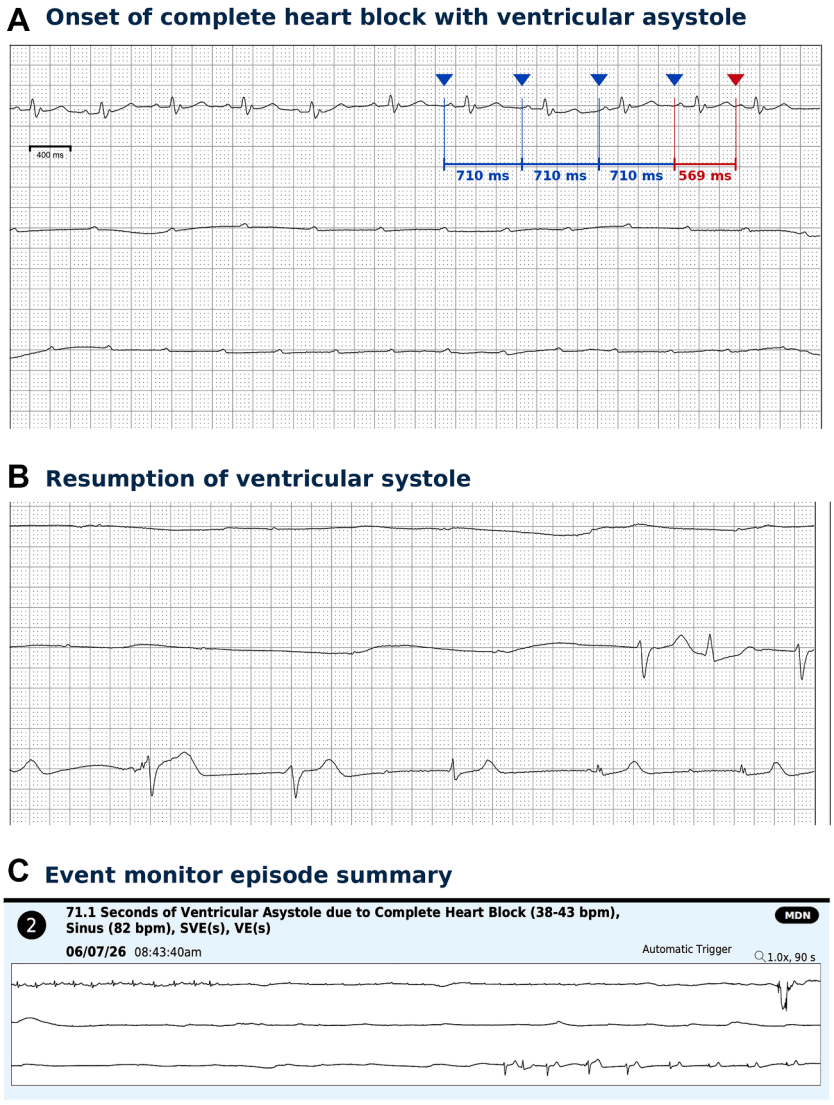
DISCUSSION

This case shows that the conventional predictors used to triage conduction risk after TAVR can be absent in a patient who develops delayed, life-threatening paroxysmal CHB. Contemporary expert-consensus pathways stratify patients largely by the baseline and postprocedural ECG. Relevant markers include preexisting right bundle branch block, PR prolongation, a new or widening LBBB, and high-grade block on inpatient monitoring after TAVR. These are supplemented by anatomic and procedural markers such as heavy calcification, deep implantation, and oversizing.[2,3] The American College of Cardiology expert-consensus decision pathway recommends permanent pacemaker implantation for high-grade AV block after TAVR, whereas an isolated new LBBB is managed with ambulatory monitoring or electrophysiology study.[3] Our patient lacked nearly all of these. Her sole marker was LBBB, which is a weaker predictor of permanent pacing and surprisingly regressed after the procedure. By every postprocedural criterion, she would be classified as low risk.

The crux of this case is that paradoxical QRS narrowing after TAVR is concerning, not reassuring. The His bundle and left bundle run through the membranous septum just beneath the aortic annulus, where expansion of the valve can cause direct injury. During valve deployment, the QRS widened transiently to 140 ms, indicative of acute His-Purkinje injury. We attribute the subsequent narrowing of our patient's QRS before discharge to the gap phenomenon. Periprocedural injury caused slow conduction in the His bundle. This proximal delay gave the diseased left bundle time to recover and conduct, narrowing the QRS.[4] This means the baseline LBBB was not a fixed anatomic block but rather a functional block in a diseased left bundle. The same principle underlies longitudinal dissociation in the His bundle, in which LBBB can be affected by conduction changes in the His bundle.[5] Because no invasive His-bundle electrograms were obtained, this mechanism is inferred from the surface ECG and clinical course rather than directly demonstrated. This mechanism is illustrated as a ladder diagram (**Figure 4**).

Phase 4 (bradycardia-dependent) block explains the features of asystole captured on the event monitor. In diseased Purkinje fibers, abnormal spontaneous diastolic depolarization drifts the membrane toward less negative potentials during prolonged diastole. When a premature atrial contraction is followed by a compensatory pause, the next sinus impulse arrives to find fast sodium channels in the diseased conduction system partially inactivated. This abolishes the action potential upstroke and causes conduction failure. Unlike phase 3 (tachycardia-dependent) block, phase 4 block is facilitated by bradycardia and pauses created by an extrasystole. The premature beat need not conduct aberrantly. It simply generates the pause that

FIGURE 2 Real-Time Ambulatory Monitor at the Event



(A) Onset of complete heart block with ventricular asystole: regular sinus P waves march at a P-P interval of 710 ms (blue calipers); the terminal P-wave is premature (red caliper; coupling interval: 569 ms, measured against the 400-ms calibration bar) and is followed by complete heart block with an absent escape rhythm and prolonged ventricular asystole. (B) Resumption of ventricular systole with ventricular escape. (C) Zio event-monitor episode summary: an automatically triggered 90-second report documenting 71.1 seconds of ventricular asystole due to complete heart block.

permits phase 4 depolarization of the diseased His-Purkinje system and conduction failure.

The duration of asystole in phase 4 block is intrinsically unpredictable. It is governed less by the inciting pause than by the latency and stability of a subsidiary escape rhythm, so symptoms range from none to syncope.[6,7] In prior reports of phase 4 block, the documented asystolic intervals are typically only a few seconds, enough to cause presyncope or syncope.[6,7] To our knowledge, no prior report describes a pause approaching the 71 seconds captured here.

Remarkably, our patient tolerated the documented 71-second pause and was neurologically intact, despite the potential for hypoxic-ischemic injury from asystole of this duration.

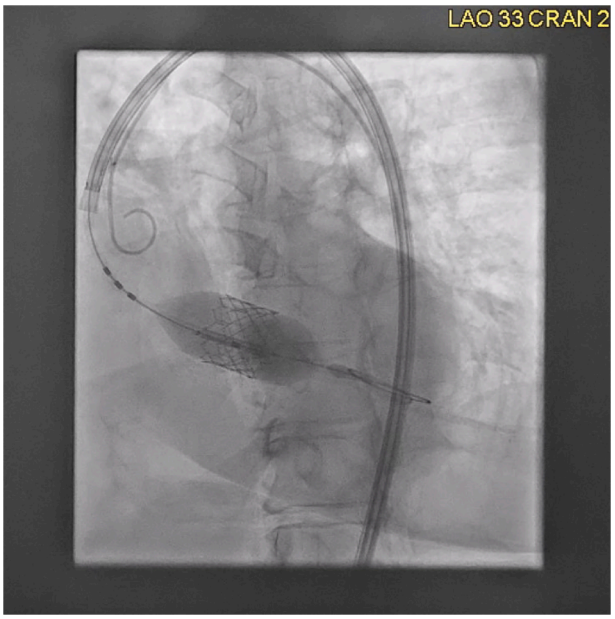
The management implications are 2-fold. First, this patient is difficult to triage. Her reassuring postprocedural ECG and absent anatomic and

telemetry predictors are falsely reassuring. The intraprocedural QRS widening and subsequent gap phenomenon QRS narrowing confound assessment of post-TAVR CHB risk. Second, the diagnosis depended on real-time ambulatory monitoring. Studies using implantable loop recorders show that high-grade AV block can be detected weeks after TAVR and is frequently missed by in-hospital telemetry.[8] This underscores the need for longitudinal rhythm monitoring in these patients. Different outpatient monitoring strategies after TAVR should be prospectively evaluated to expedite rescue pacing in those with delayed complete AV block and unstable bradyarrhythmias.[9]

CONCLUSIONS

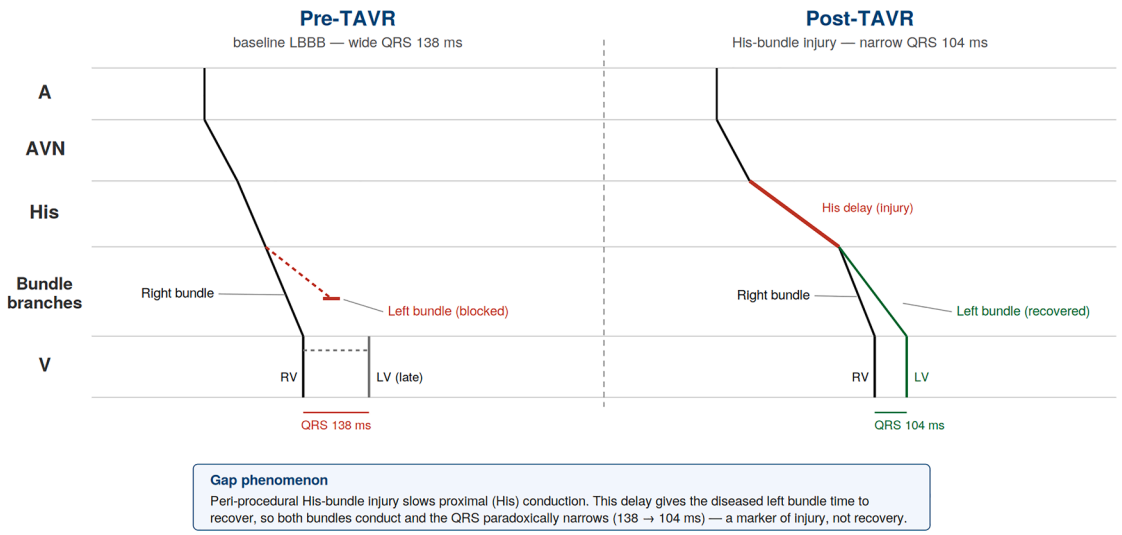
Paroxysmal CHB after TAVR can occur despite the absence of conventional predictors through bradycardia-dependent (phase 4) block in an injured His-Purkinje system. In a patient with baseline LBBB, a paradoxically narrowed QRS after TAVR can be concerning rather than reassuring. It may reflect His-bundle injury producing a gap phenomenon, and it highlights a diseased conduction system at risk for phase 4 block. Real-time ambulatory monitoring enables prompt recognition and treatment.

FIGURE 3 Fluoroscopic Image (LAO 33°, Cranial 2°) During Transcatheter Aortic Valve Replacement Showing Deployment of the 23-mm Edwards SAPIEN 3 Ultra RESILIA Balloon-Expandable Bioprosthesis Across the Aortic Annulus



LAO = left anterior oblique.

FIGURE 4 Ladder Diagram of the Gap Phenomenon in This Case



(Left) Pre-TAVR: The diseased left bundle is functionally blocked while the right bundle conducts, so the left ventricle is activated late and the QRS is wide (baseline left bundle branch block, 138 ms). (Right) Post-TAVR: Periprocedural His-bundle injury slows proximal conduction; this delay allows the diseased left bundle to recover so that both bundles conduct, paradoxically narrowing the QRS to 104 ms—a marker of His-bundle injury rather than recovery. AVN = atrioventricular node; LBBB = left bundle branch block; LV = left ventricle; QRS = QRS complex; RV = right ventricle; TAVR = transcatheter aortic valve replacement.

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KEY WORDS ambulatory rhythm monitoring, complete heart block, His-Purkinje conduction, phase 4 (bradycardia-dependent) block, premature atrial contraction, transcatheter aortic valve replacement