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EDITORIAL COMMENT

Precision Medicine With Individualized Clinical Surveillance After TAVR

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Conduction disorders and need for permanent pacemakers are more frequent after transcatheter aortic valve replacement (TAVR) than after surgical aortic valve replacement.[1,2] The atrioventricular bundle of His surfaces along the transition from the membranous to the muscular part of the interventricular septum underneath the noncoronary cusp.[3] Conceivably, depending on the position of the transcatheter valve stent frame, direct contact pressure and trauma can impinge on the conduction system after TAVR. Deeper transcatheter valve implantation and a shorter membranous septum (<3 mm), calcium distribution in the left ventricular outflow tract, and pre-existing conduction disorders (in particular right bundle branch block) predispose to new conduction disorders and high-grade atrioventricular blocks that require permanent pacemaker implantation following TAVR.[4,5]

New-onset left bundle branch block (LBBB) and need for permanent pacemaker implantation after TAVR have been linked to cardiovascular mortality and rehospitalization.[6] However, these conduction disorders may be transient and arguably become clinically irrelevant after resolution. Indeed, 2 of 3 patients who developed new LBBB or received new pacemakers after TAVR appear to have restored QRS duration or are no longer pacemaker dependent at 1 year follow-up.[7,8]

In this issue of *JACC: Cardiovascular Interventions*, Benavent-García et al[9] report on the clinical impact of new-onset persistent LBBB (NOP-LBBB) after TAVR. NOP-LBBB was determined by electrocardiography (ECG) at hospital discharge. Patients with LBBB or permanent pacemakers at baseline and those who died or received permanent pacemakers within the first 30 days after TAVR were excluded. The final study cohort included 1,052 patients who underwent TAVR between 2007 and 2020. The mean age was 78 ± 8 years, there was an equal distribution between men and women, and the median Society of Thoracic Surgery Predicted Risk of Mortality was 4.3% (Q1-Q3: 2.8%-6.6%). One-quarter of the study population underwent TAVR with self-expanding valves and the remainder with balloon-expandable valves. Approximately 30% of the overall TAVR cohort developed NOP-LBBB. The median follow-up time was 5 years (Q1-Q3: 3-6 years). Patients with NOP-LBBB had a higher incidence of the primary composite endpoint of cardiovascular mortality or heart failure (HF) hospitalization (37.5% vs 31.5%; adjusted HR [aHR]: 1.37; 95% CI: 1.09-1.73; $P = 0.007$), which was driven by an incremental cardiovascular mortality risk (30.5% vs 23.8%; aHR: 1.46; 95% CI: 1.12-1.89; $P = 0.004$). Sudden death (4.2% vs 1.5%; aHR: 3.52; 95% CI: 1.53-8.14; $P = 0.003$) and left ventricular ejection fraction (LVEF) decline by ≥10% occurred more in patients with vs without NOP-LBBB. Of note, there was no difference in all-cause mortality or HF hospitalization between patients with vs without NOP-LBBB.

The overall elderly patient cohort with significant comorbidities, intermediate operative risk, and the use of alternative-access TAVR in one-quarter of cases differs from contemporary TAVR practice. The 60% all-cause mortality rate at the median follow-up of 5 years attests to the higher risk phenotype and may further blur the effects NOP-LBBB may have in a lower risk and younger cohort.

The association between LBBB and cardiovascular mortality, sudden death, and LVEF decline makes

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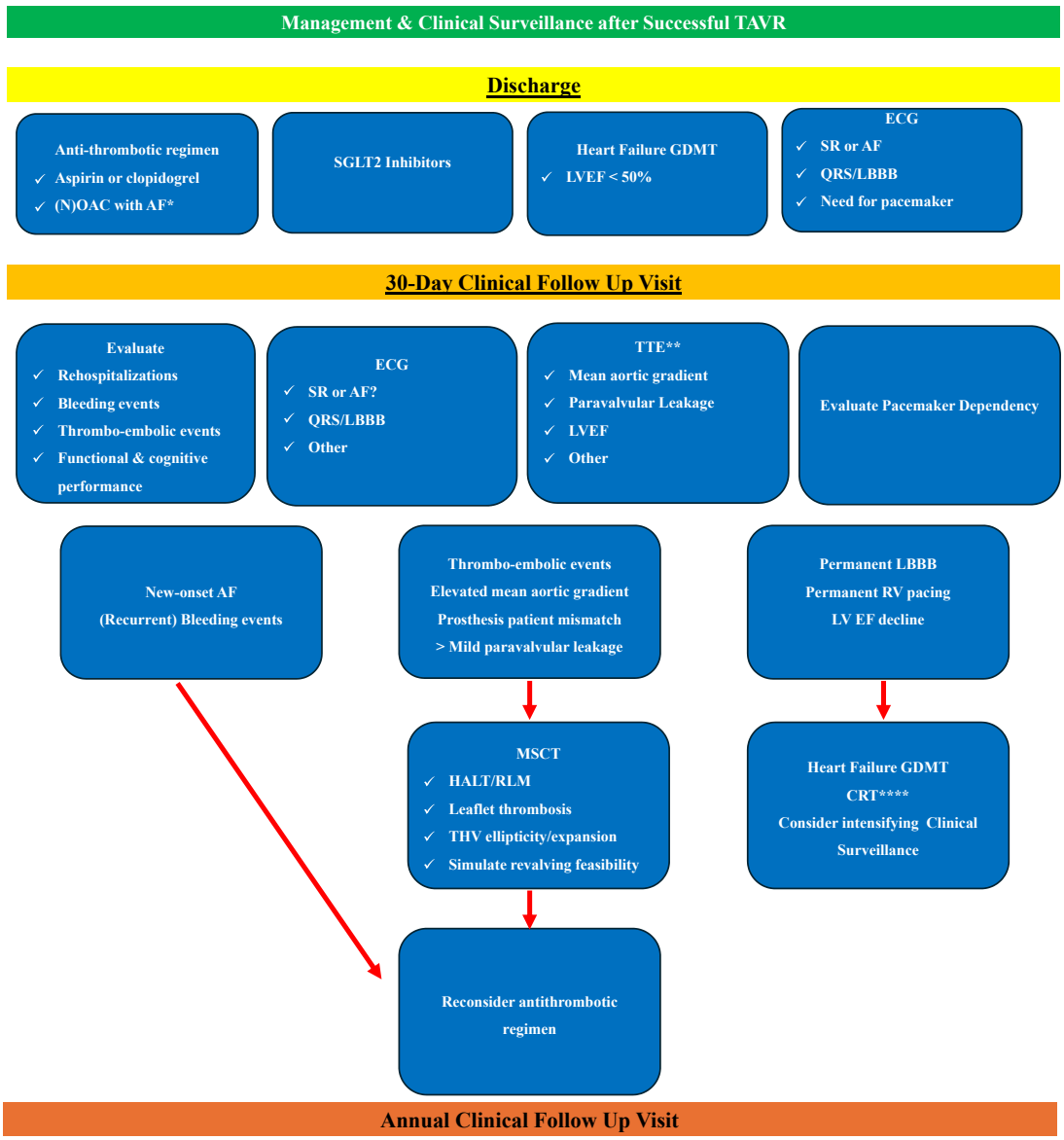
FIGURE 1 Management and Clinical Surveillance After TAVR



AF = atrial fibrillation; CRT = cardiac resynchronization therapy; ECG = electrocardiography; GDMT = guideline-directed medical therapy; HALT = hypoattenuated leaflet thickening; LBBB = left bundle branch block; LV EF = left ventricular ejection fraction; MSCT = multislice computed tomography; (N)OAC = (non-vitamin K) oral anticoagulant agent; OAC = oral anticoagulant agent; RLM = reduced leaflet mobility; RV = right ventricular; SGLT2 = sodium-glucose cotransporter 2; SR = sinus rhythm; TAVR = transcatheter aortic valve replacement; THV = transcatheter heart valve; TTE = transthoracic echocardiography. *Or other formal indication for oral anticoagulation (eg, mechanical valve prosthesis, thromboembolic disease). **Optional and mandatory with clinical suspicion (eg, heart failure symptoms, thromboembolic event). ***Simulate redo TAVR. ****Consider CRT when LVEF is <40% and LBBB is permanent.

total sense from a mechanistic point of view and is in keeping with observations related to incident LBBB in the general population.[10] However, the patient cohort could have been refined further. NOP-LBBB was determined according to ECG before discharge after the index TAVR procedure, even though ECG was performed at the 30-day visit in the clinic. Yet the literature suggests that acquired LBBB after TAVR will persist on ECG at 30-day follow-up in only 35% to 45% of patients.[7,8] LBBB that is detected on ECG at 30-day follow-up will likely be permanent. This dissociation between the definition of NOP-LBBB on the basis of predischarge ECG and the landmark analysis of clinical events starting only at 30 days is a pity, as more than 50% of the patients with NOP-LBBB in this study are expected to have normal QRS duration by 30 days, which obviously has clinical consequences. The detrimental effects of LBBB due to the consequent electrical dys-synchrony with abnormal septal motion, increased wall stress, asymmetrical hypertrophy, and impaired reverse remodeling obviously require a long time to emerge clinically.

Conceivably, the data presented by Benavent-Garcia et al[9] may underestimate the clinical impact of truly permanent NOP-LBBB (adjudicated at 30 days), which may indeed have significantly higher risk not only for cardiovascular and sudden death but even for all-cause mortality and HF hospitalization. There seems to be an opportunity to further improve quality of life and long-term survival by identifying permanent NOP-LBBB, especially when the LVEF declines. These patients should be universally considered HF patients who may benefit from the 4 pillars of guideline-directed medical therapy and cardiac resynchronization therapy.[11] Favorable effects of angiotensin-converting enzyme inhibition and sodium-glucose cotransporter 2 inhibition highlight the importance of continued medical optimization after TAVR.[12,13] The value of cardiac resynchronization therapy and left bundle branch pacing in patients with permanent LBBB after TAVR, especially in combination with LVEF decline, merits further investigation in properly powered randomized controlled trials.

Other potential targets to improve longer term outcomes after TAVR but beyond the topic of acquired conduction disorders revolve around transcatheter valve implantation techniques and early leaflet thickening. A quest for optimal transcatheter valve expansion and circularity and refined antithrombotic regimens to overcome premature leaflet thickening may preserve overall valve performance and postpone structural valve degeneration.

Finally, there remains a void in common language and consensus definitions for acquired conditions after TAVR. Three consecutive Valve Academic Research Consortium efforts achieved an established track record in harmonizing endpoint definitions, which have been widely adopted and have facilitated study interpretation and comparison.[14] Herein lies a new task for a fourth Valve Academic Research Consortium initiative to provide a proper framework and guidance on how to define, treat, and organize clinical follow-up of acquired clinical entities with the likes of new conduction disorders, pacemaker dependency, and leaflet thickening. In 2026, precision medicine should be reactive and flexible to accommodate changing clinical realities and trigger patient-tailored clinical surveillance and clinical management after TAVR (**Figure 1**). The continued goal is to maximize transcatheter durability and patient longevity, from timely patient recruitment, procedural planning, transcatheter valve platform selection, and implantation on through the continued journey during follow-up.

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