


EDITORIAL

Conduction Abnormalities after Transcatheter Aortic Valve Replacement: Pretty Common, Fairly Predictable, Barely Avoidable

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Editorial related to the article: Incidence of Conduction Disorders and Requirements for Permanent Pacemaker After Transcatheter Aortic Valve Implantation

The cardiac conduction system is commonly diseased in patients with aortic valve disease, and the site of conduction defect in those with severe calcific aortic valve stenosis is most commonly the His bundle or infra-Hisian.^{1,2} The bundle of His traverses the membranous septum towards the left ventricular outflow tract (LVOT) running superficially over the crest of the ventricular septum, originating the left bundle branch. This anatomical course makes the His bundle and its left branch susceptible to mechanical injury during transcatheter aortic valve replacement (TAVR), which involves multiple mechanical manipulations with wires, catheters, balloons, and the transcatheter heart valve (THV) inflow within that vicinity. Atrioventricular and intra-ventricular conduction abnormalities are indeed a common finding in patients with severe aortic valve stenosis referred to TAVR and new onset persistent conduction abnormalities (NOPCAs) are a common complication arising during/ after TAVR.

Unlike other periprocedural complications of TAVR, whose incidence has decreased over time, a regression in the incidence of NOPCAs after TAVR was neither seen with the introduction of new TAVR technologies/ techniques³ nor it is foreseeable in the near future.

Keywords

Aortic Valve Stenosis/ complications; Aortic Valve Stenosis/ physiopathology; Arrhythmias, Cardiac; Bundle-Branch Block/ etiology; Transcatheter Aortic Valve Replacement/ adverse effects; Pacemaker, Artificial.

The realization of the prognostic value of NOPCAs and new permanent pacemaker implantation (NPPI) after TAVR went through different phases that correspond to the main phases of TAVR evolution. When TAVR was introduced as a bail out option for extremely morbid patients who – otherwise – have no definitive therapeutic options, NOPCAs/NPPI were seen as an affordable price to pay compared with the extremely high mortality of those patients if managed conservatively. When TAVR then became an option to those who can undergo surgery – albeit at high risk – NOPCAs/NPPI stimulated some scrutiny, but the high risk of surgery led to continued underestimation of the problem. Today, as TAVR is considered an option even in low-risk patients, NOPCAs/NPPI are seen as a clear downside of TAVR as compared to surgery. Accumulating evidence suggests that NOPCAs (especially left bundle branch block — LBBB) and NPPI are still common complications of contemporary TAVR and are associated with impaired left ventricular reverse remodeling and increased heart failure hospitalizations and cardiac death after TAVR.^{4,5} In this issue of the International Journal of Cardiovascular Sciences, the study by Santos et al.,⁶ confirms that NOPCAs and NPPI continue to occur at exceedingly high rates after TAVR, especially with the Medtronic CoreValve. Similar to previous studies that involved relatively small sample size of (very) high risk patients and relatively short follow-up, the authors did not find a significant negative impact of NOPCAs on survival. Beyond incidence and prognostic value, the study explored the predictors of NOPCAs. Identification of the causes/ predictors of post-TAVR NOPCAs is of

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paramount importance for establishing strategies to reduce the incidence of NOPCAs/NPPI after TAVR. However, this process is challenging, basically due to the diversity and the interplay of offending factors. Those factors/mechanisms can be broadly classified into patient-related, THV-related, and implantation technique-related. Patient-related factors include landing-zone anatomical features (e.g. LVOT calcium load and distribution) and anatomical and functional characteristics of the conduction system (e.g. length and course of the His bundle and pre-existing atrioventricular or intraventricular conduction abnormalities). Device-related factors include THV platform (e.g. self- vs. balloon- vs. mechanically-expanding), device size, and extent of LVOT overstretch (a function of device oversizing). Implantation technique factors include balloon pre-dilatation and device implantation depth. Among those factors, baseline conduction defects (especially first degree heart block and right bundle branch block) and THV platform are the two most consistently identified predictors of NOPCAs/NPPI across different cohorts.⁷ Beyond those causative/predisposing factors, a major “confounding” factor contributing to NPPI rate is the clinical threshold of the operator. Operators with low thresholds to implant a permanent pacemaker (e.g. those adopting preemptive pacemaker implantation to facilitate early discharge in patients with transient atrioventricular block, new onset LBBB, or tachy-brady syndrome) usually have a considerably higher NPPI rate than their conservative peers, while adherence to guidelines on cardiac pacing leads to reduced NPPI rate after TAVR.⁸

Back to the real “causative/predisposing” factors, as clinicians, we should always focus more on the modifiable factors. Amongst these, a deeper device implant is an established risk factor for the development of NOPCAs and need for NPPI. Although not addressed by Santos et al.,⁶ deeper (more ventricular) implantation has been shown to increase the rate of NOPCAs and NPPI with most THV platforms. While previous

studies have suggested cut-points for desirable implantation depth, the concept of absolute cut-points may be overly simplified. A more logical concept would rather entail an individualized “optimal” implantation depth, which considers the anatomical vulnerability of the conduction system to mechanical injury during procedural steps. For example, in patients with left-sided atrioventricular bundle and/or short membranous septum (denoting a short distance between the aortic annulus and the bundle of His and its left branch), a shallower implant is required to avoid NOPCAs, while those with no such vulnerability may tolerate a relatively deeper implant. Beyond optimizing implantation depth, avoidance of unnecessary negative chronotropic agents periprocedurally, omitting unnecessary balloon pre-dilatation, and refraining from excessive THV oversizing (leading to overstretch of the LVOT) are further “pacemaker-sparing” strategies. The administration of anti-inflammatory/anti-edematous agents to prevent/relieve edema, hematoma, and ischemic damage of the conductive system is an appealing approach, though supportive evidence is scarce.⁹⁻¹¹ As mentioned earlier, watchful waiting for 3–7 days before deciding upon the need for permanent pacemaker with strict adherence to pacing guidelines can effectively reduce the number of NPPIs.

TAVR has been a story of success, backed with enthusiastic motivation of clinicians to improve techniques and of manufacturers to improve technologies in order to address the limitations of TAVR. The progress in dealing with the problem of NOPCAs/NPPI has lagged behind those achievements, largely due to protracted uncertainty regarding its prognostic relevance. Today, as the interventional community is – after all – aware of the magnitude and importance of this problem, effective handling of this shortcoming is awaited. This will require motivation of the operators to adopt “pacemaker-sparing” practices and of the manufacturers to develop technologies that address this remaining limitation of contemporary TAVR.

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