

Permanent pacemaker implantation is never a benign complication after aortic valve replacement

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Since the first report of an in-man transcatheter aortic valve replacement (TAVR) in 2002 by Cribier et al. (1), its role in the management of severe aortic stenosis (AS) has grown rapidly. Recent interest in minimally invasive approaches, driven not only by cardiac surgeons and cardiologists but also by patients, has facilitated the widespread use of TAVR; its indications have now expanded to include intermediate-risk and even low-risk surgical patient groups (2-4). Numerous randomized controlled trials (RCTs) have been performed or are still on-going; most studies demonstrated that there were no significant differences in all-cause mortality in patients with high or intermediate surgical risk (2,5-7). One RCT even showed a significantly lower mortality rate in the TAVR group than in the surgical AVR (SAVR) group at 1 year after AVR (14.2% vs. 19.1%, P=0.04) (8).

However, procedural drawbacks of these 'minimally invasive' aortic valve procedures include higher risks of paravalvular leak and atrioventricular block requiring permanent pacemaker implantation (PPI) after procedures. Both balloon-expandable and self-expanding valves pose greater risks of PPI than does SAVR because of increased mechanical interaction between the devices and the conduction system generated by radial force (9). The incidence of PPI after TAVR varies among studies from 2% to 50% (10). Despite recent development of newergeneration devices and increased practitioner awareness of this serious complication, the incidence of PPI after procedures remains over 10% (9). Previous studies demonstrated conflicting results regarding the impact of PPI on mid-term outcomes after TAVR. A recent study showed a significantly increased hazard of 1-year all-cause mortality after PPI in TAVR patients (11). Another study insisted that PPI was just a benign complication that did not affect outcomes and was even a protective factor against the occurrence of unexpected death (12).

The negative impact of PPI on long-term clinical outcomes after SAVR or TAVR has several explanations (13-15): (I) ineffective mechanical work due to asynchronous activation of ventricular segments; (II) resultant regional hypo-perfusion in the septal area and progressive ventricular remodeling; (III) reduced cardiac output owing to atrioventricular dyssynchrony; (IV) late occurrence of mitral regurgitation induced by right ventricular pacing; and (V) infectious complications such as pocket site infection and lead infection.

In this recent study by Mehaffey and his colleagues (16), the authors raised this important issue once again. Because long-term data after TAVR are lacking, this study aimed to evaluate the detrimental impact of PPI on the longterm clinical outcomes in SAVR patients that could be extrapolated to the TAVR population. Their excellent results of overall PPI rate after SAVR of 2.7% should be congratulated. The authors showed that the PPI was

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associated with a 50% increase in long-term all-cause mortality. Main strengths of their study include the followings: (I) the authors included a sufficient sample size of 2,600 patients over a 15-year study period and (II) a median follow-up duration of 7.5 years is long enough to clearly elucidate long-term effects of PPI. There is nevertheless a caveat in this study; preoperative risk factors that could affect long-term all-cause mortality were adjusted in their multivariable analysis only by patient STS score. Therefore, there remains a possibility that their main findings were affected by confounding variables. Despite this issue, this study delivers a clear message: "Permanent pacemaker implantation after TAVR is not at all a benign complication".

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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