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INVITED EDITORIAL

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Pacemakers after valve replacement: Just because we can, should we?

"TO DO NOTHING IS ALSO A GOOD REMEDY"

Hippocrates

Permanent pacemaker (PM) implantation after heart valve surgery continues to be an important postoperative complication (Figure 1). The mechanism, time course, and degree of permanence for significant conduction system impairment is poorly-studied and poses challenges to the care of surgical patients. Posited mechanisms of injury include direct trauma, ischemia, or edema of the conduction system due to its proximity to the heart valves[1] (Figure 2).

Especially in the case of AV block, there has been considerable debate as to the timing of the PM implant. Pressured for shorter hospitalization times, we are less inclined to allow a patient to languish with temporary wires for 10 or even 15 days. As PM implantation becomes less of an ordeal, the threshold to implant earlier lowers. This effect is likely to be intensified as transcatheter PMs evolve. However, committing a patient to a permanent device is not risk-free. Transvenous leads have up to a 20% fracture rate at 10 years,[6] are a risk factor for recurrent valve deterioration after index valvular surgery,[2] and may be associated with increased long-term mortality after valve surgery.[7] When do we commit? Current recommendations regarding how long to wait are vague. European societies suggest waiting for 5 days before implantation, while U.S. guidelines only state to implant "when conduction is expected not to resolve."[3] One retrospective study examined the issue of implant timing and found that a patient with complete AVB within 24 h after surgery which persists for >48 h will have a very low likelihood of recovery and PM implantation would be necessary.[4]

In this issue, Ghodsi et al. randomize those with AV block after valvular surgery to dexamethasone 4 mg/kg for 3 days or normal care.[8] Steroids, as the authors indicate, have been shown to reduce ischemia-reperfusion injury, reduce inflammation and edema, and alter molecular/channel effects resulting in enhanced conduction. The rationale for this intervention was to hasten the recovery or "unmask" reversibly-injured conduction tissue in hopes of enhancing clinical decisions for PM implantation in the future. The intervention group, consisting of 69 patients (51% female with a majority undergoing either mitral or aortic valves) had a significantly higher recovery of conduction at postoperative days 5 and 7 (82% vs. 63% and 88% vs. 61%), had a shorter total amount of time in AVB, and shorter time in the ICU. Recovery rates at 10 days were similar (83% vs. 78%) suggesting that the steroids *hastened*, but did *not absolutely increase*, recovery of conduction. Notably, this trial did not have pre-specified guidance on PM implantation, which was left to physician discretion. As a result, PM implantation rates between the two groups were similar. Major adverse events, which astutely included uncontrolled hyperglycemia and a host of other potentially steroid-related complications, did not differ significantly between the two groups.

A busy clinical electrophysiologist may ask, "Why not just put in the PM on day 5 and be done with it without steroids?" The data from this prospective study would argue that an additional 16% (62.9% vs. 78.6%) of patients that received the PM with this strategy would have recovered conduction. Only when given steroids would there be a relative assurance that implanting at Day 5 would not "condemn" any patients who may then go on to recover at Day 10 onward (82.6% vs. 83%) to live with a permanent pacer. The findings from this study demonstrate that a course of dexamethasone accelerates the recovery of reversibly-injured conduction systems and could thus clarify and, more importantly, expedite a decision to implant a device. For example, a course of steroids would be given for postoperative heart block for 3 days and, if there were no recovery, a device could be implanted on or before Day 5. Alternatively, if there were recovery the implant could be deferred. Before putting this to practice, waiting for replication and for data from a prespecified PM implant strategy after steroid may be prudent.

Ghodsi et al. should be commended for the randomized prospective study design—a feature lacking in many clinical arrhythmia investigations, especially for this topic. Limitations include the absence of data available on the subsequent long-term PM dependence of those undergoing implant and the absence of a prespecified clinical decision rule to implant systems based on response to steroids. Other questions this study raises include the issue of applicability to the expanding transcatheter valvular population and at what point a patient can be reasonably assured AV block will not recur. Nonetheless, the creative use of an old medicine to an important complication sheds brighter, and earlier, light on an ongoing postoperative dilemma.

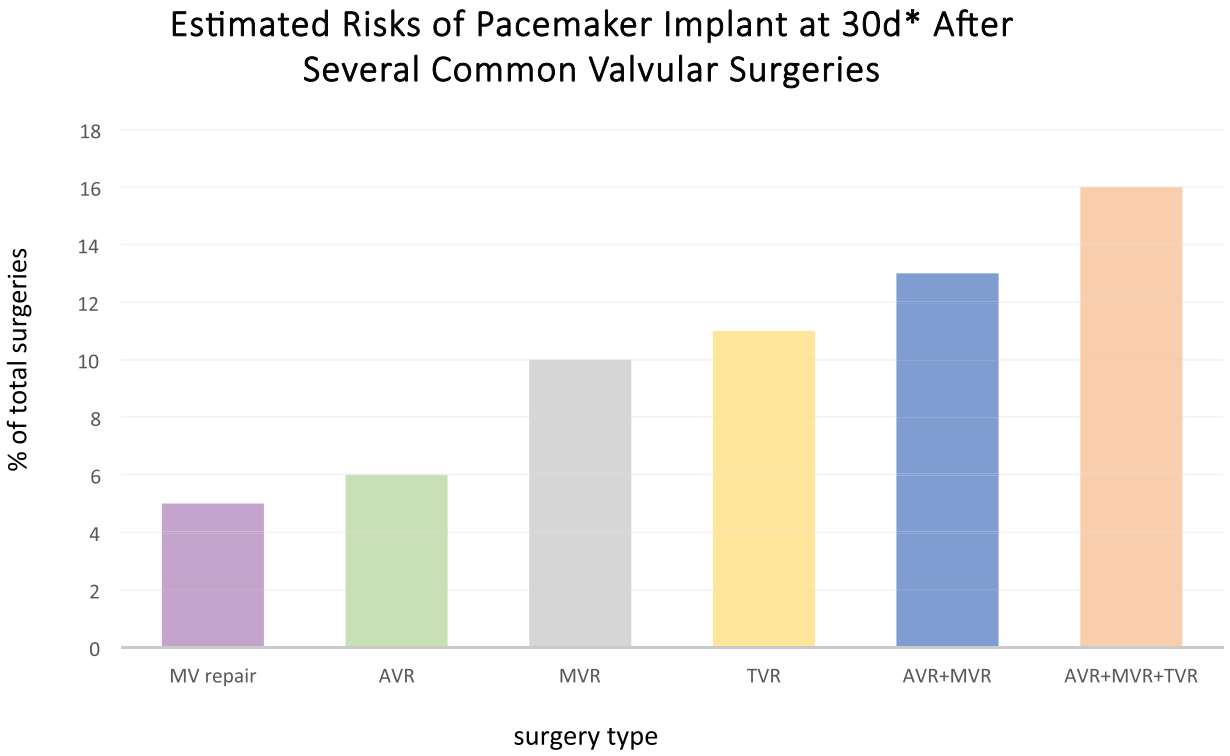

FIGURE 1 Aggregate estimates of risks for permanent pacemaker implantation according to surgery type. Clinical risk factors have also been identified, including a twofold risk for any valvular surgery that includes tricuspid valve ring repair or replacement. *These are approximate risks aggregated from various studies[1–4]

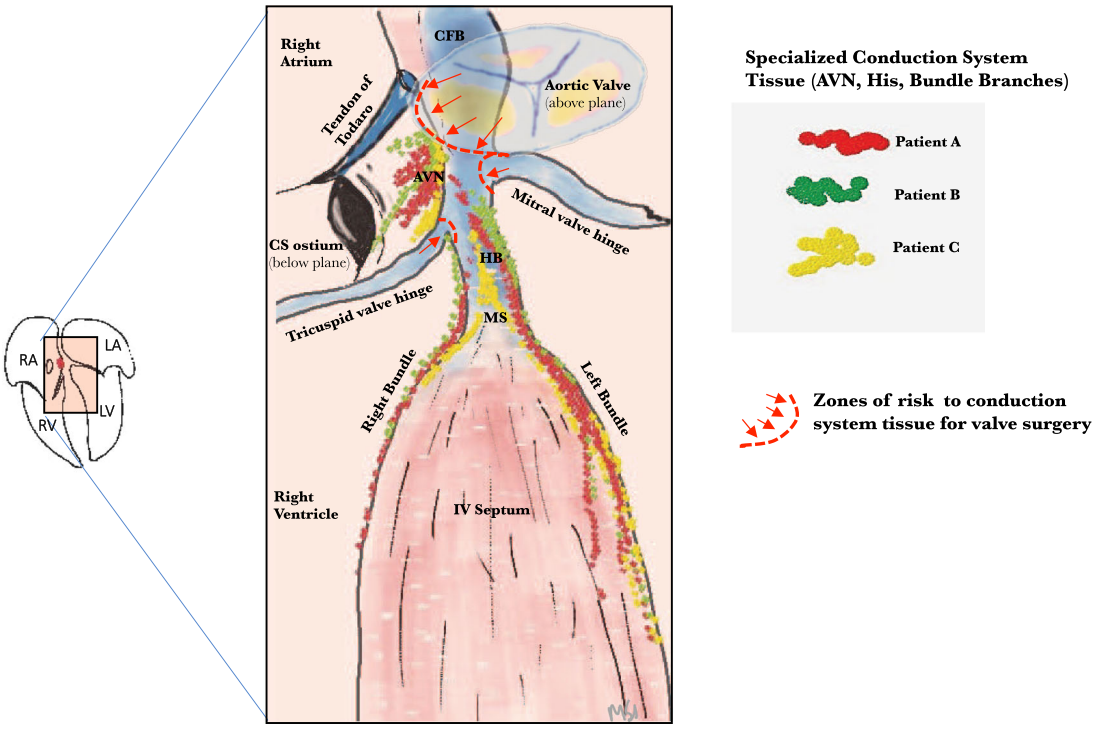

FIGURE 2 Vulnerability of the specialized conduction system to cardiac valvular surgery. The enlarged schematic shows the proximity of specialized conduction tissue to the mitral, tricuspid, and aortic (above plane) valves. There is marked variability in the course of the conduction system and its course through the ventricular portion of the membranous septum (MS; three example courses in red, yellow, green) and in the length and position of the central fibrous body (CFB) and MS.[5] CS, coronary sinus; HB, His bundle; IV, interventricular

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