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COMMENTARY

Global longitudinal strain as a risk factor for pacing-induced cardiomyopathy: another step toward mechanistic insight and prevention strategies

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Pacing-induced cardiomyopathy (PICM) is common in patients with a high burden of right ventricular (RV) pacing, with the reported incidence ranging from 6 to 39% [1] Despite its high incidence, there remains little data on the predictors of PICM in patients who have a preserved ejection fraction at the time of permanent pacemaker implantation. A lower left ventricular ejection fraction before pacemaker implantation, male sex, wider native and paced QRS durations, and increased in left ventricular dimensions on transthoracic echocardiography has been associated with the development of PICM. [2, 3].

The study by Cho et al. in this issue of the *Journal of Interventional Cardiac Electrophysiology* identified reduced global longitudinal strain (GLS) as an additional risk factor for development of PICM in patients with a preserved ejection fraction. The single-center observational study identified 168 patients undergoing pacemaker implantation with a preserved ejection fraction and an expected right ventricular pacing burden of more than 40%. PICM was defined as a > 10% decrease in LVEF, with a resultant LVEF of less than 50%. At 3 years follow-up, PICM developed in 16 (9.5%) of patients. Reduced GLS and a prolonged paced QRS duration were identified as independent predictors of PICM.

Conduction system pacing (CSP) and cardiac resynchronization therapy (CRT) have been proposed to prevent PICM as both pacing strategies result in a more physiological and narrower paced QRS. While they have been shown to improve clinical outcomes in patients with heart failure and reduced ejection fraction, clinical benefit in those with a preserved ejection fraction is less robust. [4, 5] These strategies come at an increased cost and procedural complexity and may not be feasible in all patients. Thus, identifying patients at high risk of developing PICM is essential, as most patients maintain preserved ventricular function despite chronic RV pacing. [6].

In patients at risk for developing PICM with an existing pacemaker, clinicians could consider early titration of heart failure therapy, altering of device parameters to reduce burden of RV pacing, or an early upgrade to cardiac resynchronization therapy (CRT) or conduction system pacing (CSP). In those undergoing a new implant risk stratification could direct the decision between implant of CSP or CRT device and standard RV pacing. Multiple risk factors for PICM have been identified, however, with insufficient discriminatory value to choose pacing strategy. Identification of addition risk factors for PICM, ideally those that can be measured prior to implant of a pacing device, is needed to improve the risk stratification.

Cho et al. found that a paced QRS duration of more than 170 ms was an independent predictor of PICM, but that there was no difference in burden of ventricular pacing or other device-related parameters between the 2 groups. This finding is consistent with previous studies [3, 6], and it has been suggested that the development of LV dysfunction is at least in part due to dyssynchronous contraction as a direct result of RV pacing. However, a wider-paced QRS could also be an indicator of underlying intraventricular conduction delay due to myopathic process.

Global longitudinal strain may be more reliable than LV ejection fraction in identifying patients with myocardial damage as it is less sensitive to changes in loading conditions and may unmask patients with myocardial damage in the absence of a change in ejection fraction. [7] The findings in this study are consistent with previous studies

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demonstrating a reduced GLS is associated with poorer outcomes post pacemaker implantation. [8] A reduced GLS is also an independent predictor of cardiovascular mortality in other patient cohorts who do not have a permanent pacemaker. [9] Reduced GLS could be a marker of an underlying myopathic process in these patients that ultimately leads to heart failure with reduced ejection fraction. The key question is if the development of left ventricular dysfunction is related to ventricular pacing, and subsequently if it could be prevented by a different pacing modality.

Though the current study was single center, observational and had a highly selected group of patients, the population and clinical characteristics were well defined and the outcome parameter of GLS was robustly measured and adds important information on the utility of reduced GLS as a predictor for PICM.

Routine measurements of GLS on echocardiography prior to PPM implantation are feasible and inexpensive. Combined with other established clinical and intra-procedural risk factors for PICM, it may assist in determining the appropriate pacing strategy to prevent PICM. However, further research is needed to determine if CRT or CSP can prevent PICM in this patient group.

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