EDITORIAL COMMENT

RV Electrical Activation in Heart Failure During Right, Left, and Biventricular Pacing*

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Right Ventricular (RV) Activation Mapping for Patients With Heart Failure: Back to the Basics

Since its approval by the U.S. Food and Drug Administration in 2001 for selected patients with systolic dysfunction, cardiac resynchronization therapy (CRT) has generated tremendous interest and a large body of literature. The mere idea of addressing the maladaptive electrical phenomena associated with heart failure and their hemodynamic consequence seems logical but has been fraught with limitations. For those who respond, the potential benefits of improved quality of life, New York Heart Association functional class, and hemodynamic and echocardiographic parameters such as left ventricular (LV) dimensions and LV ejection fraction have been well documented.

Unfortunately, identification of general inclusion criteria for patients likely to benefit from CRT has been generally disappointing. Most trials have been plagued with 30% of nonresponders and often more. The reasons for such a high rate of failure may involve a number of factors, namely, the heterogeneity of the population of patients, the ischemic versus nonischemic nature of cardiomyopathy, the incomplete correlation between electrocardiographic findings and the presence of mechanical dyssynchrony, and technical factors such as programming and lead positioning at the time of implantation.

Particularly interesting are the findings of improved LV dimensions or ejection fraction without consistent correlation with improved New York Heart Association functional class. A number of trials, such as REVERSE (REsynchronization re-VErses Remodeling in Systolic left vEntricular dysfunction) (1) and PROSPECT (Predictors of Response to Cardiac Resynchronization Therapy) (2,3), illustrate the difficulty in using LV echocardiographic parameters to predict the response to CRT. The reasons for that are potentially many, as mentioned previously. It remains a constant, however, that there is no reliable way to predict successful CRT. In the ongoing quest to improve CRT and decrease the large proportion of nonresponders, there has been growing interest in looking at RV parameters during CRT. Could the right ventricle be a significant player in the clinical evolution of patients with LV systolic heart failure?

In this issue of JACC, Varma et al. (4) present a comparison of RV activation during intrinsic conduction and ventricular pacing. Patients with systolic heart failure and cardiac resynchronization devices were compared with healthy control subjects. Measure of the rS duration was used as a surrogate measure of intact right bundle branch conduction. Patients with native right bundle conduction, owing to native atrioventricular conduction (in the control population) or to careful programming of timed LV pacing, generally had a shorter RV activation time, as defined by electrocardiographic imaging. This benefit persisted when they were compared with patients who were paced in the right ventricle or had biventricular pacing programmed.

It seems intuitive that using the specialized cardiac conduction tissue to depolarize the right ventricle would lead to a more rapid and organized RV activation compared with the slow concentric cell-to-cell activation that follows myocardial pacing from an RV apical site. In this regard, this work is

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reminiscent of previous ex vivo models and in vivo studies performed by our team using direct intraoperative measurements of ventricular depolarization. Patterns of propagation of the depolarization wavefront and breakthrough from the His-Purkinje system have been documented in patients with normal conduction systems or bundle branch block (5). These studies have taught us the association between electrical conduction and organized mechanical activation of the ventricles.

The interest of the work by Varma et al. (4) is that it attempts to link mechanistic work and its clinical implications and substantiates the potential mechanism for the deleterious effect of RV pacing in patients who do have native right bundle conduction, via a noninvasive novel approach.

It is appealing to conclude that a shorter RV activation duration will be beneficial to patients. Given the incomplete correlation between electrical and mechanical events, concluding that a shorter RV activation time will translate into improved function is a leap of faith that we cannot make.

Although we know that long-term RV apical pacing is probably detrimental to LV function, little is known about the long-term effects of isolated LV pacing on RV function or other parameters such as RV dimensions. The concerns over the potential for myocardial remodeling also remain. In the end, one question that will arise is whether timed LV pacing will be associated with any adverse long-term effect on the left ventricle.

We know that the population of patients with heart failure is very heterogeneous. In the study by Varma et al. (4), only 1 patient had a dilated right ventricle. RV involvement as a consequence of LV systolic failure is a common occurrence. One can only wonder whether a similar study in patients with RV involvement and conduction disease involving the terminal right bundle branch could lead to different conclusions.

Obviously, the conclusions of a small study such as the one presented here should be used as a proof of concept and for hypothesis generation. The good correlation of electrocardiographic imaging with previous invasive measures of ventricular activation is an interesting finding that suggests that we may be able to use this new tool when tailoring CRT in individual patients. Perhaps it will turn out that timed LV pacing will prove to be superior to biventricular pacing in a selected population of patients.

At a time when we are dealing with rapidly increasing health care costs, any tool that would help to better tailor therapy and identify patients who are likely to benefit from CRT is welcome. Recent trials such as MADIT-CRT (Multicenter Automatic Defibrillator Implantation Trial With Cardiac Resynchronization Therapy) (6) have looked at extending the application of CRT devices to a wider population of patients with mitigated results. We need to do better in selecting patients for CRT.

It might just be that there are no universal general criteria that can identify patients who will benefit from CRT. The work presented in this issue of JACC has the merit of bringing the focus back to the level of the mechanism and to potentially add a useful noninvasive tool to the armamentarium available with which to identify patients who will benefit from CRT.

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