HF patients, and appropriate rate-adaptive pacing should be considered in patients in whom CRT response is suboptimal owing to concurrent CI.

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Role of Septal to Posterior Wall Motion Delay in Cardiac Resynchronization Therapy

In a recent issue of the *Journal*, Marcus et al. (1) report that septal to posterior wall motion delay (SPWMD) fails to predict reverse remodeling or clinical improvement in patients having heart failure and treated with cardiac resynchronization therapy (CRT), and they conclude that SPWMD should not be used to guide the selection of candidates for CRT. Because these findings and recommendations are contrary to our published reports and have significant clinical implications, it is important for your readership to understand that the method used to determine SPWMD in their study is not the one we demonstrated previously to predict both reverse remodeling and prognosis (2,3). Apart from the limitations and biases inherent in retrospective analyses, we will focus on the differences between the two methods, the one we and others (4–6) have successfully used and the present one, and which we believe led to the disparity in outcomes. We believe that the key to the prognostic value of our method is the measuring of the SPWMD in the short axis. The rationale of measuring the SPWMD by using the short axis view at the level of the papillary muscles, and is related to histological and anatomic relationships not shared with the longitudinal axis.

Specifically, the factors determining the measure of the delay between the septum and the posterior wall obtained by using the two methods differ in several ways:

1. The direction of the myocardial fibers differs between the basal and midventricular levels. At the level of the midventricle, the fibers have a circular course.
2. The sequence of myocardial activation differs between the basal and midventricle levels in the presence of left bundle branch block (7), is variable (8), and cannot be predicted by the electrocardiogram. In particular, the ventricular conduction delay resulting from an area of conduction failure (due to scarred tissue or functional block) or from slow but homogeneous myocardial propagation gives rise to an activation of the basal segments of the left ventricle that is different from that of mid left ventricular level. This different pattern of activation might be responsible for the fragmented mechanical activity that is seen more frequently at the level of the basal septum than at the mid left ventricular level.
3. The long axis evaluation of dyssynchrony is limited by the inherent difficulties to define accurately which area (i.e., lateral, posterolateral) is visualized and obtain a perpendicular to the long axis of the ventricle view (this is the case of both images shown in the study by Marcus et al. [1] in which the midseptum is compared to the basal free wall).
4. Basal segments being tied to fibrous skeleton of the heart and their movements are strongly influenced but in variable and unpredictable ways by the movements of the aortic root and mitral annulus. These influences could cause an irregular contraction of the septal wall, thus leading to a more difficult calculation of maximum septal displacement and to a less reproducible measure.

We hope that this explanation helps in understanding that taking the measurement in another way cannot a priori be expected to yield equivalent physiological or clinically relevant information; therefore, the parameter used by Marcus et al. (1), although given the same name as ours, is not the same as ours and that the conclusions drawn by their study cannot be applied to the SPWMD parameter we have described.

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