Spotlight on left ventricular dyssynchrony: Part 2—Prognostic value

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Temporal differences in activation and contraction of various myocardial segments of the left ventricle (LV) is referred to as LV dyssynchrony (LVD). In the previous issue we discussed the potential mechanisms that result in LVD and techniques for assessment of LVD and its severity. LVD is prevalent among patient with heart failure (HF) and in cardiomyopathies that result in LV dysfunction – both global or regional. As such, LVD has been shown to predict all-cause mortality and arrhythmic outcomes among patients with chronic HF. Patients presenting acutely, with decompensated heart failure, experience myocardial stretch and abnormalities in myocardial contractility. It is conceivable that acute changes in myocardial contractility and the resultant acute LVD—induction or worsening, is likely to influence clinical prognosis among patients with acute HF.

In this issue of the Journal, Zhou et al. present their results on the relationship between LVD and clinical outcomes among 92 patients admitted with acute decompensated HF. Among these patients, 28% experienced the end-point of all-cause mortality or heart transplantation during a mean follow of 28 months. While LV ejection fraction was not different among those experiencing events vs. those who did not (34.7% vs 34.2%), those experiencing events had a greater prevalence of abnormally elevated histogram bandwidth (HBW; 85% vs 61%)—defined as $HBW > 77.76^\circ$, derived as mean + 2SD of HBW of a normal cohort. Though HBW was an independent predictor of clinical events, prediction of events was further improved by QRS duration. When both electrical (QRD duration > 120 ms) and mechanical ($HBW > 77.76^\circ$) LVD were

present in 55% of the patients who experienced an adverse event, as opposed to none when both were absent. Thus, mechanical and electrical LVD provide incremental prognostic value among acute HF patients.

The prognostic value of LVD in chronic HF has been described in several observational studies. Patients in these studies consisted of both ischemic and non-ischemic cardiomyopathies. However, those with infiltrative cardiomyopathies were not specifically evaluated. Infiltrative cardiomyopathies, specifically those with underlying inflammatory etiology, such as sarcoidosis can result myocardial scarring and conduction abnormalities, and thus can induce LVD. Koyanagawa and colleagues specifically evaluated the prognostic value of LVD among a cohort of patients with diagnosed cardiac sarcoidosis, who had undergone FDG PET/CT and gated SPECT MPI. HBW on SPECT MPI inversely correlated with LV ejection fraction and positively with infarct size. Patients with a HBW > 56° (cut-off for median for the population) had a greater occurrence of major adverse cardiovascular events (MACE)—all-cause mortality, life-threatening ventricular arrhythmias, heart blocks and HF hospitalization. HBW, along with phase standard deviation (PSD) and entropy were significant independent predictors of MACE in these patients with confirmed diagnosis of cardiac sarcoidosis. Thus, assessment of LVD in these unique populations provides additional prognostic value and has the potential to guide HF and disease-specific therapies. In these populations, it would be interesting to know if treatment mediated improvement in HF, translates into improved LV synchrony on serial imaging, which could be used as a predictor for the need of advanced HF therapies or heart transplantation in the future.

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