Koilpillai C., Quinones, M., Greenberg, B. et al. Relation of ventricular size and function to heart failure status and ventricular dysrhythmia in patients with severe left ventricular dysfunction.


Patients with severe left ventricular (LV) dysfunction may or may not have overt heart failure and ventricular dysrhythmia. To study factors behind this variability, we examined a subset of 311 patients from the Studies of Left Ventricular Dysfunction-95 with a history of moderate heart failure (treatment trial) and 216 with no failure (prevention trial), all with ejection fractions <0.35. Echocardiographic variables were compared between trials and also correlated with dysrhythmia in 258 patients, and with neurohormones in 199 patients. Compared with prevention patients, treatment patients had larger LV end-diastolic diameter, end-systolic volume, sphericity index, and ratio of early to late diastolic filling velocity by Doppler (E/A ratio), lower LV ejection fraction and atrial contribution to ventricular filling, and similar LV mass, end-diastolic volume, and estimates of systolic wall stress. With prevention and treatment patients combined, the prevalence of abnormally elevated atrial natriuretic peptide was 92% in the highest tertile of E/A ratio compared with 55% in the lower tertiles (p=0.006). Across tertiles of LV end-diastolic volume, there was an increase in the prevalence of nonsustained ventricular tachycardia (24%, 45%, and 45%; p=0.007) and premature ventricular complexes >10/hour (48%, 62%, and 80%; p<0.001). Thus, in severe LV dysfunction, ventricular filling indexes suggestive of high filling pressures, along with larger and more spherical ventricles, are particularly common in patients with overt heart failure, thus suggesting that diastolic properties and the degree of ventricular remodeling affect clinical status. Once ejection fraction is significantly reduced, the prevalence of ventricular dysrhythmia correlates with LV size rather than systolic function. This observation lends support to previous experimental findings on the role of myocardial stretch and scar in the genesis of dysrhythmia.