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Atrial Strain Assessment in Left Ventricular Diastolic Dysfunction

We read with great interest the work by Mordi et al. (1), which was concerned with the ventricular mechanics in patients with heart failure and preserved ejection fraction (HFpEF) in the current issue of *iJACC*. The investigators nicely demonstrated the relevance of echocardiographic global longitudinal strain (GLS) at rest and cardiac magnetic resonance (CMR)–derived extracellular volume (ECV) in the identification of patients with left ventricular (LV) diastolic dysfunction. Both parameters possess additional value beyond commonly established parameters like LV ejection fraction and transmitral velocities, such as, E/E′ in the differentiation among patients with hypertensive heart disease, patients with HFpEF, and healthy control subjects (1).

While the investigators valued ventricular mechanics, evaluation of atrial physiology, with its 3 distinct phases (2) was not approached, although mounting evidence has suggested a pivotal role of atrial physiology in ventricular diastolic dysfunction (2). In addition to ventricular measurements, strain and strain rate parameters are available to assess atrial reservoir function (collection of pulmonary venous return during ventricular systole), conduit function (early diastolic blood passage for ventricular filling), and booster pump function (late diastolic augmentation of ventricular filling). Although the investigators demonstrated a strong correlation of GLS with exercise capacity as expressed by peak volume of oxygen and ventilatory response/volume of carbon dioxide, atrial conduit function was shown to be the most precise predictor of exercise intolerance, beyond invasively measured ventricular parameters of stiffness and relaxation (3). In addition, there is evidence to suggest an important role of stress testing in HFpEF because diagnostic accuracy of guideline-recommended echocardiographic assessments during stress was significantly increased, mainly due to improved sensitivity (4). Furthermore, Melenovsky et al. (5) identified an impaired atrial booster pump function during stress in HFpEF as a potential mechanism to explain cardiopulmonary decompensation in this condition.

In conclusion, the investigators should be commended for expanding the existing body of literature on assessment of ventricular mechanics and tissue characterization by a multiparametric imaging approach with echocardiography and CMR in diastolic dysfunction. Both resting GLS and ECV might improve diagnostic accuracy to detect early changes in subclinical diastolic dysfunction, which potentially could be further increased by assessments during physiological exercise and the application of additional left atrial strain assessments. A complete evaluation of diastolic dysfunction should therefore incorporate ventricular and atrial physiology at rest, and ideally, also under stress, to gain further insights into the complicated and heterogeneous nature of HFpEF.

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THE AUTHORS REPLY:

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