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EDITORIAL COMMENT

# Time to Revisit Left Ventricular Wall Stress as a Tool for Heart Failure Prediction?

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Heart failure (HF) continues to be a common cardiac problem in the elderly.[1] With the increased life expectancy of our population, the prevalence of HF burden is certainly expected to burgeon. Irrespective of left ventricular (LV) ejection fraction, HF carries significant morbidity and mortality and risk for hospitalization. Transthoracic echocardiography (TTE) is a widely available test for evaluation of patients with suspected or established HF. In addition to estimation of LV ejection fraction, several additional findings from TTE can inform the clinician about adverse long-term prognosis. Left atrial volume index, indicators of advanced diastolic dysfunction, elevated right and left-sided filling pressures, elevated right ventricular systolic pressure, and regurgitation of mitral and tricuspid valves have all been shown to be associated with poorer outcomes in patients with HF. These findings from TTE are frequently used by clinicians caring for HF patients for initiation of various therapies (drug and/or device) as well as prognostication.

According to the Laplace law, ventricular wall thickness increases in response to elevated pressures and/or chamber dimensions as an adaptive mechanism to decrease wall tension.[2] While the most accurate measurement of wall stress requires invasive hemodynamics, LV systolic and diastolic wall stress (LVEDWS and LVESWS) by echocardiographic derivation is feasible and correlates well with invasively derived measurements.[3] Despite its relatively easy availability, LV wall stress is not clinically tracked or used widely for patient management in HF by most institutions. Consequently, LVEDWS and LVESWS are also not used as prognostic markers for future development of HF by clinicians.

In this issue of *JACC: Advances*, White et al[4] describe their findings from a retrospective analysis of 4,601 patients from the Atherosclerosis Risk in Community (ARIC) database. This cohort was a community dwelling elderly group of patients (median age 75 years, 58% women, and 18% self-identifying as Black) who underwent detailed TTE and were followed sequentially. The median follow-up duration of the study group was 4.6 years. All TTEs were analyzed by a core lab. HF hospitalization and HF mortality were tracked through review of hospitalizations, death certificates, and phone calls. All clinical events were adjudicated by a special committee. The authors found that LVEDWS was significantly correlated with development of HF and HF-related events over the study period.

The authors of this study need to be congratulated for looking at the carefully collected data from the ARIC study of community-dwelling individuals. Missing data elements were very few, which strengthens this study. Both the clinical outcomes adjudication and analysis of TTE findings by a core echocardiography lab were additional positive features of the study. Regarding the weakness of the study, the authors correctly point out that their use of Doppler parameters instead of direct LV end-diastolic pressure measurements could lead to errors in both LVEDWS and LVESWS estimations. While the easily obtained ratio of mitral inflow "E" velocity and the mitral annular "e" velocity can serve as a surrogate for LV end-diastolic pressure, there is still debate about the accuracy of this method even when

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obtained during sinus rhythm. While all clinical events were adjudicated, the diagnosis of HF in medical records is not always supported by data, and it is possible that there was some degree of overestimation of HF events during the follow-up.

This paper raises the very valid possibility regarding the value of LVEDWS in the prognostication and risk assessment for future development of HF in community-dwelling elderly individuals without overt clinical symptoms from ischemic heart disease. Additional questions regarding the mechanism by which increased LV wall thickness contributes to this risk deserve further exploration. In this age group, the possibilities of an infiltrative process such as amyloidosis, myocardial scarring related to ischemic heart disease, or atrial scarring are relevant. Multimodality imaging can provide answers to these questions. Finally, earlier detection of risk for future development of HF will be a worthwhile pursuit if appropriate therapies instituted early could slow disease progression. Thus, we are left with a few unanswered questions but many possibilities for meaningful next steps regarding future research avenues in this field.

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**KEY WORDS** ARIC study, echocardiography, heart failure, LV wall stress