REPLY: Speckle-Tracking Echocardiography-Derived Longitudinal Dysfunction

A Novel Starting Point of the Hypertensive Patient's Journey Toward Heart Failure

We are pleased to learn of the interest demonstrated in our recent work published in *JACC* by Drs. Galderisi and Trimarco who raise a very important issue related to the mechanisms underlying cumulative hypertension-related myocardial dysfunction demonstrated by our group on behalf of the community of CARDIA (Coronary Artery Risk Development in Young Adults) participants and investigators. This has been a central research issue for our laboratory in the past 2 decades (1), and although we do not have direct evidence to demonstrate such a mechanism in the CARDIA cohort yet, our overall experience with other cohort studies (2) and others (as highlighted in the letter to *JACC*) suggest that indeed myocardial fibrosis either focally as localized scarring or more diffuse as interstitial fibrosis (more likely in the case of the CARDIA cohort and other population studies) could be the underlying mechanism for the observed alterations of global longitudinal shortening seen in our study. Díez et al. (3) demonstrated the association of chronic hypertension with fibrosis histologically, and in the MESA (Multi-Ethnic Study of Atherosclerosis Study), chronic hypertension was associated with increased left ventricular mass over 10 years of follow-up and greater myocardial fibrosis (both diffuse interstitial and focal) (4). Additionally, increased myocardial fibrosis and increased left ventricular mass were demonstrated to correlate with myocardial dysfunction measured by magnetic resonance imaging tagging as reduced circumferential shortening, a less sensitive but perhaps also more specific measure of myocardial dysfunction (5). Considered together, these observations, as suggested by Drs. Galderisi and Trimarco, indeed point to hypertrophy and fibrosis as the pathophysiological pathway linking myocardial dysfunction and hypertension.

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