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# Left Atrial Mechanics in Hypertensive Patients

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## *Dear Editor,*

The left atrium (LA) plays an important role in the overall cardiovascular performance by contributing to the left ventricular (LV) filling with its reservoir, conduit, and contractile functions (1). Until the early 2000s, the noninvasive study of the LA was performed using the so-called "classic" echocardiography-derived parameters (LA area, maximum volume, phasic volumes, and blood flow assessment of the transmitral or the pulmonary flow), but these parameters were subject to a number of limitations (2). In the past decade, new imaging modalities have emerged, providing new insights into the noninvasive assessment of the LA. Echocardiography-based automated techniques for sophisticated analysis of myocardial displacement have emerged such as Doppler tissue imaging (DTI) or speckle tracking (ST) which enable the quantification of regional and global myocardial mechanics such as displacement, velocity, strain (ε), and strain rate (SR) (3). New information on the clinical relevance of the LA ε and SR analysis is constantly emerging. The LA mechanics is a promising tool in several clinical contexts, either for diagnostic or therapeutics decision-making process. Moreover, the LA mechanics seems to have a prognostic importance across several clinical cardiovascular and systemic diseases (4). Previous studies have demonstrated an important association between the LA reservoir phase mechanical indices and the LA histology (5) and the LA fibrosis assessed with cardiac magnetic resonance (6), supporting the use of the LA reservoir phase mechanics as an index of the LA stiffness. Systemic arterial hypertension is associated with morphological and functional LA abnormalities. An increase in the LA size is a hallmark of the LA remodeling process and is a common finding in hypertensive patients (7, 8). It is well known that the increase in the LA size can predict target organ damage (9) and adverse cardiovascular outcomes (10, 11).

Several authors have raised the question of whether the changes in the LA mechanics occur before or after the morphological ones. Sahebjam et al. (12) compared the LA mechanics in hypertensive patients with normotensive age-matched controls, using DTI to derive the LA reservoir phase ε and SR. Both these groups showed a normal LA size. The authors were able to demonstrate significantly lower values of ε and SR for the LA reservoir phase in the hypertensive patients when compared with the normotensive group. It is possible to speculate that a chronic pressure overload of the LA leads to a reduction in the LA reservoir phase performance, as was assessed with DTI. That was probably related to the LV diastolic dysfunction and temporary elevation of the LV filling pressures in the context of hypertensive heart disease. Unfortunately, the authors do not present any estimation of the LV filling pressures (such as E/e'). In the group of hypertensive patients, there was a significantly higher number of diabetics, which can be an important confounding factor, since coexisting diabetes in hypertensive patients can further impair the LA mechanics (13, 14). With respect to the assessment of the LA mechanics, it is well established that DTI only represents a few points in the LA wall (4 in the present paper). This is substantially fewer than the 12- or 15-segment model proposed for the LA mechanics assessment with 2D-ST echocardiography (3). Another important limitation of the paper is the construction of the multivariate model. More than 10 variables were included in the linear regression model, raising the possibility that the models were overfitted. In the linear regression model, the authors included both continuous and categorical variables! It would also be important to present the quality of the model to predict the LA reservoir phase SR. Finally, we would also suggest a multivariate analysis to the LA reservoir phase ε. It is important to acknowledge that the LA reservoir ε and SR are influenced not only by chamber stiffness, LA volume, and pressures changes but

also by the downward movement of the mitral annulus towards the apex, as a result of the LV contraction, just before the opening of the mitral valve (4). Borges et al. (15) demonstrated that in hypertensive patients, the longitudinal mitral annulus velocity was reduced. Since the LA is inseparable from the LV and from the mitral annulus movement, it may be of interest to combine the evaluation of the LA mechanics with the LV longitudinal systolic function. This study of Sahebjam et al. (12) was of interest insofar as it lends support to the use of the LA reservoir phase mechanics in hypertensive patients, but further studies are needed to assess its clinical and therapeutic usefulness.

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