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Comparison of Left Atrial Function Between Hypertensive Patients With Normal Atrial Size and Normotensive Subjects Using Strain Rate Imaging Technique

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Dear Editor,

I read with great interest the study by Sahebjam et al. (1), who compared the left atrial (LA) function between hypertensive patients with a normal atrial size and normotensive subjects, using strain rate imaging (SRI) by strain Doppler. They found that patients with hypertension showed significantly lower values of strain (S), strain rate (SR), and velocity during reservoir function compared with controls, although the LA dimensions were not increased. These results agree with those of a previous study by Kokubu et al. (2) (2007), reporting that LA S and SR values were lower in patients with hypertension when compared with normal subjects, irrespective of the presence of LA enlargement or left ventricular (LV) hypertrophy.

The methods and the interpretations of the results, however, raise several concerns. Although it was not specified, I expect that hypertension was of recent onset or the patients had suffered from hypertension for a few years, because not only all the patients have diastolic dysfunction (grade one) in the early phase, but also the LV mass index average was still in the range of normality (except for 22 out of 75 patients that had LV hypertrophy) and all the patients had a normal atrial size. These findings emphasize that SI is a useful tool for the early detection of LA dysfunction, when traditional echocardiographic parameters are still normal, as several studies have demonstrated previously. An important consideration is that some of the patients (17 out of 75) and 2 out of 45 controls had diabetes mellitus. Diabetes mellitus is known to cause atrial fibrosis, which leads to the impairment of the LA function.

In fact, numerous studies have demonstrated that patients with diabetes show an early LA dysfunction (3, 4). Consequently, the low values of S, SR, and velocity in the study by Sahebjam et al. may have been due to diabetes rather than only hypertension.

Mondillo et al. (3) employed speckle tracking and showed that LA longitudinal S, during ventricular systole and early and late diastole, was lower in patients with diabetes and in patients with hypertension than in controls and that it was further reduced in patients with coexisting diabetes and hypertension. The association of diabetes and hypertension with LA S abnormalities was independent of clinical and echocardiographic variables (LA dimension and volume as well as LA ejection fraction), which were similar. Also, our preliminary data on 30 diabetic patients without coronary artery disease (5) demonstrated abnormalities of atrial reservoir function according to speckle tracking. Such abnormalities are an expression of early pathological changes in the atrial walls, which are thinner than the LV walls, when LV global and segmental systolic function is still normal. I believe that the presence of diabetes mellitus, as well as hyperlipidemia, could have influenced the results of their study.

Then, Sahebjam et al. report findings that are contradictory to those in the study by Kokubu et al. (2) as regards the effect of renin–angiotensin system inhibitors on S and SR parameters. Whereas the former found that 31 out 75 hypertensive patients, who were using renin-angiotensin system inhibitors, had lower S and SR values than did those not receiving this therapy (even if this difference did not constitute statistical significance), the latter found that deformation parameters tended to normalize

after renin-angiotensin system inhibition, indicating a therapeutic effect on the LA function. Moreover, Tsang (6) reported that LA structural remodeling appeared reversible with Quinapril.

The most surprising result of the Sahebjam et al. study is that S and SR values, in both hypertensive and control groups, were much lower than those obtained in numerous previous studies (7-9). Indeed, while the values of atrial myocardial deformation properties range from 65.4 ± 19.5% to 82 ± 19% for LA systolic S and from 3.4 ± 1 S^{-1} to 4.4 ± 1.6 S^{-1} for LA systolic SR in different studies on normal subjects, Sahebjam et al. reported 27.1 ± 11.94% for LA S and 2.28 ± 0.49 S^{-1} for LA SR, despite the fact that all the studies used the same echocardiographic machine (Vivid 7, GE) and the sample volume was placed on the same side. Furthermore, the previous studies have observed that the LA systolic S and SR values of hypertensive patients are surprisingly lower than those obtained in patients with atrial fibrillation (AF) (10-12) and mitral stenosis (8, 13).

Because of LA remodeling and dilation due to myocyte cell loss as well as changes in the extracellular matrix composition and fibroblast proliferation and differentiation into myofibroblasts, with both diffuse interstitial and patchy fibrosis, AF creates lower S and SR values compared to other diseases. Owing to the disorganization of the atrial muscle bundles and atrial fibrosis, more severe impairment of LA deformation properties has been reported in mitral stenosis patients with AF (8, 14). Probably, the authors did not continuously perform manual tissue tracking, frame by frame, during the cardiac cycle, to maintain the sample volume's position within the atrial wall and, as a result, the sample volume did not follow the movements of the myocardial wall and, thus, recorded lower values.

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