

## Letter to the Editor

### Noninvasive Assessment of Left Atrial Function by Strain Rate Imaging in Patients with Hypertension: A Possible Beneficial Effect of Renin-Angiotensin System Inhibition on Left Atrial Function

To the Editor:

I read with great interest the study by Dr. Kokubu *et al.* (1), which showed that the mean systolic strain rate (SR) of left atrial (LA) segments (SR-LAs) in a dilated LA group and non-dilated LA group was significantly lower than that in normotensive (NT) subjects, irrespective of the presence or absence of left ventricular hypertrophy (LVH); that the mean SR-LAs in hypertensive (HT) patients was lower than that in NT subjects; that the mean SR-LAs in a non-dilated LA group administered renin angiotensin system (RAS) inhibitors was equivalent to that in NT subjects; and that in HT patients, the mean SR-LAs, as an index of LA reservoir function, decreased before development of LA enlargement and LVH. These results suggested that treatment with RAS inhibitors preserves LA reservoir function in HT patients without dilated LA, and that SR imaging can detect LA dysfunction in HT patients and is useful for evaluation of the therapeutic benefit on LA reservoir function. The methods and interpretation of the results, however, raise several concerns.

Kokubu *et al.* (1) report that 80 patients with HT were studied, in whom relevant history included hypercholesterolaemia, diabetes, and paroxysmal atrial fibrillation, which could affect LA systolic or diastolic function. Despite this fact, the relations of these diseases to LA systolic or diastolic function were not well described. This raises the question: was there any relation between these aspects of the patient histories and the results of this study?

In addition, the HT patients had greater body mass index (BMI) than did the NT patients, which could have affected LA systolic or diastolic function. However, Kokubu *et al.* (1) did not adequately describe the relation of BMI to LA systolic or diastolic function. Here again, did the relation between BMI and these functions affect the results?

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1. Kokubu N, Yuda S, Tsuchihashi K, *et al.*: Noninvasive assessment of left atrial function by strain rate imaging in patients with hypertension: a possible beneficial effect of renin-angiotensin system inhibition on left atrial function. *Hypertens Res* 2007; **30**: 13–21.

### Response to: Noninvasive Assessment of Left Atrial Function by Strain Rate Imaging in Patients with Hypertension: A Possible Beneficial Effect of Renin-Angiotensin System Inhibition on Left Atrial Function

To the Editor:

We thank Dr. Song for his interest in our article on the assessment of left atrial (LA) function by strain rate imaging in patients with hypertension (HT). It has been that the simultaneous presence of diabetes mellitus (DM), hypercholesterolemia, and obesity is associated with the development of left ventricular (LV) dysfunction (1–3) and is likely to influence not only LV, but also LA function. However, LA function in patients with DM, hypercholesterolemia and/or obesity has not been fully investigated due to the lack of an appropriate method for the assessment of LA function. Inaba *et al.* (4) have recently demonstrated that patients with persistent atrial fibrillation (AF) have significantly lower LA strain rate during systole than that in aged-matched controls. In our preliminary data, strain rate during systole and early diastole was decreased in patients with DM and HT (5). Thus, we agree that the presence of paroxysmal AF, DM hypercholesterolemia and obesity may have affected the LA function in this study.

In the HT group in our study, only 7 of patients (9%) had a history of paroxysmal AF, and 15 (19%) had DM in the HT group. The mean SR-LAs ( $2.06 \pm 0.68$  vs.  $2.10 \pm 0.53$  s<sup>-1</sup>, n.s., respectively) and mean SR-LAa ( $-2.49 \pm 0.63$  vs.  $-2.98 \pm 1.09$  s<sup>-1</sup>, n.s., respectively) in hypertensive patients with a history of paroxysmal AF were comparable to those in hypertensive patients without a history of paroxysmal AF. Furthermore, the mean SR-LAs ( $1.93 \pm 0.44$  vs.  $2.13 \pm 0.56$  s<sup>-1</sup>, n.s., respectively) and mean SR-LAa ( $-2.82 \pm 0.79$  vs.  $-2.97 \pm 1.11$  s<sup>-1</sup>, n.s., respectively) were similar in hypertensive patients with and without DM. The prevalence of hypercholesterolemia in the HT group was significantly greater than that in the normotensive group (33% vs. 16%,  $p < 0.05$ , respectively). No significant difference in the mean SR-LAs ( $2.09 \pm 0.62$  vs.  $2.11 \pm 0.51$  s<sup>-1</sup>, n.s., respectively) or mean SR-LAa ( $-2.99 \pm 0.91$  vs.  $-2.94 \pm 1.14$  s<sup>-1</sup>, n.s., respectively) was found between hypertensive patients with and those without hypercholesterolemia. In normotensive subjects, neither the mean SR-LAs nor the mean SR-LAa was significantly different between the subjects with and those without hypercholesterolemia.

As shown in our Results section, the body mass index (BMI) showed no correlation with either mean SR-LAs ( $p = 0.08$ ) or mean SR-LAa ( $p = 0.88$ ). The prevalence of obesity, which was defined as BMI > 25 kg/m<sup>2</sup>, was comparable between the HT group and normotensive group (32% vs.

48%, respectively). Neither the mean SR-LAs nor the mean SR-LAa was not significantly different between the hypertensive patients with and without obesity.

In short, we believe that the presence of DM, hypercholesterolemia, obesity and/or a history of paroxysmal AF could have negligibly influenced the results of our study. We agree that other large-scale prospective studies will be needed to examine whether the presence of these factors adversely effects LA function as assessed by strain rate imaging.

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