In this issue of the Journal, Oishi et al report on the use of 2-dimensional speckle tracking echocardiography (2D-STE) to demonstrate the negative effect of cardiovascular risk factors on left atrial (LA) and left ventricular (LV) function related to aortic stiffness.1 Their study showed that myocardial strain measurement by 2D-STE clearly identified the effects of cardiovascular risk factors on LA and LV function, and the correlation with arterial stiffness. They conclude that 2D-STE is a useful diagnostic modality for earlier detection of abnormal LA and LV function related to increased aortic stiffness in asymptomatic patients with cardiovascular risk factors.

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Previous studies have revealed that the use of strain assessment by 2D-STE enables noninvasive and accurate assessment of myocardial strain, not only of the LV myocardium,2,3 but also the right ventricular myocardium,4 but only recently has it been used for the assessment of LA strain5 and arterial circumferential deformation.6 Unlike conventional echocardiography, 2D-STE can evaluate the longitudinal strain and strain rates of the LA. It is well known that the myocardial layer of the LA has a 2-part composition; that is, longitudinal and circumferential fibers.7 Although evaluation of LA function is challenging because of its thin myocardium, the feasibility and excellent reproducibility of LA strain measurement has been reported.8 The contractile, conduct, and reservoir functions of the LA can be estimated from analysis of the strain curve.9 Recent study showed that the ratio of early diastolic transmural flow to mitral annular velocity (E/e')/peak systolic LA strain (S-LAs) was an index of LA stiffness.10 The E/e'/S-LAs was significantly higher in patients with diastolic heart failure compared with those with diastolic dysfunction but not in heart failure.

Aortic stiffness has recently been recognized as an important marker of cardiovascular events in patients with cardiovascular risk factors11 and those with heart failure with preserved ejection fraction.12 To date, the effect of cardiovascular risk factors on LA and LV dysfunction, and the relationship with changes in aortic stiffness (LA-LV-arterial coupling) has not been fully elucidated. Oishi et al evaluated abdominal aortic stiffness by calculating its circumferential deformation by 2D-STE, and investigated the effect of cardiovascular risk factors on LA-LV-arterial coupling.1 In their multivariate linear regression analysis, peak early diastolic LV longitudinal strain rate (SR-LVe) and E/e'/S-LAs were independent predictors related to abdominal aortic stiffness in patients with cardiovascular risk factors. On the other hand, only age contributed to abdominal aortic stiffness in the control subjects. The authors state that the subendocardial LA and LV layers in the longitudinal direction, and the intima and media of large artery are simultaneously impaired by cardiovascular risk factors in subclinical patients. In addition, they also state that LA and LV myocardial fibrosis and medial degeneration of the abdominal aorta might be important mechanisms of impaired LA-LV-arterial coupling.1

For the detection of myocardial fibrosis, cardiac magnetic resonance imaging (MRI) is the reference method. Late gadolinium-enhanced MRI (LGE-MRI) can detect small subendocardial infarction with high spatial resolution compared with myocardial single-photon-emission computed tomography (SPECT).13 There are some comparative studies of strain measurement by 2D-STE and cardiac MRI. As for the evaluation of LV myocardial scar, LV myocardial strain evaluation by 2D-STE can detect segments with myocardial infarction, and global LV strain is closely associated with infarct size on LGE-MRI.14 In regard to the assessment of myocardial fibrosis in the LA, Oakes et al reported a method that uses LGE-MRI for assessing LA wall enhancement, which is presumed to be fibrosis.15 They also showed that LA wall fibrosis on LGE-MRI is inversely related to LA strain and the strain rate measured by 2D-STE. As cardiac MRI is useful for tissue characterization, a comparative study with cardiac MRI might be of value to investigate whether or not the fibrotic process is associated with abnormal LA-LV-Ao coupling. In addition, as the current authors point out, further interventional study is required to clarify whether angiotensin II receptor blockers and statin treatment have a beneficial effect for patients with abnormal LA-LV-arterial coupling.

### References

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