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Editorial

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The Role of Systolic Blood Pressure Reduction in Diastolic Dysfunction: RAAS Inhibition versus Non-RAAS Blood Pressure Lowering

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▸ See the article "Addition of Amlodipine or Valsartan for Improvement of Diastolic Dysfunction Associated with Hypertensi" in volume 28 on page 174.

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Hypertension significantly contributes to cardiovascular morbidity and mortality by causing substantial structural and functional adaptations, including left ventricular (LV) diastolic dysfunction (DD).[1)2)] LVDD is characterized by alterations in LV diastolic filling, which may include impairments in myocardial relaxation and abnormal distensibility of the myocardium. Importantly, LVDD is considered a critical link between hypertension and heart failure (HF), particularly in individuals with HF preserved ejection fraction (HFpEF), which is quite prevalent, accounting for up to one-half of patients with HF, and is associated with substantial morbidity and mortality.[3)4)] The prevalence of HFpEF has progressively increased over the last decades, but death rates have not changed substantially.[5)] These trends highlight the importance of understanding the pathophysiologic alterations that precede the development HFpEF, particularly hypertension-induced LVDD.

Several studies have demonstrated consistently that lowering blood pressure (BP) with antihypertensive medications improves LVDD.[6-10)] This effect was seen with several antihypertensive classes, indicating that BP reduction rather than the use of specific antihypertensive agents is important to improve LVDD including a rennin-angiotensin-aldosterone system (RAAS) inhibitor or non-RAAS BP lowering agents. Until now, there is no randomized trial to compare the effect of RAAS inhibitor versus non-RAAS BP lowering agents on improving DD in hypertensive patients.

In this issue of *Journal of Cardiovascular Imaging*, Oh et al.[11)] tested addition of amlodipine to standard antihypertensive therapy for superiority to addition of valsartan in improving DD by lowering systolic BP more effectively in hypertensive patients. In this prospective, multicenter, open-label, randomized trial at four centers in Korea, 104 controlled, hypertensive patients with DD were randomly assigned to receive either amlodipine 2.5 mg (amlodipine arm) or valsartan 40 mg (valsartan arm) in addition to antihypertensive therapy. Systolic BP decreased significantly from baseline in both treatment groups ($p < 0.001$). E/E′ decreased significantly from 13.0 ± 2.2 to 12.0 ± 2.7 in the amlodipine arm and from 14.4 ± 4.3 to 12.7 ± 3.7 in the valsartan arm ($p < 0.01$ in both groups). The change of E/E′ was not significantly different between treatment groups ($p = 0.25$). There were also no significant

between-group differences regarding the changes in systolic BP, LV mass index, and left atrial volume index.

This study has several limitations. This is a mechanistic study, not an outcome trial. Thus, this study results cannot conclude which class of antihypertensive agents improves cardiovascular outcome in hypertension with DD. The authors defined DD as the ratio of mitral inflow velocity to annular relaxation velocity (E/E′) > 10 instead of the recent definition of DD.[12] Also, in this study, novel echocardiographic indices of longitudinal and circumferential strain and diastolic strain rate by speckle tracking echocardiography (STE) were not used. Although there are several limitations, this study was a well-designed, randomized trial to compare the effect of amlodipine versus an angiotensin receptor blocker on improving DD in hypertensive patients.

In conclusion, Oh et al.[11] are to be congratulated for their work that describes addition of low-dose calcium channer blocker or angiotensin-converting enzyme inhibitors associated with a significant improvement of DD in controlled hypertension patients.

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