A rterial hypertension is a major risk factor for congestive heart failure. Left ventricular (LV) hypertrophy is often associated with arterial hypertension and characterizes subjects with a particularly elevated risk of untoward cardiovascular events, including heart failure. LV hypertrophy is, in turn, associated with impaired LV myocardial contractility and LV diastolic dysfunction. LV hypertrophy, impaired LV myocardial contractility, and LV diastolic dysfunction predict heart failure in population-based studies. 

LV hypertrophy regression, proven to be protective in LV hypertrophy. The study tested the hypothesis that lower in-treatment blood pressure (BP) target could result in greater LV diastolic function regression. Accordingly, Doppler parameters of LV diastolic function were compared between a regimen defined “intensive,” because of a predefined systolic BP (SBP) target of <130 mm Hg, and a second regimen defined “standard,” because of a prespecified SBP target of <140 mm Hg. Combinations of valsartan, either 160 or 320 mg, plus amlodipine, either 5 or 10 mg, were used; additional antihypertensive medications were added if needed to reach the BP targets. The main finding of the study was that changes in Doppler parameters of LV diastolic function were comparable, on average, between the 2 treatment arms. Hence, on the matter of the impact of aggressive or standard antihypertensive treatment on LV diastolic dysfunction regression, there is apparently no good news.

However, the study cannot be simply “dismissed” as a negative result. As may be seen in Table 4 and Figure 4 of the study, BP reduction by either treatment regimen was associated with improvement in the Doppler parameter of LV diastolic function. Given the SDs seen in Table 3, within-group variability of end-study systolic BP prevailed on between-group differences, generating a significant overlap in final BP levels in the 2 groups. In fact, 50% of the patients in the intensive treatment arm did not reach the systolic BP target of systolic BP <130 mm Hg, whereas 25% of the patients in the standard treatment arm actually had systolic BP <130 mm Hg at 6-month follow-up. Nevertheless, isovolumic relaxation time, which is a measure of active LV relaxation, was shorter with intensive treatment and was so even when normalized by heart rate, which is important, because β-blockade was allowed in the study and was used in more than half of the patients in the intensive treatment arm. Table 4 showed that reduction in isovolumic relaxation time was consistent and progressively greater with a greater percentage reduction in systolic BP in the study. Hence, the impact of the 2 treatment regimens on LV diastolic function may not be as similar as it appears.

Whether changes reported by Solomon et al indicate “true” LV diastolic function improvement could be disputed. Doppler echocardiography can be a reproducible method for assessment of LV diastolic function. However, assessment of intrinsic LV diastolic properties, that is, LV relaxation and chamber compliance, cannot be derived immediately from traditional Doppler parameters and may require more sophisticated approaches. This is because traditional Doppler echocardiography, such as the popular isovolumic relaxation time, E deceleration time, and E/A, are “load dependent.” In

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contrast, tissue Doppler imaging and color M-mode–derived parameters of LV diastolic function, such as E’ and E-wave propagation rate, are significantly less preload dependent, and their combination with traditional Doppler parameters allow for better representation of LV relaxation and LV chamber compliance. Solomon et al found that E’ increased and E/E’ decreased with BP lowering, suggestive of improved LV relaxation and increase in LV compliance. Therefore, increase in E and in E/A and shorter isovolumic relaxation time and E deceleration time reported at follow-up could be put in the right context of true improvement in LV diastolic relaxation (and LV suction capability) with BP lowering.

On a separate issue, the study by Solomon et al showed that final brachial and central systolic BPs were slightly lower in patients in the intensive treatment arm compared with those in the standard treatment arm; in contrast, LV mass and left atrial volume index regressions, augmentation index reduction, and LV ejection fraction increase were similar in the 2 treatment regimens. A seducing hypothesis is that LV mass regression and arterial stiffness reduction in hypertension may be more relevant (or more reliable) than BP lowering as predictors of LV diastolic function improvement during treatment in arterial hypertension. Previously, LV hypertrophy regression was identified as a relevant marker of cardiovascular risk prediction has been on the basis of observational studies. However, a new paradigm is emerging in arterial hypertension. For now, the fact that isolated LV diastolic dysfunction may regress by lowering BP in patients with uncontrolled arterial hypertension is in itself good news indeed.

Disclosures

None.

References

Treatment of Isolated Left Ventricular Diastolic Dysfunction in Hypertension: Reaching Blood Pressure Target Matters
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