

Decreased Aortic Inertance Increases Susceptibility of Late-Systolic Left Ventricular Ejection to Arterial Wave Reflections

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Background: Left ventricular (LV) afterload patterns consisting of late-systolic loading has been linked to LV remodeling and fibrosis in a number of studies. The contributions from arterial wave reflections (WR) has therefore garnered much interest. Aortic dilation may facilitate the adverse effects of WRs through its effect on aortic inertance. Decreased aortic inertance from aortic dilation is particularly important in late-systole, when the LV-aortic pressure gradient generally reverses and ejection decelerates until time of aortic valve closure.

Hypothesis: Decreased aortic inertance from aortic dilation is associated with LV hypertrophy.

Methods: We measured carotid-femoral pulse wave velocity (PWV; a measure of arterial stiffness) and LV mass (LVM) with SSFP-MRI in 409 subjects (mean age = 61 years). Aortic geometry was measured using SSFP-MRI, with a novel 3D aortic analyzer (Medical Imaging Applications, Coralville, Iowa). We computed compliance and inertance from PWV and geometric measurements. Reflection magnitude (RM) was calculated from pressure-flow analysis of calibrated carotid tonometry and aortic flow (PC-MRI).

Results: A non-linear relationship between inertance and LVM was found, with a more pronounced slope at lower inertance values (Figure). After log-transformation of LVM and adjusting for age, height, weight, sex, and area compliance of the thoracic aorta, decreased aortic inertance was independently associated with increased LVM (standardized $b=-0.382$; $P<0.001$). Aortic inertance was the strongest predictor of LVM in this model, whereas area compliance was not predictive. There was significant interaction between inertance and RM ($P=0.029$) such that the negative relationship between inertance and LVM was stronger for greater RM.

Conclusions: Reduced inertance from aortic dilation is independently associated with LV hypertrophy. This is consistent with the principle that reduced inertance diminishes the buffer between pressure gradient transients and aortic flow. In late-systole, augmentation of the negative LV-aortic pressure gradient by WRs imposes a greater deceleration force on LV ejection.

