Greater Early and Late Arterial Loading with Advancing Age is Associated with Impaired Hemodynamic Efficiency in a Community Dwelling Population

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Objective:
Aortic elastic properties are pivotal for proper arterial-ventricular coupling and optimal hemodynamic efficiency, minimizing wasted, potentially damaging, energy. Major alterations in arterial properties ensue with aging, potentially reducing hemodynamic efficiency. Therefore, we hypothesized that hemodynamic efficiency is reduced with advancing age in a community dwelling population free of cardiovascular disease; this decline is explained in part by alterations in arterial loading parameters.

Methods:
We studied 382 participants (185 men, age range 26-95) from the Baltimore Longitudinal Study of Aging who had carotid tonometry and left-ventricular outflow Doppler testing performed. Pressure and flow waveforms were analyzed using custom-designed Matlab software to calculate total and steady state power. Efficiency was defined as the quotient of mean and total power. Early loading was assessed by characteristic impedance (Zc), while late loading was assessed by reflected wave transit time (RWTT), and reflection coefficient (RC); nonpulsatile loading was assessed by total vascular resistance (TVR).

Results:
Efficiency declined with advancing age (β=-0.1, P<0.0001), adjusting for gender, race, weight hypertension, and diabetes. Hemodynamically, efficiency was directly associated with heart rate, RWTT, and RC while it was inversely associated with Zc and TVR. In standardized multivariate analysis, RWTT had the highest standardized coefficient (β=34.2, P=0.0002) followed by RC (β=21.5, P<0.0001), Zc (β=-19.9, P=0.0024), TVR (β=-5.4, P<0.0001), and HR (β=0.2, P<0.0001). The hemodynamic variables explained 27% of the reduced efficiency with advancing age.

Conclusion:
Hemodynamic efficiency is reduced with advancing age. This decline is associated with greater nonpulsatile, early arterial loading, and shorter reflection time. Such a decline in efficiency implies greater wasted energy with aging that is potentially dissipated in the central arteries and high-flow organs, contributing to arterial remodeling and chronic diseases of aging. Further prospective analyses of larger samples is needed to examine whether hemodynamic efficiency and wasted energy parameters better predict the longitudinal decline in end-organ function.