Large Artery Stiffness in Health and Disease

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The aorta exerts a critically important cushioning function to limits arterial pulsatility due to the intermittent left ventricular ejection. This cushioning function protects the microvasculature from excessive fluctuations in pressure and blood flow. Aortic stiffening, which occurs with aging and various disease states, impairs this cushioning function, and has important deleterious consequences on cardiovascular health. These include isolated systolic hypertension, excessive penetration of pulsatile energy into the microvasculature of target organs that operate at low vascular resistance, and unfavorable ventricular-arterial interactions that promote left ventricular remodeling, fibrosis, dysfunction, and heart failure.

Given its important role in arterial hemodynamics and cardiovascular physiology, it is not surprising that large-artery stiffness independently predicts the risk of new onset hypertension, as well as hard cardiovascular events. In addition to the brain and the kidney, other high-flow target organs may be susceptible from excessive pressure and flow pulsatility, including the testicles, the pancreatic islets and the liver. Recent data indicate that arterial stiffness precedes and predicts the onset diabetes. Aortic stiffening represents a high-priority therapeutic target to ameliorate the global burden of cardiovascular disease. We will review key physiologic aspects related to arterial stiffness, the impact of aortic stiffening on target organs, noninvasive methods for the measurement of large artery stiffness, mechanisms leading to aortic stiffening, and clinical applications of arterial stiffness measurements.

Pusatile hemodynamics in Heart Failure with Preserved Ejection Fraction

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Heart failure with preserved ejection fraction (HFpEF) is a growing clinical problem. Hypertension has long been known to be a powerful risk factor for HFpEF. However, patients with identical BP may have substantial different afterload patterns, which can be assessed via analyses of the time-varying pressure and blood flow generated by the left ventricle (LV).

Accumulating data indicate that arterial wave reflections are key for left ventricular afterload. During systole, the LV generates a forward-traveling wave which travels with a given speed along the wall of the aorta and more distal conduit arteries, and is partially reflected at sites of impedance mismatch (branching points, lumen diameter tapering, change in local stiffness). Innumerable small reflections from distributed reflection sites are transmitted back toward the heart and merge into a discrete reflected wave, which travels back to the LV. In adolescents, the bulk of reflected waves arrive at the aortic root during diastole, augmenting coronary perfusion pressure. With advancing age, PWV increases, and reflected waves arrive at the heart during mid-to late systole. Under these circumstances, reflected waves can exert important unfavorable effects, including myocardial fibrosis, dysfunction and failure. Accumulating evidence links wave reflection to important cardiac abnormalities. Wave reflections at rest and during exercise are increased in HFpEF. Therapeutic approaches to address malignant afterload patters in HFpEF will be discussed.