Arterial stiffness

Arterial stiffness	
Biological system	arteries

Arterial stiffness occurs as a consequence of biological aging and arteriosclerosis. Inflammation plays a major role in arteriosclerosis development, and consequently it is a major contributor in large arteries stiffening.[1] Increased arterial stiffness is associated with an increased risk of cardiovascular events such as myocardial infarction and stroke, the two leading causes of death in the developed world. The World Health Organization predicts that in 2010, cardiovascular disease will also be the leading killer in the developing world and represents a major global health problem.

Several degenerative changes that occur with age in the walls of large elastic arteries are thought to contribute to increased stiffening over time, including the mechanical fraying of lamellar elastin structures within the wall due to repeated cycles of mechanical stress; changes in the kind and increases in content of arterial collagen proteins, partially as a compensatory mechanism against the loss of arterial elastin and partially due to fibrosis; and crosslinking of adjacent collagen fibers by advanced glycation endproducts (AGEs).[2]

Contents

Background

When the heart contracts it generates a pulse or energy wave that travels through the circulatory system. The speed of travel of this pulse wave (pulse wave velocity (PWV)) is related to the stiffness of the arteries. Other terms that are used to describe the mechanical properties of arteries include elastance, or the reciprocal (inverse) of elastance, compliance. The relationship between arterial stiffness and pulse wave velocity was first predicted by Thomas Young in his Croonian Lecture of 1808 [3] but is generally described by the Moens–Korteweg equation[4] or the Bramwell–Hill equation.[5] Typical values of PWV in the aorta range from approximately 5 m/s to >15 m/s.

Measurement of aortic PWV provides some of the strongest evidence concerning the prognostic significance of large artery stiffening. Increased aortic PWV has been shown to predict cardiovascular, and in some cases all cause, mortality in individuals with end stage kidney disease,[6] hypertension,[7] diabetes mellitus[8] and in the general population.[9][10] However, at present, the role of measurement of PWV as a general clinical tool remains to be established. Devices are on the market that measure arterial stiffness parameters (augmentation index, pulse wave velocity). These include Complior, CVProfilor, PeriScope, Hanbyul Meditech, Mobil-O-Graph NG, BP Plus (Pulsecor), PulsePen, BPLab Vasotens, Arteriograph, Vascular Explorer, and SphygmoCor.[11]

Pathophysiological consequences of increased arterial stiffness

The primary sites of end-target organ damage following an increase in arterial stiffness are the heart, the brain (stroke, white matter hyperintensities (WMHs)), and the kidneys (age-related loss of kidney function). The mechanisms linking arterial stiffness to end-organ damage are several-fold.

Firstly, stiffened arteries compromise the Windkessel effect of the arteries.[12] The Windkessel effect buffers the pulsatile ejection of blood from the heart converting it into a more steady, even outflow. This function depends on the elasticity of the arteries and stiffened arteries require a greater amount of force to permit them to accommodate the volume of blood ejected from the heart (stroke volume). This increased force requirement equates to an increase in pulse pressure.[12] The increase in pulse pressure may result in increased damage to blood vessels in target organs such as the brain or kidneys.[13][14] This effect may be exaggerated if the increase in arterial stiffness results in reduced wave reflection and more propagation of the pulsatile pressure into the microcirculation.[13]

An increase in arterial stiffness also increases the load on the heart, since it has to perform more work to maintain the stroke volume. Over time, this increased workload causes left ventricular hypertrophy and left ventricular remodelling, which can lead to heart failure.[15] The increased workload may also be associated with a higher heart rate, a proportionately longer duration of systole and a comparative reduction of duration of diastole.[16] This decreases the amount of time available for perfusion of cardiac tissue, which largely occurs in diastole.[12] Thus the hypertrophic heart, which has a greater oxygen demand, may have a compromised supply of oxygen and nutrients.

Arterial stiffness may also affect the time at which pulse wave reflections return to the heart. As the pulse wave travels through the circulation it undergoes reflection at sites where the transmission properties of the arterial tree change (i.e. sites of impedance mismatch). These reflected waves propagate backwards towards the heart. The speed of propagation (i.e. PWV) is increased in stiffer arteries and consequently reflected waves will arrive at the heart earlier in systole. This increases the load on the heart in systole.[17]

See also

- John R. Cockcroft, notable researcher on the subject
- Pulse wave velocity
- Compliance (physiology)

Notes

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