

## **The Arterial Pulse in Aging**

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Typically, changes in the arterial tree with age are inferred from the brachial cuff sphygmomanometer, and comprise a moderate increase in systolic pressure (25 mmHg), decrease in diastolic pressure (5 mmHg) and increase in pulse pressure (30 mmHg). Aging changes in the central arteries are much higher (increase in pulse pressure by ~ 200%). These central pressure changes are accompanied by like increase in left ventricular systolic pressure and reduction in left ventricular perfusion pressure during diastole (1).

Changes in central hemodynamics can be explained and monitored from changes in the arterial pressure pulse waveform. Marey (2) and Mahomed (3) were the first to describe these pressure waveform changes with mechanical sphygmograms and emphasised the typical changes as being increase in or augmentation of the late systolic peak and disappearance in the secondary diastolic pressure wave. Mahomed noted similar changes in persons with high blood pressure, and their association with heart failure, stroke, and early death

Using accurate electronic tonometry, Kelly et al (4) showed similar changes in the radial and carotid artery pressure waveforms, with changes more marked in the carotid. Others at cardiac catheterisation noted even greater augmentation in the ascending aorta, with magnitude of the secondary late systolic wave approaching the magnitude of the initial pressure wave so that augmentation index (magnitude of the augmented wave ÷ pulse pressure approached 50%) (5). These changes were attributed to increased stiffening of the proximal aorta (measured as increase in characteristic impedance and pulse wave velocity) with increase in the augmented pressure wave attributed to this and early wave reflection from peripheral arterioles. Similar changes were seen in hypertension (3) and attributed to the same mechanism (6).

Changes in shape and amplitude of the arterial pressure wave with aging have been measured by others including the Framingham investigators. Early results (7) have been interpreted to show reduction in wave reflection over the age of 60 years so that increase in central aortic pressure is caused principally by increased aortic stiffness and decrease in secondary late systolic augmentation. In other work, Mitchell has attributed such change to decrease in "effective" diameter of the aorta with age.

Mitchell's work has been challenged (8). The concept of decreasing effective aortic diameter with age has not been confirmed in a longitudinal analysis of Framingham data (9). Analysis of the ACPP normal cohort by Namasivayam et al (10) shows that augmentation from early wave reflection remains dominant over age 60, as it is in earlier life. Potential problems with Mitchell's work remain identification of an inflection point on the aortic pressure wave for measurement of augmentation, errors in measuring flow in the LV outflow tract and relating this to carotid pressure for determination of aortic impedance. Other potential problems include assumption of identical aortic flow waveforms with aging and failure to consider the curvilinearity of augmentation index with age when components of this (pulse pressure and augmented pressure) rise linearly with age, but have different intercepts on the pressure axis.

These issues will require resolution in the near future if aortic pressure is to attain a position as superior to brachial systolic pressure, as a better predictor of cardiovascular events and as a target for therapy.

Michael O'Rourke is a founding director of AtCor Medical, manufacturer of systems for analysing the arterial pulse.

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