

Exercise hypertension is related to aortic reservoir function: a first in-human exercise central haemodynamic study

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Exercise hypertension is prognostically important, but little is known on central haemodynamic factors associated with this response. We hypothesized that increased exercise central blood pressure (BP) would be related to increased forward wave travel and proximal aortic reservoir function. This study aimed to determine this using wave intensity analysis (WIA) for the first time during exercise. Following routine diagnostic coronary angiography, simultaneous pressure and Doppler flow velocity waveforms were recorded in the ascending aorta *via* sensor-tipped intra-arterial wires in 10 participants (mean age 54 ± 10 years, 70% male) with normal left-ventricular function and who were free of coronary artery disease. Measures were recorded at baseline and during light-moderate intensity cycle exercise at 60% of age-predicted-maximum heart rate (HR). Aortic reservoir pressure was calculated by subtraction from the central pressure waveform. Using WIA we identified dominant wave types throughout the cardiac cycle (forward and backward, compression and decompression waves). From rest to exercise, HR and maximal central BP increased significantly (+ 28% and + 15%, $P < 0.001$ for both respectively). The strongest correlate of the change in maximal BP was the change in peak aortic reservoir pressure ($r = 0.755$, $P = 0.012$). During exercise, there were significant increases in forward compression waves during early systole (baseline $27.02 \times 10^6 \pm 15.71 \times 10^6 \text{ W.m}^{-2}\text{s}^{-2}$ vs exercise $41.01 \times 10^6 \pm 21.09 \times 10^6 \text{ W.m}^{-2}\text{s}^{-2}$, + 62% $P = 0.014$, corresponding to peak ejection) and forward decompression waves in late systole (baseline $10.96 \times 10^6 \pm 4.60 \times 10^6 \text{ W.m}^{-2}\text{s}^{-2}$ vs exercise $20.88 \times 10^6 \pm 5.79 \times 10^6 \text{ W.m}^{-2}\text{s}^{-2}$, + 127% change $P < 0.001$, corresponding to myocardial deceleration pre-aortic valve closure), but no change in backward travelling waves ($P > 0.05$ for all). We conclude that increased central BP with exercise may be due to impaired aortic reservoir function and major increases in aortic forward travelling waves that occur in early systole. These findings have relevance to understanding the pathophysiology of exercise hypertension.