

Obese young adults exhibit early peripheral arterial remodelling but preserved conduit artery function

Scott T. Chiesa, Marietta Charakida, Alicja Rapala, Devina J. Bhowruth, Helen C. Nguyen, Frida Dangardt, George Davey Smith, Debbie A. Lawlor, and John E. Deanfield

Background:

Elevated BMI in young adulthood is a causal driver of increased left ventricular mass and blood pressure, but appears to have little effect on peripheral vascular function (Wade et al 2017).

Purpose:

To investigate whether chronically increased resting blood flows and shear rates caused by an obesity-driven hyperaemic state protect the peripheral vasculature at this early age via structural and functional changes within the conduit arteries.

Methods:

We studied 412 young adults (23.4 ± 0.5 years; 272 female) from the Avon Longitudinal Study of Parents and Children (ALSPAC) and those with $\text{BMI} \geq 30 \text{ kg/m}^2$ were classed as obese. Resting brachial arterial diameters and flows were measured and shear rates were calculated as $4 \times$ time-averaged mean velocity / diameter. Blood pressure, endothelial function (flow-mediated dilation: FMD) and arterial stiffness (carotid-radial pulse wave velocity: PWV) were also measured. In a subset of patients ($n=164$), 50MHz very high-resolution ultrasound (VHRUS) was used to measure radial artery diameter, intima-media thickness (IMT), and individual intima and media thicknesses (IT and MT, respectively).

Results:

14% of participants were obese. Brachial artery diameter and blood flow were increased in obese vs. normal weight individuals (3.51 ± 0.07 vs. $3.15 \pm 0.03 \text{ mm}$ and 49 ± 3 vs. $35 \pm 1 \text{ ml} \cdot \text{min}^{-1}$, respectively; $p < 0.001$ for both). Brachial artery diameter was unrelated to blood pressure ($r = 0.14$; $p = 0.44$) but was associated with resting brachial blood flow ($r = 0.68$; $p < 0.001$), resulting in identical resting vessel shear rates between groups (93.4 ± 4 vs. $93.4 \pm 2 \text{ s}^{-1}$; $p = 0.99$). FMD was improved in obese participants ($+1.2\%$; $p = 0.04$) due to greater post-ischaemic reactive hyperaemia. Arterial stiffness, measured by PWV, was not different ($p = 0.38$). In contrast to the brachial artery, radial artery diameter was not different (1.88 ± 0.08 vs. $1.81 \pm 0.04 \text{ mm}$; $p = 0.42$), and radial IMT was increased in obese individuals (0.17 ± 0.01 vs. $0.15 \pm 0.01 \text{ mm}$; $p = 0.014$). This response was driven entirely by increases in media thickness (0.10 ± 0.01 vs. $0.08 \pm 0.01 \text{ mm}$; $p = 0.004$), was associated with increased systolic blood pressure ($r = 0.40$; $p = 0.04$), and resulted in an increase in the MT/diameter ratio (0.056 ± 0.005 vs. 0.047 ± 0.002 ; $p = 0.023$).

Conclusion:

Chronic elevations in arterial blood flow in young obese individuals may preserve conduit artery function via increased vessel shear stress and physiological structural remodelling, resulting in no evident relationship with adverse cardiovascular risk factors at this age. The observation of increased media thickening in smaller radial arteries suggests that early pressure-driven pathological changes in arterial structure and function at this age may be best studied using VHRUS in more distal vascular beds.