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3.8

CHILDHOOD OBESITY: DOES IT HAVE ANY EFFECT ON YOUNG ARTERIES?

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Background: Prevalence of overweight (OW) and obesity (O) in children and adolescents has been increased in the past three decades. Obese children are prone to develop early cardiovascular (CV) morbidity in their adult life. Impaired arterial stiffness might be detected in this population. The aim of our study was to compare the arterial function parameters (AFPs) in O/OW patients and healthy subjects.

Methods: 6.816 subjects (3.668 boys) aged 3–18 years were recruited and categorised by their body mass index (BMI) into normal weight (N), OW and O groups regarding their age and sex. AFPs were measured by occlusive-oscillometric device. Propensity score matching was carried as statistical test.

Results: 19.9% (n = 1.356) of the population were OW/O, 911 (516 boys) were OW and 445 (273 boys) were O. PWV_{ao} did not differ significantly between N (5.9 ± 0.8 m/s) and OW patients (5.9 ± 0.8 m/s); and N (6.0 ± 0.7 m/s) and O patients (6.0 ± 0.8 m/s). Aix_{ao} was significantly lower in OW (9.3 ± 7.4% vs 7.6 ± 7.0%, p < 0.00001) and in O patients (9.7 ± 8.1% vs 6.6 ± 7.2%, p < 0.00001) compared to controls. No significant difference was found regarding SBP_{ao} values between controls and OW and O groups (N = 110.7 ± 12.4 mmHg vs OW = 110.3 ± 11.9 mmHg; N = 115.6 ± 14.0 mmHg vs O = 114.3 ± 12.8 mmHg).

Conclusions: Aortic stiffness – expressed by PWV_{ao} – did not differ between N and O/OW children and adolescents, however Aix_{ao} was remarkably, significantly lower in O/OW patients. We may conclude that the pathophysiological consequences in the circulatory system due to childhood OW/O are compensated hemodynamically in these patients, presumably by decreasing total peripheral vascular resistance.

Oral Session IV – Models, Methodologies and Interventions

4.1

PROBING ARTERIAL STIFFNESS AT THE NANO-SCALE USING THE INTERNAL MAMMARY ARTERY AS A NOVEL TARGET

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Introduction: Arterial stiffening is associated with structural and biomechanical alterations in the aorta. However, there are still gaps in our understanding as to how the structure and properties of arteries across the vasculature are altered with high PWV. Objective: To determine whether altered ultrastructural and nanomechanical properties are exhibited in the internal mammary artery (IMA) in high PWV patients.

Methods: Human IMA biopsies were obtained from patients with known carotid-femoral PWV. Patients were grouped as low PWV (8.5 ± 0.7 ms⁻¹, n = 8) and high PWV (13.4 ± 3.0 ms⁻¹, n = 9). With Peakforce QNM atomic force microscopy (AFM) the nanomechanical (elastic modulus) and morphological properties (collagen fibril diameter and D-Period) of the IMA were measured. Principal component analysis (PCA) was used to determine the relationship of nanomechanical and structural data with proteomics data (small leucine rich proteoglycans, SLRPs) [1] and patient metadata.

Results: PCA analysis shows that the nano-scale elastic modulus was one of the key variables which separated low and high PWV groups and was correlated with PWV. Furthermore, nano-scale alterations in adventitial collagen fibrils were evident. D-Period and collagen fibril diameter were found to be negatively correlated. Most SLRPs were closely grouped in the PCA analysis.

Conclusions: Although the IMA is not involved in the carotid-femoral pathway, patients with high PWV exhibited distinct alterations in the IMA at the nano-scale relative to those with low PWV. Our approach provides new insight into systemic structure-property changes in the vasculature, and also provides a novel method for characterizing small biopsy samples for arterial stiffening studies.

Reference

[1] Hansen ML, Beck HC, Irmukhamedov A, Jensen PS, Olsen MH, Rasmussen LM. Proteome analysis of human arterial tissue discloses associations between the vascular content of small leucine-rich repeat proteoglycans and pulse wave velocity. 2015 Arterioscl. Thrombosis Vasc. Biol. 35:1896-1903.

4.2

DISCREPANCY BETWEEN IN-VIVO MEASURE AND EX-VIVO CALCULATION OF PULSE WAVE VELOCITY IN RETINAL ARTERIES

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Background: Pulse wave velocity (PWV) in large arteries is a pressure-dependent marker of arterial stiffness. The retinal vasculature provides unique access to the microcirculation. There is inconsistency between reported values of retinal PWV (rPWV). The pressure dependency of rPWV was measured in-vivo and calculated ex-vivo using retinal artery material properties to investigate the inconsistencies.

Methods: High-speed fundus videos (125 fps) from three Sprague Dawley rats were recorded simultaneously with electrocardiogram and blood pressure. rPWV was measured using the cardiac component of retinal artery diameter waveforms at two retinal sites across a physiological range (70–130 mmHg) of mean arterial pressure (MAP). Ex-vivo tensile testing was performed on bovine retinal arteries, rat retinal arteries being too small for myography. Diameter and wall thickness of the retinal artery adjacent to the optic disc were measured using optical coherence tomography. Tensile testing was performed using a wire myograph in 9 bovine retinal artery specimens. **Results:** In-vivo results showed a significant positive correlation between rPWV (4.9 ± 1.8 mm/s) and MAP (R² = 0.58, p < 0.001) as expected. Ex-vivo, calculated rPWV using material stiffness and geometry measurement ranged between 4.6 and 7.0 m/s at effective distending pressures between 70 and 100 mmHg.

Conclusions: Ex-vivo and in-vivo results differed by three orders of magnitude but should be the same. Ex-vivo results are in the same order as measured in-vivo in large arteries. In-vivo rPWV was lower than expected yet was responsive to changes in MAP. Further studies are required to uncover what rPWV is a measurement of, if not arterial stiffness.

4.3

WHOLE-BODY VS. REGIONAL ARTERIAL STIFFNESS: IMPLICATIONS FOR A SINGLE WINDKESSEL MODEL OF THE CIRCULATION

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Introduction: We questioned whether a single Windkessel (WK) adequately describes the circulation by estimating the radial arterial diastolic pressure-decay constant (tau) and combining this with systemic hemodynamic and arterial stiffness measurements.

Methods: In the non-invasive cardiac laboratory, we performed echocardiography with simultaneous cuff BP, heart-femoral [hf] and femoral-ankle [fa] PWV (Colin VP1000), and radial tonometry (Sphygmocor). Tau was calculated by photo-digitizing the radial pulse contour and fitting pressures (at 20 ms intervals) to: $P = A + ([\text{systolic BP}] - A) \cdot \exp(-(t - t_0)/\tau)$, where P = pressure, A = modeled minimum diastolic BP, and t₀ = mono-exponential decay start time. Systemic vascular resistance (SVR) = mean pressure/[cardiac output]; WK stiffness (1/[WK capacitance]) = SVR/tau; central and peripheral