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### **P3.02: MORNING BLOOD PRESSURE SURGE, BLOOD PRESSURE VARIABILITY AND AORTIC STIFFNESS IN ESSENTIAL HYPERTENSION**

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ranged from 501-861  $\mu\text{m}$  (humans) and 40  $\mu\text{m}$  (rabbit pups) and the mean IMT ranged 543-1011  $\mu\text{m}$  (humans) and 88  $\mu\text{m}$  (rabbit pups). The table shows a summary of the obtained coefficients of variation. The figure shows the diameter and IMT during four cardiac cycles measured with ARTIC. In conclusion ARTIC can be a useful tool to automatically characterize the arterial wall in both humans and small animals.

### P3 Population Science

#### P3.01

##### EFFECT OF BODY SIDE ON ATHEROSCLEROTIC PLAQUE DISTRIBUTION IN THE CAROTID AND FEMORAL ARTERIES

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**Objective:** Atherosclerotic disease is caused by a combination of systemic, and local factors. The influence of local geometry (affecting local flow conditions) is often neglected. In contrast to the carotid artery, at the iliac-femoral artery region, a large degree of bilateral asymmetry exists. Therefore, our aim was to determine the influence of body side on the prevalence of atherosclerosis (i.e. plaque and intima-media thickening; IMT) at the carotid and femoral arteries.

**Methods:** Data were used from the ASKLEPIOS study, including 2524 apparently healthy subjects with a mean age of 46 year (range 35-55). Echographic images were obtained bilaterally at the common carotid and common femoral arteries. A single observer approach was used for the acquisition and quantification of plaques and IMT.

**Results:** The carotid artery displays almost no left-right difference in IMT values nor plaques. In contrast, the femoral artery displays substantially more atherosclerosis on the right side. Specifically, the IMT distribution at the right common femoral artery is more skewed (Percentile 90 right: 1.11 mm, left 1.01 mm;  $p < 0.001$ ), which is mirrored by a significantly higher plaque prevalence (right 21.9 vs left 15.7 %;  $p < 0.001$ ).

**Conclusions:** Atherosclerotic lesions are more prevalent at the right than at the left femoral artery. This finding highlights the role of local arterial geometry on the development of atherosclerosis, and underscores the importance of the choice of body side when assessing vascular health.

#### P3.02

##### MORNING BLOOD PRESSURE SURGE, BLOOD PRESSURE VARIABILITY AND AORTIC STIFFNESS IN ESSENTIAL HYPERTENSION

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Morning blood pressure surge is associated with increased cardiovascular risk, possibly due to its ability to reflect short-term BP variability and/or its link with arterial stiffness. The link between morning BP surge and vascular stiffness is not defined.

689 untreated hypertensives ( $48 \pm 10$  years, BP  $149/92 \pm 17/10$  mmHg) underwent c-f PWV and 24-h ambulatory BP measurement. Morning surge was calculated as: sleep-rough surge (STS, 2-hour average SBP after wake-up minus average of 3 SBP centered on the lowest nighttime reading), pre-awakening surge (PAS, 2-hour average SBP after wake-up minus 2-hour average SBP before wake-up), and rising BP surge (RBS, SBP on rising minus the lowest SBP in the 30' before). Average real variability (ARV, 24h average of the absolute differences between consecutive SBP), was considered a measure of short-term SBP variability.

STS and RBS were directly correlated to c-fPWV ( $r = 0.17$  and  $r = 0.12$ ,  $p < 0.01$ ) and ARV ( $r = 0.28$  and  $r = 0.23$ ,  $p < 0.001$ ), while PAS had no such relationships. Patients in the top quartile of STS ( $> 39$  mmHg) had higher age- and 24-h mean BP-adjusted c-fPWV ( $9.73 \pm 2$  vs  $9.29 \pm 2$  m/s,  $p = 0.004$ ), while no difference was found for the top quartile of PAS or RBS. In a multivariate regression, high STS values predicted a high c-fPWV ( $\beta = 0.08$ ,  $p = 0.038$ ), independently of age, sex, 24-h mean BP and nocturnal BP reduction. After adding ARV ( $\beta = 0.17$ ,  $p < 0.001$ ) to the model, the relationship between STS and c-fPWV was no longer significant ( $\beta = 0.05$ ,  $p = 0.17$ ).

Morning SBP surge, calculated as STS, has a positive relation with aortic stiffness in hypertension, which is no longer significant after adjusting for 24-h short-term BP variability.

#### P3.03

##### INTERNATIONAL COMPARISON OF ESTIMATED AORTIC PULSE WAVE VELOCITY (APWV) IN 3 SETTINGS OF AFRICAN- AND EUROPEAN- ORIGIN PATIENTS AND CONTROLS – GHANA, BRITAIN AND BARBADOS

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**Objectives:** In resource-poor settings, aPWV's prognostic power, independent of blood pressure (BP), has potential as an intervention target against 'hypertension' and diabetes (T2DM). We compared simple determinants of aPWV in 3 African-origin settings and British Europeans.

**Methods:** Using methods standardised across 3 hospital sites in Ghana, Britain and Barbados, aPWV was estimated over 8 cardiac cycles up to 3 times by the single cuff-based Arteriograph, whose prognostic impact was recently reported. Sub-samples had repeatability measured separately. With basic anthropometry, data were analysed by regression (MRA), adjusting for temperature (co-linear with site).

**Results:** 527 people, (Ghana 296; UK 128 (35% African-origin); Barbados 103) 54% women, had satisfactory measurements, in 125 hypertensives without T2DM, 248 T2DMs and 154 apparently healthy controls, mean $\pm$ SD age  $50 \pm 15$ , range 20-87y, BMI  $28.1 \pm 6$  kg/m<sup>2</sup>. Cross-site age-/sex / adjusted mean (SD) aPWV were  $8.8 \pm 1.6$ ,  $9.3 \pm 2.3$  and  $8 \pm 1.8$  m/sec for Ghana, UK & Barbados respectively, with sys/diasBP  $147 \pm 23 / 85 \pm 13$ ,  $136 \pm 17 / 82 \pm 13$  and  $126 \pm 26 / 72 \pm 15$  mmHg. Forced into the MRA model, patient group, BMI and temperature did not contribute while systolic BP (standardized Beta (=B) 0.28), age (0.26) site (0.22) or ethnicity as West African, African-Caribbean  $>$  European (0.3), heart rate (0.21) and gender (0.17), were all related to aPWV,  $p < 0.002$ . Pulse, rather than systolic, pressure did not contribute, but central sysBP did (0.3,  $p < 0.0001$ ).

**Conclusions:** These data suggest that aPWV is not only a robust prognostic indicator but a potential treatment target, across the range of BP less affected by anthropometry and these 'disease' states. Are ethnic effects confounded by BP?

#### P3.04

##### GENETIC AND ENVIRONMENTAL CONTRIBUTIONS TO ARTERIAL STIFFENING, ATHEROSCLEROTIC PLAQUE AND CALCIFICATION

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**Introduction:** The prognostic importance of aortic calcification is often attributed to its close association with atherosclerotic plaque but it may relate to its effects on aortic stiffness and be driven by processes distinct from those of atherosclerosis. The aim of the present study was to determine the individual heritability of atherosclerosis plaque, calcification and aortic stiffness.

**Methods:** For heritability we examined 900 female twins from the Twins UK cohort aged 21-81 years and measured pulse wave velocity (PWV) and calcified and non-calcified plaque by carotid ultrasound. Total aortic plaque burden and calcium score were measured in a sub-sample of 100 women who underwent computed tomography (CT) and magnetic resonance imaging (MRI). Heritability and effect of environmental factors were estimated using structural equation modelling

**Results:** In the total cohort, adjusted heritability estimates of PWV and calcified plaque were 38% (confidence interval (CI): 19-59%) and 0.61 (CI: 4-83%), respectively. In contrast heritability of non-calcified plaque was only 5% (CI: 0-61%). Shared genetic factors accounted for 92% of the correlation between PWV and calcified plaque. In a sub-sample of twins with CT/MRI imaging an additive genetic component accounted for 77% (CI: 32%-89%) of variation in total aortic calcification. Shared genetic factors accounted for  $> 99\%$  of the correlation between PWV and calcification.

**Conclusion:** Our findings suggest calcification is driven by processes that are, at least in part, distinct from atherosclerosis. The association between aortic stiffening and calcification can be accounted for by common genetic factors, which contribute little to atherosclerosis.