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P7.16: VALIDATION OF AN OSCILLOMETRIC BRACHIAL CUFF METHOD TO DERIVE CENTRAL BLOOD PRESSURE USING DIFFERENT CALIBRATION MODES

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pressure-independent index of stiffness of the aorta, femoral and tibial artery (CAVI) is associated with TDI of diastolic function.

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SERUM INFLAMMATORY MARKERS ARE POOR PREDICTORS OF VASCULAR INFLAMMATION AND VASCULAR INFLAMMATION DOES NOT DETERMINE AORTIC STIFFNESS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

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Background: COPD is independently associated with increased cardiovascular events. Arterial stiffening and systemic inflammation are postulated aetiological factors. We hypothesised that vascular inflammation links systemic inflammation with vascular stiffening and sought to test this in a cohort of COPD subjects undergoing baseline FDG PET/CT either as part of the EVOLVE observational study or EVOLUTION trial (NCT 01541852).

Methods: 85 COPD subjects underwent assessments including spirometry, arterial stiffness (aortic pulse wave velocity (aPWV)), inflammatory biomarkers (fibrinogen and hsCRP) and FDG PET/CT imaging (lungs, aorta and carotids) to evaluate inflammation and aortic calcification.

Results: 66% of the cohort were male, median age was 68 (IQR 63-73) years, 87% were ex-smokers. Mean aPWV was 9.9 (SEM 0.2) m/s, aortic calcification volume 7156 (1461) mm³, hsCRP 5.2 (0.8) mg/dl, fibrinogen 3.4 (0.08) g/l. Log hsCRP correlated only with carotid FDG uptake (R=0.23, p=0.04) and log fibrinogen did not correlate with FDG uptake in any vascular region. Systemic inflammatory markers were positively associated with aortic inflammation but only weakly. The estimated change in FDG uptake was 0.2 (95% CI 0.11-0.29) and 0.07 (0.06-0.08), for each log unit change in fibrinogen and hsCRP respectively.

Aortic inflammation was not a significant determinant of aPWV, but aortic calcification was, adjusted for age, supine HR, MAP and years smoked (p=0.02, β =0.26).

Conclusion: HsCRP and fibrinogen are weak predictors of vascular inflammation and therefore likely unsuitable stratification biomarkers of vascular inflammation in COPD. Calcification rather than inflammation appears to be the dominant pathophysiological mechanism underlying arterial stiffness in COPD.

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REACTIVE HYPEREMIA INDEX AND FLOW MEDIATED DILATION WITH UPPER- AND LOWER-ARM CUFF OCCLUSION: ARE THEY MEASURING THE SAME?

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Objective: Methodological issues are major reasons preventing the use of endothelial function testing in clinical practice This study aimed to address the relationship between two non-invasive techniques, brachial artery flow-mediated dilation (FMD) and as reactive hyperemia index (RHI), comparing also lower (forearm, L) and upper (arm, U) cuff occlusion.

Methods: In 17 young healthy subjects (9 males, age 29 ± 4 years) FMD (Cardiovascular Suite, Quipu s.r.l., Pisa, Italy). and RHI (EndoPAT 2000, Itamar Medical, Israel) were measured simultaneously in two separate occasions using 5 minutes of L- or U-ischemia. Baseline and Hyperemic Shear rate (SR) were also computed.

Results: L-FMD $(7.32\pm4.87\%)$ and L-RHI $(0.61\pm0.29\%)$ were significantly lower (p<0.05) and p<0.01, respectively) as compared to U-FMD $(10.48\pm5.67\%)$ and U-RHI $(0.86\pm0.23\%)$. L-RHI and U-RHI tended to be related (r=0.49; p=0.06), while L-FMD and U-FMD were not (r=0.39; p=0.12),

L-FMD was significantly related to L-SR (r=0.62; p<0.01), but not to L-RHI (r=0.17; p=0.54). L-RHI was not significantly correlated with L-SR (r=0.24; p=0,38). U-RHI was related to U-FMD (r=0.50; p<0.05) and to U-SR

(r=0.50, p<0.04). In multiple regression analysis (full model: r2=0.23) U-FMD but not U-SR was associated with U-RHI (r2=0.20; p=0.05).

Conclusions: In healthy subjects, the assessment of FMD and RHI with lower and upper cuff occlusion is not equivalent. L-FMD, but not U-FMD is related to SR increase, thus possibly representing a better marker for conduit artery endothelial function. U-RHI and U-FMD possibly provide similar information on vascular reactivity. Caution is deemed in interpreting studies conducted with different methodologies.

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VALIDATION OF AN OSCILLOMETRIC BRACHIAL CUFF METHOD TO DERIVE CENTRAL BLOOD PRESSURE USING DIFFERENT CALIBRATION MODES

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Background: There is interest in measuring central blood pressure (BP) from non-invasive upper-arm cuff devices, the accuracy of which may be influenced by different calibration modes. The aim of this study was to determine the validity of an upper-arm cuff oscillometric device to estimate central BP by comparison to invasively acquired aortic BP, using different calibration modes.

Methods: 122 patients (mean age 63 ± 13 years) undergoing coronary angiography had simultaneous measurement of ascending aortic BP (via fluid-filled catheter) and non-invasive upper-arm cuff oscillometry (Sphygmocor Xcel) to estimate central BP. A 'derivation' cohort (n=60, 117 simultaneous measures) was randomly selected to produce different calibration modes to estimate central systolic BP. These different calibration modes were then applied to the remaining 'validation' cohort (n=62, 119 simultaneous measures).

Results: Conventional calibration with brachial systolic and diastolic BP underestimated central systolic BP (mean difference -7.2 \pm 9.6 mmHg) with evidence of bias at higher BP values (r=-0.50; p<0.001). The same was observed for oscillometric mean arterial pressure and diastolic BP calibration, but with greater underestimation (mean difference -19.6 \pm 11.9 mmHg) and bias (r=-0.72; p<0.001). A refined calibration mode significantly improved central systolic BP estimation (mean difference 1.0 \pm 11.0 mmHg) and removed all bias (r=0.07; p=0.45). Moreover, this method had greater sensitivity (79.5%) and specificity (80.0%) for predicting central hypertension (invasive aortic systolic BP \geq 130 mmHg) compared to other methods.

Conclusions: Significant improvements in accuracy for estimating central BP are achieved through refinement of standard, non-invasive calibration modes using an oscillometric brachial cuff device.

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DISASSOCIATION OF BLOOD PRESSURE FROM AORTIC RESERVOIR CHARACTERISTICS BETWEEN THE AORTA AND RADIAL ARTERIES

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Background: Aortic reservoir pressure (RP) and excess pressure (XSP) predict cardiovascular events independent of clinic blood pressure (BP). It is unknown whether RP and XSP change in magnitude from the central to peripheral large arteries where conventional BP is measured. This information has implications for understanding the arterial pathophysiology. This study aimed to determine the change in RP and XSP from the aorta to the brachial and radial arteries, as well as associations of these indices with BP.

Methods: 23 participants (aged 65 ± 9 years, 70% male) undergoing clinically indicated cardiac angiography had intra-arterial pressure waveforms measured via fluid-filled catheter in the ascending aorta, brachial (mid-humorous) and radial arteries (wrist) by catheter pull-back. RP and XSP (using previously published algorithms), and BP were derived from pressure waveforms at each location.

Results: There was a non-significant decrease in RP from the aorta to the brachial and radial arteries (112 ± 22 , 109 ± 18 , 103 ± 17 mmHg respectively,