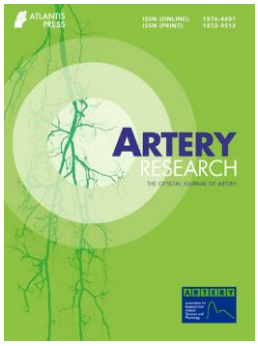

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P7.17: DISASSOCIATION OF BLOOD PRESSURE FROM AORTIC RESERVOIR CHARACTERISTICS BETWEEN THE AORTA AND RADIAL ARTERIES

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

P7.14
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) mm^3, 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 ($p=0.02$, $\beta=0.26$).
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.

P7.15
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±4.87%) and L-RHI (0,61±0.29%) were significantly lower ($p<0.05$ and $p<0.01$, respectively) as compared to U-FMD (10.48±5.67%) and U-RHI (0.86±0.23%). 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.

P7.16
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±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±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±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 ≥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.

P7.17
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,

p=0.242). On the other hand, XSP significantly increased from the aorta to the brachial and radial arteries (21±8, 41±15, 58±19 mmHg respectively, p<0.001). However, neither RP or XSP (either measured by peak or integral) were significantly associated with either systolic BP or pulse pressure at any arterial location (p>0.05 all).
Conclusion: RP is relatively constant between the aorta and radial arteries, whereas XSP increases significantly. Neither indices are related to BP, thus supporting the independent pathophysiological relevance of aortic reservoir characteristics.

P7.18
VALIDATION TESTING FOR THE NON-INVASIVE MEASUREMENT OF AORTIC RESERVOIR CHARACTERISTICS FROM BRACHIAL CUFF OSCILLOMERIC PRESSURE WAVEFORMS

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Background: Aortic reservoir pressure (RP) and excess pressure (XSP) derived non-invasively from radial tonometry independently predict cardiovascular events and mortality. However, whether RP and XSP can be derived non-invasively from brachial oscillometric cuff pressure waveforms has never been undertaken. This study sought to determine the validity of measuring aortic reservoir characteristics from non-invasive oscillometric cuff waveforms.
Methods: 97 participants (aged 62±11 years, 67% male) undergoing coronary angiography had simultaneous measurement (n=247) of ascending aortic pressure (via fluid-filled catheter) and oscillometric brachial cuff pressure (via SphygmoCor XCEL). RP and XSP derived non-invasively from cuff waveforms were compared with invasive measures.
Results: There were small mean differences between non-invasive and invasive methods for both RP (1.42±18.16 mmHg) and XSP (-1.45±25.52 mmHg), with significant corrleations observed between methods (p<0.001 both).
Conclusion: Aortic reservoir characteristics of RP and XSP can be derived non-invasively from oscillometric brachial pressure waveforms, thus providing a mean for widespread research and clinical use.

P7.19
ARTERIAL STIFFNESS AND DISEASE-RELATED ORGAN DAMAGE IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Hypothesis: increased arterial stiffness has been reported in subjects with systemic lupus erythematosus (SLE) compared with healthy controls. In SLE, indexes of organ damage are related to a poor clinical status and worse prognosis independently from the activity of the disease. Data are controversial about the association between SLE-related organ damage and arterial stiffness.
Methods: 40 subjects with history of SLE (mean age 45±12 years, 90% women) and a median disease duration of 12 years (IQR 5-19), underwent assessment of carotid-femoral pulse wave velocity (cf-PWV) by means of applanation tonometry (SphygmoCor). A comprehensive clinical, metabolic and immunological assessment was performed. Irreversible organ damage, not related to active inflammation, was assessed through the Systemic Lupus International Collaborating Clinics (SLICC) damage index.
Results: mean BP was 128/75±16/10 mmHg. 9 subjects (23%) were on antihypertensive treatment, 4 (10%) had had previous cardiovascular events, 17 (42%) were treated with steroids, 29 (71%) with hydroxychloroquine, 15 (37%) with other immunosuppressants. Median SLICC index was 2 (IQR 1-3), average cf-PWV was 7.5±1.9 m/s. cf-PWV significantly increased across SLICC damage index categories (F=3.141, p<0.019). The association between cf-PVW and SLICC index persisted after adjustment for age, sex, mean arterial pressure, height, heart rate, disease duration, anti-hypertensive treatment, number of drugs for SLE therapy, C-reactive protein and previous cardiovascular events (p=0.031).
Conclusions: in subjects with SLE under active treatment, SLICC damage index had a significant independent association with cf-PWV. Further studies are needed to explore the role of arterial stiffness as a predictor of disease-related organ damage in SLE.

P7.20
THE IMPACT OF OBSTRUCTIVE SLEEP APNEA ON ARTERIAL STIFFNESS IS INDEPENDENT OF GENDER IN PATIENTS WITH HYPERTENSION

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Introduction: In men with hypertension, obstructive sleep apnea (OSA) is associated with increased arterial stiffness. However, it is not clear if the impact of OSA on patients with hypertension is similar in women.
Methods: We recruited consecutive patients with established diagnosis of hypertension under a standardized antihypertensive treatment (hydrochlorothiazide plus enalapril or losartan). All patients were submitted to full polysomnography and carotid-femoral pulse wave velocity (PWV). We performed analysis according to the presence of OSA (defined by an apnea-hypopnea index ≥15 events/hour of sleep) and by gender (male and females).
Results: Ninety-five patients were studied (14males without OSA; 28 males with OSA, 29 females without OSA and 24 females with OSA). OSA frequency was 66% in males group versus 45% in females group (p=0.02). The age of female with OSA (59±10yrs) was significantly higher than female without OSA(52±10yrs), while the age did not differ between the male with(58-±10yrs) or without OSA(56±8yrs). The BMI was also significantly greater in female with OSA (32.8±5 vs. 28.7±5kg/m^2), while was similar in male with(30.5±4.5) or without OSA(29.5±2.5). The blood pressure was not different in the patients with or without OSA. PWV was significantly higher in both male(12.7m/s) and female(13.2m/s) with OSA than the couterparts without OSA(male-11 m/s, female-11.7 m/s) even after adjustments by age. The multivariate linear regression showed that OSA was independently associated with PWV(p=0.008).
Conclusions: In patients with hypertension, the presence of OSA is associated with higher PWV regardless of gender.

P8.1
CENTRAL HEMODYNAMICS IN SYSTEMIC SCLEROSIS: A CASE-CONTROL STUDY

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Background: Although a few studies have suggested an alteration in aortic stiffness in patients with systemic sclerosis (SS), a disease characterized by immunological and microvascular changes and by tissue fibrosis, the functional properties of the large arteries have been understudied in SS.
Methods: 34 women with SS [age 60±14 years, BP 123/70±17/10 mmHg] and 34 healthy age- and BP-matched women underwent determination of carotid-femoral pulse wave velocity (PWV, a direct measure of aortic stiffness) and aortic augmentation (SphygmoCor, AtCor). All participants also underwent determination of carotid-radial PWV, as a measure of stiffness of upper-limb arteries. We excluded participants with overt cardiovascular disease and concomitant important disease.
Results: Age and brachial BP were nearly identical in the 2 groups. Patients and controls did not differ by carotid-femoral PWV (9.2±3 vs 9.1±2 m/s, p=0.91) or carotid-radial PWV. Aortic augmentation, was higher in women with SS; unadjusted: 16.1±8 vs 11.5±7, p=0.014; adjusted for pulse pressure and heart rate (AIx@75): 30.9±16 vs 22.2±12, p=0.012). SS independently predicted AIx@75 in a multivariate analysis. Among patients with SS, age, brachial mean BP and serum C-reactive protein all predicted carotid-femoral PWV. Age and mean BP were the only predictors of AIx@75. Organ damage scores had no significat correlation with central hemodynamics parameters.
Conclusions: SS is associated with an increase in aortic augmentation (as a measure of the contribution of reflected wave to central waveform), but not in aortic or upper-limb arterial stiffness. Microvascular involvement might occur earlier than stiffening of the large arteries in SS.