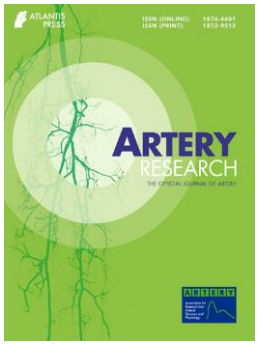

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2.1: A METHOD FOR THE MEASUREMENT OF PRESSURE SENSITIVITY OF CAROTID-FEMORAL PULSE WAVE VELOCITY IN HUMANS

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time elapsed since smoking (<5 years, 5–15 years and > 15 years). Subjects had at the beginning and end of the study determinations of carotid-femoral pulse wave velocity (PWV) and common carotid intima-media thickness. Based on these measurements the annual absolute changes were calculated.
Results: Smoking at baseline was not associated with statistically significant differences in PWV and CIMT. However, the annual change of PWV was statistically different between the groups of smokers, non-smokers and the 3 groups of ex-smokers ($p = 0.041$) after adjustment for relevant confounders. Specifically, smokers had 0.23 m/s/year (95% CI: 0.10 to 0.35), non-smokers 0.17 m/s/year (95% CI: 0.08 to 0.25), quitters (<5 years) had 0.28 m/s/year (95% CI: 0.07 to 0.49), quitters (5–15 years) had 0.35 m/s/year (95% CI: 0.11 to 0.59) and quitters (>15 years) −0.07 m/s/year (95% CI: −0.26 to 0.13). Similar trend for slower progression was observed for CIMT in past smokers (>5 years) but this was not statistically significant.
Conclusions: Quitting smoking slows down progression of vascular aging after many years, implying a period of adjustment for former deleterious effects of smoking.

1.4
CHILDHOOD DETERMINANTS OF EARLY ADULT ARTERIAL STIFFNESS IN DIFFERENT ETHNIC GROUPS

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Childhood determinants of aortic pulse wave velocity [PWV] are poorly understood. We tested how factors measured twice previously in childhood in the MRC 'DASH' study, particularly body mass (BMI) components and BP, affected PWV in young adults.
Methods: Of 6643 London children, aged 11–13 y, from 51 schools in samples of about 1000 in 6 ethnic groups, with markedly different adult cardiovascular risk, 4785 (72%), were seen again at 14–16 y. In 2013, 666 (97% of invited) took part in a young-adult pilot (21–23 y) follow-up. With psychosocial, anthropometric and BP measures, PWV was recorded via an upper arm cuff on the calibrated Arteriograph device. In a subsample (n = 334) PA was measured over 5 days via the ActivPal.
Results: Unadjusted PWVs in Black Caribbean and White UK young men were similar (mean+SD 7.9 + 0.3 vs 7.6 + 0.4 m/sec) and lower in other groups at similar systolic (s)BPs (120 mmHg) and BMIs (24.6 kg/m^2). In fully adjusted regression models, independent of BP effects, while Black Caribbean (higher BMIs and waists), Black African and Indian young women had lower PWV (by 0.5–0.8, 95%CI 0.1–1.1 m/sec) than White UK women (6.9 + 0.2 m/sec), values were still increased by age, BP, powerful impacts from waist/height and time spent sedentary but a reducing impact of time walking >100steps/min (both $p < 0.01$), with a racism effect (+0.4 m/sec) in women. Childhood effects of waist/hip were also detectable.
Conclusion: Even by young adulthood, increased waist/height ratios, lower physical activity, BP and psychosocial variables (eg: perceived racism) are independent determinants of arterial stiffness, likely to increase with age.

1.5
RELATION OF ARTERIAL STIFFNESS WITH LEFT VENTRICULAR DIASTOLIC FUNCTION IN GENERAL POPULATION

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Left ventricular diastolic function declines with aging and hypertension. It is well known that elevated blood pressure results in increased arterial stiffness. The study aims to determine the relationship between arterial stiffness and left ventricular diastolic function in general population.
Methods: We recruited 303 (mean age, 46.9 years; 167 women, 155 normotensives) members of randomly recruited families. Normotension and hypertension were diagnosed based on both office and ambulatory blood pressure measurements, or history of antihypertensive treatment. Pulse wave velocity (PWV), peripheral and central pulse pressure (pPP; cPP) were evaluated by means of pulse wave analysis. Left ventricle (LV) diastolic function was determined by measuring transmitral (early (E) and late (A) diastolic peak velocities and E/A ratio) and pulmonary (peak systolic (S) and diastolic (D) velocity and S/D ratio) flow velocities and diastolic velocities of septal and lateral mitral annulus (E' wave and E/E' ratio) obtained in tissue doppler. Additionaly we measured left atrium diameter (LAd).
Results: After adjusting for relatedness, pPP, cPP and PWV were negatively associated with E/A, and positively with E/E' and S/D ($P < 0.001$). In multivariate analysis the most closely related parameters were: cPP with E/E'($\beta = 0.04$, $P = 0.001$), cPP with S/D ($\beta = -0.004$, $P = 0.011$), and pPP with E/E' ($\beta = -0.03$, $P = 0.003$). Additionally pPP was associated with LAd ($\beta = 0.058$, $P = 0.011$). In hypertensives pPP and cPP related both to E/E' and S/D ($p < 0.01$).
Conclusions: Our study suggested that increased arterial stiffness as estimated by pulse pressure measurement might be considered as a determinant of left ventricular diastolic dysfunction.

1.6
THE BODE INDEX PROGNOSTIC SCORE IS AN INDEPENDENT DETERMINANT OF ARTERIAL STIFFNESS IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

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Introduction: COPD is associated with increased cardiovascular risk, independent of established risk factors. Arterial stiffness is a surrogate of cardiovascular risk and we sought to determine its relationship with COPD severity and prognosis in the ERICA (Evaluation of role of inflammation in airways disease) multi-site UK study: the largest cohort study focusing on cardiovascular manifestations in COPD.
Methods: Spirometry and haemodynamic measures (aortic pulse wave velocity (aPWV), augmentation index (AIx)) were performed in 729 COPD subjects aged ≥40 years. COPD severity was classified by BODE Index [BMI (low BMI worse prognosis), Obstruction (FEV1), Dyspnoea (mMRC score), Exercise tolerance (6-minute walk distance) high BODE index: worse outcome], a validated score based on clinical variables and an independent predictor of mortality in COPD.
Results: Mean aPWV was 10.3 (SD 2.6) m/s, AIx 27 (10)%. BODE correlated with aPWV ($R = 0.2$, $p = 0.0001$) and this was maintained when adjusted for study site, age, supine HR and MAP, years smoked and cardiovascular comorbidities (MI, stroke, diabetes, peripheral vascular disease), ($\beta = 0.2$, $p = 0.0001$). BODE was also a determinant of AIx when adjusted for site, age, seated HR and MAP, years smoked and cardiovascular comorbidities ($\beta = 0.1$, $p = 0.02$).
Conclusions: BODE is associated with arterial stiffness in COPD, independent of traditional risk factors. Its composite variables are not on the causal pathway for vascular stiffness, so its association likely reflects patient susceptibility to smoke injury in the lungs and vasculature. BODE may also enhance cardiovascular risk stratification in COPD, since its relationship with stiffness was independent of self-reported cardiovascular comorbidities.

2.1
A METHOD FOR THE MEASUREMENT OF PRESSURE SENSITIVITY OF CAROTID-FEMORAL PULSE WAVE VELOCITY IN HUMANS

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Background: Carotid-femoral pulse wave velocity (cfPWV), a marker of cardiovascular disease, is modified by both blood pressure and changes in

arterial wall properties. Current cfPWV measurement does not differentiate between effects of blood pressure and arterial wall properties. Animal studies show that the blood pressure sensitivity of arterial PWV is indicative of blood vessel remodeling. Measurement of this parameter in humans requires a forced change in blood pressure, as can be achieved by Valsalva maneuver. This study investigated a simplified method of measurement of pressure dependency of cfPWV.

Methods: Aortic blood pressure was measured using a validated transfer function from a brachial cuff waveform together with cfPWV in 27 subjects (15 female, 36 $\pm$ 19 years) in both the standing and supine position. The additional change in hydrostatic pressure across the carotid-femoral path length was estimated using body surface distances.

Results: Diastolic blood pressure changed for all subjects (standing 83 $\pm$ 8 mmHg, supine 70 $\pm$ 8 mmHg, $p < 0.001$). Hydrostatic change in pressure across the carotid-femoral path added a further difference of 19 $\pm$ 2 mmHg ($p < 0.001$). Standing cfPWV was 7.3 $\pm$ 2.2 m/s and supine cfPWV 5.2 $\pm$ 1.3 m/s ($p < 0.001$). The resulting pressure sensitivity of cfPWV ranged from 2.7 to 39.4 cm/s/mmHg and had a correlation with age (0.2 cm/s/year, $R^2 = 0.35$, $p < 0.001$).

Conclusions: Measuring cfPWV and blood pressure in the standing and supine position provides a method of calculation of pressure sensitivity of cfPWV that could be easily implemented in any research laboratory or clinic and may provide predictive information beyond either cfPWV or blood pressure alone.

2.2
RE-REFLECTION OF BACKWARD PROPAGATING WAVES LEADS TO AMPLIFICATION OF THE FORWARD PRESSURE WAVE IN WAVE SEPARATION ANALYSIS

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Introduction: In wave separation analysis, the pressure wave is decomposed into a single forward and backward component, which actually compounds all forward and backward propagating waves. We hypothesize that, in particular in presence of early reflections as in aortic coarctation, re-reflection of backward propagating waves at the ventricular-arterial interface amplifies the forward wave component.

Methods: We set up a 3D fluid-structure interaction model of the aorta based on MRI scans of a healthy volunteer. With the healthy model as reference, we introduced a 25 mm narrowing section in the descending thoracic aorta to model an aortic coarctation, with coarctation index (CI) 0.65 and 0.5. Inflow and outflow boundary conditions were kept constant to allow studying the isolated effect of the coarctation. Aortic root pressure and flow waveforms were extracted and subjected to wave intensity and wave separation analysis.

Results: The presence of the coarctation increased systolic pressure by 10 mmHg and 41 mmHg for CI 0.65 and 0.5, respectively. Wave separation analysis indicated that this increase in blood pressure was about equally due to an increase in the amplitude of both the forward and backward pressure wave. Wave intensity analysis - though only after separating into forward and backward wave intensity - revealed that the amplification of the forward pressure wave is caused by re-reflection of backward waves at the level of the aortic valve.

Conclusion: We conclude that wave separation analysis might overestimate the incident pressure wave component because of re-reflection of backward waves at the aortic valve.

2.3
TESTING RIVA-ROCCI'S BASIC ASSUMPTIONS BY SYSTEMATIC REVIEW AND META-ANALYSIS TO DETERMINE THE TRUE DIFFERENCE BETWEEN AORTIC AND BRACHIAL INVASIVE BLOOD PRESSURE

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Background: The Riva-Rocci brachial blood pressure (BP) method purported to measure aortic BP, and this remains the cornerstone thesis of clinical BP measurement. However, few studies have confirmed this thesis with direct BP measurements. This study aimed to determine the true differences in aortic and brachial BP by systematic review and meta-analysis of invasive (intra-arterial) data.

Methods: Five online databases and several offline techniques were used to search for studies that reported simultaneous or sequentially recorded intra-arterial aortic and brachial BP. Differences in systolic BP (SBP) and diastolic BP (DBP) were calculated as brachial minus aortic values.

Results: Data from 12 studies (from 1956 to 2013), totalling 399 participants (aged 57.3 [95% CI: 52.2, 62.4] years, 76.9% male) met inclusion criteria. Brachial SBP was significantly higher than aortic SBP (pooled SBP difference estimate = 7.99 [95% CI: 5.30, 10.7] mmHg, $p<0.001$; $I^2 = 93.3\%$). However, there was only a minimal decrease in DBP between the aorta and brachial artery (pooled DBP difference estimate = -0.67 [95% CI: -1.67, 0.32] mmHg, $p = 0.18$; $I^2 = 79.7\%$). Heterogeneity in SBP differences between studies was modestly explained by age ($R^2 = 5.7\%$), but not by sex, measurement method (simultaneous or sequential) or type of catheter (fluid-filled or micromanometer [$R^2 = 0\%$ all]).

Conclusion: Although only minimal difference in DBP, brachial SBP is significantly higher than aortic SBP, with substantial variability in the magnitude of SBP difference. This questions the Riva-Rocci assumption of brachial BP being representative of aortic BP, and could have accuracy implications for BP assessment using the brachial cuff method.

2.4
AMBULATORY AORTIC STIFFNESS, INDEPENDENTLY OF STATIC, ASSOCIATES WITH NARROWER RETINAL ARTERIOLAR CALIBERS IN HYPERTENSIVES: THE SAFAR STUDY

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Background: Arterial stiffness measured under static conditions reclassifies significantly cardiovascular (CV) risk and associates with organ damage, including narrower retinal arterioles. However, arterial stiffness exhibits diurnal variation, thus single static stiffness recordings do not correspond to the "usual" 24 hr, awake and asleep average arterial stiffness. We aimed to test the hypothesis that ambulatory 24 hr, awake and asleep aortic (a) pulse wave velocity (PWV) associate with retinal vessel calibers, independently of confounders and of static arterial stiffness, in hypertensive individuals free from diabetes and CV disease.

Methods: Digital retinal images were obtained (181 individuals, age: 53.9 $\pm$ 10.7 years, 55.2% men) and retinal vessel calibers were measured with validated software to determine central retinal arteriolar and venular equivalents (CRAE and CRVE, respectively); ambulatory (24 hr, awake, asleep) and static office aPWV were estimated by Mobilo-O-Graph; and static office carotid-femoral (cf) PWV by SphygmoCor.

Results: Regression analysis performed in 320 gradable retinal images showed that, after adjustment for confounders: (i) ambulatory aPWV was significantly associated with narrower retinal arterioles but not with venules; (ii) asleep aPWV had stronger associations with CRAE than awake aPWV; (iii) both ambulatory aPWV and cfPWV were associated mutually independently with narrower retinal arterioles; aPWV introduction in the model of cfPWV, improved model's R^2 ($p = 0.012$). Similar discriminatory ability of 24 hr aPWV and of cfPWV to detect the presence of retinal arteriolar narrowing was found.