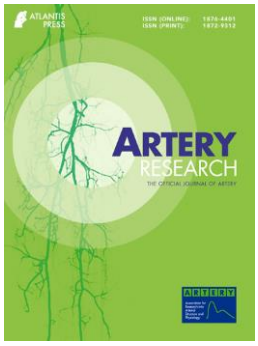

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2.3: DETERMINANTS OF INAPPROPRIATELY HIGH PULSE WAVE VELOCITY IN HYPERTENSIVE PATIENTS: A RETROSPECTIVE CROSS-SECTIONAL COHORT STUDY

Marina Di Pilla, Rosa Maria Bruno, Simona Buralli, Melania Sgro', Piero Amedeo Modesti, Stefano Taddei, Lorenzo Ghiadoni

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indices independently predict cardiovascular events. Aim of this study is to investigate whether central haemodynamics predict major adverse cardiovascular events (MACEs) in ED patients beyond traditional risk factors.
Methods: MACEs in relation to aortic pressures and Augmentation index (AIx) were analyzed with proportional hazards models in 398 patients (mean age, 56 years) without established cardiovascular disease (CVD).
Results: During the mean follow-up period of 6.5 years, a total of 29 (6.5%) MACEs occurred. The adjusted relative risk (RR) of MACEs was 1.062 (95% CI 1.016−1.117) for a 10-mmHg increase of aortic systolic pressure, 1.117 (95% CI 1.038−1.153) for a 10-mmHg increase of aortic pulse pressure (PP), and 1.191 (95% CI 1.056−1.372) for a 10% absolute increase of AIx. The based on categories for 10-year coronary heart disease risk and adapted at 6.5 years overall net reclassification index (NRI) showed marginal and indicative risk reclassification for AIx (15.7%, P=0.12) and aortic PP (7.2%, P=0.20) respectively.
Conclusions: Our results show for the first time that higher central pressures and wave reflections indices are associated with increased risk for a MACE in patients with ED without known cardiovascular disease. Considering the adverse prognostic role of central haemodynamics on outcomes, the present findings may explain part of the increased cardiovascular risk associated with ED.

2.1
THE RELATIVE IMPORTANCE OF CENTRAL AND BRACHIAL BLOOD PRESSURE IN PREDICTING CARDIOVASCULAR EVENTS: AN INDIVIDUAL PARTICIPANT META-ANALYSIS OF PROSPECTIVE OBSERVATIONAL DATA FROM 22,433 SUBJECTS

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Systolic blood pressure (SBP) differs between the brachial artery and aorta. Prospective data suggest that central pressure predicts future cardiovascular events, but it is unclear if it is superior to brachial pressure.
Methods and Results: A systematic review and individual participant data meta-analysis from 15 studies was undertaken. Study-specific associations of central and brachial pressure with cardiovascular outcomes, with and without mutual adjustment, were determined using Cox proportional hazard models, and random effect models to estimate pooled estimates. Of 22,433 participants, 908 had a myocardial infarction (MI) and 641 a stroke. The pooled age, sex, height and heart rate adjusted hazard ratio (HR) [95% CI] per SD increase in brachial SBP was 1.17 [1.03, 1.32] for MI and 1.28 [1.13, 1.46] for stroke and 1.16 [1.02, 1.33] and 1.33 [1.15, 1.53] for central SBP, respectively. Mutual adjustment attenuated the HRs for MI: brachial SBP (1.16 [0.90, 1.48]), central SBP (1.09 [0.87, 1.38]) and stroke: brachial SBP (1.18 [0.97, 1.42]), central SBP (1.19 [0.99, 1.44]). However, associations between central SBP and stroke, after adjustment for brachial SBP, were higher in those aged <61 years than in older individuals (1.83 versus 1.08 p-interaction <0.001).
Conclusion: Brachial and central SBP have similar associations with future CV events. Larger studies are required to test whether central SBP may be a more powerful predictor of stroke risk in younger individuals.

2.2
CENTRAL-TO-PERIPHERAL DIASTOLIC BLOOD PRESSURE ATTENUATION IN HEALTHY ADOLESCENTS AND THE EFFECTS OF HEART RATE. THE MACISTE STUDY

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Background: Heart rate (HR) is directly associated to central-to-peripheral pulse wave amplification. We aimed at evaluating the associations between heart rate and each BP component in a cohort of healthy adolescents.
Objective: 470 healthy adolescents (17±1.4 years, 56% boys, brachial BP 123/67±11/7 mmHg, HR 72±12 bpm) were enrolled in the present study. Brachial BP was measured on 3 occasions by validated devices. Central BP was estimated by radial and brachial applanation tonometries, and calibrated to brachial MAP/DBP (SphygmoCor).
Results: Brachial and central BP were 123/67±11/7 mmHg and 105/69±9/8 mmHg. SBPamp was 1.17±0.04, PPamp was 1.57±0.13, while DBP amplification was 0.97±0.01 (DBP attenuation). HR had a direct correlation with brachial and central DBP (r=0.38 and r=0.46, both p<0.01) and central SBP (r=0.09, p=0.04), but not with peripheral SBP (p=0.59), and a negative one with brachial and central PP (r=-0.24 and r=-0.37, both p<0.01). HR had a positive association with PPamp (r=0.38, p<0.01), and a negative one with SBPamp (r=-0.14, p<0.01) and DBPamp (r=-0.55, p<0.01). The slope of BP change for each 10-bpm HR increase was steeper for central DBP (2.8±0.3 mmHg), than for peripheral DBP (2.2±0.3 mmHg, p for difference between regression coefficients <0.01), and for central and brachial DBP than for central SBP (0.7±0.3 mmHg, both p<0.01).
Conclusions: HR is associated with more pronounced changes in DBP than in SBP, and in central than peripheral DBP. Increasing HR may attenuate DBP from centre to periphery. The assumption that DBP is constant along the arterial tree may not be valid during dynamic conditions.

2.3
DETERMINANTS OF INAPPROPRIATELY HIGH PULSE WAVE VELOCITY IN HYPERTENSIVE PATIENTS: A RETROSPECTIVE CROSS-SECTIONAL COHORT STUDY

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Background: Age and blood pressure (BP) are known to be the main determinants of large artery stiffness. However other factors may lead to an inappropriately high pulse wave velocity (PWV). We investigated the determinants of inappropriately high PWV in hypertensive patients and their possible role in causing organ damage accrual.
Methods: Hypertensive patients were selected among those attending a visit in our Hypertension Outpatient Clinic and undergoing carotid-femoral PWV by applanation tonometry, and cardiac and carotid ultrasound during a 5-year period (2006-2011). Inappropriately high pulse wave velocity (PWV) was calculated as the ratio between the observed value and the values predicted according to the formula derived from international reference values stratified by age and mean BP (oPWV/pPWV)[1,2].
Results: 731 hypertensive patients were selected (age 30-88 years, 42% women, 57% taking BP-lowering drugs). Median oPWV/pPWV was 10±2% (range 6±1-19±6%). In a multiple linear regression model, independent determinants of oPWV/pPWV were: daylight hours (β -1.59, SE 0.33), age (β -0.65, SE 0.08), BMI (β 0.64, SE 0.20), blood glucose (β 0.19, SE 0.05), carotid atherosclerosis (β 2.48, SE 1.20). Though oPWV/pPWV was significantly higher in men and current smokers, the association disappeared in the

multiple regression model. There was no association between oPWV/pPWV and any antihypertensive drugs.
Conclusion: Younger age, obesity, dysglicemia are associated with inappropriately elevated PWV in hypertensive patients. A more advanced atherosclerotic process might also contribute to excess aortic stiffness. Whether an inappropriately high PWV translates into an increased cardiovascular risk should be determined in longitudinal studies.
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2.4
SODIUM CONSUMPTION, CENTRAL AND PERIPHERAL BLOOD PRESSURE, AND FOOD HABITS IN A POPULATION OF HEALTHY ADOLESCENTS. THE MACISTE STUDY

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Objective: The relationship between sodium consumption, central BP and the main dietary sources of daily sodium intake in adolescence has been poorly explored. We have evaluated sodium intake, central and peripheral BP in a population of Italian adolescents.
Methods: 401 healthy adolescents aged 17±1 years (58% boys, average brachial/central BP: 124/67±11/7 mmHg, and 105/69±9/8 mmHg), attending a High School, Terni, Italy, were evaluated. Daily sodium intake was estimated from a single fasting urine by a validated formula. Sources of daily sodium intake were investigated by a self-administered food frequency questionnaire. Central BP was estimated by radial and brachial applanation tonometries, and calibrated to brachial MAP/DBP (SphygmoCor).
Results: 24-h estimated urinary sodium (24-hUNa) was 13530 mmol/d (3.116 g/d). The 89% of the population showed excess sodium intake. 24-hUNa was directly correlated to brachial and central SBP ($r=0.14$ and $r=0.15$, both $p<0.01$), to brachial and central PP ($r=0.19$ and $r=0.24$, both $p<0.01$), and to central-to-peripheral PP amplification ($r=-0.13$, $p<0.01$), but not to central-to-peripheral SBP amplification ($r=-0.01$, $p=0.85$). In a fully-adjusted multivariate regression model, 24-hUNa ($b=0.10$, $p=0.04$) was independently related to central-to-peripheral PP amplification, but not to other measures of both peripheral and central BP. In a factorial analysis, the main daily dietary sources of sodium were bread, biscuits, and salt added to foods.
Conclusions: Sodium intake has a direct relationship with both central and peripheral SBP and PP, and shows an independent association with central-to-peripheral PP amplification. The adverse effects of an excess of sodium intake are more pronounced in central than in peripheral PP.

2.5
THE EFFECT OF RENAL DENERVATION ON CENTRAL BLOOD PRESSURE AND ARTERIAL STIFFNESS IN TREATMENT RESISTANT ESSENTIAL HYPERTENSION: A SUBSTUDY OF A RANDOMIZED SHAM-CONTROLLED DOUBLE-BLINDED TRIAL (THE RESET TRIAL)

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Background: A recent sham-controlled trial (ReSET) showed no sustained effect of renal denervation (RDN) on 24-hour ambulatory blood pressure (24hA-BP) measurements in patients with treatment resistant hypertension.[1] The aim of this substudy was to investigate, whether RDN affects central blood pressure (C-BP) and arterial stiffness independently of brachial artery BP-levels.
Methods: ReSET was a randomized, sham-controlled, double-blinded single-center trial. Main inclusion criteria were: daytime systolic 24hA-BP ≥145mmHg following 1 month of stable medication and 2 weeks of compliance registration. RDN was performed by a single experienced operator using the unipolar Medtronic Flex catheter[1]. C-BP and carotid-femoral pulse wave velocity (PWV) were obtained at baseline and after 6 months with the SphygmoCor®-device.
Results: Fifty-three patients (77% of the ReSET cohort) were included in this substudy. The groups were similar at baseline (SHAM/RDN): $n=27/n=26$; 78/65% males; age 59±9/54±8 years (mean±SD); systolic brachial BP 158±18/154±17 mmHg; systolic 24hA-BP 153±14/151±13 mmHg; systolic C-BP 146±20/143±17 mmHg; diastolic C-BP 92±14/94±10 mmHg; augmentation index (AIx) 26±9/28±13 %; PWV 10.7±2.1/10.1±2.2 m/s. Changes in systolic C-BP (-2±17 (SHAM) vs. -8±16 (RDN) mmHg), diastolic C-BP (-2±9 (SHAM) vs. -5±9 (RDN) mmHg), AIx (0.7±7.0 (SHAM) vs. 1.0±7.4 (RDN) %), and PWV (0.1±1.9 (SHAM) vs. -0.6±1.3 (RDN) m/s) were not significantly different after six months ($P>0.13$ in all tests). Changes in brachial BP and 24hA-BP were also not significantly different.
Conclusions: In a sham-controlled setting, there were no significant effects of RDN on C-BP or arterial stiffness. Thus, the idea of BP-independent effects of RDN on large arteries is not supported.
References
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2.7
NON-INVASIVE EVALUATION OF END SYSTOLIC LEFT VENTRICULAR ELASTANE ACCORDING TO PRESSURE-VOLUME CURVE MODELLING DURING EJECTION IN ARTERIAL HYPERTENSION

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Objective: Non invasive methods have been proposed to assess end systolic left ventricular (LV) elastance (Ees), but clinical application remains complex. The aim of the present study was to 1) estimate Ees according to modeling of LV pressure-volume (P-V) curve during ejection and validate our method with existing published LV P-V loop data 2) test clinical applicability to detect a difference in Ees between normotensive and hypertensive subjects.
Methods: Based on P-V curve and a linear relationship between LV elastance and time during ejection, we fitted the systolic pressure curve (non linear least square method). We then computed slope and intercept of time varying elastance, and calculated Ees as LV elastance at the end of ejection. As a validation, 22 P-V loops obtained from previous invasive studies were digitized and analyzed with our method. To test clinical applicability, P-V curve was obtained from 32 normotensive and 33 hypertensive subjects, using carotid tonometry and real-time 3D echocardiography.
Results: A strong univariate relationship ($r^2=0.92$, $p<0.005$) and good limits of agreement were found between previous invasive measurement of Ees and our new proposed Ejection P-V Curve method. In hypertensives, when compared to normotensives, the increase in arterial elastance (Ea, 1.83±0.80 vs 1.45±0.41 mmHg/mL, $p<0.001$) was compensated by an increase in Ees (2.65±1.07 vs 1.88±0.54 mmHg/mL, $p<0.001$) without change in Ea/Ees (0.76±0.19 vs 0.85±0.23, $p=0.09$).
Conclusions: Ees can be estimated non invasively from modeling of P-V curve during ejection. This approach was found sensitive enough to detect an expected difference in LV contractility in hypertensive patients.