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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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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