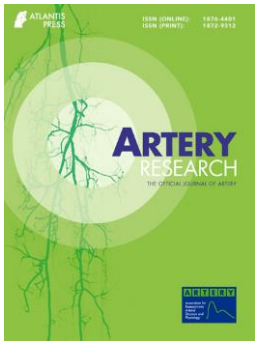

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2.7: NON-INVASIVE EVALUATION OF END SYSTOLIC LEFT VENTRICULAR ELASTANE ACCORDING TO PRESSURE-VOLUME CURVE MODELLING DURING EJECTION IN ARTERIAL HYPERTENSION

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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.