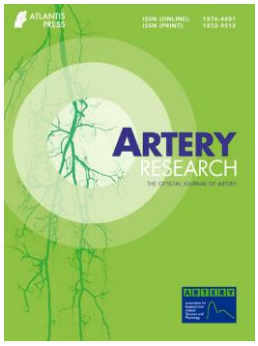

ATLANTIS PRESS

Artery Research

ISSN (Online): 1876-4401 **ISSN (Print): 1872-9312**
Journal Home Page: https://www.atlantis-press.com/journals/artres

2.6: NON-INVASIVE ESTIMATION OF EXERCISE CENTRAL BLOOD PRESSURE BY RADIAL TONOMETRY MAY BE UNDERESTIMATED DUE TO BRACHIAL-TO-RADIAL-SYSTOLIC-BLOOD-PRESSURE-AMPLIFICATION AND IS RELATED TO UPPER LIMB BLOOD FLOW VELOCITY

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To cite this article: D. Picone, R. Climie, M. Keske, J. Sharman (2014) 2.6: NON-INVASIVE ESTIMATION OF EXERCISE CENTRAL BLOOD PRESSURE BY RADIAL TONOMETRY MAY BE UNDERESTIMATED DUE TO BRACHIAL-TO-RADIAL-SYSTOLIC-BLOOD-PRESSURE-AMPLIFICATION AND IS RELATED TO UPPER LIMB BLOOD FLOW VELOCITY, Artery Research 8:4, 124–125, DOI: https://doi.org/10.1016/j.artres.2014.09.062

To link to this article: https://doi.org/10.1016/j.artres.2014.09.062

Published online: 7 December 2019

The short resonance inside the sensor circuit crosses the frequency of the externally applied field and shifts the impedance measured at the excitation coil. As the distance between the two sensors is known the PWV can be determined. The stiffer or the narrower a vessel is the higher is the PWV. A model based approach determines out of the PWV signal the restenosis degree inside the implanted stent. All further measurements are referenced to the first initial value done after the stent implantation. This approach is robust and has mean cross dependences because no absolute pressure measurement is required.

2.3
LOCAL STIFFNESS OF THE CAROTID ARTERY IS ASSOCIATED WITH INCIDENT CARDIOVASCULAR EVENTS AND ALL-CAUSE MORTALITY—A SYSTEMATIC REVIEW AND META-ANALYSIS

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Introduction: It has been suggested that local carotid stiffness is independently associated with cardiovascular (CV) events and mortality. However, consistent evidence for such an association is lacking. We therefore performed a systematic review and meta-analysis of longitudinal studies on the association between local carotid stiffness, incident CV events and all-cause mortality.
Methods: Medline and Embase were searched for articles published up to June 1, 2014. All studies were included which evaluated the association between local carotid stiffness (as determined by ultrasonography) on the one hand and incident CV events and mortality on the other. We used random-effects models to calculate hazard ratios (HRs) and 95% confidence intervals (95%CIs) for pooled data.
Results: We included 10 studies with data for 19,919 participants and 177,136 person-years of follow-up. The pooled HRs (95%CIs) for one SD higher carotid elastic modulus were: for CV events (fatal and nonfatal combined) 1.19 (1.06-1.33; 10 studies, n=19,496); for CV mortality 1.34 (1.15-1.55; 4 studies, n=3,083) and for all-cause mortality 1.26 (1.14-1.40; 5 studies, n=3,501). All results were adjusted for age, sex, blood pressure (SBP and/or MAP), and CV factors. Results were qualitatively similar when HRs were pooled for lower carotid distensibility and compliance instead of higher elastic modulus.
Conclusion: The present meta-analysis shows a strong association between local carotid stiffness and incident CV events, CV mortality and all-cause mortality. In a next step, we will do an individual participant meta-analysis to evaluate whether the association between local carotid stiffness and CV events and mortality is independent of carotid-femoral pulse wave velocity.

2.4
RELATIONSHIP BETWEEN ADULT TRANSFER FUNCTION DERIVED CENTRAL AORTIC SYSTOLIC PRESSURE AND MEASURED SYSTOLIC PRESSURE IN THE HEALTHY CHILDREN POPULATION

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Background: A non-invasive method, that used a general transfer function (TF), has been shown to accurately predict central aortic pressure from peripheral tonometry measurements in the adult population. However applying the same TF to estimate central aortic systolic pressure (aSP) in children has not yet been fully evaluated. This study aimed to assess the usage of adult TF to estimate aSP in children (aSP_{est_ch}) by establishing and testing different linear single/multivariate regression models between the adult TF estimated aSP (aSP_{TF_ad}) and the measured aSP (aSP_{meas_ch}).
Methods: 218 healthy, pre-pubescent children aged 8 years (113 male) had tonometer measured radial and carotid arterial pressure waveform recorded using the SphygmoCor device. Central aSP_{TF_ad} was estimated from the radial pressure using the TF (SphygmoCor, AtCor Medical), and the carotid systolic pressure taken as a surrogate for central pressure (aSP_{meas_ch}). The study group was divided into two groups: one to estimate the models (n=50, 19 male); another to test the models (n=168, 94 male). Models 1 and 2 were two simple linear regression models, whilst models 3 and 4 were two multivariate regression models.
Results: In the tested group, the aSP_{est_ch} from all models showed high correlations and low average differences with aSPmeas_ch (model 1 $R^2=0.88$, difference=1.6+2.6 mmHg; model 2 $R^2=0.88$ difference=1.8+3.4 mmHg; model 3 $R^2=0.89$ difference=-0.7+2.5 mmHg; model 4 $R^2=0.89$ difference=-1.2+2.7 mmHg, all $p<0.0001$).
Conclusion: Central aSP in children can be estimated accurately using the adult TF from the radial pulse by incorporating the now defined linear relationship between aSP_{TF_ad} and aSP_{meas_ch}.

2.5
COMPARISON OF NON-INVASIVE AND INVASIVE MEASUREMENTS OF CENTRAL BLOOD PRESSURE IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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Background: The blood pressure (BP) in the ascending aorta can be estimated non-invasively by pulse wave analysis using commercially available devices such as the SphygmoCor. However, this device has not been validated for use in patients with chronic kidney disease (CKD).
Objectives: Comparison of non-invasively obtained estimates of central BP with invasively measured central BP in CKD patients.
Methods: Patients with stable CKD stage 3-5 corresponding to an estimated glomerular filtration rate (eGFR) under 60 ml/min undergoing elective coronary angiography were included. Central BP was estimated by the SphygmoCor using four different calibrations: Invasive aortic diastolic and mean BP (invasive), systolic and diastolic brachial BP (sys-dia), brachial diastolic and mean arterial BP (form factor 0.33) (ff-0.33), and brachial diastolic and mean arterial BP (form factor 0.4) (ff-0.4). Brachial artery BP was measured simultaneously with a validated oscillometric BP device.
Results: Forty-nine patients, 65% males, age 61±13 (mean±SD) with a median eGFR of 18 ml/min (range 5-59 ml/min) were enrolled. Invasive BP was 150.8±22/ 75.4±11 mmHg while brachial BP was 147.3±19 /86.0±10 mmHg (mean differences: -3.6±9.3/10.6±7.9 mmHg ($P<0.012$).
Mean differences (invasive minus estimated central BP) with the four calibrations used were: -5.9±6.1/1.3±1.0 mmHg, $P<0.001$ (invasive); -16.0±9.3/11.5±8.0 mmHg, $P<0.001$ (sys-dia); -17.5±10.5/11.5±7.9 mmHg, $P<0.001$ (ff-0.33); -7.5±10.2 /11.6±7.9 mmHg, $P<0.001$ (ff-0.4).
Conclusion: In CKD patients, we found a systematic difference between estimated and invasively measured central BP. Surprisingly, brachial systolic BP was very close to invasively measured central systolic BP and was even more accurate than estimates based on calibration with invasively obtained BP.

2.6
NON-INVASIVE ESTIMATION OF EXERCISE CENTRAL BLOOD PRESSURE BY RADIAL TONOMETRY MAY BE UNDERESTIMATED DUE TO BRACHIAL-TO-RADIAL-SYSTOLIC-BLOOD-PRESSURE-AMPLIFICATION AND IS RELATED TO UPPER LIMB BLOOD FLOW VELOCITY

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Objectives: Both central blood pressure (BP) and light-moderate exercise BP are independently associated with cardiovascular risk, and measuring central BP during exercise may have clinical relevance. Brachial-to-radial-systolic-blood-pressure-amplification (B-R-SBP_{AMP}) could influence the accuracy of central SBP estimation by radial tonometry during exercise. This study aimed to determine the influence of light-moderate exercise on B-R-SBP_{AMP} and consequent central SBP estimation. Independent correlates of B-R-SBP_{AMP} were also explored.
Methods: Sixty healthy participants (39±16 years, 50% male) underwent testing during light-moderate intensity (40W, 50RPM) semi-recumbent cycling. SBP was identified by brachial and radial ultrasound (1st Doppler flow inflection = 1st Korotkoff sound during cuff deflation). Haemodynamics were recorded by ultrasound and tonometry. Bra-Rad-SBP_{Amp} was defined as radial minus brachial SBP.
Result: Exercise radial SBP was significantly higher than brachial SBP (144±21 versus 134±17 mmHg; $p<0.001$). Exercise Bra-Rad-SBP_{Amp} was 10±11 mmHg and increased with advancing age ($r=0.360$, $p=0.005$). Exercise central SBP was significantly higher when radial tonometry was calibrated with radial SBP (accounting for Bra-Rad-SBP_{Amp}) versus brachial SBP (117±16 versus 110±13 mmHg, $p<0.001$). Low brachial peak flow velocity relative to radial velocity was negatively associated with exercise B-R-SBP_{Amp} ($r=-0.439$, $p=0.001$), independent of age, sex, heart rate and mean arterial pressure ($\beta=-0.389$, adjusted $R^2=0.273$, $p=0.003$).

Conclusions: Significant Bra-Rad-SBP_{Amp} exists during light-moderate exercise. This will result in underestimation of central SBP unless Bra-Rad-SBP_{Amp} is considered and is influenced by variation in peak blood flow velocity magnitude between the brachial and radial arteries.

3.1
QUANTIFICATION OF THREE-DIMENSIONAL VASCULAR SMOOTH MUSCLE ORIENTATION AND ITS DISPERSION IN MURINE CAROTID ARTERIES

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Introduction: Vascular smooth muscle cells (SMCs) play a pivotal role in regulating vascular tone in arteries, and are therefore an essential part of constitutive models of the arterial wall. Since an SMC exerts its contractile force mainly along its longitudinal axis, SMC orientation is a major determinant of arterial biomechanical behaviour. To our knowledge, all current constitutive models assume SMC orientation to be perfectly circumferential. Moreover, the mechanical effect of any dispersion in SMC orientation is neglected. In this study, we developed a method to quantify in 3D the orientation of SMCs in the intact arterial wall.
Methods: SMC nuclei of six excised and mounted murine carotid arteries were imaged using two-photon laser scanning microscopy. 3D image processing was used to identify individual nuclei and their orientations. Subsequently, SMC locations and orientations were converted to cylindrical and spherical coordinate systems, respectively. We found SMCs to be arranged in distinct inner and outer layers. For each layer, SMC orientations were described by a Bingham distribution, a statistical distribution to describe axis orientations in 3D.
Results and Conclusion: Distributions show a statistically significant ($p<0.05$) helical angular component in both layers (inner: $\Theta_h=2.7\pm2.7^\circ$, outer: $\Theta_h=5.1\pm2.7^\circ$, mean±SD), and considerable dispersion. We conclude that vascular SMC orientation 1) can be quantified in 3D, 2) has a distinct helical component, and 3) shows considerable dispersion. The acquired distribution data are essential to improve constitutive modelling of the arterial wall, by quantitatively describing physiological SMC orientation and dispersion.

3.2
INACTIVE MATRIX GLA PROTEIN IS CAUSALLY RELATED TO HEALTH OUTCOMES: A MENDELIAN RANDOMIZATION STUDY IN A FLEMISH POPULATION

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Background: Matrix Gla-protein is a vitamin K-dependent protein that strongly inhibits arterial calcification. Vitamin K deficiency leads to production of inactive nonphosphorylated and uncarboxylated MGP (dp−ucMGP). The risk associated with dp−ucMGP in the population is unknown.
Methods: In a Flemish population study, we measured circulating dp−ucMGP at baseline (1996−2011), genotyped *MGP* and recorded adverse health outcomes until December 31,2012. We assessed the multivariable-adjusted association of adverse health outcomes with dp−ucMGP and we applied a Mendelian randomization analysis based on *MGP* genotypes.
Results: Among 2318 participants, baseline dp−ucMGP averaged 3.61 μg/liter. Over 14.1 years (median), 197 deaths occurred, 58 from cancer and 70 form cardiovascular disease, and 85 participants experienced coronary events. The risk of death and non-cancer mortality curvilinearly increased ($P\leq0.008$) by 15.0% (95% confidence interval, 6.9−25.3) and by 21.5% (11.1−32.9) for a doubling of the nadir: 1.43 and 0.97 μg/liter, respectively. With higher dp−ucMGP, cardiovascular mortality log-linearly increased (hazard ratio for dp−ucMGP doubling, 1.14[1.01−1.28]; $P=0.027$), but coronary events log-linearly decreased (0.93 [0.88−0.99]; $P=0.021$). dp−ucMGP levels were associated ($P\leq0.001$) with *MGP* variants *rs2098435*, *rs4236* and *rs2430692*. For non-cancer mortality and coronary events ($P\leq0.022$), but not for total and cardiovascular mortality ($P\geq0.13$), the Mendelian randomization analysis suggested causality. In a nested case-control study, 64 patients with coronary events had lower dp−ucMGP than 107 matched controls (3.51 *vs.* 4.54 μg/liter; $P=0.012$).
Conclusions: Higher dp−ucMGP predicts total, non-cancer and cardiovascular mortality, but lower coronary risk. For non-cancer mortality and coronary events, these associations are likely causal.

3.3
ASSOCIATIONS BETWEEN PLASMA INCRETIN HORMONE RELEASE AND AORTIC STIFFNESS AND BLOOD PRESSURE IN INDIVIDUALS WITHOUT KNOWN DIABETES: THE ADDITION-PRO STUDY

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The role of incretin hormone release on vascular structure and function remains unclear. Hence, in 836 Danish individuals without know diabetes and without antihypertensive treatment, we examined the associations of glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP) release during a 3-point 75 g oral glucose tolerance test (0, 30, 120 minutes) with carotid-femoral pulse wave velocity (PWV), and brachial and central blood pressure. For GLP-1 and GIP, total area under the curve (AUC) reflecting circulating incretin levels, and incremental AUC (iAUC) reflecting the incretin response were calculated and transformed by the base 2 logarithm. Associations were analysed by linear regression adjusting for age and sex, and for heart rate and mean blood pressure in the PWV analyses. Mean age was 55.1 (SD: 7.4) years, and 52% were men. A doubling in $iAUC_{GLP\text{-}1}$ was associated with a PWV (95% CI) of -0.04 (-0.12;0.04) m/s, and a central systolic and diastolic blood pressure and pulse pressure (95% CI) of -1.2 (-2.1;-0.3), -0.8 (-1.3;-0.2), and -0.4 (-1.0;0.2) mmHg, respectively. Associations with brachial blood pressure were of similar magnitude as central blood pressure. For $AUC_{GLP\text{-}1}$, only brachial diastolic blood pressure showed a statistically significant association (-1.0 (95% CI: -1.9;-0.2) mmHg for an $AUC_{GLP\text{-}1}$ doubling). Besides a positive association between $iAUC_{GIP}$ and brachial pulse pressure, GIP was not associated with PWV or blood pressure. Although cross-sectional, these results indicate that a high GLP-1 response has a beneficial impact on the vascular function but not on vascular structure, whereas GIP does not affect the vasculature.

3.4
EFFECT OF VITAMIN D SUPPLEMENTATION ON AORTIC STIFFNESS AND CENTRAL HAEMODYNAMICS IN OLDER INDIVIDUALS WITH VITAMIN D DEFICIENCY: PROMISING OBSERVATIONAL DATA IS NOT SUPPORTED WHEN TESTED BY DOUBLE-BLIND, PLACEBO-CONTROLLED, RANDOMISED TRIAL DESIGN

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Background: Observational studies report a relationship between increased aortic stiffness and low vitamin D levels. This suggests that aortic stiffness may be improved with vitamin D supplementation, but there is limited intervention data to support this hypothesis. This study aimed to determine the effect of vitamin D supplementation on aortic stiffness and associated central haemodynamic indices.
Methods: In a double-blind, placebo-controlled trial, 265 individuals (63±7, female 50%) with vitamin D deficiency were randomized to 12-months intervention (vitamin D 50,000 IU/month; n=132) or matching placebo (n=133). Aortic stiffness (carotid-femoral pulse wave velocity; aPWV), central haemodynamic indices (augmentation index, augmented pressure, central pulse pressure) and brachial blood pressure were measured at baseline, six and 12 months.
Results: At baseline there were no significant differences between the groups in aPWV (9.0±0.3 vs 9.1±0.2 m/s; P=0.6), nor any other central hemodynamic indices or brachial blood pressure (all $P > 0.05$). Intervention failed to make any clinical or statistically significant changes to aPWV