2.2: INACCURACIES WITH BRACHIAL AND RADIAL APPLANATION TONOMETRY: SIMILAR IN MAGNITUDE, BUT IN OPPOSITE DIRECTION

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To cite this article: T.L. de Backer, S.J. Vermeersch, B. Drieghe, Y. Taeymans, P. Segers, L. van Bortel (2012) 2.2: INACCURACIES WITH BRACHIAL AND RADIAL APPLANATION TONOMETRY: SIMILAR IN MAGNITUDE, BUT IN OPPOSITE DIRECTION, Artery Research 6:4, 143–143, DOI: https://doi.org/10.1016/j.artres.2012.09.014

To link to this article: https://doi.org/10.1016/j.artres.2012.09.014

Published online: 21 December 2019
chromatography/mass spectrometry and untargeted metabolomics profiling were used to identify metabolites (endogenous and/or drug metabolites) that were significantly different after spironolactone treatment compared with placebo.

**Results:** Spironolactone significantly reduced aortic PWV ($P = 0.047$), but this change was no longer significant after adjustment for the change in daytime systolic BP ($P = 0.132$). Metabolomics profiling identified 43 features (nothing endogenous) that corresponded to downstream spironolactone metabolites, including canrenoate, but none of these correlated with the change in aortic PWV ($P < 0.05$ for all). However, canrenoate was significantly related to the reduction in daytime systolic BP ($r = -0.355$, $P = 0.017$) and 24 hour PP ($r = -0.332$, $P = 0.026$). These associations remained significant on multiple regression analysis after adjustment for sex and body mass index.

**Conclusions:** The downstream spironolactone metabolite canrenoate appears to be an active metabolite with BP-dependent effects on the attenuation of aortic stiffness in people with HRE. This finding, together with the lack of change in endogenous metabolites relating to fibrosis, suggests that the antifibrotic effects of spironolactone could be BP-dependent.

**Oral session 2**

Free Communication Oral Presentations

In association with the North American Artery Society

2.1 WALL TENSILE STRESS BUT NOT ATHEROSCLEROTIC RISK FACTORS PREDICTS 3-YEAR CAROTID INTIMA-MEDIA THICKNESS PROGRESSION RATE IN HEALTHY SUBJECTS

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In carotid IMT is considered marker of early atherosclerosis. However, experimental and clinical studies suggest that changes in arterial wall thickness may also represent an adaptive response to chronic changes in vessel diameter and/or intraluminal BP aimed to normalize wall tensile stress (WTS).

**Aim of the study:** to assess whether in healthy young-to-middle-aged population a short-term common carotid (CCA) IMT progression rate ($\Delta$IMT) is associated with WTS at baseline.

**Methods:** study population consisted of 749 healthy subjects at low-average Framingham risk (327 men, age=45±8 years) undergoing carotid US, fasting blood test (LDL-and HDL-cholesterol, triglycerides), oral glucose tolerance test and questionnaire regarding smoking and family history of cardiovascular diseases at baseline and after 3-year follow-up. WTS was calculated as (diastolic BP/CCA radius)/IMT.

**Results:** during a 3-year period, BMI, waist girth, systolic and diastolic BP and plasma glucose increased ($p < 0.001$), HR decreased ($p < 0.001$), and plasma lipids and insulin did not change significantly. CCA diameter ($+0.09±0.30$ mm, $p < 0.0001$) and IMT ($+18±58$ mm, $p < 0.0001$) increased, whereas WTS ($-0.27±6.70$ kPa, $p = 0.36$) remained unchanged. $\Delta$IMT significantly increased by WTS quartiles at baseline ($p < 0.0001$). In multiple regression model, adjusted for gender, smoking, family history and atherosclerotic risk factors (or their 3-year changes), the only independent determinants of $\Delta$IMT were male gender, age and WTS at baseline ($\beta$:SE: $0.12±0.04$, $0.17±0.04$ and $0.32±0.04$, cumulative $R^2 = 0.20$, $p < 0.0001$).

**Conclusions:** In healthy young-to-middle-aged population, the strongest predictor of short-term IMT progression rate was baseline WTS, followed by age and gender. Thus, IMT increase cannot be always considered marker of atherosclerosis.

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In the studied cohort, applanation tonometry tended to overestimate BA FF (i.e. measuring a flattened curve) by 2.1%, yet it also tended to underestimate RA FF (i.e. measuring a more peaked profile) by 2.5%. Combined, these inaccuracies may amplify the RA/BA ratio.

**Conclusions:** Both RA and BA hand-held applanation tonometers introduce inaccuracies of similar magnitude but in opposite direction. These inaccuracies may lead to errors in the assessment of central pressure and brachial-to-radial amplification. Whether these inaccuracies may be improved by investigator-independent automated applanation tonometry should be investigated.

2.3 CLINICALLY APPLICABLE APPROACH TO DISCRIMINATE BLOOD PRESSURE DEPENDENT AND INDEPENDENT CHANGES IN ARTERIAL STIFFNESS


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**Background:** Monitoring of changes in arterial stiffness in hypertensive patients requires consideration of blood pressure (BP) dependent effects. Based on non-invasive measurements of carotid artery BP and cross-sectional area waveforms, we assessed the BP-dependent change in stiffness in 24 patients referred to our outpatient hypertension clinic, irrespective of antihypertensive treatment.

**Methods and Results:** In 13 women and 11 men (mean±SD; SBP/DBP: 157±26/92±10 mmHg, age: 53±15 yrs, BMI: 29±6 kg/m²) tonometric foot-to-foot carotid-femoral pulse wave velocity (cPWVCF) and common carotid artery cross-sectional area (wall-track) and local pressure waveforms were measured at baseline and at 3-month follow-up. Carotid stiffness was expressed as pulse wave velocity based on the distensibility coefficient (cPWVDC). Per individual, a mono-exponential curve was fitted through baseline carotid diastolic (d), dicrotic notch and systolic (s) pressure-area points to enable prediction of the BP-dependent change in stiffness expressed as cPWVDC*. In patients in whom diastolic blood pressure (DBP) was significantly reduced, cPWVDC* and (measured) cPWVDC were reduced by 1 m/s, similar to the predicted BP-dependent change in stiffness (Table). Patients showing no change in BP showed no change in stiffness. Correlations were cPWVDC* versus cPWVDC: $r = 0.45$ ($p = 0.037$), while cPWVDC and cPWVDC: $r = 0.26$ ($p = 0.22$).

**Conclusion:** Quantitative assessment of carotid stiffness as function of pressure enables prediction of BP-dependent changes in stiffness. Our clinically applicable approach could help to discriminate pressure dependent and independent effects of antihypertensive treatment on arterial stiffness.