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3.2: VARIABILITY IN MEAN ARTERIAL PRESSURE AND DIASTOLIC BLOOD PRESSURE FROM CENTRAL TO PERIPHERAL LARGE ARTERIES: RELEVANCE TO ARTERIAL PHYSIOLOGY AND ESTIMATED CENTRAL BLOOD PRESSURE

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3.1 REDUCING ARTERIAL STIFFNESS INDEPENDENTLY OF BP: PROOF OF CONCEPT? CAVI, PWV AND CARDIAC DATA IN THE 6-MONTH VASERA TRIAL

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Purpose/ background/ objectives: People with or at risk of Type II diabetes (T2DM) are at increased risk of vascular disease and arterial stiffness (AS). We hypothesized that spironolactone and dietary nitrate (beetroot juice) separately and together would reduce AS, measured as cardiac-ankle vascular index (CAVI Fukuda Denshi, Japan mainly BP-independent) or aortic pulse wave velocity (PWV).

Methods: 126 (60% T2DM) were randomized, double-blind to spironolactone (≤ 50 mg) or doxazosin (control ≤ 16 mg) and active/ placebo juice ($\leq 9/0$ mmol) daily. AS and echocardiographic measures (on a subgroup) were performed. Intention-to-treat analysis adjusted for between-group blood pressure (BP) change over time was performed using SAS.

Results: Change in (Δ)BP was not different between spironolactone and doxazosin (mean -6.7 mmHg), nor between the juices. Δ CAVI was marginally reduced on doxazosin compared to spironolactone ($-0.11[-0.30,0.08]$ vs. $0.14[-0.06,0.34]$ units, $p=0.080$) but more for aortic PWV ($-0.44[-0.69,-0.20]$ vs. $-0.07[-0.32,0.18]$ ms⁻², $p=0.04$). Dietary nitrate had no impact, but did rise in plasma.

Spironolactone improved Δ relative wall thickness vs. doxazosin ($0.01[-0.02,-0.0]$, $p<0.01$). Dietary nitrate decreased left ventricular (LV) end diastolic and systolic volume ($-6.3[-11.1,-1.6]$ mL and $-3.2[-5.9,-0.5]$ mL, $p<0.05$) and increased end diastolic mass/volume (EDMV) ratio ($0.04[0,0.7]$ g/mL, $p<0.05$) vs. placebo. There were no drug–juice interactions.

Conclusions: Contrary to our hypothesis, spironolactone did not reduce AS, rather central PWV declined on doxazosin. Spironolactone enhanced LV remodelling, while dietary nitrate improved LV volumes and the EDMV ratio, perhaps indicating improved LV strain.

3.2 VARIABILITY IN MEAN ARTERIAL PRESSURE AND DIASTOLIC BLOOD PRESSURE FROM CENTRAL TO PERIPHERAL LARGE ARTERIES: RELEVANCE TO ARTERIAL PHYSIOLOGY AND ESTIMATED CENTRAL BLOOD PRESSURE

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Background: Mean arterial pressure (MAP) and diastolic blood pressure (DBP) are thought to consistently decline approximately 1-3 mmHg from the aorta to peripheral large arteries, thus providing a small pressure gradient to aid blood flow. The magnitude of this gradient is important for correct waveform calibration and central BP estimation. However, there is little invasive data determining the variability in MAP and DBP from central to peripheral arteries, which was the goal of this study.

Methods: 52 patients (mean age 62 ± 11 years) undergoing cardiac angiography had intra-arterial BP measured via catheter in the ascending aorta, brachial and radial arteries by sequential pull-back. MAP was calculated by integration of ensemble averaged waveforms, and DBP from the foot of the waveforms.

Results: On average, MAP and DBP decreased from the aorta-to-brachial (MAP -1.5 ± 3.9 mmHg DBP -2.7 ± 4.1 mmHg) and brachial-to-radial (MAP -2.0 ± 4.4 mmHg DBP -1.8 ± 3.3 mmHg) arteries. However, changes in aortic-to-radial MAP (range -14.9 to 6.8 mmHg) and DBP (range -13.1 to 2.1 mmHg) were highly variable, including increases in MAP among 23% of patients. Importantly, the relationship between MAP and DBP changes were synergistic, with DBP decreasing if MAP increased and vice versa. The

magnitude of aorta-to-radial MAP and DBP differences were significantly related to height and age.

Conclusions: Although MAP and DBP are reduced on average from central to peripheral large arteries, the magnitude of change is variable and related to patient characteristics. These new observations are highly relevant to understanding arterial hemodynamic (patho)physiology and accurate non-invasive estimates of central BP.

3.3 DISCOVERY OF A NEW BLOOD PRESSURE PHENOTYPE FROM INVASIVE CENTRAL-TO-PERIPHERAL RECORDINGS: IMPLICATIONS FOR BRACHIAL CUFF ACCURACY AND CARDIOVASCULAR RISK ASSESSMENT

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Background: Accuracy of brachial cuff blood pressure (BP) may be influenced by individual variability in central-to-peripheral systolic BP (SBP)-amplification, but this has never been determined. We aimed to achieve this by characterising SBP-amplification phenotypes and examining associations with cuff BP accuracy.

Methods: Intra-arterial BP was measured at the ascending aorta, brachial and radial arteries in 77 patients (aged 61.5 ± 10.3 years 68% male) following coronary angiography. Cuff BP was measured bilaterally by oscillometric devices before catheterisation, and then simultaneously with intra-arterial brachial BP. SBP-amplification was defined by ≥ 5 mmHg SBP increase between the aorta-to-brachial or brachial-to-radial arteries.

Results: Average aortic-to-brachial and brachial-to-radial SBP-amplification were 8.5 ± 9.5 mmHg and 6.4 ± 9.4 mmHg respectively. However, four distinct SBP-amplification phenotypes were observed: 1) both aortic-to-brachial and brachial-to-radial SBP-amplification ($n=24$) 2) only aortic-to-brachial SBP-amplification ($n=24$) 3) only brachial-to-radial SBP-amplification ($n=16$) 4) no aortic-to-brachial or brachial-to-radial SBP-amplification ($n=13$). Compared with the first three phenotypes, patients with no SBP-amplification had elevated aortic SBP (143.1 ± 23.0 mmHg versus 122.4 ± 18.3 126.0 ± 19.5 and 134.8 ± 12 mmHg respectively $p=0.0066$) that was significantly underestimated by brachial cuff BP (-11.7 ± 8.7 mmHg, $p=0.004$), despite no differences in clinical characteristics or cuff BP between phenotypes ($p>0.1$ all).

Conclusions: These are the first data to describe distinctive central-to-peripheral SBP-amplification phenotypes, and includes discovery of a phenotype in which cardiovascular risk is likely to be elevated because of significantly increased aortic SBP that is not detected by conventional cuff BP methods.

3.4 WAVE INTENSITY ANALYSIS PROVIDES NOVEL INSIGHTS INTO PULMONARY HYPERTENSION

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Background: The objective of the study was to apply wave intensity analysis (WIA) in the pulmonary artery to characterise the magnitude, origin, type and timing of arterial waves in individuals with and without pulmonary hypertension (PH).

Methods: Right heart catheterisation was performed using a pressure and Doppler flow sensor tipped catheter to obtain simultaneous pressure and