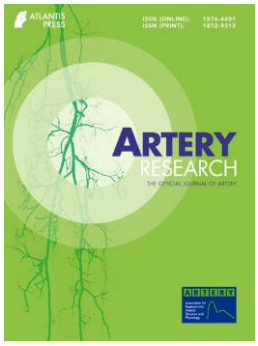

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P1.7: PARAMETERS OF ARTERIAL STIFFNESS DIFFER BETWEEN ATRIAL, VENTRICULAR, AND ATRIAL-VENTRICULAR CARDIAC PACING MODES

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Conclusions: During exercise increased sympathetic tone could be the main reason for the decreased c-rtT, but other mechanisms should contribute to the regulation of the finger tip skin microcirculation, where termoregulation plays a major role.

P1.7
PARAMETERS OF ARTERIAL STIFFNESS DIFFER BETWEEN ATRIAL, VENTRICULAR, AND ATRIAL-VENTRICULAR CARDIAC PACING MODES

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An interrelation between heart rate and arterial stiffness is established. However, the relationship between cardiac pacing mode and the stiffness of arterial vessels is not. This study investigated arterial stiffness parameters such as carotid-femoral pulse wave velocity (cfPWV) and aortic augmentation index (AIx) in pacemaker subjects (n=46) paced via atrial (Ap), ventricular (Vp), or atrial ventricular (ApVp) modes at 60, 70, 80, 90 and 100 bpm in the supine position. At each heart rate, brachial blood pressure was measured, the central aortic pressure waveform derived using a validated transfer function applied to brachial cuff waveforms (SphygmoCor XCEL), and cfPWV measured using simultaneous acquisition of the carotid (tonometer) and femoral (thigh cuff) pulse. Aortic and brachial systolic, diastolic, and mean pressure did not differ between pacing modes. However, AIx was lower with ApVp (24±9 %) and Vp (19±11 %) pacing than Ap pacing (34±10 %, $p<0.001$), with Vp being lower than ApVp ($p<0.01$). Ejection duration followed the exact pattern of AIx. Aortic pulse pressure was also lower with ApVp (37±9 mmHg) and Vp (36±11 mmHg) pacing than Ap pacing (42±12 mmHg, $p<0.01$). However, cfPWV was greater with ApVp pacing (10.6±1.9 m/s, $p<0.05$) and Vp pacing (11.0±2.1 m/s, $p<0.01$) than Ap pacing (9.8±1.7 m/s). This study showed differences in vascular stiffness with cardiac pacing modes. Further research is required to investigate the opposing changes in AIx and cfPWV and to determine if pacing mode drives differences in arterial stiffness or differences are characteristic of the subjects assigned to different pacing modes.

P1.8
ANTIHYPERTENSIVE MEDICINES OF UP TO 4-DRUG COMBINATIONS IN A LARGE, COMMUNITY-BASED STUDY: DIFFERENTIAL RELATIONSHIPS WITH BRACHIAL BLOOD PRESSURE AND AORTIC WAVEFORM PARAMETERS

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Background: Comparing relationships that antihypertensives have with brachial blood pressure (BP) and aortic waveform parameters helps clinicians to predict the effect on the latter in brachial BP-based antihypertensive therapy. We aimed to make such comparisons with new waveform measures and a wider range of antihypertensive regimens than examined in previous research.
Methods: Cross-sectional analysis of 2915 adults (61% male; aged 50-84 years): 1619 on antihypertensive treatment and 1296 untreated hypertensives. Sixteen medicine regimens of up to 4 combinations of drugs from six antihypertensive classes were analysed. Aortic systolic BP (SBP), augmentation index (AIx), excess pressure integral, reflection index (RI), backward pressure amplitude (Pb) and pulse wave velocity (PWV) were calculated from aortic pressure waveforms derived from suprasystolic brachial measurement.
Results: For all regimens, brachial SBP was lower with antihypertensive use. However, while brachial SBP did not differ across the 16 regimens ($P=0.17$), RI ($P<0.0001$), Pb ($P=0.0001$) and AIx ($P<0.0001$) did. This was predominantly due to beta-blocker associations: forest plots of single-drug class comparisons across regimens with the same number of drugs (for between 1- and 3-drug regimens) revealed that AIx, Pb and RI were higher with the use of a beta-blocker compared with vasodilators and diuretics, despite no differences in brachial SBP. Compared to those untreated, beta-blocker use was associated with greater percentage differences in brachial BP than aortic waveform parameters.
Conclusions: Beta-blocker use has weaker associations with wave reflection measures than brachial SBP, suggesting that effects on these may be overestimated with brachial BP-based antihypertensive therapy.

P1.9
EVALUATION OF AFFECTIVE TEMPERAMENTS AND ARTERIAL STIFFNESS IN TREATED HYPERTENSIVE PATIENTS

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Background: Hypertension has widely studied psychosomatic connections, there are, however, only limited data about the influence of affective temperaments. The aim of our study was to evaluate arterial stiffness in hypertensive patients with dominant (DOM) or subdominant affective temperaments (SDOM).
Methods: 152 hypertensive patients, free of treated psychiatric diseases, completed the TEMPS-A, Beck Depression Inventory and Hamilton Anxiety Scale in two GP practices. Of those 11 DOM and 11 SDOM patients and 22 hypertensive controls (matched for age, sex and the presence of diabetes) were included for arterial stiffness measurements.
Results: Pulse wave velocity and augmentation index did not differ significantly among the groups studied. Compared to controls, in the combined DOM+SDOM group brachial systolic (130.5 (121.9-138.5) vs. 122.8 (114.4-129.6) mmHg), diastolic (72 ± 1.5 vs. 66.9 ± 1.8 mmHg) and mean blood pressure (91.9 ± 1.4 vs. 86 ± 1.9 mmHg) as well as central diastolic (69 ± 1.45 vs. 64.98 ± 1.83 mmHg) and mean blood pressure values (89.4 ± 1.58 vs. 84.65 ± 1.99 mmHg) were significantly lower. Beck and Hamilton scores were significantly higher in the DOM+SDOM group.
Limitations: The cross-sectional design of the study precludes the evaluation of causality.
Conclusion: The similar arterial stiffness parameters besides lower blood pressure values and the increased depression and anxiety scores in the DOM+SDOM group might refer the presence of increased cardiovascular risk. Affective temperaments may play a substantial role in such health-related behaviours, therefore they may be an important factor in developing strategies related to cardiovascular health management.

P1.10
LONGITUDINAL CHANGES IN GEOMETRIC AND FUNCTIONAL ARTERIAL PROPERTIES IN VASCULAR EHLERS-DANLOS SYNDROME WITH CELIPROLOL

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Objective: Vascular Ehlers-Danlos (vEDS) syndrome is a rare disease (1/100,000), due to mutations in the collagen type III (COL3A1). vEDS is characterized by early spontaneous arterial rupture or dissection. Celiprolol, a beta1 antagonist beta2 partial agonist conferred protection against CV events with paradoxical stiffening effects (Ong et al, Lancet 2010). Our aim was evaluate celiprolol effect on arterial properties during a long term longitudinal follow-up of a large population of patients.
Methods: 63 patients (age 35±10, 57% females) having at least 2 visit were followed 5 years during 6±3 visits. Carotid internal diastolic diameter (Di), intima-media thickness (IMT), arterial wall cross-sectional (WCSA), circumferential wall stress, distensibility and Young's elastic modulus (Einc) were measured. The evolution over time in response to celiprolol was studied using mixed models.
Results: 46 patients were exposed to celiprolol. SBP increased with time under celiprolol (0.79 mmHg/y, $p<0.001$), so did central SBP (0.89 mmHg/y; $p=0.002$) and central PP (1.24 mmHg/y, $p<0.001$), without changed heart rate. Di and IMT increased (+36μm/y, $p<0.001$ and +4.4 μm/y, $p<0.001$, respectively). Einc increased (29.92 kPa/y, $p<0.001$) and distensibility decreased (-0.003 kPa-1/y, $p<0.001$). In unexposed patients (n=17), brachial BP did not change significantly, whereas changes in arterial wall properties were similar to those exposed to celiprolol.
Conclusion: The effect of time on large arteries properties seems similar whether patients are treated with celiprolol or not. Changes might thus be due to aging process rather that to pharmacologically induced changes.