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


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Conclusions: During exercise increased sympathetic tone could be the main reason for the decreased c-tT, 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 (cPFWV) 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 cPFWV 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 (24:19.5%) than Ap (24:19.5%) pacing (34:10.0%, 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 (36:81:11 MMHg) pacing than Ap pacing (42:12:12 MMHg, p<0.01). However, cPFWV 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 (9.6±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 cPFWV 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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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. Celprolol, a beta1 antagonist beta2 partial agonist conferred protection against CV events with paradoxical stiffening effects (Ong et al, Lancet 2010). Our aim was evaluate celprolol 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 (WCAS), circumferential wall stress, distensibility and Young’s elastic modulus (Einc) were measured. The evolution over time in response to celprolol was studied using mixed models.

Results: 46 patients were exposed to celprolol. SBP increased with time under celprolol (0.79 MMHg/y, p<0.001), so did central SBP (0.89 MMHg/y, p=0.002) and central AP (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 celprolol.

Conclusion: The effect of time on large arteries properties seems similar whether patients are treated with celprolol or not. Changes might thus be due to aging process rather that to pharmacologically induced changes.