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Although in-vitro biomechanical behaviour of arteries is considered to differ substantially, we developed a set-up to characterize mouse carotid artery biaxial mechanics using high-frequency ultrasound to track diameter and two-photon laser scanning microscopy (TPLSM) to capture the microstructure. We aimed to (1) quantify reproducibility and (2) compare quasi-static and dynamic elastic behaviour.

**Design and method:** After euthanasia, eight carotid arteries from four male surplus mice were mounted between glass micropipettes. Four carotids were stretched to in-vivo length and exposed to quasi-static pressure inflation from 0–200 mmHg. Second, axial stretch (lz) was varied for constant pressures of 60/100/140 mmHg to determine an axial stiffness coefficient. Third, vessels were exposed to pulsatile pressures (systolic pressures of 80/120/160 mmHg, at 5 Hz) and at frequencies (f) of 2.5/5/10 Hz at 120 mmHg. Single-point pulse wave velocity (PWV; Bramwell-Hill) was determined and compared with corresponding PWVs calculated from the quasi-static pressure-diameter curve. The axial stiffness coefficient was obtained as the local slope of the F-lz curve. Fourth, information on adventitial collagen-structure deformation was obtained using second-harmonic generation TPLSM. The first three protocol steps were performed in duplicate to determine coefficients of variation (CVs).

**Results:** CVs for PWV were ~9% for low/medium and 27% for high pressure. For f2.5 and f10 Hz these were 16% and 26%. Dynamic PWVs were higher than quasi-static PWVs for all test conditions (p < 0.012, mean ± SD, at f5 Hz: PWVP80 2.4 ± 0.2 vs. 2.1 ± 0.1 m/s, PWVP120 6.3 ± 0.6 vs. 5.0 ± 0.5 m/s, PWVP160 13.3 ± 1.9 vs. 10.3 ± 2.2 m/s, and at P120 mmHg: PWVP2.5 3.9 ± 0.7 vs. 3.5 ± 0.3 m/s, PWVP10 3.1 ± 0.9 vs 2.5 ± 0.2 m/s.). Axial stiffness coefficients increased with pressure 60–140 mmHg (1.3 ± 0.4, 2.4 ± 1.0, 4.9 ± 2.8 cm/s). Measurements from fresh and non-fresh vessels were not significantly different (p > 0.009). From 60–100 mmHg undulation of adventitial collagen strands disappeared, whereas from 100–140 mmHg orientation changed (Figure).

**Conclusions:** Our innovative set-up shows well-acceptable reproducibility and demonstrates the importance of quasi-static and dynamic conditions when studying arterial mechanics.
brachial blood pressure (BP). Whether it can be changed independently of BP in the short term is uncertain but recent observations suggest that AS may be specifically modulated by the autonomic nervous system (ANS). Here we compared effects of device guided breathing (DGB, known to reduce sympathetic activity) with nifedipine on cfPWV.

**Design and method:** Patients with essential HT on pharmacological treatment (mean ± SD age 48 ± 14 years, n = 19) had cfPWV (SphygmoCor) and brachial BP (Omron) measurements performed before and after DGB and oral administration of nifedipine 10 mg. The two interventions were performed consecutively in single visit with the patient lying in supine position. DGB is a biofeedback technique which slows the breathing rate to <10 breaths/minute and decreases BP via its action on the ANS. Nifedipine is a short acting peripheral arterial vasodilator that tends to increase sympathetic activity.

**Results:** Baseline systolic BP (SBP) 150.4 ± 12.6 and diastolic BP (DBP) 89.8 ± 8.7 mmHg, heart rate (HR) 65.9 ± 11.2 bpm; cfPWV 10.3 ± 2.4 m/s. Compared to nifedipine, DGB caused less reduction of both brachial SBP and DBP: decrease of 12.3 (95%CI 8.4, 16.1) vs. 16.1 (12.7, 19.5) mmHg in SBP for DGB and nifedipine and 5.9 (3.1, 8.7) vs. 10.4 (7.1, 13.5) mmHg in DBP (P < 0.05 for difference between DGB and nifedipine). DGB caused a greater reduction in cfPWV: decrease of 1.2 (0.8, 1.8) and 0.7 (0.1, 1.3) m/s for DGB and nifedipine respectively (P = 0.02 for difference between DGB and nifedipine). HR decreased during DGB by 3.6(1.2, 5.9) bpm and increased after administration of nifedipine: 7.6(3.9, 11.3) bpm.

**Conclusions:** DGB had a greater impact on AS despite a smaller effect on BP. These results demonstrate that cfPWV can be changed independently from BP in the short-term and support a specific role of the ANS in regulating cfPWV.