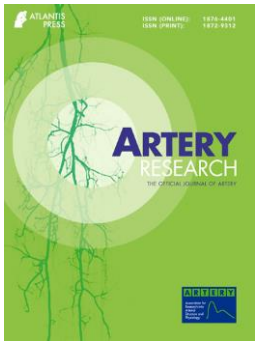

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8.8: SYMPATHETIC VASOCONSTRICTOR RESPONSE TO LOWER BODY NEGATIVE PRESSURE IN YOUNG OBESE ADULTS: THE PRELIMINARY FINDING

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Results: No differences in age (39+/-9 v 39+/-9 years P 0.87) or BMI (24.85+/-3.29 v 25.75+/-3.68 kg.m^2 P 0.24) were observed. No differences in IVSd (0.86+/-0.15 v 0.85+/-0.18 cm P 0.64), LVIDd (4.98+/-0.55 v 4.96+/-0.42 cm P 0.95), LVPWd (0.81+/-0.17 v 0.90+/-0.21 cm P 0.05) or LV Mass (168.86+/-56.85 v 182.61+/-61.70 g P 0.43) were observed. However, MV E/A (1.85+/-0.51 v 1.48+/-0.51 P 0.0004), but not LV IVRT (0.09+/-0.02 v 0.09+/-0.01 P 0.25), was different.
Conclusions: Changes in cardiac function are observed before alterations in cardiac structure in healthy subjects with premature vascular stiffening.
References
1. Mansour AS, YannoutsosA, MajahalmeN, AgnolettiD, Safar ME, OuerdaneS, BlacherJ. Aortic stiffness and cardiovascular risk in type 2 diabetes. JHypertens. 2013; 31(8):1584-92.

8.8
SYMPATHETIC VASOCONSTRICTOR RESPONSE TO LOWER BODY NEGATIVE PRESSURE IN YOUNG OBESE ADULTS: THE PRELIMINARY FINDING

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Background: Elevations in muscle sympathetic nerve activity (MSNA) and sympathetic vasoconstrictor responsiveness to sympathoexcitation are associated with increased cardiovascular risks, which affect hemodynamics, and have been reported in obese adults with metabolic syndrome (1-3). It remains unclear whether this observation may also be present in young metabolically healthy obese adults.
Purpose: To compare sympathetic vasoconstrictor and hemodynamic responsiveness to lower body negative pressure (LBNP, -20 mmHg) in young normal-weight (NW) vs. obese (OB) adults.
Method: Eleven NW (female=6; 25±2 yrs; 22.4±0.6 kg/m2) and 13 OB adults (female=6; 27±1 yrs; 32.7±0.6 kg/m2) underwent 2-min of LBNP in the supine position. Ultrasonography [brachial diameter, forearm blood flow (FBF), forearm vascular conductance (FVC)], MSNA [burst frequency, total MSNA, sympathetic vascular transduction], and beat-to-beat hemodynamics [heart rate (HR), mean arterial pressure (MAP), total peripheral resistance (TPR), cardiac output (CO), stroke volume (SV), systemic compliance (SC)] were reported. FBF and FVC were normalized to lean forearm mass, and TPR, CO, SV, and SC to body surface area.
Results: Baseline MAP was lower in OB ($P<0.05$). In response to LBNP, normalized FBF, FVC, SV, CO, and SC decreased whereas TPR increased similarly in both groups ($P<0.05$). Brachial diameter and HR did not change in both groups. MAP decreased similarly by ~2-4 mmHg, but the values were lower in the OB group ($P<0.05$). Burst frequency, total MSNA, and sympathetic vascular transduction increased similarly in both groups ($P<0.05$).
Conclusion: Young metabolically healthy obese adults did not exhibit altered sympathetic vasoconstrictor responsiveness under resting condition.
References
1. Straznicky NE, Grima MT, Sari CI, Eikelis N, Lambert EA, Nestel PJ, et al. Neuroadrenergic dysfunction along the diabetes continuum: a comparative study in obese metabolic syndrome subjects. Diabetes. 2012; 61(10): 2506-16.
2. Huggett RJ, Burns J, Mackintosh AF, Mary DA. Sympathetic neural activation in nondiabetic metabolic syndrome and its further augmentation by hypertension. Hypertension. 2004; 44(6):847-52.
3. Kuniyoshi FH, Trombetta IC, Batalha LT, Rondon MU, Laterza MC, Gowdak MM, et al. Abnormal neurovascular control during sympathoexcitation in obesity. Obesity research. 2003; 11(11):1411-9.

8.9
REDUCTION IN MYOCARDIAL WALL STRESS AND DELAYED MYOCARDIAL RELAXATION DURING EXERCISE

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Introduction: Myocardial wall stress (MWS) is thought to be the mechanical stimulus to ventricular hypertrophy (1,2). The objective of this study was to examine the effect of exercise on time-varying MWS (3).
Methods: Twelve subjects, aged 42.0 ± 16.8 (mean ± SD) years, systolic blood pressure (BP) (128 ± 11mmHg), were studied before and during peak bicycle exercise (85% of target heart rate). We estimated MWS from 3D transthoracic echocardiographic imaging of the left ventricle (LV) and LV pressure was derived from carotid tonometry during systole. Carotid pressure calibrated by mean and diastolic BP was used to calculate time-varying LV wall stress from endocardial and epicardial volumes obtained from Philips 3DQ analysis package. Time of onset relaxation (TOR) was defined as percentage of time to peak wall stress to ejection duration.
Results: There was a significant reduction in peak and mean MWS during exercise (rest 435.3±25.3 VS exercise 385.9±22.5, $p=0.001$ and 387.3±24.2 VS 368.7±19.6 kdynes/cm2, $p=0.016$), despite significant increase in systolic BP (128±3 VS 210±6 mmHg, $p<0.001$). LV end-diastolic volume (EDV) (119.3±9.4 VS 95.2±7.8ml, $p<0.001$) and volume at time of peak MWS (86.5±7.0 VS 68.3±6.3ml, $p=0.001$) were reduced significantly during exercise, but TOR was delayed (24.5±1.2 VS 31.0±1.6%, $p=0.003$).
Conclusion: Peak and mean MWS were reduced during peak exercise as a result of reduction in pre-load, despite of significant increase in systolic BP. But there was evidence of delayed myocardial relaxation during exercise.
References
1. Grossman W, Jones D, McLaurin LP. Wall stress and patterns of hypertrophy in the human left ventricle. The Journal of Clinical Investigation 1975; 56:56-64.
2. Ganau A, Devereux RB, Pickering TG et al. Relation of left ventricular hemodynamic load and contractile performance to left ventricular mass in hypertension. Circulation 1990; 81:25-36.
3. Chirinos JA, Segers P, Gupta AK et al. Time-varying myocardial stress and systolic pressure-stress relationship: role in myocardial-arterial coupling in hypertension. Circulation 2009; 119:2798-807.

8.10
BRACHIAL ARTERY FLOW-MEDIATED DILATATION: DIFFERENT PATTERNS OF WALL SHEAR RATE INCREASE DURING REACTIVE HYPERAEMIA

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Background: Wall shear rate (WSR) is considered an important stimulus for flow-mediated dilatation (FMD). However, its estimation by conventional ultrasound is challenging due to inherent difficulties of velocity estimation near the arterial wall. To evaluate how WSR influences brachial artery FMD, we used a prototype Doppler ultrasound system which provides simultaneous estimates of WSR at near and far walls and continuous arterial diameter tracking.
Methods: Data from 33 young healthy individuals (27.5±4.9yrs, 19F) were analysed. FMD was assessed with a conventional reactive hyperaemia technique using Ultrasound Advanced Open Platform (ULA-OP). All acquired raw data were post-processed using custom-designed software to obtain WSR and diameter parameters.
Results: Baseline diameter and FMD were 3.29±0.45 mm and 6.54±3.54 %, respectively. During hyperaemia, we observed two distinct patterns of increased WSR: monophasic (MOP, n=15 fast increase reaching peak WSR at once) and biphasic (BIP, n=18 fast followed by slow increase before reaching peak WSR). In BIP, peak WSR (657±153 sec-1 vs 522±132 sec-1) and WSR area under the curve until peak dilation (20398±6265 au vs 13530±5592 au) were significantly greater than in MOP (both $p<0.05$). Absolute diameter increase was significantly greater in BIP (0.24±0.10 mm) than in MOP (0.15±0.09 mm, $p<0.05$). Percentage diameter increase tended to be greater in BIP (7.6±3.3 %) than MOP (5.3±3.5 %, $p=0.08$).
Conclusions: These results demonstrate that there are distinct WSR increase patterns during hyperaemia, and that these patterns are associated with differences in the magnitude of hyperaemic WSR. Our observations suggest that