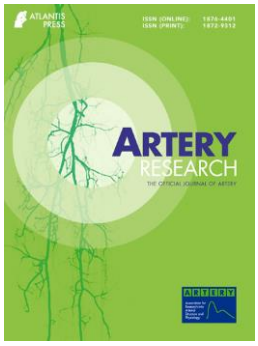

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12.8: VENTRICULAR-ARTERIAL UNCOUPLING DOES NOT DEPEND ON ARTERIAL ELASTANCE AFTER MYOCARDIAL INFARCTION

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the loading phase of this relationship (elastic energy stored during the cardiac cycle, W_E).
Results: At baseline, W_V and W_E were higher in HT than in NT subjects (W_V: 1.06±0.78 versus 0.66±0.49mmHg.mm^2, $p<0.01$ W_E: 2.33±1.47 versus 1.69±1.15mmHg.mm^2, $p<0.05$) but W_V/W_E was similar (43.0%±10. 1% versus 39.4%±11.8%). Heating did not modify significantly W_E in both groups but induced an increase in W_V only in HT patients (HT: +0.39±0.67mmHg.mm^2, $p<0.05$ NT: +0.24±0.43mmHg.mm^2, $p=0.14$ HT versus NT: $p=0.09$). Subsequently, W_V/W_E increased in HT but not in NT subjects (HT: +9.2%±9.1%, $p<0.01$ NT: +3.9%±9.9%, $p=0.22$ HT versus NT: $p<0.01$). Midwall stress, used as index of wall loading conditions, similarly increased in both groups (HT: +19.0±7.8 kPa, $p<0.001$ NT: +28.1±7.7 kPa, $p<0.01$ HT versus NT: $p=0.13$).
Conclusions: AWV is maintained during flow increase in NT subjects but increases in HT subjects. Excessive loss of energy may contribute to impair cardiovascular coupling during hypertension.

12.6
THE ROLE OF NEURONAL NITRIC OXIDE SYNTHASE IN YOUNG ADULTS

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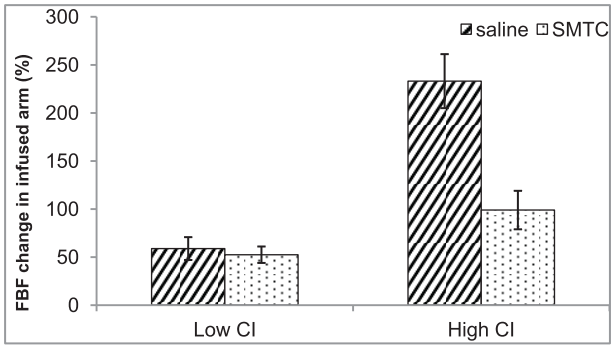
Background: Early elevation in blood pressure are characterised by a hyperkinetic circulation, with an elevated cardiac index (CI) being the dominant feature. Neuronal NOS is a key regulator of vascular tone during mental stress and is attenuated in patients with established hypertension. However, the role of nNOS has not yet been examined in young adults with a hyperactive response to stress.
Methods: 20 subjects (M:11, 28±6 years) were dichotomised into high and low CI. Forearm blood flow (FBF) was measured using strain gauge plethysmography at rest and during a word interference test (Stroop); before and after the infusion of the nNOS-specific inhibitor, S-methyl-l-citrulline (SMTC).
Results: Cardiac index was 2.88±0.7 versus 4.32±0.9 L/min/m^2 in the low and high groups, respectively. Mental stress induced a marked increase in FBF in subjects with high CI versus low CI, which was signficantly blunted after infusion of SMTC ($P<0.05$ for Two-way repeated measures ANOVA).
Figure 1: FBF response to mental stress during saline or SMTC in subjects with low versus high CI



Conclusions: The vasodilatory response to mental stress is enhanced in individuals with elevated cardiac index and nNOS appears to play a key role in this response. This may be a protective response in individuals in whom sympathetic activity may be high.

12.7
THE RELATIONSHIP BETWEEN FUNCTIONAL ARTERIAL RESPONSE AND CIRCULATING BIOMARKERS OF PATIENTS WITH FIBROMUSCULAR DYSPLASIA

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Background: Fibromuscular dysplasia (FMD) is a rare idiopathic, non-atherosclerotic non-inflammatory vascular disease. This work represents the first study of the pathophysiology of FMD. We investigated the relationships between circulating biomarkers and the non-invasive vascular parameters.
Methods: We included 50 patients with FMD, 50 essential hypertensive patients (HT) and 50 healthy subjects (NT) matched for age, sex, ethnicity and blood pressure. We determined circulating levels of total microparticles (MPs) (annexinV+MPs), endothelial MPs (CD144+MPs, CD62E+MPs and CD31+CD41-MPs) and SMC-MPs by flow cytometry analysis. We measured forearm endothelial function by post-ischemic flow dependent vasodilation. Shear stress was estimated using the formula of Weaver (1-3). *Aortic stiffness* was assessed by measuring carotid-*femoral pulse wave velocity*. Triple signal score was assessed from 15-MHz echotracking system.
Results: There are no significant differences between rates of total MPs, endothelial MPs (CD144+MPs, CD62E+MPs and CD31+CD41-MPs) and SMC-MPs between 3 groups (with p-value 0.38 0.52 0.65 0.17 and 0.25 respectively). Endothelial MPs were not correlated with the endothelial dysfunction, nor with the shear stress, whether in FMD, NT or HT. We observed a strong negative correlation between aortic stiffness and *nitroglycerin*-mediated dilation in the group NT, HT and whole population ($r= -0.43$, $p= 0.001$ $r= -0.29$, $p= 0.03$ $r = -0.35$, $p<0.001$ respectively), but not in FD ($p=0.5$). SMC-MPs were not associated with the triple signal or any arterial parameter in the group FMD nor in the whole population.
Conclusions: The number of MPs was not correlated with large artery properties. Arterial stiffness is negatively related to endothelium-independent dilatation.
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12.8
VENTRICULAR-ARTERIAL UNCOUPLING DOES NOT DEPEND ON ARTERIAL ELASTANCE AFTER MYOCARDIAL INFARCTION

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Objective: Ventricular remodeling may occur following myocardial infarction (MI) of the left ventricle (LV) and such remodeling has been shown to be correlated with increased patient morbidity and mortality. It is important to estimate the likelihood of remodeling from the state of the infarcted LV. The aim of the study was to assess the ventricular-arterial coupling (VAC) in patients with ST segment elevation (STEMI) and non ST segment elevation MI (NSTEMI) treated with percutaneous coronary intervention (PCI).
Methods: In 93 patients with acute coronary syndrome and PCI (70% male, age 61.5±10.1 years (M±SD), 57 (61.3%) with STEMI, smokers 25%, arterial hypertension 20.4%, blood pressure 129±6/82±7 mmHg) 2-dimentional echocardiography was performed to assess arterial elastance (Ea) and end-systolic LV elastance (Ees) on admission and in 4 weeks. VAC was assessed as the ratio Ea/Ees.
Results: Baseline LV ejection fraction (LVEF) was 47.4±4.3%, E/A 0.95±0.18, Ea 1.9±0.3 mmHg/ml/m^2, Ees 2.1±0.4 mmHg/ml/m^2, VAC 0.89±0.1. At baseline all patients had LVEF >40% and VAC in optimal range. In 4 weeks after PCI VAC >1.2 (upper optimal level) was revealed in 19% of patients with STEMI and 44% with NSTEMI. In patients with achieved VAC >1.2 Ees (from 2.1±0.4 to 1.5 ±0.3 mmHg/ml/m^2, $p<0.001$), stroke work (SW) (from 6585±1059 to 6919±2131 mmHg*ml/m^2, $p<0.05$), potential energy (PE) (from 1976±371 to 3025±1127 mmHg*ml/m^2, $p<0.001$), pressure-volume area (PVA) (from 6647±1060 to 6977±2136 mmHg*ml/m^2, $p<0.001$), LV work efficiency (SW/PVA) (from 78 to 89% , $p<0.001$) significantly decreased while Ea (1.9±0.3 and 2.1±0.4 mmHg/ml/m^2, $p>0.05$) did not

change. In patients with VAC in optimal range in 4 weeks Ees decreased from 2.3±0.3 to 2.1±0.4 mmHg/ml/m^2 ($p<0.001$), Ea (from 1.87±0.29 to 1.64±0.17 mmHg/ml/m^2, $p<0.001$) and VAC (from 0.82±0.12 to 0.81±0.19, $p<0.04$) did not change.
Conclusions: Impairment of functioning of cardio-vascular system assessed by increased value of VAC >1.2 was revealed in 30% of patients with acute coronary syndrome. Increase of VAC is associated predominantly with decrease of Ees and LV work efficiency (SW/PVA). Increased VAC index >1.2 indicating LV-arterial uncoupling may be an early marker of unfavorable cardiac remodeling.

12.9
VENTRICULAR ARTERIAL COUPLING IN ISOMETRIC HANDGRIP TEST IN UNTREATED HYPERTENSIVE PATIENTS

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Aim: To evaluate cardiovascular adaptation to increased afterload during handgrip isometric exercise (HIE) in untreated hypertensive patients.
Methods: 75 untreated hypertensive patients (age 54±7years, 44 males, BP 153/93 mmHg) underwent simultaneous EchoCG and blood pressure (BP) acquisition at rest and during HIE. End-systolic pressure was determined as 0,9 x brachial systolic BP (SBP). Arterial elastance (Ea) and LV elastance (Ees) were calculated as end-systolic pressure (ESP) /stroke volume (SV) and ESP/end-systolic volume (ESV). Ventricular-arterial coupling index was assessed as Ea/Ees. Efficiency of left ventricle (ELV) was evaluated by stroke work (SW)/pressure-volume area (PVA) ratio. SW=ESPчSV, PVA=SW+PE (ESPчESV/2-end diastolic pressure xESP/4). $p<0,05$ was considered significant.
Results: Ea/Ees<0,5 was found in 76% (n=57, 18 female) before HIE. In 38% (n=22, 4 (23%) female) Ea, Ees, Ea/Ees and SW/PVA did not change significantly. In 11% there was further decrease of Ea/Ees associated with significant increase of ELV. In 51% (n=29, 14 (49%) female) Ea/Ees increased due to increase of Ea from 1,98±0,32 to 2,35±0,41 ($p<0,05$) while Ees increased from 5,95±2,2 to 4,58±1,0 ($p<0,05$). Ea/Ees increase was associated with decrease of ELV from 0,89±0,02 to 0,84±0,02 ($p<0,05$) indicating cardiovascular misadaptation to HIE.
In subjects (n=18, 3 female) with normal Ea/Ees 0,5-1,2 before HIE Ea/Ees and ELV did not change in 8 (49%, all males). In 10 subjects (3 female) Ea/Ees decreased due to significant increase of Ees (from 3,15±0,68 to 5,02±1,34 ($p<0,05$), and ELV increased from 0,81±0,03 to 0,88±0,01 ($p<0,05$).
Conclusion: Cardiovascular misadaptation to afterload is the most prevalent type of reaction to HIE in subjects with decreased baseline Ea/Ees and may be also observed in subjects with normal baseline ventricular-arterial coupling. This misadaptation in subjects with baseline ventricular arterial uncoupling is associated with female gender.

12.10
REDUCED VENTRICULAR-ARTERIAL COUPLING AS AN EARLY MARKER OF CARDIOVASCULAR REMODELING IN HYPERTENSIVE MEN

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Objective: To evaluate ventricular-arterial coupling (VAC), left ventricular hypertrophy (LVH), diastolic function and arterial stiffness in young and middle-aged men with uncomplicated arterial hypertension
Methods: 97 young men aged 18-27 years (21,2±1,9 years, BP 156,5±14,0/98,5±9,1 mmHg) and 68 middle-aged men aged 40-60 years (n=68, age 53,9±7,2 years, BP 152,7±9,6/94,8±7,8 mmHg) (M) underwent simultaneous EchoCG, blood pressure (BP) and pulse wave velocity measurement. VAC index was calculated arterial elastance (Ea) and left ventricular elastance (Ees) ratio. Ea=end-systolic pressure/stroke volume, Ees=end-systolic pressure/end-systolic volume. LVH was diagnosed if LV mass index was >115 g/m2, Increased arterial stiffness -if PWV >10 m/s, diastolic dysfunction – if E/A <1,0 and E/E'<7 m/s.
Results: In young men Eea 1,86±0,32 and index VAC (0,52±0,10) was similar to that in middle-aged (1,9±0,47 and 0,48±0,19, respectively), despite that Ees in young men was significantly lower (3,67±0,85) than in middle-aged men (4,6±2,1, $p<0,01$). VAC index <0,5 was found in 34% young men and in 57% middle-aged men ($p<0,05$), LVH in 7,4% and 67% ($p<0,05$), diastolic dysfunction 4,1% and 62%, respectively. VAC index was similar in those with and without LVH or diastolic dysfunction in the both age groups. In young men with LVH VAC index was 0,63±0,26, without LVH 0,54±0,12, in middle-aged patients with LVH – 0,45±0,16, without LVH – 0,49±0,21. In young men with diastolic dysfunction VAC index was 0,61±0,13, without diastolic dysfunction – 0,58±0,16. In middle-aged men 0,45±0,14 and 0,48±0,16, respectively. PWV>10 m/s was found in 22,7% of young men and in 80,1% of middle-aged ($p<0,05$). No significant correlation between VAC index and BP, LVMI, PWV, E/A, E/E' was found.
Conclusion: Decrease in the VAC index<0,5 indicating LV-arterial uncoupling may be an early marker of cardiovascular remodeling in hypertensive men that may be observed before development of LVH, diastolic dysfunction or increased arterial stiffness.

12.11
SARCOPENIA AND VASCULAR RISK IN A HEALTHY ELDERLY UK POPULATION (BRAVES STUDY)

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Introduction: Sarcopenia, the loss of skeletal muscle mass and strength that occurs with advancing age [1] is correlated with functional decline and disability but little is known about its relationship with cardiovascular risk. Bioimpedence analysis (BIA) is a validated technique for measuring muscle mass, convenient for use in large cohort studies. Arterial stiffness (compliance) is an independent predictor of cardiovascular events.
Methods: The BRAVES study was designed to compare cardiovascular risk between two healthy elderly cohorts in the UK and in Italy. We used data from the UK cohort to investigate the relationship between sarcopenia and vascular compliance.
Participants were eligible if aged 65-85 years, lived within the Brighton area and had weight loss of no more than 5% in the last month. All underwent physical exam, BIA assessment of skeletal mass index (SMI) and two measures of arterial compliance. Pulse wave velocity (PWV) was measured between carotid-femoral and carotid-radial arteries and the augmentation index (AIx) derived from carotid and radial arteries. A bivariate correlation was performed.
Results: Ninety patients (64 female; 26 male) had mean age 73, mean FFM 46.84kg (range 34.7-74.7) and mean SMI 6.77 (range 4.84-10.09). There was a negative relationship between SMI and Radial AIx (R=0.-542, p=0.000) as well as Carotid AIx (R=-0.391, p=0.002) but not PWV. Using multiple regression to control for the effects of age and gender, SMI was independently related to radial AIx (p=.013).
Conclusions: Skeletal muscle mass index is strongly negatively correlated with augmentation index, a measure of vascular stiffness. This finding suggests that elderly patients with higher muscle mass have a more compliant aorta and hence lower cardiovascular risk. Whether sarcopenia acts as a marker for CV risk or plays an active role in cardiovascular disease progression is not yet established and deserves further investigation.
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13.1
THE EFFECTS OF ALPHA 1-ADRENOCEPTOR-BLOCKADE AND ANGIOTENSIN CONVERTING ENZYME-INHIBITION ON INDICES OF AORTIC STIFFNESS MEASURED BY AN OSCILLOMETRIC SINGLE CUFF METHOD IN HYPERTENSION: THE DOXAZOSIN RAMIPRIL STUDY

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Objectives: To study whether inhibition of the renin-angiotensin-aldosterone system has effects on arterial stiffness beyond blood pressure (BP) reduction alone.
Methods: Hypertensive patients (age 54±12 years, 34% women) were randomized double-blind to ramipril (10 mg od, n=32) or doxazosin (8 mg od, n=26) for 12 weeks. Central aortic BP and pulse pressure (PP), aortic pulse wave velocity (PWV), and augmentation index (AIx) were assessed by a single