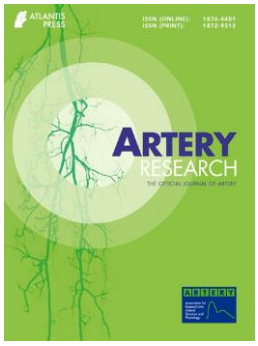

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12.7: THE RELATIONSHIP BETWEEN FUNCTIONAL ARTERIAL RESPONSE AND CIRCULATING BIOMARKERS OF PATIENTS WITH FIBROMUSCULAR DYSPLASIA

Thi-Phuong-Nhung Hoang, Pierre Boutouyrie, Hakim Hettab, Louise Marais, Aurelien Lorthioir, Michael Frank, Ralph Niarra, Jean-Marie Renard, Yann Chambon, Xavier Jeunemaitre, Pierre-François Plouin, Stéphane Laurent, Chantal Boulanger, Michel Azizi

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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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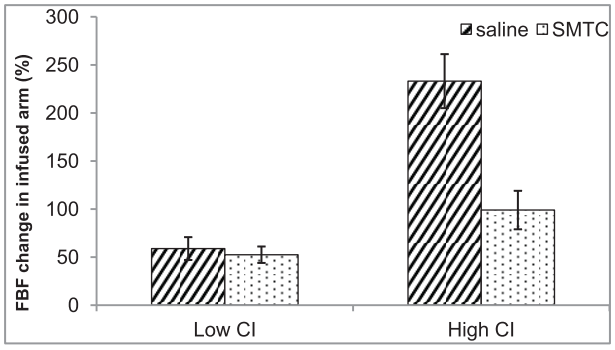
Background: Early elevation in blood pressure are characterised by a hyperkinetic circulation, with an elevated cardiac index (CI) being the dominant feature. Neuronal NOS is a key regulator of vascular tone during mental stress and is attenuated in patients with established hypertension. However, the 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

Thi-Phuong-Nhung Hoang [1], Pierre Boutouyrie [4], Hakim Hettab [2], Louise Marais [1], Aurelien Lorthioir [4], Michael Frank [4], Ralph Niarra [4], Jean-Marie Renard [2], Yann Chambon [2], Xavier Jeunemaitre [3], Pierre-François Plouin [3], Stéphane Laurent [4], Chantal Boulanger [1], Michel Azizi [4]
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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