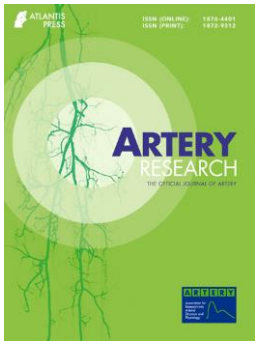

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12.5: IMPAIRED REGULATION OF ARTERIAL WALL VISCOSITY DURING CHANGES IN BLOOD FLOW IN ESSENTIAL HYPERTENSIVE PATIENTS

Frederic Roca, Michele Iacob, Guillaume Feugray, Christian Thuillez, Jeremy Bellien, Robinson Joannides

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12.1
EVALUATION OF ACUTE EFFECTS OF COFFEE CONSUMPTION ON ARTERIAL STIFFNESS IN HEALTHY ADULT PEOPLE USING AN OSCILLOMETRIC DEVICE

Pedro Forcada [1], Dario Echeverry [2], Alejandro Pizano [2], Felix Montes [2], Daniel Acuña [2]
[1]*Instituto De Investigaciones Cardiologicas, Buenos Aires, Argentina*
[2]*Laboratorio Funcion Vascular Instituto De Cardiologia F. De Medicina, Bogota, Colombia*

Environment/Objectives: Several studies in different populations and conditions shown contradictory results about the effect of coffee on arterial stiffness (AS). Coffee consumption is high around the world and it is very important to define its CV effects.
To evaluate the acute effects on haemodynamic parameters and AS, after consumption of regular coffee or, decaffeinated coffee.
Methods: In a prospective, self controlled cohort study we included 32 healthy p. (46.2±10.4y.o., 16 men (53.5±18) and 16 women (43.0±21)(p=0,186)). Fourteen regular coffee consumers (87.5%) (p=NS). Haemodynamic parameters and AS were assessed non invasively using oscilometric Arteriograph® (TensioMed Budapest, Hungary Ltd.). Each subject received 14 gr. of excelso coffee (151.2 mg caffeine) and two weeks apart, 14 gr of decaf coffee (3.92 mg) in random order. Baseline, 30 and 60 min parameters are reported.
Results: SBP increased at 30 and 60 min 3.9 mmHg (p=0.013) y 3.8 mmHg (p=0.002) respectively, la DBP increased 4.1 mmHg (p=0.001) y 3.2 mmHg (p=0.003), MAP 4.0 mmHg (p<0.001) y 3.3 mmHg (p=0.001), Heart rate decreased 3.2 (p=0.002) and 5 latidos/minuto (p<0.001) and aortic SBP increased 5.8 mmHg (p=0.002) and 7.6 mmHg (p=0.003) only with caffeine. Brachial Aix increased 19.9% at 30 (p<0.001) and 20.0% at 60 minutes (p<0.001). Aortic Aix increased 10.05% (p<0.001) y 10.2% (p<0.001) only with caffeine. PWV was not affected by caffeine (p=0.861). The shift of these parameters was mainly driven by changes in women.
Conclusions: Caffeine at usual doses (two expresos) increased peripheral AS but not aortic PWV, specially in women.

12.2
HIGH PULSE WAVE VELOCITY IS ASSOCIATED WITH INCREASED VISIT-TO-VISIT SYSTOLIC BLOOD PRESSURE VARIABILITY IN CONTROLLED ARTERIAL HYPERTENSION

Elena Troitskaya, Yulia Kotovskaya, Zhanna Kobalava
Peoples' Friendship University of Russia, Moscow, Russia

Background: Visit-to-visit blood pressure variability (BPV) is associated with adverse cardiovascular outcomes in different patients' populations[1-2]. Arterial stiffness is a potential mechanism of increased visit-to-visit BPV[3]. Carotid—femoral PWV has become increasingly important for total cardiovascular risk estimation.
Materials and methods: 52 pts (20 men, age 58.9±9.0 yrs 4 smokers 6 diabetics) were treated to target BP<140/90mmHg with a RAAS-inhibitor/amlodipine combination for 14 months. Baseline brachial BP was 163.4±8.1/100.9±4.2mmHg achieved-123.7±9.7/76.8±6.7mmHg. Central BP and PWV were measured at baseline and after 14 months. Individual values of PWV were assessed according to age and BP categories[4]. BPV was calculated as SD for 5 visits during 8 months after target BP achievement.p<0.05 was considered significant.
Results: Baseline central BP was 137.8±17.3/86.6±12.0mmHg, achieved 125.2±13.5/80.3±6.6mmHg (p<0.05). Baseline PWV varied from 7.6 to 19.2 m/s (median 12.2 m/s), achieved — from 9.9 to 17.4 m/s (median 13.4 m/s),p>0.05. Normal values of PWV according to individual reference values were observed in 25.5% of patients (group1, mean PWV 10.0±1.5 m/s), increased — in 74.5% (group2, mean PWV 13.8±2.4 m/s). Groups were similar by age, gender, metabolic risk factors, baseline and achieved BP and visit-to-visit BPV. SBPV range was 1.79-16.79 mmHg (tertile I<5.38 II 5.38 — 7.78 III>7.78 mmHg). Increased PWV value was more often observed in the III tercile of visit-to-visit SBPV (90.3% comparing to 58.8% in tercile I and 73.3% in tercile II, Pearson (χ^2)=5.9,p<0.05).
Conclusion: Elevation of PWV above individual reference values in patients with uncomplicated AH is associated with higher visit-to-visit SBPV. This finding confirms role of arterial stiffness in visit-to-visit BPV increase.

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12.3
24 HOUR AMBULATORY BLOOD PRESSURE MONITORING AND PULSE WAVE VELOCITY PATTERNS IN KENYAN ADOLESCENTS

Anthony Etyang [1,2,3,4]
[1]*KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya*
[2]*London School of Hygiene and Tropical Medicine, London, UK*
[3]*African Population and Health Research Centre, Nairobi, Kenya*
[4]*King's College, London, UK*

Background: There are no data on ambulatory blood pressure monitoring (ABPM) and arterial stiffness parameters in sub-Saharan African children. We performed 24-hour ABPM and pulse wave velocity (PWV) measurements in adolescents living in 2 slums in Nairobi, Kenya.
Methods: We selected 1,100 11-17 year olds who from birth had been continuous residents of the Nairobi Urban Health and Demographic Surveillance System (NUHDSS) to participate in the study. Participants underwent anthropometric measurements (weight, height, mid-upper arm circumference [MUAC]) and answered questions on their socioeconomic status (SES). A clinic BP measurement was then taken using an automated Omron ™ M10-IT monitor (mean of 2 from 3 readings). Participants then underwent 24-hr ABPM and PWV measurement using an Arteriograph ™24 monitor.
Results: 500 (90%) of 558 children recruited between December 2015 and June 2016 had acceptable ABPM readings (≥ 20 daytime and ≥7 nighttime readings). Mean (SD) clinic BP, and 24 hour-ABPM values were 98(11) and 117(12) systolic and 63(8) and 64(7) mmHg diastolic respectively. Mean clinic PWV and 24 hour-PWV were 7.3(1.5) ms^{-1} and 7(0.8) ms^{-1}respectively. In multivariate regression analyses age (p=0.004), BMI (p=0.033) and PWV (p<0.001) were strong independent predictors of 24-hour BP values. Blood indices (hemoglobin, white cell and platelet count), gender, MUAC and SES had no independent influence on 24hr BP and PWV.
Conclusions: These are to our knowledge the first 24hr ABPM and PWV data generated from sSA adolescents. Long-term cardiovascular outcome studies are needed to determine the predictive ability of ABPM and PWV measurements.

12.5
IMPAIRED REGULATION OF ARTERIAL WALL VISCOSITY DURING CHANGES IN BLOOD FLOW IN ESSENTIAL HYPERTENSIVE PATIENTS

Frederic Roca, Michele Iacob, Guillaume Feugray, Christian Thuillez, Jeremy Bellien, Robinson Joannides
Department of Pharmacology, Rouen University Hospital, Rouen, France

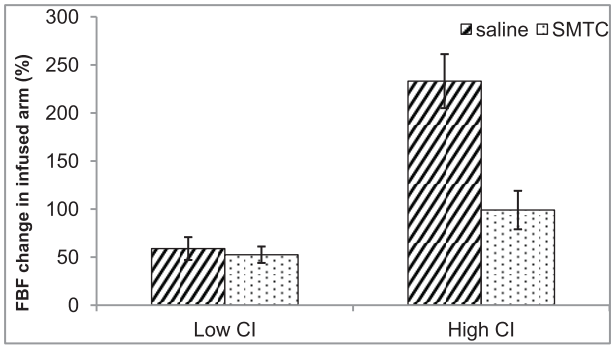
Background: Arterial wall viscosity (AWV) is a major source of cardiac energy dissipation along the arterial tree. Evolution of AWV during increase in blood flow and the impact of essential hypertension on this evolution have never been studied.
Methods: Radial artery diameter, wall thickness and arterial pressure were simultaneous measured in 18 untreated essential hypertensive (HT) subjects and 14 frequency matched normotensive (NT) controls at baseline and during a sustained blood flow increase induced by hand skin heating. AWV was estimated by the ratio of the area of the hysteresis loop of the pressure-diameter relationship (viscous energy dissipated, W_V) to the area under

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

Jessica Middlemiss, Sarah Evans, Joseph Cheriyan, Lucy Yang, Ian Wilkinson, Carmel McEniery
University of Cambridge, UK

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]
[1]*University Paris Descartes, Paris, France*
[2]*INSERM U970 Paris Cardiovascular Research Center, Paris, France*
[3]*Clinical Investigation Center, Paris, France*
[4]*Department of Genetics and Hypertension Unit, Paris, France*

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

Elena Zharikova, Svetlana Villevalde, Zhanna Kobalava
Peoples Friendship University of Russia, Moscow, Russia

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