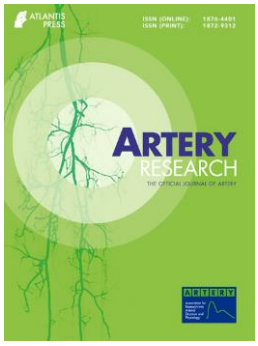

Artery Research

ISSN (Online): 1876-4401 **ISSN (Print): 1872-9312**
Journal Home Page: https://www.atlantis-press.com/journals/artres

P13.01: THE IMPACT OF ACUTE SYSTEMIC INFLAMMATION ON ARTERIAL FUNCTION OF PATIENTS WITH STABLE ANGINA PECTORIS: ADDING FUEL TO THE FIRE WITHIN?

P. Xaplanteris, C. Vlachopoulos, I. Dima, D. Terentes-Printzios, N. Alexopoulos, C. Stefanadis

To cite this article: P. Xaplanteris, C. Vlachopoulos, I. Dima, D. Terentes-Printzios, N. Alexopoulos, C. Stefanadis (2010) P13.01: THE IMPACT OF ACUTE SYSTEMIC INFLAMMATION ON ARTERIAL FUNCTION OF PATIENTS WITH STABLE ANGINA PECTORIS: ADDING FUEL TO THE FIRE WITHIN?, Artery Research 4:4, 183–183, DOI: https://doi.org/10.1016/j.artres.2010.10.133

To link to this article: https://doi.org/10.1016/j.artres.2010.10.133

Published online: 21 December 2019

P12.08
THE ASSESSMENT OF ARTERY STIFFNESS IN YOUNG POPULATION WITH PRIMARY ARTERIAL HYPERTENSION

M. Walczak, K. Hoffmann, M. Cymerys, D. Pupek-Musialik
University of Medical Sciences, Poznan, Poland

Objectives: Young patients with primary arterial hypertension (PAH) should be thoroughly diagnosed and screened for the presence of other cardiovascular risk factors. The aim of this study was to assess the artery stiffness in young adults with PAH.
Methods: In the study there were 33 men with PAH, age 21.52 ± 3,15. 15 patients were treated with ACE-inhibitors or beta- blockers; 18 remained in clinical observation. The anthropometric and blood pressure measurements were taken. The arterial stiffness was estimated by Pulse Trace PCA2 which is a pulse contour analysis system using a photo-plethysmography transducer to obtain signal of the Digital Volume Pulse (DVP) waveform. It calculates time from systolic inflection point (if present) or systolic peak to diastolic inflection point (PPT), Stiffness Index (SI) defined as the subjects height divided by PPT.
Results: In the whole examined population the range of SI was 4-9 m/s; mean SI was 7.35 ± 1.32 m/s. In subjects treated pharmacologically mean SI was 7.64 ± 0.86 m/s, whereas in patients treated non- pharmacologically mean SI was 7.28 ± 1.56 m/s.
Conclusions: 1. There is no statistically significant difference in SI in young patients with PAH. 2. SI is correlated with age and blood pressure.

P12.09
VASCULAR FUNCTION IN PREECLAMPSIA. ANALYSIS OF DIGITAL VOLUME PULSE BY PHOTOPLETHYSMOGRAPHY

C. Fava [1], E. Nesta [1], G. Amen [1], F. Dalle Vedove [1], G. Zanconato [2], P. Minuz [1]
[1]*University of Verona, Department of Medicine, Verona, Italy*
[2]*University of Verona, Department of Life and Reproduction Science, Verona, Italy*

To test whether vascular function is altered in normal pregnancy and preeclampsia (hypertension and proteinuria after the 20th week of pregnancy), we performed a cross-sectional study comparing 15 preeclamptic women (PE) with 31 normotensive pregnant -11 pair matched for gestational age (MGA-NP) and 20 at term (T-NP)- and 20 non-pregnant women (HC). All subjects were matched for age and BMI ($<27\ Kg/m^2$). Women with obesity, diabetes, pre-existing arterial hypertension were excluded. Women with PE received antihypertensive drugs. Stiffness index (SI) and Reflection Index (RI) were obtained by digital volume pulse analysis using photoplethysmography. Mean arterial pressure (MAP), plasma creatinine and uric acid were higher in PE ($p<0.001$ vs. NP and HC), whereas heart rate (HR) was lower in PE ($p<0.05$ vs. NP). RI was higher in PE respect to NP (median [min-max] 66.5%[37-80]) vs. 51.5% [31-80]; $p<0.001$) whereas SI tended to increase in PE (7.6 m/sec [5.6 13.5]; MGA_NP 6.5 m/sec [5.4-8.7]; $p=0.10$). When normalized for MAP, both SI/MAP and RI/MAP were similar in PE and NP, whereas SI/HR and RI/HR normalized for HR were higher in PE ($p<0.05$ vs. NP and MGA_NP). SI ($p<05$) was higher in PE respect to HC. Correlation between SI and RI ($r_s=0.56$ $p<0.001$), MAP and both SI and RI ($r_s=0.48$ $p=0.001$; $r_s=0.50$ $p<0.001$), HR and RI ($r_s=0.58$ $p<0.001$), uric acid and RI ($r_s=0.41$ $p=0.007$) was observed in PE+NP. The present results confirm that vascular function is altered in PE with higher RI suggesting peripheral vasoconstriction and a tendency toward higher SI, suggesting diminished compliance.

Physiopathology 7
P13.01
THE IMPACT OF ACUTE SYSTEMIC INFLAMMATION ON ARTERIAL FUNCTION OF PATIENTS WITH STABLE ANGINA PECTORIS: ADDING FUEL TO THE FIRE WITHIN?

P. Xaplanteris, C. Vlachopoulos, I. Dima, D. Terentes-Printzios, N. Alexopoulos, C. Stefanadis
Hippokrateion Hospital, 1st Department of Cardiology, Athens Medical School, Athens, Greece

Purpose: According to recent evidence, acute inflammation is associated with transiently impaired vascular function and aortic compliance in healthy adults. The relationship between acute inflammatory stimuli and arterial stiffness in patients with stable angina pectoris (SAP) has not been investigated yet.
Methods: We studied the effect of an acute inflammatory stimulus on wave reflections and aortic stiffness, in 13 patients with SAP (mean age 49.4 years, 13 men) and in a control group consisting of 13 healthy adults (mean age 47.2 years, 7 men). We used Salmonella Typhi vaccine to induce an acute, mild, transient and systemic inflammatory response in both groups. cfPWV and AIx were measured at baseline and 8h after vaccination.
Results: Inflammation led to a decrease in AIx, 8h after vaccination in both groups (from 26.2% to 20% in SAP patients, $P<0.05$, from 31.2% to 22.9% in healthy adults, $P<0.05$). cfPWV increased in the healthy group after 8h (from 6.70m/sec to 6.96m/sec, $P<0.05$), however in the SAP group there was no change in aortic stiffness 8h after vaccination (from 8.33m/sec to 8.01m/sec, P=ns).
Conclusions: Acute systemic inflammation has an effect on wave reflection in both healthy and SAP patients, however there is a significant increase of large artery stiffness only in the healthy group, indicating a different behaviour of large arteries in the SAP group, probably due to the atherosclerotic burden present on their arteries. This finding is particularly important for determining possible links between inflammation and arterial function in the setting of coronary heart disease.

P13.02
VENTRICULO-VASCULAR COUPLING IS IMPAIRED IN PATIENTS WITH TYPE-II-DIABETES MELLITUS AND RESISTANT HYPERTENSION

T. K. Sønder [1], B. B. Løgstrup [3], J. Lambrechtsen [2], L. M. Van Bortel [4], P. Segers [5], K. Egstrup [1]
[1]*Department of Medical Research, University Hospital of Odense, Svendborg Hospital, Svendborg, Denmark*
[2]*Department of Cardiology, Svendborg Hospital, Svendborg, Denmark*
[3]*Department of Cardiology, University Hospital of Aarhus, Skejby Hospital, Aarhus, Denmark*
[4]*Heymans Institute of Pharmacology, Ghent University, Ghent, Belgium*
[5]*Institute for Biomedical Technology (IBITECH), Ghent University, Ghent, Belgium*

Objective: To examine if ventriculo-vascular coupling (VVC) is impaired in patients with resistant hypertension (RH) and type-II-diabetes mellitus.

Characteristic	Controlled hypertension	Resistanthy pertension	P	Adjusted P
Sex (male/female)	**15/9**	**27/7**		
Age (years)	62±10	64±9	0.49	
Body mass index (kg/m^2)	34±7	35±5	0.311	0.205
Length of disease (years)	10.4±6.3	14±7	0.023	0.028
Pulse pressure (mmHg)	50±8	66±9	<0.0001	<0.0001
Mean arterial pressure (mmHg)	88±4	96±7	<0.0001	<0.0001
Heart rate (bpm)	74±11	71±13	0.312	0.238
Pulse wave velocity (m/s)	9.7±3	12.1±5	0.042	0.385
Characteristic impedance	0.07±0.03	0.1±0.1	0.031	0.045
E_A (mmHg/ml)	1.63±0.5	1.86±0.6	0.124	0.057
E_{MAX} (mmHg/ml)	2.7±1	2.1±0.9	0.023	0.077
E_A/E_{MAX}	0.7±0.3	1.1±0.5	0.003	0.005
K_{LV} (mmHg/ml)	0.16±0.1	0.3±0.2	0.015	0.04
Ejection fraction (%)	55±10	45±11	<0.0001	0.001
E/Ê	9±3	11±4	0.006	0.007

Ventriculo-vascular coupling is impaired in patients with type-II diabetes melitus and resistant hypertension P 13.02

Methods: We included 87 patients. RH was defined according to guidelines from the American Heart Association.
Echocardiography was performed using GE Vivid 7and pulse wave analysis using Sphygmocor. All examinations were done under standardized conditions. All analyses were done blinded offline using Echopac and customized software.
VVC was estimated from stroke volume, end systolic pressure and —volume using the formula E_A/E_{MAX}.
Left ventricular chamber stiffness (K_{LV}) was estimated from mitral valve deceleration time (MV dect) using the formula 70(MV dect-20)^2.
All statistical analyses were adjusted for sex, age, length of disease and heart rate using multiple linear regression.
Results: 34 patients had RH and 24 had controlled hypertension (CH) leaving 29 with uncontrolled hypertension. See table 1 for patient characteristics. Patients were comparable with regards to age and BMI. Pulse pressure, mean arterial pressure and length of disease varied significantly between patients with RH and CH.
Patients with RH had higher E_A/E_{MAX} ($P=0.005$) and lower EF ($P=0.001$). They also had higher K_{LV} ($P=0.04$) and E/É ($P=0.007$) as well as higher characteristic impedance ($P=0.045$). Pulse wave velocity was not statistically significantly higher when adjusted for covariates ($P=0.385$).
Conclusion: Patients with resistant hypertension have dysfunctional VVC most likely due to stiffening of the left ventricle, which could be due to increased afterload and stiffness of the arterial system.