



## Artery Research

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### **P.045: REVERSIBLE LEFT VENTRICULAR DYSFUNCTION AND BRAIN NATRIURETIC PEPTIDE (BNP) PLASMA LEVELS IN PATIENTS WITH TRAUMATIC BRAIN INJURY**

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non-invasively by carotid-femoral pulse wave velocity (PWV) and central augmentation index (AI).

**Design:** Twenty two subjects with pheochromocytoma (PHEO), 34 subjects with essential hypertension (EH) and 40 healthy normotensive controls (C) were investigated using an applanation tonometer (Sphygmocor). Twelve patients with pheochromocytoma were studied after tumor removal.

**Results:** The gender, age, body mass index and lipid profiles were comparable among all the groups. Fasting plasma glucose levels in PHEO were higher in comparison to the other groups ( $6.1 \pm 1.7$  vs.  $EH 4.9 \pm 1.1$  vs.  $C 4.8 \pm 0.8$  mmol/l;  $p < 0.001$  for all comparisons). Brachial blood pressure values in PHEO were lower in comparison to EH ( $135 \pm 24/77 \pm 12$  vs.  $153 \pm 6/90 \pm 11$  mmHg;  $p < 0.001/0.001$ ) and higher in comparison to C ( $135 \pm 24/77 \pm 12$  vs.  $121 \pm 11/72 \pm 9$  mmHg;  $p < 0.001/n.s.$ ). The pulse wave velocity in pheochromocytoma did not differ from EH ( $7.1 \pm 1.3$  vs.  $7.3 \pm 1.5$  m.s<sup>-2</sup>, ns.) and was significantly higher than in controls ( $7.1 \pm 1.3$  vs.  $5.9 \pm 0.7$  m.s<sup>-2</sup>;  $p < 0.001$ ). No differences were found in AI among all groups. In multiple regression the only significant variables independently associated with PWV in pheochromocytoma were 24h urine norepinephrine excretion ( $\beta = 0.566$ ,  $p < 0.001$ ) and fasting plasma glucose levels ( $\beta = 0.346$ ,  $p = 0.015$ ). Successful tumor removal led to a significant decrease in PWV ( $6.9 \pm 0.8$  vs.  $5.5 \pm 0.7$  m.s<sup>-2</sup>;  $p < 0.001$ ).

**Conclusion:** Catecholamine excess in pheochromocytoma is accompanied by an increase in pulse wave velocity, which is reversed by the successful tumor removal. Pulse wave velocity in subjects with pheochromocytoma is positively associated with 24h urine norepinephrine levels and fasting plasma glucose levels.

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#### P.044

##### EFFECT OF MILD INCREASE OF PHYSICAL ACTIVITY ON MICROVASCULAR REACTIVITY IN OBESE SUBJECTS WITH DIABETES MELLITUS TYPE 2

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Microangiopathy, well known in diabetic patients as a cause of late complications, develops mainly due to chronic exposition to elevated glucose and triglyceride level. Physical training acts as a protective factor even if no changes in metabolic parameters are observed. It's supposed, that lifestyle modifications leads to the improvement of endothelial dysfunction and microvascular reactivity, in healthy subjects it has already been proven experimentally.

In 8 patients with type 2 diabetes mellitus was measured microvascular reactivity and perfusion of skin in lower limbs by laser-doppler flowmetry and transcutaneous oximetry. First before the study, second after 3-week's period of habitual physical activity, third after 3-week's period of mild increased physical activity and finally after next 3-week's period of habitual activity. Training intensity was objectified (non sport-practiced subjects) by pedometers. Results were evaluated by Friedman and pair Wilcoxon test.

After mild aerobic activity (walk about 800 [560 - 1400] meters/day) microvascular reactivity was increased in both tests (increase after heating from  $4,9x [4,4-5,4]$  to  $6,1x [5,7-6,8]$ ,  $p < 0.01$ , shorten half time to reach maximum perfusion from  $4,1 [2,7-5,4]$  s to  $3,1 [2,4-4,0]$  s,  $p < 0.05$ . The increased perfusion lasted after following four weeks of habitual activity in smaller extent (microvascular reactivity increase after heating  $5.2 [4.8-6.1]$  s, half time to reach maximum perfusion  $3.8 [2.7-5.0]$ , this increase was not significant in comparison with habitual activity in the first period). Metabolic and anthropometric parameters and transcutaneous oxygen tension didn't change significantly.

#### P.045

##### REVERSIBLE LEFT VENTRICULAR DYSFUNCTION AND BRAIN NATRIURETIC PEPTIDE (BNP) PLASMA LEVELS IN PATIENTS WITH TRAUMATIC BRAIN INJURY

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**Aim:** We investigated the possibility of myocardial dysfunction and pertinent alterations of BNP plasma levels in patients with traumatic brain injury (TBI).

**Patients and Methods:** This study included 30 critically care patients (20 males, mean age  $30 \pm 8$  years old) who were admitted to the intensive care unit with TBI (Glasgow Coma Scale upon admission  $< 8$ ). Patients with a previous history of cardiovascular disease, chest trauma, sepsis and/or other critical illness known to be associated with myocardial dysfunction were excluded. Left ventricular (LV) function was assessed by transoesophageal echocardiography. BNP plasma concentrations were measured in all patients. Echocardiographic and BNP measurements were performed on a weekly basis.

**Results:** Eight patients progressed towards brain death. Five patients presented global reversible LV dysfunction during the first week after the TBI, which normalized over time (within 2 weeks upon presentation). Ten patients exhibited segmental contractility disturbances during the first 2 weeks after the TBI, which normalized over time (within 4 weeks upon presentation). All the above patients presented electrocardiogram changes that normalized in line with the echocardiographic changes. The initial BNP plasma concentrations in the 15 patients with the reversible cardiovascular dysfunction were significantly increased as compared with those without cardiovascular dysfunction ( $105 \pm 53$  pg/ml vs.  $53 \pm 26$  pg/ml,  $p < 0.001$ ). BNP concentrations were positively correlated with diffuse subarachnoid hemorrhage ( $r = 0.85$ ,  $p < 0.001$ ) and poor outcome ( $r = 0.88$ ,  $p < 0.001$ ).

**Conclusion:** Reversible cardiac disturbances developed in almost 50% of patients with TBI. Increased BNP concentrations are associated with a poor outcome in the above patients.

#### P.046

##### IMPAIRED ARTERIAL ELASTIC PROPERTIES IN HUMAN IMMUNODEFICIENCY VIRUS INFECTED NAÏVE PATIENTS. THE ROLE OF SUBCLINICAL INFLAMMATION

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**Introduction:** Subclinical inflammation has been associated with impaired arterial elastic properties. Human immunodeficiency virus (HIV) infection per se represents a model of chronic inflammation, possibly related to progression of atherosclerosis.

**Purpose:** To investigate the impact of HIV infection on arterial elastic properties in HIV naïve patients.

**Methods:** We studied 32 HIV infected naïve patients (aged 32 years, 28 males, 18 smokers) and 26 healthy individuals matched for age, sex and smoking status. Aortic augmentation index (AIx) and augmented pressure (AP) were assessed using assessment applanation tonometry of the radial artery. Carotid femoral pulse wave velocity (PWV) was estimated as an index of aortic stiffness by means of a computerized method (Complior SP).

**Results:** HIV patients compared to controls had increased levels of pulse pressure ( $52$  vs.  $44$  mmHg,  $p = 0.004$ ), while there was no difference regarding body mass index and metabolic profile. AP and AIx were significantly decreased in HIV patients ( $0.3$  vs.  $4$  mmHg,  $p = 0.01$  and  $1\%$  vs.  $12\%$ ,  $p = 0.005$ , respectively) even after correction for heart rate ( $0.6\%$  vs.  $8.3\%$ ,  $p = 0.043$ ). HIV patients and controls exhibited similar values of PWV ( $6$  vs.  $6.1$  m/s, respectively,  $p = 0.83$ ).

**Conclusions:** HIV infected naïve patients compared with controls are characterized by decreased wave reflections and similar values of large artery stiffness. Peripheral vasodilatation is suggested to be the predominant mechanism, induced probably by the chronic subclinical inflammation in this setting.

#### P.047

##### EXAMINATION OF PRESSURE AND VOLUME PULSE WAVES

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Measurements of the arterial pulse were made at the brachial, carotid, and radial artery. The pressure and volume waveforms were obtained using piezoelectric and optical sensors, respectively. The data were digitized using a digital storage oscilloscope interfaced with a personal computer. The time between velocity maxima of the two waveforms was computed after Savitzky-Golay filtering. In the absence of arterial expansion, the derivative