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### **P12.04: THE ROLE OF OXIDATIVE STRESS IN ACETYLCHOLINE-INDUCED RELAXATION OF DEENDOTHELIZED ARTERIES**

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Carotid wave speed was higher in men ( $p = 0.03$ ) and increased with age in both sexes although the rise was more marked in women.

**Conclusions:** Cardiac wave intensity falls with age in healthy individuals and is lower in women. Wave reflections from the head are reduced in healthy older people.

1. Hughes & Parker *Med Biol Eng Comput.* 2009;47:207-10.

#### P12.04

##### THE ROLE OF OXIDATIVE STRESS IN ACETYLCHOLINE-INDUCED RELAXATION OF DEENDOTHELIZED ARTERIES

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Nitric oxide (NO) produced by endothelium in response to vasorelaxants, such as acetylcholine, induces vasorelaxation of vascular smooth muscle cells (VSMC). It has been found that VSMC express NO-synthase, however, the principal question remained unanswered, if it is physiologically relevant. Because injury of endothelium triggers free-radical production which decreases NO availability we hypothesized that the destruction of arterial anatomical integrity by rubbing off endothelial layer made vessels insensitive to vasodilators as a consequence of oxidative stress. We examined acetylcholine-induced vasorelaxation in deendothelized thoracic aorta (TA), mesenteric artery (MA) and pulmonary artery (PA) of Wistar rats under protection against oxidative stress. Acetylcholine produced vasorelaxation in arteries with intact endothelium, whereas the relaxation in endothelium-denuded rings was inhibited. Pretreatment of TA, MA and PA denuded rings with tempol, free-radical scavenger, improved relaxation to acetylcholine compared to untreated rings. The improved relaxation in all denuded rings was inhibited when ODC, an inhibitor of guanylate cyclase, or L-NAME, an inhibitor of NO-synthase, were administered contemporary with tempol. Chemiluminescence method revealed that endothelial denudation of TA and PA increased the production of superoxides. Immunohistochemical staining confirmed expression of NOS3-isoform in both intimal and medial cells in all arteries. Results revealed that deendothelized arteries under protection against oxidative stress exerted relaxation to acetylcholine which was mediated by NO and cGMP. The study suggests that VSMC can release NO in amounts sufficient to account for the vasorelaxation. This finding challenges the concept of the exclusive role of endothelial cells in the relaxation of VSMC.

#### P12.05

##### IMPACT OF IGF-1 ON ARTERIAL STIFFNESS IN PATIENTS WITH ACROMEGALY: COMPARISON OF MEASURES OF APWV AND AASI

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**Introduction:** Acromegaly, caused by excess of growth hormone, has high cardiovascular mortality and morbidity. Aortic pulse wave velocity (aPWV) and Ambulatory arterial stiffness index (AASI) are known measures of arterial stiffness. We evaluated the factors influencing aPWV and AASI in patients with acromegaly.

**Method:** Patients with acromegaly at various stages in their disease were assessed for disease activity by IGF-1, presence of hypopituitarism, other co-morbidities, aPWV and measurement of 24 hour ambulatory blood pressure and thereby AASI.

**Results:** 55 patients (mean age:  $52.8 \pm 14.0$  years, aPWV:  $11.7 \pm 3.6$  m/sec, AASI:  $0.31 \pm 0.27$ ), 32.7% female, took part in the study. 36.4% had hypopituitarism. Their blood pressure was  $126.0 \pm 16.7 / 78.1 \pm 11.8$  mm Hg and 45.5% were dippers. The IGF-1 was in the age related normal range in 54.5% of patients. There were no significant differences in aPWV or AASI between patients with high or normal IGF-1. Multivariate analysis including pulse pressure, heart rate, gender, presence of hypopituitarism, nocturnal dipping of blood pressure, proteinuria, cigarette smoke exposure, diabetes and hypertension, identified Age ( $\beta$ : 0.25, 95% CI of  $\beta$  0.11 to 0.38,  $p = 0.002$ ) and hyperlipidaemia ( $-4.9$ ,  $-9.1$  to  $-0.7$ , 0.026) independently influenced aPWV, while IGF-1 ( $\beta$ : 0.001, 95% CI of  $\beta$ : 0.0 to 0.002,  $p = 0.042$ ) independently influenced AASI.

**Conclusion:** In patients with acromegaly, IGF-1 independently influences AASI but not aPWV which is affected by traditional cardiovascular risk factors, namely age and hyperlipidaemia. These findings suggest that IGF-1, by its effects on vascular smooth muscle cells, affects the stiffness of muscular arteries but not that of the elastic arteries.

#### P12.06

##### DOES A 6-MONTH MILITARY MISSION IN AFGHANISTAN HAVE AN IMPACT ON INFLAMMATION MARKERS, VITAMIN D LEVEL, AND ARTERIAL STIFFNESS?

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**Background:** Excessive workload may have transient detrimental effects on left ventricular function and arterial stiffness. Furthermore, heavy endurance physical exercise has been shown to induce short-term systemic inflammation response. Recently, vitamin D has attracted increased attention owing to its potential anti-inflammatory effects. The aim of this study was to investigate the impact of a 6-month military mission on arterial stiffness, inflammation, and vitamin D level.

**Methods:** Sixty-five soldiers (age  $26 \pm 4$  years) deployed to a peacekeeping mission in Afghanistan for 6 months were examined before and after the mission. We assessed arterial stiffness by carotid-femoral pulse wave velocity and pulse wave analysis using the Sphygmocor device. A set of inflammation-related markers was assessed in the blood using biochips. Serum 25-hydroxyvitamin D level was measured using a radioimmune assay.

**Results:** Arterial stiffness and brachial and central blood pressure did not differ significantly before and after the mission. Vitamin D level increased by 2.6 times ( $40 \pm 15$  vs  $104 \pm 24$  nmol/L,  $p < 0.001$ ). Significant increases were observed in high-sensitivity C-reactive protein ( $0.68 \pm 0.7$  vs  $1.47 \pm 3.55$  mg/L,  $p = 0.03$ ), leukocyte count  $5.4 \pm 1.1$  vs  $6.3 \pm 1$  ( $\times 10^9$ /L,  $p < 0.001$ ), as well as in various pro-inflammatory cytokines, including IL-1 $\alpha$  ( $0.13 \pm 0.18$  vs  $0.2 \pm 0.23$  pg/mL,  $p < 0.001$ ), IFN- $\gamma$  ( $2.6 \pm 2.4$  vs  $5.3 \pm 3.4$  pg/mL,  $p < 0.001$ ), and MCP-1 ( $151 \pm 61$  vs  $229 \pm 95$  pg/mL,  $p < 0.001$ ).

**Conclusion:** Arterial stiffness was not altered by arduous conditions during the deployment. However, there were significant changes in the spectrum of inflammation markers and vitamin D levels. We speculate that elevated vitamin D levels may have ameliorated the possible inflammation-induced changes in arterial stiffness.

#### P12.07

##### LACK OF RELATION BETWEEN ENDOTHELIAL FUNCTION AND CAROTID ARTERY STIFFNESS IN YOUNG, HEALTHY MALE SUBJECTS

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The endothelium was shown to reduce vascular stiffness of muscular arteries by producing vascular smooth muscle relaxing, vasodilative factors. Stiffening of arteries with advancing age and risk factor exposure predominantly involves the elastic segments of the arterial tree. It is not known to what extent the stiffness of large elastic arteries is under endothelial control. This study was designed to investigate the relationship between endothelial function and stiffness of the carotid artery, a representative of central elastic arteries.

Conduit artery endothelial function was assessed in 58 subjects by measuring brachial artery flow mediated dilatation (FMD). Carotid artery elastic parameters were calculated from carotid pulse pressure measured by local tonometry and from pulsatile distension determined by echo wall-tracking. Systemic arterial stiffness was assessed by aorto-femoral pulse wave velocity (PWV). Relations between variables were determined by univariate correlation analysis.

All measured values fell within age related normal ranges. FMD was inversely related to age and DBP ( $r = -0.49$  and  $-0.48$ , respectively;  $p < 0.01$  for both). FMD was also significantly and inversely related to PWV ( $r = -0.46$ ;  $p < 0.05$ ), but was not related to any parameter of carotid artery elasticity.

We suggest that age-related impairment in large elastic vessel function may not be significantly influenced by the loss of vasodilative capacity of the endothelium.