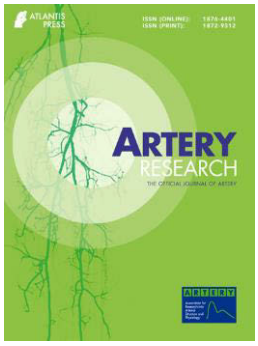

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P2.36: INTERRELATIONSHIPS OF URIC ACID LEVELS, ARTERIAL STIFFNESS, PERIPHERAL AND CENTRAL PRESSURES IN HEALTHY, NORMOTENSIVE INDIVIDUALS

P. Xaplanteris, C. Vlachopoulos, I. Dima, N. Ioakeimidis, K. Baou, C. Stefanadis

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from the changes in neurohormonal regulatory systems, kidney function and cardiovascular system, such as the decreased overall systemic vascular resistance and the reduced arterial blood pressure.

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P2.35
ROLE OF SYMPATHETIC ACTIVATION ON BRACHIAL ARTERY ENDOTHELIAL FUNCTION DURING HYPERINSULINEMIA IN HEALTHY SUBJECTS

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Aim: Hyperinsulinemia worsens brachial artery endothelial function in healthy subjects, while in vitro and in vivo (in other vascular districts) evidence show that insulin facilitates nitric oxide release and endothelium-dependent dilatation. We evaluated role of sympathetic activation during hyperisulinemia on brachial artery endothelial function.
Methods: In 20 healthy male volunteers (age: 27±5 yrs), endothelium-dependent (flow-mediated dilation, FMD) and -independent (sublingual 25 μg glyceryl trinitrate, GTN) dilation were evaluated by ultrasound and computerized analysis of brachial artery diameter. Measures were taken at -60, -10, 120 and 240 minutes during euglycemic hyperinsulinemic clamp (insulin infusion at 0.25 mU.min-1.kg-1 and 20% glucose at variable rates), in absence (n=10) or presence (n=5) of infusion of clonidine (0.0052 μg.min-1.kg-1).
Results: Insulin infusion raised plasma concentrations from 63±4 to 210±22 pmol/l, without changes in blood pressure or heart rate. Insulin raised plasma noradrenaline (from 260±40 to 333±62 pg/ml, $p<0.05$). This increase was not observed in the presence of clonidine infusion. No change in FMD was observed during insulin infusion (from 7.2+-0.7 to 7.2±0.5%), while response to GTN was decreased (from 9.1±1.0 to 6.8±0.8%; $p<0.05$). Infusion of clonidine alone did not modify blood pressure, heart rate, FMD and response to GTN. During insulin clamp in the presence of clonidine infusion, FMD did not change (from 7.4±1.8 to 6.9 ±2.9%, p=n.s.), while response to GTN was increased (from 9.4±1.0 to 12.2±0.8, $p<0.05$).
Conclusions: In healthy subjects, a modest 4-hour hyperinsulinemia does not alter brachial artery endothelial function, but impairs endothelium-independent response. This effects disappears blocking sympathetic nervous system.

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P2.36
INTERRELATIONSHIPS OF URIC ACID LEVELS, ARTERIAL STIFFNESS, PERIPHERAL AND CENTRAL PRESSURES IN HEALTHY, NORMOTENSIVE INDIVIDUALS

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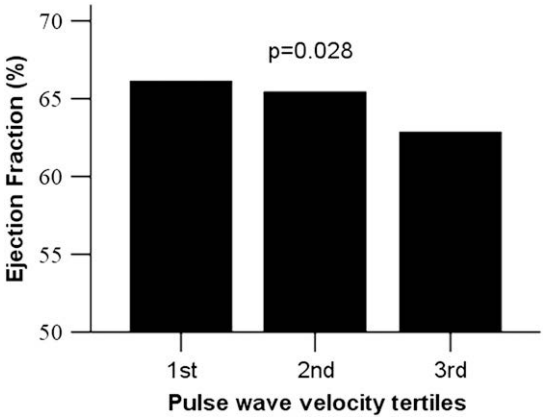
Purpose: Uric acid (UA) has been associated with cardiovascular disease, hypertension and endothelial dysfunction. The relationship between UA and arterial stiffness, peripheral/central pressures in normotensive individuals has not been addressed.
Methods: The study included 120 normotensive individuals (79 males, mean age 40.9 years). UA levels were determined from blood samples; peripheral pressures were measured by an electronic sphygmomanometer; aortic pressures were measured using a validated device, while carotid-femoral pulse wave velocity (PWV) was measured as an index of aortic stiffness. The distribution of serum uric acid (UA) was split by the median (4.5 mg/dL) and subjects were divided in low (n=59) and high (n=61) UA group. Comparisons were performed using the independent samples t-test.
Results: UA levels were positively correlated with peripheral systolic (pSP, $r=0.373$, $p<0.001$) and diastolic (pDP, $r=0.362$, $p<0.001$) blood pressures, as well as central systolic (cSP, $r=0.296$, $p<0.001$), and diastolic (cDP, $r=0.359$, $p<0.001$) pressures. When compared to low UA subjects, high UA subjects demonstrated significantly higher levels of pSP (110.9±12.3 vs 118.5±8.7 mmHg, $p<0.001$), pDP (66.5±11 vs 74.05±7 mmHg, $p<0.001$), cSP (100.6±12.4 vs 107.2±8.9 mmHg, $p=0.001$) and cDP(67.6±10.9 vs 75.1±7.3 mmHg, $p<0.001$). As regards to PWV, it was positively correlated with UA levels ($r=0.242$, $p<0.01$), with significantly higher levels observed in the high UA group (6.03±1.06 vs 6.55±1.18 m/sec, $p=0.01$).
Conclusion: Increased levels of UA are associated with higher levels of peripheral/central pressures and herald arterial stiffening, as estimated by PWV, even in healthy, normotensive individuals. Our findings further elucidate the interplay of UA and arterial function.



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P2.37
HUMAN-SPECIFIC GRAVITATIONAL DAMAGE OF VASCULAR SYSTEM

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Objectives: To present a concept of human-specific gravitational damage of vascular system.
Methods: Application of Newton theory of gravitation to Guytońs cardio-vascular(CV) physiology supported by analysis of published research.
Results: In upright posture gravitation creates significant gradient of gravitational potential(GP) across human body. This gradient moves blood downward within CV system. CV system must actively respond to emptying of the upper body in upright posture. Guytońs CV physiology with passively filling heart determines two basic ways to prevent gravitation-induced downward blood shift: 1)low body vasoconstriction squeezing blood to the upper parts — well demonstrated in tilt studies by powerful increase of peripheral vascular resistance during head up tilt(precisely, feet-to-head gradient of GP requires exactly opposite head-to-feet gradient of additional vasoconstriction), 2)water retention to indirectly increase intravascular volume in the upper body — shown in space crews during postflight adaptation. The price is significant elevation of intravascular pressure and mechanical stress on vascular walls. This stress, however, is naturally prevented during walk when activated calf muscle pumps effectively return blood into upper body. From this analysis modern lifestyle with reduced walking and prolonged high upright sitting causes excessive gravitation-induced mechanical stress in vascular system. Mechanical wall stress has been widely shown to promote atherosclerosis in large arteries and hypertrophy/remodeling in small arteries while in severe cases also may cause wall rupture/dissection.
Conclusion: Gravitation may seriously damage human vascular system in modern sitting lifestyle.

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P2.38
THE ANRIL LOCUS ON CHROMOSOME 9P21 AFFECTS STIFFNESS OF THE ABDOMINAL AORTA

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Coronary artery disease (CAD) is the leading cause of death worldwide. Recently, several genome wide association studies have reported associations between a region on chromosome 9p21 and a broad range of arterial diseases, including CAD and intracranial aneurysms. However, no clear associations with intermediate phenotypes have been described. In order to investigate the possible influence of the CAD-associated SNPs on arterial wall integrity, we analyzed associations between SNPs and stiffness of the abdominal aorta.
400 subjects, 212 men and 188 women (70-88 years) were studied. The pulsatile diameter of the abdominal aorta was examined at the midpoint between the renal arteries and the bifurcation, using a wall track system. Blood pressure was taken from the brachial artery (Dinamap). Two CAD- and aneurysm-associated SNPs (rs10757274 and rs2891168) and one T2D-