P4.61: ASSOCIATION BETWEEN ENDOTHELIUM-DEPENDENT VASODILATATION AND SERUM PULMONARY SURFACTANT PROTEIN D CONCENTRATION IN PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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T allele is low, further study with sufficient number of subjects is warranted.

* Salvi E et al., Hypertension 2012; 59: 248.

P4.60 DEFECT VENOUS WALL PROPERTIES AS WELL AS ARTERIOLAR REGULATION IN PATIENTS WITH VASOVAGAL SYNCOPE

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The initiating trigger in vasovagal syncope (VVS) is unclear but impaired venous return has been considered as a central factor. A greater leg venous compliance (Vc) may augment the central hypovolemia during standing. The aim of the study was to evaluate Vc in VVS patients and controls. 15 VVS females (24.8 ± 4.9 years) and 15 age-matched female controls (22.9 ± 3.2 years) were studied. Venous occlusion plethysmography was used to measure calf volume changes. A thigh cuff was inflated to 60 mmHg for 8 min with a subsequent linear decrease of 1 mmHg/s in cuff pressure (P). Vc was determined using the first derivative of a quadratic regression equation describing the volume-pressure relationship [Compliance = β1+2β2(P)]. The capillary filtration was subtracted from the volume curve to correct for the effect on Vc.

Vc was reduced in VVS females (P < 0.05). No differences were found in the venous capacitance response or capillary filtration. Resting arterial blood flow was lower and the peripheral resistance higher in VVS females (P < 0.05). The time for 50% of the capacitance response to be developed was increased in VVS females (P < 0.05), and the rate of the capacitance response correlated to the reduced arterial inflow of blood (r = -0.64, P < 0.01).

The study shows defects both in venous wall properties and arteriolar regulation in patients with VVS. A reduced blood flow to the lower limb with a concomitant reduction in filling rate of the capacitance vessels during standing might change the central baroreceptor response with a defect hemodynamic adjustment as a consequence.

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Pulmonary surfactant protein D (SP-D) is considered as a candidate biomarker for the functional integrity of the lung and for disease progression, which can be detected in serum. SP-D may function to modulate inflammation and host defense in the vasculature, which may impact the development or progression of cardiovascular disease. Circulating SP-D is supposed to be a predictor of cardiovascular morbidity and mortality and adding prognostic information to well-established risk factors.

Aims: measurement and assessment of serum SP-D level and endothelium-dependent vasodilatation in patients with chronic obstructive pulmonary disease (COPD).

Methods and Results: in 39 patients with II and III stage of COPD we have measured serum SP-D level and endothelium-dependent vasodilatation during reactive hyperemia test. Comparing II to III stage more impaired endothelium-dependent vasodilatation during reactive hyperemia test and reduced increase in brachial artery diameter in III stage were registered (median D 26% vs. D 10.9%, correlation -0.71, p < 0.05). SP-D level also correlated with COPD stage (correlation 0.75, p = 0.01). However, we didn’t find reliable correlation between SP-D level and endothelium-dependent vasodilatation. At the same time endothelium-dependent vasodilatation has shown high correlation with serum pro-inflammatory and anti-inflammatory cytokines level, negative and positive, accordingly.

Conclusion: both worsening of endothelium function and SP-D concentration strongly correlates with stage of COPD. However, can suppose that endothelium dysfunction in COPD-patients isn’t associated with SP-D serum concentration, and SP-D can’t be assessed as the significant risk factor of endothelial dysfunction. This fact requires further investigation.

P4.62 PROGNOSTIC VALUE OF ARTERIAL STIFFNESS INDICES IN PATIENTS WITH ACUTE ISCHEMIC STROKE

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It is unclear whether arterial stiffness predicts the outcome of patients with acute ischemic stroke. We aimed to assess the prognostic value of arterial stiffness in this population. We studied 280 consecutive patients (37.5% males, age 78.8 ± 6.4 years) who were hospitalized in our Department for acute ischemic stroke between September 2010 and May 2012. Arterial stiffness was assessed by measuring the augmentation index (Alx), central systolic blood pressure (cSBP) and central pulse pressure (cPP) over the radial artery with the Sphygmocor device. The severity of stroke was assessed with the National Institute of Health stroke scale (NIHSS) score at admission and the outcome was assessed with the modified Rankin scale score at exit from the hospital. None of the indices of arterial stiffness correlated with NIHSS score at admission. Alx showed a negative correlation with the modified Rankin scale score at exit from the hospital (r = -0.200, p < 0.05). cSBP and cPP correlated with the number of days of hospitalization (r = 0.180, p < 0.05 and r = 0.225, p < 0.05, respectively). Twenty-five patients (8.9%) died during hospitalization. These patients had lower Alx than patients who were discharged (18.2 ± 11.3 vs. 29.9 ± 9.8, respectively; p < 0.005). Other indices of aortic stiffness did not differ between patients who died during hospitalization and those who were discharged. In conclusion, a higher Alx was associated with better functional outcome and lower mortality rate in patients with acute ischemic stroke. Competing causes of death and the relatively beneficial effect of elevated BP during the acute phase of stroke might partly explain this apparently paradoxical finding.