assessed while the plasma level of VPO1 in patients and the expression of VPO1 in arterial tissues was measured. Cultured human aorta vascular smooth muscle cells were treated with ANGII, and the proliferation activity, VPO1 expression, H2O2 and HOCL level were examined. The effect of VPO1 RNA interference, apocynin, catalase and PD98059 on VPO1 expression and the proliferation activity of cells were observed.

Results The VPO1 level/expression was significantly increased in patients with essential hypertension and in spontaneously hypertensive rats concomitant with definite vascular remodeling by evaluating the intima-media thickness, pressure-strain elastic modulus and stiffness index of carotid artery in patients, as well as the media thickness, lumen diameter, media thickness/lumen diameter ratio and mean nuclear area in artery media in spontaneously hypertensive rats. The angiotension II-stimulated cell proliferation of human aorta smooth muscle cells was inhibited by knockdown of VPO1 using small hairpin RNA. Moreover, the NADPH oxidase inhibitor, apocynin, the hydrogen peroxide scavenger, catalase, but not the ERK1/2 inhibitor, PD98059 attenuated Ang II-mediated upregulation of VPO1 and generation of hypochlorous acid.

Conclusions VPO1 is a novel regulator of vascular smooth muscle cell proliferation via NADPH oxidase/H2O2/VPO1/ERK1/2 pathways and plays an important role in vascular remodeling during hypertension.

AngiotensinII Modulates Ion Pumps of Smooth Muscle Cells Derived from Umbilical Artery of Human Neonates with Hypertensive Family History
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Objective To investigate sodium pump and calcium pump activities and mRNA expression level and the changes after AngiotensinII (AngII) treatment in human umbilical artery smooth muscle cells (HUASMCs) isolated from neonates with positive hypertensive family history (FH+) or with negative hypertensive family history (FH).

Methods Ion pump activities in cultured HUASMCs were detected by spectrophotography. The mRNA expression of sodium pump α1-subunit and plasma membrane Ca2+-ATPase isoform 1 (PMCA1) in FH+ and FH- HUASMCs was measured by RT-PCR.

Results Sodium pump, calcium pump activities in FH+ HUASMCs were higher than those in FH- group (p < 0.05), but the mRNA expression of sodium pump α1-subunit and PMCA1 showed no difference between two groups. In FH+ group, after 24-h treatment, AngII (1×10−7 mol/L) elevated the activities of sodium pump (4.62±0.26 vs 3.52±0.33) and calcium pump (4.00±0.31 vs 3.01±0.52), and up-regulated sodium pump α1-subunit mRNA expression (0.946±0.099 vs 0.697±0.050, n=5, p=0.01), however higher concentration AngII (1×10−6 mol/L) suppressed the activities of sodium (2.47±0.27) and calcium pump (1.79±0.27), and down-regulated sodium pump mRNA expression (0.445±0.065). Whereas, in FH- groups, both concentration (1×10−6 and 10−7 mol/L) of AngII suppressed the activities of sodium pump (3.49±0.34, 2.21±0.23 vs 4.70±0.44) and calcium pump (2.85±0.31, 1.87±0.16 vs 4.27±0.48), but only AngII (10−7 mol/L) down-regulated their mRNA expression (α1-subunit: 0.515±0.133 vs 0.335±0.097, PMCA1: 0.165±0.049 vs 0.397±0.046, n=5, p<0.01).

Conclusions The activity of sodium pump and calcium pump is increased in FH+ HUASMCs. AngII inhibits both Na+ and Ca2+ ion pumps activities and mRNA expression in FH+ HUASMCs, and may have biphasic effects on ion pump activities and mRNA expression in FH+ human arterial smooth muscle cells.

Serum HDL-C Levels Correlated with the Haemodynamic and Severity in Patients with Idiopathic Pulmonary Arterial Hypertension
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Aims Although dyslipidemia was an established risk factor for cardiovascular disease, its role in the pathogenesis of pulmonary arterial hypertension (PAH) is still unclear. The aims of the current study were to elucidate the clinical significance of the serum dyslipidemia levels in patients with idiopathic PAH (IPAH).

Methods Serum dyslipidemia levels (total-cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C)) and other clinical datas collected from 90 consecutive adult patients with IPAH from April 2008 to Dec 2009 were retrospectively evaluated in our center. Right heart catheterisation was performed in all patients. Forty-five age and sex-matched healthy volunteers served as control subjects.

Results The levels of serum TC and HDL-C was significantly decreased in patients with IPAH compared with control subjects (3.77±0.86 vs 4.25±0.75 mmol/L, p<0.05; 1.04±0.31 vs 1.46±0.31 mmol/L, p<0.01 respectively). Serum LDL-C levels decreased in proportion to the severity of WHO function. Compared with the high HDL-C group, the low HDL-C group demonstrated significantly lower in 6 min walk distance (6MWD), cardiac output...
(CO), mixed venous saturation (SvO2) and PaCO2 whereas significantly higher in pulmonary vascular resistance (PVR) and serum uric acid (UA) levels. Serum HDL-C levels positively correlated with 6MWD (r=0.34, p<0.001), CO (r=0.35, p<0.001), SvO2 (r=0.40, p<0.001) and PaCO2 (r=0.289, p<0.05); negatively correlated with UA levels (r=-0.45, p<0.001) and PVR (r=-0.30, p<0.05).

Conclusion Serum HDL-C levels correlated with the clinical severity of PAH and maybe serve as a novel risk factor for the malignant disease.

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**THE RESEARCH OF THE RELATION BETWEEN THE REFRACTORY HYPERTENSION AND THE HOMOCYSТЕINE**

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**Objective** To explore the relation between the refractory hypertension and the homocysteine (HCY).

**Methods** 164 patients with hypertension were randomly divided into refractory hypertension group (n=76) and non-refractory hypertension group (n=88), and meanwhile 60 healthy persons were regarded as the control group. We checked their homocysteine valuation and had the statistics analysis.

**Results** The homocysteine valuation of refractory hypertension group was significantly higher than those of the control group (p<0.01) and was also higher than those of non-refractory hypertension group (p<0.05). On the other hand. The homocysteine valuation of non-refractory hypertension group was higher than those of the control group (p<0.05).

**Conclusion** The blood homocysteine may be considered a relationship factor with refractory hypertension.

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**THE IMPACT OF VALSARTAN ON ARTERIAL STIFFNESS IN PATIENTS WITH CORONARY HEART DISEASE AND HYPERTENSION**

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**Objective** To examine whether the angiotensin II receptor blockers valsartan would improve arterial stiffness to a greater extent than an blocker amlodipine in patients with coronary heart disease and hypertension.

**Methods** Combined with hypertension.

**Results** Systolic blood pressure, diastolic pressures were significantly decrease in both groups (p<0.05). Systolic blood pressure, diastolic pressures were signifiantly higher in severe OSA group, whereas no signifiantly differences were found when compare day-time systolic and diastolic pressures between groups. Moreover, night-time diastolic pressure was significantly higher in severe OSA group than mild OSA group (p value is 0.046 and 0.024) in severe OSA group. Whereas no significant differences were found when compare day-time systolic and diastolic pressures between groups. Moreover, night-time diastolic pressure was significantly higher in severe OSA group than mild OSA group (p value is 0.039). After adjusting the confounders including age, sex, BMI, smoking and drinking history, and cardiovascular diseases, the statistic differences still remained. However, Office blood pressure including systolic and diastolic blood pressure had no significant differences between each two groups.

**Conclusion** Severe OSA significantly increases blood pressures, especially night-time blood pressures, of hypertensive patients who receive the optimal medication for hypertension. 24 h ABPM is more accurate than office pressure to evaluate the blood pressure of hypertensive patients with OSA.

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**EFFECTS OF BMI ON ISCHAEMIC STROKE IN HOSPITAL PATIENTS WITH ESSENTIAL HYPERTENSION**

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**Objective** To investigate the association between BMI and the incidence of ischaemic stroke in essential hypertensive (EH) patients.