(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 IPAH and maybe serve as a novel risk factor for the malignant disease.

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

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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.

**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 equivalent antihypertensive medication, the calcium channel blocker amlodipine in patients with coronary heart disease combined with hypertension.

**Methods** 75 patients with coronary heart disease combined with hypertension in all accepted brachial-ankle pulse wave velocity (ba-PWV) and central aortic blood pressure measurement. They were administered to take valsartan in a dose of 160mg per day (valsartan group, n = 35) or amlodipine 5–10 mg per day (amlodipine group, n = 40) respectively soon after to ensure equivalent BP control. Measurements of ba-PWV and central aortic BP were carried out again after 24 weeks.

**Results** After 24 weeks there were no statistical differences in coronary artery Gensini score, left ventricular ejection fraction, mean heart rate and types of therapy medication between valsartan group and amlodipine group (p > 0.05). Systolic blood pressure, diastolic blood pressure and pulse blood pressure of brachial artery as well as central artery between baseline level and end of the study had no statistical differences between two groups (p > 0.05), and had significant decrease in both groups (p < 0.01). The levels of central artery BP and brachial BP controlled by valsartan were similar to amlodipine. There were no significant differences in ba-PWV between two groups at baseline (12.6 ± 2.6 vs 21.1 ± 2.6, p > 0.05), however a significant decrease were observed in valsartan group after 24 weeks (12.6 ± 2.6 vs 10.8 ± 1.9, p < 0.01), while no significant changes appeared in amlodipine group (12.1 ± 2.6 vs 11.4 ± 2.7, p > 0.05).

**Conclusion** Valsartan may improve arterial stiffness to a significantly greater extent than amlodipine despite similar central artery and brachial BP control.

**IMPACTS OF OBSTRUCTIVE SLEEP APNOEA ON THE BLOOD PRESSURE IN HYPERTENSIVE PATIENTS UNDER THE OPTIMAL MEDICATION**

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**Background and Objective** Obstructive sleep apnoea (OSA) is a prevalent disease, however only 10% OSA patients receive regular treatment. OSA is an independent risk factor for hypertension and cardiovascular diseases. We aim to investigate the impacts of obstructive sleep apnoea on the blood pressure in hypertensive patients under the optimal medication though office blood pressure and 24 h ambulatory blood pressure monitoring (24 h ABPM), respectively.

**Methods** 52 patients with hypertension were enrolled consecutively and all received the optimal medication for hypertension. An overnight polysomnography and a 24 h ABPM were performed to each patient. According to the apnoea-hypopnoea index, the patients were divided into four groups: no OSA group (AHI < 5, n = 13), mild OSA group (5 ≤ AHI < 15, n = 19), moderate OSA group (15 ≤ AHI < 30, n = 11), severe OSA group (AHI ≥ 30, n = 9). The results of 24 h ABPM and office pressure were compared respectively.

**Results** As to the 24 h ABPM results, 24 h systolic and diastolic pressures were significantly higher in severe OSA group than no OSA group (p value is 0.036 and 0.022), and night-time systolic and diastolic pressures were significantly higher too (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.

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

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