hospital were recruited. The patients were divided into three groups according to their risk factors. The patients without hypertension and diabetes were included into Group A (n=95). The patients with high blood pressure or diabetes were included into group B (n=221) and the patients with both of high blood pressure and diabetes were included into group C (n=72). Coronary artery calcification score, lipid profiles (lipoprotein (a), LDL, HDL, TG, TC) and coronary angiography were determined in each group.

Results Among the 3 groups, there is no significant difference between sex, drinking history, smoking history; there is significant difference between age and the incidence of coronary heart disease among the 3 groups (age $F=5.737$, $p=0.005$; coronary heart disease $F=6.283$, $p=0.002$). Coronary artery calcification score is significantly higher in group C than that of groups A (groups C 256.9 ± 430.199 VS group A 103.74 ± 299.85 , $p=0.011$). Coronary artery calcification score was positively correlated with lipoprotein (a) ($p=0.005$), age ($p=0.021$) in group A. Coronary artery calcification score was positively correlated with low-density lipoprotein ($p=0.018$), age ($p=0.000$) in group B. There is no significantly correlation between coronary artery calcification score and lipid profiles in group C. Summary analysis coronary artery calcification score was positively correlated with LP (a) ($p=0.013$), low-density lipoprotein ($p=0.021$), age ($p=0.000$).

Conclusion In the low-risk coronary heart disease group, lipoprotein (a) is positively correlated with coronary calcification score, which suggests lipoprotein (a) is an independent risk factor for coronary artery calcification for these apparently low risk patients. The present study may contribute to the early diagnosis and intervention of coronary artery disease for those patients.

e0346 SCREENING OF SLEEP APNOEA-HYPOPNOEA SYNDROME FROM ECG DERIVED RESPIRATION OF AMBULATORY ECG

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Objective To evaluate the feasibility of screening sleep apnoea-hypopnoea syndrome (SAHS) from ECG-derived respiration (EDR) of ambulatory ECG (AECG) monitoring.

Methods The overnight sleep investigation was administered to 80 subjects by polysomnogram (PSG) and 24 h AECG monitoring simultaneously during February through November, 2004. The ECG analysers did not know the PSG results at all. They were both asked to give the apnoea hypopnoea index (AHI) by EDR and PSG respectively. The PSG result was considered as the gold standard so as to evaluate the feasibility of screening SAHS from EDR of AECG monitoring.

Results The average age, male gender, body mass index, history of hypertension were higher in the SAHS (+) patients than those of the SAHS (-) patients. Automatic analysis was performed with software in a sensibility of 75%, 87.5% and 100% respectively. When software sensibility adjusted to 75%, the sensitivity of screening SAHS with EDR was 26.7%, with the specificity of 80%, the positive predictive value of 80%, the negative predictive value of 26.7%, the diagnose accordance rate of 40%. When software sensibility was adjusted to 87.5%, the sensitivity of screening SAHS with EDR was 55%, with the specificity of 45%, the positive predictive value of 75%, the negative predictive value of 25%, and the diagnose accordance rate of 52.5%. When software sensibility was adjusted to 100%, the sensitivity of screening SAHS with EDR was 88.3%, with the specificity of 35%, the positive predictive value of 84.1%, the negative predictive value of 50%, and the diagnose accordance rate of 75%.

Conclusion EDR technique of AECG was useful to screen the suspicious SAHS patients, sensitivity and the diagnosis coincidence rate was higher when the sensibility of automatic analysis software was adjusted to 100%.

e0347 THE CHANGES OF HEART RATE TURBULENCE (HRT) IN SLEEP APNOEA-HYPOPNOEA SYNDROME (SAHS)

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Objective We investigated the changes of heart rate turbulence (HRT) in sleep apnoea-hypopnoea syndrome (SAHS).

Methods 75 patients underwent overnight polysomnography for clinically suspected SAHS and simultaneous Holter monitoring (23:00–6:00). According to the apnoea-hypopnoea index (AHI), the patients were assigned to group SAHS (+) (AHI ≥ 5 , $n=52$) or group SAHS (-) (AHI < 5 , $n=23$). HRT (onset, slope) of two groups were compared.

Results Turbulence slope (TS) of group SAHS (+) was significantly lower in group SAHS (-) ($p<0.01$), turbulence onset of two groups all smaller than zero, turbulence onset of group SAHS (+) were higher than group SAHS (-)'s, but no significant difference, the number of ventricular premature contractions of group SAHS (+) were more than group SAHS (-)'s, but also no significant difference.

Conclusions Heart rate turbulence phenomenon diminishes in sleep apnoea-hypopnoea syndrome patients, indicating damages in cardiac autonomic activity in SAHS, turbulence slope decreasing could be considered as prognosis index of SAHS.

e0348 THE EFFECTS OF VALSARTAN ON ANGIOTENSIN II TYPE 1 AND TYPE 2 RECEPTOR IN ISOLATED REPERFUSED ISCHAEMIC RAT HEARTS

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Object To determine the effects of Angiotensin II Type 1 receptor blockade valsartan on AT₁ and AT₂ receptor during ischaemia reperfusion.

Methods The hearts of 24 SD rats were isolated, linked to Langendorff perfusion apparatus, and randomly divided into 3 equal groups: control group, perfused with modified Krebs-Henseleit (K-H) buffer for 110 min; ischaemia/reperfusion (I/R) group, perfused with K-H buffer for 20 min, exposed to ischaemia for 30 min, and then reperfused with K-H buffer for 60 min; valsartan group, perfused with K-H buffer with valsartan for 20 min, exposed to ischaemia for 30 min, and then reperfused with K-H with valsartan for 60 min. The left ventricular (LV) function including maximal uprising velocity of left ventricular pressure (+dp/dt_{max}) and maximal decreasing velocity of left ventricular pressure (-dp/dt_{max}) were monitor. The coronary effluent were measured 20 min after the stabilisation of perfusion, and 20, 40, and 60 min after reperfusion. After the stop of reperfusion, the structure were observed using electron microscope. The AT₁ and AT₂ receptor mRNA express were examined by Northern blot. The AT₁ and AT₂ receptor protein expression were examined by Northern blot.

Results Compared with control, I/R impaired left ventricular systolic and diastolic function (+dp/dt_{max} 1892 ± 231 mm Hg.s⁻¹ vs 836 ± 223 mm Hg.s⁻¹; -dp/dt_{max} -1175 ± 223 mm Hg.s⁻¹ vs -613 ± 224 mm Hg.s⁻¹, all $p<0.01$), decreased coronary effluent (5.9 ± 0.8 ml.min⁻¹ vs 3.3 ± 0.5 ml.min⁻¹, $p<0.01$) damaged the