and investigate variations of lipid, glucose, and vascular endothelial growth factor (VEGF) levels myocardial perfusion images and cardiac function, as well as the possible mechanisms to improve myocardial perfusion.

Methods Total 102 cases were selected, 5 patients were lost. Finally 97 patients included, 77 males and 20 females, aged 56 ± 13 . Of whom, 30 cases suffered from acute myocardial infarction (AMI), 48 from acute coronary syndromes (ACS), and 19 from chronic stable angina pectoris. They were randomised into three groups, A ($n=33$), B ($n=31$), and C ($n=33$) without differences in the baseline level. Normal adults were control group D ($n=30$). Groups A and B were given by Acarbose 50 mg tid and 100 mg tid, respectively, Groups C and D were given by placebo, the treatment course lasted three months, and CHD patients of each group underwent the same basic treatments after PCI. Myocardial perfusion imaging and variations of blood lipid, IGT, VEGF levels, myocardial perfusion images and cardiac function were observed.

Results 1. The IGT had no statistically differences before treatments in A, B and C groups ($p>0.05$). The changes of FPG and OGTT 2 h PG levels had statistically differences after treatments in A and B groups ($p<0.05$), especially B group ($p<0.01$); The changes of HbA1c had no statistically significant differences after treatments in the four groups ($p>0.05$); The changes of lipid levels had no statistically differences ($p>0.05$) before and after treatments in A, B, C and D groups, respectively. 2. A, B, C groups had the significantly higher plasma VEGF levels (203 ± 89 ng/l vs 77 ± 52 ng/l, $p<0.01$) than D group before treatments. The changes of VEGF levels had statistically differences after treatments in A and B groups ($p<0.05$), especially B group ($p<0.01$). The changes of VEGF levels had no statistically differences before and after treatments in C and D groups ($p>0.05$). 3. The myocardial perfusion images and cardiac function had no statistically differences before treatments in A, B and C groups ($p>0.05$), The changes of myocardial perfusion images had statistically differences after treatments in A and B groups ($p<0.05$), especially B group ($p<0.01$); The changes of cardiac function had statistically differences after treatments in A and B groups ($p<0.05$), there is no statistically differences after treatments between A and B ($p>0.05$); The changes of myocardial perfusion images and cardiac function had no statistically differences in C group ($p>0.05$).

Conclusions Acarbose can regulate IGT, improve myocardial perfusion images and cardiac function. The mechanisms may include reducing VEGF levels, suppressing endothelial hyperplasia, and improving the microcirculation.

e0343 TRIPLE VERSUS DUAL ANTIPLATELET THERAPY IN PATIENTS WITH ACUTE CORONARY SYNDROME UNDERGOING PERCUTANEOUS CORONARY INTERVENTION

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Background Following percutaneous coronary intervention (PCI), clopidogrel in addition to aspirin therapy leads to greater protection from thrombotic complications than aspirin alone. Whether triple antiplatelet therapy is superior or similar to dual antiplatelet therapy in patients with acute coronary syndrome undergoing PCI in the era of drug-eluting stents remains unclear.

Objectives To evaluate the effect of triple antiplatelet vs dual antiplatelet therapy in patients with acute coronary syndrome after PCI.

Methods and Results We collected consecutive 1203 acute coronary syndrome patients undergoing drug-eluting stents implantation. They received either dual (aspirin plus clopidogrel; dual group; $n=682$) or triple (aspirin plus clopidogrel plus cilostazol; triple

group; $n=521$) antiplatelet therapy. The triple group received additional cilostazol at least for 1 month. Various major adverse cardiac events at 1 year were compared between these 2 groups. Compared with the dual group, the triple group had a similar incidence of major bleeding events but a significantly lower incidence of in-hospital mortality. Clinical outcomes at 1 year showed that the triple group had significantly lower incidences of cardiac death and total major adverse cardiac events than the dual group.

Conclusions Triple antiplatelet therapy seems to be superior to dual antiplatelet therapy in patients ACS undergoing PCI with drug-eluting stents.

e0344 THE MECHANISM RESEARCH OF FRP INHIBITS ENDOTHELIAL CELL APOPTOSIS

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Background Atherosclerosis is the most common cause of cardiovascular diseases in the world. Although the development of atherosclerosis appears to be the result of multiple maladaptive pathways, a particularly important factor in the pathogenesis of atherosclerosis is oxidised low density lipoprotein (ox-LDL), which contributes to endothelial damage. Data from our lab and others show that Follistatin related protein (FRP), which is expressed in the vasculature, has cardioprotective effects, suggesting that loss of FRP protection might play a role in the development of atherosclerosis.

Objective In the present study, we determined whether FRP overexpression protects against endothelial cell (EC) damage, an intermediate endpoint for atherosclerosis.

Methods We bred ApoE knockout (ApoE $(-/-)$) mice that were FRP+ transgenic (they overexpressed FRP). We compared them to control mice (their littermates). Human umbilical vein endothelial cells (HUVECs) were isolated and treated with ox-LDL and recombinant FRP. FRP-induced signal transduction and Bcl2 mRNA and protein stability were analysed.

Results After 16 weeks, ApoE $(-/-)$ FRP (+) mice had significantly fewer apoptotic endothelial cells than controls. In vitro experiments showed that the effect of FRP on EC apoptosis was mediated by upregulation of expression of the antiapoptotic protein Bcl2.

Conclusion FRP overexpression maintains EC viability by preventing apoptosis via Bcl2 upregulation. FRP may be a novel therapeutic target for the prevention and treatment of vascular EC injury and of atherosclerosis.

e0345 SERUM LIPOPROTEIN (A) IS POSITIVELY CORRELATED WITH CORONARY ARTERY CALCIFICATION IN LOW RISK CHINESE PATIENTS

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Background Prior studies indicated that lipoprotein (a) is an independent risk factor for coronary atherosclerosis, but the relationship of serum lipoprotein (a) and coronary artery calcification is still poorly understood in Chinese population.

Objective The present study is to investigate the human lipid profile of a single center (lipoprotein (a), other blood lipid levels) with the relationship of coronary artery calcification.

Method 388 patients suspected with coronary artery disease under coronary CT examinations from March 2007 to June 2009 in our

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