enzyme immunoassay. The method of immunofluorescence flow cytometry was performed for measuring CD11b/CD18 expression on leukocytes in all subjects. High sensitivity- Creactive protein (hs-CRP), WBC and PMN were also measured and analysed.

Results Plasma level of MPO in CHD group was much higher than that in controls [(332.05±167.56) pg/ml vs (277.81±142.68) pg/ml, p<0.05]. CD11b/CD18 level differed significantly between CHD group and control group [(53.7±24.1) vs (23.0±10.2), p<0.01]. The levels of hs-CRP and WBC were markedly increased in cases than those in controls (p<0.05–0.01). MPO levels correlated positively with CD11b/CD18 and WBC levels (r=0.539, p<0.01and r=0.3, p<0.05, respectively), but had no significant correlation with CRP, TC, TG, LDL, HDL, IMA, cTnI.

Conclusion In conclusion, the levels of MPO, CD11b/CD18, hs-CRP and WBC are elevated in patients with CHD. Inflammation may be one of important reasons for ACS occurrence and MPO, leukocytes and their CD11b/CD18 expressions and hs-CRP were involved in the occurrence of ACS together. MPO may be an inflammation marker independent of hs-CRP.

Acknowledgements This work was supported by a grant from the Nature Science Foundation of Fujian Province (2006J0326, X0650036).

e0113 EFFECTS OF CAPTOPRIL ON MYOCARDIAL ENERGY METABOLISM IN CHRONIC PRESSURE OVERLOAD RATS

doi:10.1136/hrt.2010.208967.113

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Objective To investigate the effect of Captopril on cardiac function and levels of energy-rich phosphates in pressure overload induced left ventricular hypertrophy rats.

Methods Totally, 120 SD rats were randomly divided into three groups: sham operation group (SH) (n=40), coarctation of abdominal aorta group (CAA) (n=40) and Captopril group (CAP) (n=40). Parameters of cardiac function, levels of energy-rich phosphates and morphological changes of the myocardial mitochondria were observed at the 6^{th} and 8^{th} week after this therapy.

Results 1. At 6^{th} week, in CAA group, the cardiac function parameters (LVMI and LVEDP) were increased and $\pm dp/dt_{max}$ was decreased, while ATP and ADP were decreased and AMP was increased (p<0.01). These changes were much obvious at 8^{th} week (p<0.01). 2. Compared with that of CAA group, the parameters of heart function and energy-rich phosphates (ATP, ADP, AMP, TAN) in CAP group were improved significantly (p<0.01) at the 6^{th} and 8^{th} week. 3. In CAP group, the parameters of heart function and energy-rich phosphates (ADP, AMP, TAN) were much better at 8^{th} week than that of 6^{th} week. 4. The morphological change of mitochondria was less in CAP group than that in CAA group.

Conclusion Captopril can significantly improve the myocardial energy metabolism in pressure overload rats and can protect the function of myocardial mitochondria.

e0114 EXPRESSION OF PREGNANCY-ASSOCIATION PLASMA PROTEIN A AND INDUCIBLE NITRIC OXIDE SYNTHASE IN THE WALL OF BALLOON INJURED AND EARLY ATHEROSCLEROTIC PORCINE CORONARY ARTERY

doi:10.1136/hrt.2010.208967.114

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Purpose To investigate the role of pregnancy-associated plasma protein A (PAPP-A), a novel marker of atherosclerotic plaque activity, in the progress of injured-restenosis and atherosclerosis, and the relationship between the expression of PAPP-A and inducible nitric oxide synthase (iNOS) in the wall of coronary artery.

Methods The balloon injury procedure was done in the coronary arteries of 5 male pigs (injury group), and the artery segments were harvested in 28d after balloon injury. The expression of PAPP-A and iNOS were detected in the wall of coronary arteries by the means of immunohistochemical study and reverse transcription-polymerase chain raction. Expression of PAPP-A and iNOS were also detected in coronary artery wall of four pigs fed a high-cholesterol atherogenic diet for 15 weeks (CHOL group).

Results A marked increase in PAPP-A-positive cell number of the injury group was seen compared with the CHOL group, both in medial smooth muscle cells (PAPP-A staining: 33.2±2.9 vs 5.5±2.8, p<0.05) and neointimal (intimal) cells (PAPP-A staining: 28.3±3.1 vs 3.8±2.4, p<0.05); while iNOS-positive cell number decrease, only in neointimal (intimal) cells (iNOS stain: 1.1±0.3 vs 18.4±4.2, p<0.01). The expression of PAPP-A mRNA was higher in the injury group, compared with the CHOL group (0.81±0.08 vs 0.54±0.13, p<0.05), but nearly no expression in "normal" control vessel segements (0.03±0.01); while iNOS mRNA was lower in the injury group (0.18±0.09 vs 0.62±0.13, p<0.05).

Conclusion PAPP-A plays role in the progress of early atherosclerotic lesions and restenostic lesions.

e0115 OVEREXPRESSION AND INHIBITION OF CAMK2D GENE IN PRIMARY MYOCARDIAL CELLS BY LENTIVIRUS

doi:10.1136/hrt.2010.208967.115

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Purpose It has been found that Camk2d (calcium/calmodulin dependent protein kinase II delta) is related to E-C couple and myocardial hypertrophy and heart failure in pathological states. Moreover, its function alteration may play some role in arrhythmia. To further investigate the mechanism of Camk2d in onset and development of arrhythmia, we have built a platform for next stage by overexpressing Camk2d in myocardial cells by lentivirus transduction and inhibiting it by RNAi. Method (1) Rat Camk2d ORF was cloned by PCR, ligated into lentivirus vector and then packaged into lentivirus particles. (2) 3 shRNA sequences against Camk2d were designed and cloned. The one with highest inference efficiency was then screened by westernblot following with calcium phosphate transfection on 293 cells. (3) The selected RNAi clone was packaged into lentivirus particles. (4) Cultured myocardial cells from neonatal rats were transducted with overexpression or RNAi lentivirus and harvested for analysing Camk2d level by Realtime-PCR and westernblot.

Result Myocardial cells transducted by overexpression lentivirus exhibited an over 5-time higher level of Camk2d than normal, while in RNAi transfected cells, expression of Camk2d decreased by around 50%.

Conclusion Lentivirus can efficiently transduct primary myocardial cells with exogenous genes to obtain cells with special gene up- or down-regulated.

e0116 THE CARDIOPROTECTIVE EFFECT OF ISCHAEMIC PRECONDITIONING AND THE EXPRESSION OF ADIPONECTIN IN RAT MYOCARDIAL ISCHAEMIC PRECONDITIONING MODEL

doi:10.1136/hrt.2010.208967.116

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Background A number of recent studies have reported the protective effect on ischaemic myocardial by ischaemic preconditioning (IPC)

and adiponectin. Method and Result IPC model and MI model was set up. The protective effect of IPC was studied by measuring infarction size with Masson's Trichrome staining. The expression of protein and mRNA of adiponectin was examined by immunohistochemistry and quantitative real time RT-PCR o h, 6 h, 12 h and 24 h after IPC. And the plasma levels of adiponectin at four time points after IPC was also detected by ELISA. IPC reduced infarct size compared with control MI model (20±2%LV area vs 31±3%LV area, $p<0.05$). The expression of adiponectin mRNA 6 h and 12 h after the IPC was 2.2 and 2.1 times greater than the sham group ($p<0.05$) and the expression of adiponectin protein was siginificantly higher than non-ischaemic area ($p<0.05$). Compared to the sham groups, the plasma level of adiponectin increased significantly 0 h, 6 h and 12 h after IPC (0 h:7.40±0.47 vs 10.90±1.74;6 h:8.18±1.41 vs 10.98±1.74; 12 h:6.97±1.02 vs 9.31±0.96, $p<0.05$).

Conclusion Late IPC reduced infarction size and improved the expression of adiponectin mRNA and protein in myocardium, and also improved the concentration of adiponectin in plasma, which indicates that the adiponecitn may play a role in the protective effect of IPC.

e0117 THE FOUNDATION RESEARCH OF RENAL DENERVATION IN THE TREATMENT OF HYPERTENSION IN CANINE

doi:10.1136/hrt.2010.208967.117

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Objective To explore relationship between sympathetic activity and mechanism of hypertension, observe the treatment of renal artery denervation for hypertension, to assess the validity and safety of the treatment.

Methods 18 mongrel dogs were divided into two groups, 10 dogs were made to neurogenic hypertension model through the compression of the demyelinated vagus nerve by carotid sheath vessels pulse, another eight as the control group, two groups were operated by renal arterial radiofrequency ablation with 10 w power, no more than 60°C, at least 2 min. Blood pressure and other parameters were monitored at preoperative and 1 week, 2 weeks, 4 weeks, 10 weeks post operation. Renin activity, angiotensin II, aldosterone, and creatinine were measured at the same time.

Results we succeed in establishing the canine neurogenic hypertension model and the blood pressure were substantially reduced after sympathetic denervation. After modelling, the systolic pressure increased from 155.7±21 mm Hg to 179.5±23 mm Hg, and the diastolic pressure increased from 117.4±18.9 mm Hg to 138.2±13.4 mm Hg, there was a significant difference ($p<0.01$). Blood pressure decreased significantly in both the control group and blank group after renal denervation at 2 weeks, 4 weeks, 10 weeks, especially after ablation 10 weeks the blood pressure decreased more obviously (systolic blood pressure 179.5±23 mm Hg vs 143.9±11.7 mm Hg, diastolic blood pressure 138.2±13.4 mm Hg vs 114.9±15.5 mm Hg, $p<0.001$). Renin activity (PRA), Angiotensin II (Ang II) and aldosterone (Ald) levels were decreased after ablation, the levels of PRA detected preoperative, 1 week, 2 weeks, 4 weeks and 10 weeks after ablation were 0.26±0.09 ng/ml/h, 2.2±1.44 ng/ml/h, 0.71±0.57 ng/ml/h, 0.49±0.35 ng/ml/h, 0.24±0.12 ng/ml/h, the levels of Ang II were 76.9±14.3 pg/ml, 120±25.2 pg/ml, 97.1±21.9 pg/ml, 76.5±13.7 pg/ml, 64.4±11.1 pg/ml, the levels of Ald were 1.8±1.27 ng/dl, 7.5±1.73 ng/dl, 6.6±3.34 ng/dl, 4.6±2.59 ng/dl, 3.3±1.61 ng/dl. But the levels of Cr were not changed too much, it shows no great difference (57±12.7 umol/l, 45±7.4 umol/l, 36±19.2 umol/l, 43±8.6 umol/l, 41±21.8 umol/l $p>0.05$).

Conclusion Sympathetic nerves accelerate the development and progression of hypertension, catheter-based renal denervation causes substantial and sustained blood pressure reduction, and it cause no injury on renal, If in the future it can be widely applied in the treatment of hypertension, it will have broad application prospects and huge social benefits.

e0118 THE MYELOPEROXIDASE INHIBITOR, AMINOBENZOIC ACID HYDRAZIDE, ALTERS NEUTROPHIL-ENDOTHELIAL CELL INTERACTION

doi:10.1136/hrt.2010.208967.118

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Objective Acute myocardial infarctions (AMI) are associated with vascular inflammation, including activation of neutrophils and their adherence to vascular endothelial cells via CD11b/CD18 binding to intercellular adhesion molecule (ICAM). Myeloperoxidase (MPO) is an inflammatory biomarker, can induce CD11b surface expression in polymorphonuclear neutrophils (PMNs), but its role in regulating adhesion is not well characterised. MPO's role in regulating adhesion was further investigated by comparing the effects of aminobenzoic acid hydrazide (ABAH), an inhibitor of MPO, antibodies specific for CD11b and vehicle control on PMN adhesion to endothelial cells.

Methods Human neutrophils were isolated from the peripheral blood of patients with AMI or healthy participants using Percoll density gradient centrifugation. The effects of ABAH and anti-CD11b antibodies on neutrophil adhesion to endothelial cell were measured using adhesion assays.

Results The adhesion rate was significantly higher for neutrophils isolated from AMI patients than healthy individuals ($p<0.001$). Neutrophil adhesion was reduced upon treatment with ABAH in a dose dependent manner. The adhesion rate was significantly reduced in neutrophils treated with 10 μM and 20 μM ABAH as compared to the untreated group. Treatment with anti-CD11b antibodies also significantly reduced neutrophil adhesion compared to the untreated control group ($p<0.001$).

Conclusions MPO might enhance the neutrophils adhesions to endothelial cells in AMI patients through the upregulation of CD11b expression in the suface of neutrophils. The interference of cell adhesion by ABAH may be mediated by reduced CD11b expression in neutrophils.

Acknowledgements This work was supported by grants from the Nature Science Foundation of Fujian Province (2006J0326, X0650036) and the Science and Technology Planning Project of Fujian Province (2005Q006).

e0119 CARDIOPROTECTIVE EFFECT OF PI3K/AKT PATHWAY IN ISCHAEMIC POSTCONDITIONING AGAINST ISCHAEMIA AND REPERFUSION INDUCED INJURY IN ISOLATED RAT HEART

doi:10.1136/hrt.2010.208967.119

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Objective To explore the cardioprotection effect of co-treatment with ischaemic postconditioning and the mechanism of PI3K/Akt signal pathway in ischaemia postconditioning.

Methods 32 healthy adult male Wistar rats were assigned randomly into ischaemia/reperfusion group (I/R), ischaemia postconditioning group (IPost), IPost+Wortmannin group (IPost+W), I/R+SB216763group (I/R+SB), each group has eight rats. Rats were used for Langendorff isolated heart perfusion. The hearts were subjected to global ischaemia for 30 min followed by 60 min reperfusion. The cardia injury was evaluated by the levels of lactate dehydrogenase (LDH) and Creatine kinase (CK) in the coronary effluent.