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 significantly 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±7.46;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 adiponectin may play a role in the protective effect of IPC.

**e0117** THE FOUNDATION RESEARCH OF RENAL DERENATION IN THE TREATMENT OF HYPERTENSION IN CANINE

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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℃, 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, 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±0.44 ng/ml/h, 0.71±0.57 ng/ml/h, 0.49±0.38 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, 64.2±11.1 pg/ml, the levels of Ald were 1.8±1.27 ng/dl, 7.5±2.73 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, 45±6.6 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

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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 (PMN), 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 surface of neutrophils. The interference of cell adhesion by ABAH may be mediated by reduced CD11b expression in neutrophils.

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

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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 (I/Pst), I/Pst+Wortmannin group (I/Pst+W), I/R+SB216765 group (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.
Ventricular haemodynamic parameters were also measured, include HR, LVSFP. Left ventricular myocardial was separated and cut to five slices. After experiment, the myocardium was used for myocardial infarction size evaluated with TTC stained. Immunohistochemical staining for Phosphorylation Akt and GSK-3β expression.

**Results** Ischemic postconditioning reduced LDH, CK and improved the haemodynamic parameters, and reduced myocardial infarction size (29.5% vs 47.3%). Phospho-Akt and phospho-GSK-3β expression increased markedly in IPost group. Wortmannin may reduced Phospho-Akt expression, and phospho-GSK-3β expression increased in I/R+SB group.

**Conclusion** Ischemic postconditioning may synergically protect myocardium in isolated rat heart. Wortmannin, an inhibitor of Akt, may weaken the cardioprotection effect of postconditioning. SB216763, as an inhibitor of GSK-3β, can simulate cardioprotection effect of postconditioning. Akt and GSK-3β play important role in the mechanism of signal pathway in ischaemia postconditioning.

**Objective** To elucidate the effects of postconditioning on ischaemia/reperfusion cardiac and the role of reperfusion injury salvage kinase pathway in type 2 diabetic rats.

**Methods** The type 2 diabetic rats were induced by the intravenous injection of streptozotocin and high caloric diet. 60 Wistar rats were divided into three groups randomly. Ischaemia-reperfusion in normal rats (A group), ischaemia postconditioning in normal rats (B group), ischaemia postconditioning in diabetic rats (C group). Rats were used for Langendorff isolated heart perfusion with 30 min of global ischaemia and 60 min of reperfusion, then the models of Ischaemia-reperfusion (A) were made. But to B and C, rat hearts were subjected to six cycles of 10 min of globe ischaemia and 10 min of reperfusion (A). The SB216763, as an inhibitor of GSK-3β, was used for Langendorff isolated heart perfusion with 30 min of globe ischaemia and 60 min of reperfusion, then the models of Ischaemia-reperfusion in diabetic rats (C) was established. 27 SD rats were randomly divided into four groups: Normal group (N, n=5); Disease group (D, n=6). Beginning of reperfusion after 30 min of MI; MI/R post-intervention group (T, n=8). Intraperitoneal injection of CT-1 C-terminal peptide (100 μg/kg) was performed after intraperitoneal injection of CT-1 C-terminal peptide (100 μg/kg) at same time of beginning of reperfusion after 30 min of MI; MI/R pre-intervention group (O, n=8). The serum CK activity and drive the cell apoptosis was measured.

**Results** At the end of 30 min of MI, the average survival time of the disease group of SD rats was 93.17 ± 24.7 min, that of MI/R pre-intervention group was 87.88 ± 18.3 min. The average survival time of MI/R post-intervention group was 155.5 ± 50.13 min, significantly longer than that of the disease and MI/R pre-(chronic) intervention group (p > 0.01). The serum CK activity and drive the cell apoptosis was measured.

**Conclusion** Ischemic postconditioning may significantly protect myocardium in isolated normal rat hearts. But in diabetic rats the mechanism of Ischaemic postconditioning has no effect, the mechanism of this phenomena maybe connected with gsk-3β in the condition of diabetic.