

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 0 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 ( $20 \pm 2\%$ LV area vs  $31 \pm 3\%$ 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:  $7.40 \pm 0.47$  vs  $10.90 \pm 1.74$ ; 6 h:  $8.18 \pm 1.41$  vs  $10.98 \pm 1.74$ ; 12 h:  $6.97 \pm 1.02$  vs  $9.31 \pm 0.96$ ,  $p < 0.05$ ).

**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 DENERVATION 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°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  $155.7 \pm 21$  mm Hg to  $179.5 \pm 23$  mm Hg, and the diastolic pressure increased from  $117.4 \pm 18.9$  mm Hg to  $138.2 \pm 13.4$  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  $179.5 \pm 23$  mm Hg vs  $143.9 \pm 11.7$  mm Hg, diastolic blood pressure  $138.2 \pm 13.4$  mm Hg vs  $114.9 \pm 15.5$  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  $0.26 \pm 0.09$  ng/ml/h,  $2.2 \pm 1.44$  ng/ml/h,  $0.71 \pm 0.57$  ng/ml/h,  $0.49 \pm 0.35$  ng/ml/h,  $0.24 \pm 0.12$  ng/ml/h, the levels of Ang II were  $76.9 \pm 14.3$  pg/ml,  $120 \pm 25.2$  pg/ml,  $97.1 \pm 21.9$  pg/ml,  $76.5 \pm 13.7$  pg/ml,  $64.4 \pm 11.1$  pg/ml, the levels of Ald were  $1.8 \pm 1.27$  ng/dl,  $7.5 \pm 1.73$  ng/dl,  $6.6 \pm 3.34$  ng/dl,  $4.6 \pm 2.59$  ng/dl,  $3.3 \pm 1.61$  ng/dl. But the levels of Cr were not changed too much, it shows no great difference ( $57 \pm 12.7$  umol/l,  $45 \pm 7.4$  umol/l,  $36 \pm 19.2$  umol/l,  $43 \pm 8.6$  umol/l,  $41 \pm 21.8$  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 (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  $\mu$ M and 20  $\mu$ M 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 (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.