Ventricular haemodynamic parameters were also measured, include HR, LVSF. Left ventricular myocardial was separated and cut to five slice. After experiment, the myocardial 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, S8216763, as a 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.

**e0120** **THE IMPACT OF DIABETES ON THE ROLE OF REPERFUSION INJURY SALVAGE KINASE PATHWAY**

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**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 globe 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 as ischaemia postconditioning during the early minutes of reperfusion. Phosphorylation of akt and gsk-3β were analysed by western blotting and immunohistochemical staining.

**Results** phospho-akt and phospho-gsk-3β expression increased markedly in B group. But compared A group, there were no parently deference in C group. phospho-akt and phospho-gsk-3β expression in C group is more less than in B group.

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

**e0121** **THE EFFECTS OF ENDOTHELIN-1 AND BQ-123 ON ATPASE ACTIVITY AND MRNA EXPRESSION IN AORTIC SMOOTH MUSCLE CELLS FROM SPONTANEOUSLY HYPTERTENSIVE RATS**

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**Aim** To study the effects of endothelin-1 (ET-1) and BQ-123 (ETα receptor antagonist) on activities and mRNA expression of ATPase in aortic smooth muscle cells (ASMCs) from spontaneously hypertensive rats (SHR) and Wistar-Kyoto (WKY) rats.

**Methods** The ASMCs were isolated from SHR and WKY rats. The ATPase activities of cultured ASMCs were determined by spectrophotography. The mRNA levels of Na+, K+-ATPase α-subunit and plasma membrane Ca2+-ATPase isoform 1 (PMCA1) were measured by semiquantitative reverse transcription PCR (RT-PCR).

**Results** 3 different concentrations of ET-1 (1×10⁻⁹, 1×10⁻⁸ and 1×10⁻⁷ mol/l) significantly attenuated the activities of Na+, K+-ATPase and Ca2+-ATPase and PMCA1 mRNA expression (all p<0.01) in ASMCs from SHR. Three different concentrations of BQ-123 (1×10⁻⁸, 1×10⁻⁷ and 1×10⁻⁶ mol/l) obviously prevented ET-1 mediated the inhibition of two kinds ATPase activities (all p<0.01) and downregulation of PMCA1 mRNA expression (p<0.01). But the mRNA expression level of Na+, K+-ATPase α-subunit had no alteration after intervened by ET-1 (p>0.05).

**Conclusions** ET-1 may suppress Na+, K+-ATPase, Ca2+-ATPase activities via ETα receptor. The influence of ET-1 on Ca2+-ATPase activity may partially occur in the transcriptionel level. BQ-123 can inhibit the effect of ET-1 on two kinds ATPase activities of ASMCs in SHR by blocking the ETα receptor.

**e0122** **EFFECTS OF CARDIOTROPHIN-1 C-TERMINAL PEPTIDES ON CARDIOMYOCYTE APOPTOSIS IN SD RATS FOLLOWING MYOCARDIAL ISCHAEMIA REPERFUSION INJURY**

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**Objective** Observation of CT-1 C-terminal peptides in ischaemia-reperfusion injury before and after the intervention on myocardial cell apoptosis in SD rats.

**Methods** With ligation-release SD rats left posterior descending branch of coronary artery the ischaemia reperfusion heart model was established. 27 SD rats were randomly divided into four groups: Normal group (N, n=6); Disease group (D, n=6); Beginning of reperfusion after 30 min of MI; MI/R post-intervention group (T, n=6). 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=6); MI/R experiments was performed after intraperitoneal injection of CT-1 C-terminal peptide (100 μg/kg) for 7 days. In accordance with the ECG monitoring results ended the experiment in animals dying, left the serum for results ended the experiment in animals dying, left the serum for

**Results** thereafter and the myocardial injury and the extent of oxidative damage (r values were

**Conclusion** CT-1 C-terminal peptide in ischaemia-reperfusion injury could reduce myocardial apoptosis.

**e0123** **THE IMPACT OF DIABETES ON THE ROLE OF REPERFUSION INJURY SALVAGE KINASE PATHWAY**

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**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 globe 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 as ischaemia postconditioning during the early minutes of reperfusion. Phosphorylation of akt and gsk-3β were analysed by western blotting and immunohistochemical staining.

**Results** phospho-akt and phospho-gsk-3β expression increased markedly in B group. But compared A group, there were no parently deference in C group. phospho-akt and phospho-gsk-3β expression in C group is more less than in B group.

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