Ventricular haemodynamic parameters were also measured, include HR, LVSP. 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. SB216763, 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.

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**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 difference 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 phenomina maybe connected with gsk-3β in the condition of diabetic.

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

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**Aim** To study the effects of endothelin-1 (ET-1) and BQ-123 (ET<sub>A</sub> 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<sup>+</sup>, K<sup>+</sup>-ATPase α subunit and plasma membrane Ca<sup>2+</sup>−ATPase isoform 1 (PMCA) were measured by semiquantitative reverse transcription PCR (RT-PCR).

**Results** 3 different concentrations of ET-1 (1×10<sup>−9</sup>, 1×10<sup>−8</sup> and 1×10<sup>−7</sup> mol/l) significantly attenuated the activities of Na<sup>+</sup>−K<sup>+</sup>-ATPase and Ca<sup>2+</sup>−ATPase and PMCA mRNA expression (all p<0.01) in ASMCs from SHR. Three different concentrations of BQ-123 (1×10<sup>−9</sup>, 1×10<sup>−7</sup> and 1×10<sup>−6</sup> mol/l) obviously prevented ET-1 mediated the inhibition of two kinds ATPase activities (all p<0.01) and downregulation of PMCA mRNA expression (p<0.01). But the mRNA expression level of Na<sup>+</sup>−K<sup>+</sup>-ATPase α subunit had no alteration after intervened by ET-1 (p>0.05).

**Conclusions** ET-1 may suppress Na<sup>+</sup>−K<sup>+</sup>-ATPase, Ca<sup>2+</sup>−ATPase activities via ET<sub>A</sub> receptor. The influence of ET-1 on Ca<sup>2+</sup>−ATPase activity may partially occur in the transcriptional level. BQ-123 can inhibit the effect of ET-1 on two kinds ATPase activities of ASMCs in SHR by blocking the ET<sub>A</sub> receptor.