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A COMPARATIVE STUDY ON THE MECHANISMS OF INHIBITING ISCHEMIA-REPERFUSION MYOCARDIUM APOPTOSIS THROUGH IGF-1 POSTCONDITIONING AND ISCHEMIC POSTCONDITIONING

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Objective This study aims to investigate the influence of IGF-1 postconditioning on ischemia-reperfusion myocardium via the comparison with ischemia postconditioning and explore their possible inhibiting-mechanisms on myocardium apoptosis.
Methods Total of 48 SD rats were randomly and evenly divided into 4 groups: sham-operation group (sham), ischemic/

reperfusion group (I/R), ischemic postconditioning group (I-POST), and IGF-1 postconditioning group (IGF-1-POST), and the models were established through repeated ligating. Except for the sham group, rats in the other three groups remained ischemic for 45 min and then they were reperfused for 120 min. During the first 30 min of the reperfusion period, the ECG states were recorded, and hemodynamic parameters were monitored at the end of the time span. The TNFα, IL-6 and IL-10 level in the serum were detected by ELISA. The degree of myocardial damage in pathology was observed by HE staining. The apoptosis was detected by TUNEL.P $^{ser\text{-}133}$ -CREB and the expression of Bcl-2 protein was monitored through Western Blot and immunohistochemical methods.

Results Compared with that of I/R group, the cardiac function of rats in I-POST group and IGF-1-POST group was improved; TNFα and IL-6 level in the serum got lower while IL-10 level became higher; The elevation degree of ST segment, the ventricular premature beats and the duration of ventricular arrhythmia decreased at the same time; The degree of myocardial damage in pathology lessened and the number of apoptosis reduced; P $^{ser\text{-}133}$-CREB and the expression of Bcl-2 protein increased significantly ($p<0.05$). In comparison with the I-POST group, TNFα decreased but IL-10 increased ulteriorly in IGF-1-POST group ($p<0.05$). Moreover, the elevation degree of ST segment, the ventricular premature beats and the duration of ventricular arrhythmia increased slightly ($p<0.05$) in IGF-1-POST group other than the I-POST group. At other aspects mentioned above there was no significant difference between these two groups ($p>0.05$).

Conclusion IGF-1 has postconditioning protective function, which can protect myocardium from I/R injury. And this protecting mechanism might be similar to that of ischemic postconditioning. The anti-apoptosis of IGF-1 postconditioning and ischemic postconditioning might be related with P $^{ser\text{-}133}$-CREB and the high level expression of Bcl-2 protein.