GW23-e1870 **THE EFFECTS OF ISCHAEMIC POSTCONDITIONING ON THE EXPRESSION OF BCL-2 AND BAX PROTEIN IN RATS FOLLOWING ISCHAEMIA/REPERFUSION INJURY**

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Objectives Recently, many researchers have discovered that ischaemic postconditioning (IP) was a neuroprotective factor on cerebral ischaemia/reperfusion injury, which could have many mechanisms. Our researches purpose is going to observe the effects of ischaemic postconditioning on the expression of bcl-2 and Bax protein in rats following middle cerebral artery occlusion (MCAO) and to prove neuroprotective effect by ischaemic postconditioning.

Methods A rat model of focal cerebral ischaemia/reperfusion injury was established by middle cerebral artery occlusion using modified filament method. Male SD rats were randomised into 3 groups (n=10): sham-operate (sham) group, ischaemia/reperfusion (I/R) group and ischaemic postconditioning (IP) group.

Ischaemic postconditioning was induced by three repeated cycles of carotid artery occlusion for 5 min and reperfusion for 5 min. Rats were Sacrificed at 24 h after reperfusion, Neurological functional deficits were evaluated at 3 h,12 h and 24 h after ischaemia/reperfusion. At 24 h after the reperfusion, then infarct size and functional neurological outcome were measured. The brains were obtained for TTC staining and oedema examination and the brains were obtained for bcl-2 and Bax protein expression by immunohistochemistry method.

Results Their infarcted brain volumes were measured after 24 h. No infarct were found in the brains of rats in the sham group. The percentage of infarcted brain volumes in the I/R and I/P groups were 42±l0%, 27±11%, respectively. The infracted volumes of I/P groups was reduced compared to the I/R group, and there was significant difference ($p<0.01$). And brain oedema of rats in IP group decreased compare to that of I/R group ($p<0.05$). The rats of IP group had better neurological performance than that of I/R group. The expression of Bcl-2 of brain tissues in IP group were markedly increased compare to that of I/R group at all time points. While the expression of bax protein in IP group were markedly diminished compare to that of I/R group.

Conclusions The functional neurological outcome was improved and the cerebral infarct size and oedema were reduced by ischaemic postconditioning. The expression of Bcl-2 was up-regulated by ischaemic postconditioning. While the expression of Bax was down-regulated by ischaemic postconditioning that might be associated with the mechanism of ischaemic postconditioning protection on brain.