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Enhanced Neuroprotective Effects by Inter-Ischemia Hypothermia in Cerebral Stroke

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ABSTRACT

Background and Purpose. Studies have shown that inter-ischemia hypothermia is able to reduce the size of myocardial infarctions and improve their clinical outcomes. The present study determined whether inter-ischemia hypothermia induced by pharmacological approach induced stronger neuroprotection in ischemic brains.

Methods. Adult male Sprague-Dawley rats were studied in 4 groups: (1) sham; (2) stroke; (3) stroke treated with pharmacological hypothermia before reperfusion (inter-ischemia hypothermia); and (4) stroke treated with pharmacological hypothermia after reperfusion is initiated (inter-reperfusion hypothermia). The combination of chlorpromazine and promethazine with dihydrocapsaicin was used to induce hypothermia. To compare the neuroprotective effects of drug-induced hypothermia between the groups, brain damage was evaluated using infarct volume and neurological deficits. In addition, mRNA expressions of NADPH oxidase subunits

and glucose transporter subtypes were determined by real-time PCR. ROS production was measured by Flow cytometry assay at the same time points.

Results: In both hypothermia groups, cerebral infarct volumes and neurological deficits were reduced. ROS production and the expressions of NOX subunits and glucose transporter subtypes were also significantly reduced in both hypothermia groups as compared to the ischemic group. While there were no statistically significant differences between the two hypothermia groups at 6 h reperfusion, brain damage was significantly further decreased by inter-ischemia hypothermia at 24 h.

Conclusion: Inter-ischemia hypothermia and inter-reperfusion hypothermia after stroke induced neuroprotection by reducing oxidative injury, while neuroprotion was more effective with inter-ischemia hypothermia. This study provides a new avenue and reference for a stronger neuroprotective hypothermia before vascular recanalization in stroke patients.