Remote ischemic perconditioning

- Effects of different remote ischemia perconditioning methods on cerebral infarct volume and neurological impairment in rats
- Promising Cerebral Blood Flow Enhancers in Acute Ischemic Stroke
- Remote Ischemic Perconditioning for the Treatment of Acute Ischemic Stroke
- Ischemic Tolerance of the Brain and Spinal Cord: A Review
- Neuroprotective delivery platforms as an adjunct to mechanical thrombectomy
- Limb remote ischemic conditioning increases Notch signaling activity and promotes arteriogenesis in the ischemic rat brain
- Glucagon-like peptide-1 (GLP-1) mediates cardioprotection by remote ischaemic conditioning
- Limb Ischemic Perconditioning Attenuates Blood-Brain Barrier Disruption by Inhibiting Activity of MMP-9 and Occludin Degradation after Focal Cerebral Ischemia

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Remote Ischemic Preconditioning (RIPC) is a technique that involves limiting blood flow to a remote organ (such as an arm or leg) in order to induce a state of “ischemic tolerance” in a critical organ (such as the heart or brain), making it more resistant to injury during a subsequent ischemic event. This is achieved by applying brief, repeated cycles of ischemia (restriction of blood flow) followed by reperfusion (restoration of blood flow) to the remote organ. RIPC has shown promise in reducing the risk of ischemic injury in various medical settings, including heart surgery, cardiac arrest, and stroke.

Remote ischemic perconditioning (RIPerC) is a novel neuroprotective method against cerebral infarction that has shown efficacy in animal studies but has not been consistently neuroprotective in clinical trials.

Otsuka et al. focused on the temporal regulation of ischemia-reperfusion by RIPerC to establish an optimal method for RIPerC. Rats were assigned to four groups: 10 min ischemia, 5 min reperfusion; 10 min ischemia, 10 min reperfusion; 5 min ischemia, 10 min reperfusion; and no RIPerC. RIPerC interventions were performed during ischemic stroke, which was induced by a 60-min left middle cerebral artery occlusion. Infarct volume, sensorimotor function, neurological deficits, and cellular expressions of brain-derived neurotrophic factor (BDNF), B-cell lymphoma 2 (Bcl-2), Bcl-2-associated X protein (Bax), and caspase 3 were evaluated 48 h after the induction of ischemia. Terminal deoxynucleotidyl transferase-mediated dUTP-biotin nick-end labeling (TUNEL) was also performed. RIPerC of 10 min ischemia/10 min reperfusion, and 5 min ischemia/10 min reperfusion decreased infarct volume, improved sensorimotor function, decreased Bax, caspase 3, and TUNEL-positive cells, and increased BDNF and Bcl-2 expressions. Our findings suggest RIPerC with a reperfusion time of approximately 10 min exerts its neuroprotective effects via an anti-apoptotic mechanism. This study provides important preliminary data to establish more effective RIPerC interventions [1)]

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Otsuka S, Itashiki Y, Tani A, Matsuoka T, Takada S, Matsuzaki R, Nakanishi K, Norimatsu K, Tachibe Y, Kitazato R, Nojima N, Kakimoto S, Kikuchi K, Maruyama I, Sakakima H. Effects of different remote ischemia perconditioning methods on cerebral infarct volume and neurological impairment in rats. Sci Rep. 2023 Feb 7;13(1):2158. doi: 10.1038/s41598-023-29475-2. PMID: 36750711.