Abstract: P456

Xanthine oxidase suppression mediates anti-oxidative effect of somatic nerve stimulation in cardiac IR injury

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OBJECTIVES: The cardioprotective effects of somatic afferent nerve stimulation known as electroacetupuncture (EA) on PC6 and ST36, activating median nerve has been suggested in ischemic reperfusion (IR) injury. The anti-oxidative effect of EA in cardiac IR injury remains unclear. We explore the role of EA stimulation via regulate oxidative stress in IR injury rat model.

METHODS: To prove the protective effect of somatic nerve stimulation in IR injured rat heart, IR injury was established by occlusion of LAD for 40 min and reperfusion for 5 days and treated with EA on PC6 and ST36 for 5 consecutive days. Transthoracic echocardiography and molecular and histological evaluations were performed. To investigate the anti-oxidative effect of nerve stimulation, DHE fluorescence, xanthine oxidase (XO) activity and acetylcholine (Ach) amount were measured. In H9c2 cells, Ach was treated to simulate nerve stimulation under hypoxia-reoxygenation (HR). Atropine, a muscarinic Ach receptor was pretreated to estimate effect of Ach on cardiac myocyte cell viability, intracellular ROS generation and XO activation.

RESULTS: Somatic afferent nerve stimulation on PC6 and ST35 limited infarct size in IR injured heart. Echocardiography suggested that the reduced ejection fraction and fractional shortening was recovered by the stimulation. Oxidative stress indicator, DHE fluorescence determined that PC6 and ST36 stimulation abrogated IR-induced ROS generation in risk region. XO activation in IR injury was abrogated and tissue acetylcholine amount was enhanced with the stimulation. In addition, intracellular ROS generation and XO activation by HR stress were decreased with acetylcholine treatment in H9c2 cells.

CONCLUSION: Our results suggest that XO inhibition is responsible for protection of somatic nerve stimulation on PC6 and ST36 against cardiac IR injury.