CONFERENCE ABSTRACT
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ASSESSMENT OF THE MONOAMINE OXIDASE INHIBITION EFFECT ON ISCHEMIC PRECONDITIONING IN ISOLATED RAT HEARTS

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Key words: monoamine oxidase inhibitors, ischemic preconditioning, isolated rat hearts

OBJECTIVES AND BACKGROUND

Sublethal generation of reactive oxygen species (ROS) mediates the cardioprotective effect of ischemic preconditioning (IPC). However, the sources of ROS are partially elucidated. Monoamine oxidases (MAOs) A and B are mitochondrial enzymes representing a constant source of hydrogen peroxide (H_2O_2) in the heart.

AIM

This study was purported to assess the effects of two MAO inhibitors (clorgyline for MAO-A and pargyline for MAO-B) on the IPC-related protection in isolated rat hearts.

MATERIALS AND METHODS

Hearts subjected to 30 min global ischemia and 120 min reperfusion were assigned to the following groups: (i) Control - no intervention; ii) IPC - 3 cycles of 5 min ischemia/5 min reperfusion before global ischemia, (iii) IPC-Clorgyline - IPC plus clorgyline (50 μM/l, iv) IPC-Pargyline – IPC plus pargyline (0.5 mM/l). Post-ischemic functional recovery was assessed by measuring the left ventricular developed pressure (LVDP) and the indices of contractility (+dLVP/dtmax) and relaxation (-dLVP/dtmax). Infarct size (IS) was quantified by triphenyl-tetrazolium chloride staining.

RESULTS

Patients IPC significantly improved functional recovery that was further enhanced in the presence of either clorgyline or pargyline. IS reduction was comparable among all preconditioned groups.

CONCLUSIONS

In isolated rat hearts MAO inhibition enhanced the IPC-related functional recovery without interfering with infarct size reduction.

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