Change of Nitric Oxide Synthasesfrom Epicardium to Endocardium in Rat Cardiac Ventricular During Ischemic Preconditioning

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Background: Ischemic preconditioning (IPC) protects the heart against ischemia and reperfusion (I/R)-induced injuries. Our previous studies investigated nitric oxide (NO) might contributes to protein kinase G (PKG), ATP-sensitive K+ (KATP) channels pathways in IPC phenomenon. Enzymatical NO production is catalyzed by endothelial, neuronal and inducible NO synthases (eNOS, nNOS, iNOS, respectively) but their roles during IPC are still unclear. The present study attempted to determine the expression of eNOS, iNOS in epicardium, midcardium and endocardium and in infracted, viable areas during IPC.

Methods & results: Isolated rat heart were perfused by Langendorff system and subjected to 30 min of ischemic solution then 60 min of reperfusion by normoxic solution (I/R group, n=5), or preconditioned 3 single-5 min with ischemic solution (IPC group, n=5), 300M SNAP (SNAP group, n=5) prior to I/R episodes. eNOS, iNOS expression in whole heart and in epi-, mid-, endocardium were detected by western blotting. 20μm in thickness of ventricular epi-mid-endocardium were sectioned and eNOS, iNOS were detected by immunofluorescence assay using confocal microscope. Infarction sizes were observed by TTC staining. The results showed that IPC and SNAP significantly reduced infarction size. Infarction areas in IPC and SNAP groups located somewhere near endocardium but were spreaded fromepicardium to endocardium in I/R

group. Both eNOS and iNOS were highly expressed in IPC groups. The results from IPC group via immunofluorescence assay showed high intensity of iNOS in endocardium area.

Conclusions: Our results showed the expression of eNOS, iNOS in epi-mid-endocardium and infarction/viableareas within ventricular. The results demonstrated that NO play important role to reduce infraction size induced by I/R and iNOS may effect the distribution of injury from epicardium to endocardium during ischemic preconditioning.

Key words : Nitric Oxide, Ischemic Preconditioning, Endocardium, inducible NOS.