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PACING POSTCONDITIONING PROTECTS THE HEART BY A PATHWAY INVOLVING NITRIC OXIDE AND NATRIURETIC PEPTIDES

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Introduction Brief periods of ventricular pacing during the early reperfusion phase (pacing-induced postconditioning, (PPC)) have been shown to protect the heart against ischemia reperfusion injury. The aim of this study was to explore the role of natriuretic peptides (atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP)), protein kinase G (PKG) and nitric oxide (NO) in the protective effect of PPC.

Methods Isolated perfused (Langendorff) rat hearts (n=6) were subjected to 30 minutes coronary occlusion and 30 minutes reperfusion. PPC consisted of 3 episodes of 30 seconds ventricular pacing alternated with 30 seconds atrial pacing during early reperfusion. Studied were control with only ischemia and reperfusion, PPC, PPC in combination with selective blockers of the ANP (H-[1,2,4]oxadiazole[4,3-a]quinoxalin-1-one (ODQ)), BNP (isatin) and NO (L-nitro-arginine-methylester (L-name)). Another sets of animals were treated with the agonists of ANP (ANP), BNP (Rat BNP-32), NO donor (S-nitroso-N-acetylpenicillamine (SNAP)) and PKG activator 8-(4-chlorophenylthio)-guanosine 3', 5'-cyclic monophosphate (CPT) at the beginning of reperfusion. Hemodynamics were computed by a data acquisition program. Infarct size and area at risk were determined using TTC and blue dye.

Results Ischemia and reperfusion resulted in a poor recovery of heart hemodynamics. PPC significantly ($p<0.03$) improved cardiac hemodynamics and decreased the infarct size ($p<0.05$). Isatin and L-name completely abrogated the protective effect of PPC. However, ODQ did affect the protection afforded by PPC. When applied exogenously Rat BNP-32, CPT and SNAP showed a significant ($P<0.03$) recovery in cardiac hemodynamics and significantly ($P<0.05$) decreased the infarct size. This protection is comparable to that produced by PPC.

Conclusions Exogenous and endogenous BNP, NO and PKG protected the heart against ischemia reperfusion injury. However, ANP is not involved in this protection. Interaction between ROS and NO did not block PPC protection to the heart.