NITRIC OXIDE- AND PROSTAGLANDIN-MEDIATED CARDIOPROTECTION BY BRADYKININ IN MYOCARDIAL ISCHEMIA AND REPERFUSION INJURY

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The aim of the present study was to investigate the involvement of nitric oxide and prostaglandin pathways in the cardioprotective actions mediated by bradykinin via the combined inhibition of angiotensin converting enzyme and aminopeptidase P in an in vivo rat model of acute ischemia (30 min) and reperfusion (4 h). Myocardial infarction was produced by occlusion of the left anterior descending coronary artery for 30 min followed by 4 h of reperfusion. Infarct size was measured by using the staining agent TTC (2,3,5-triphenyl-tetrazolium chloride). Lipid peroxide levels in serum and in heart tissue were estimated spectrophotometrically. A lead II ECG was monitored at various intervals throughout the experiment. Infarct size expressed as percent of left ventricle was found to be 50.5 ± 3.5 in control animals and was reduced to 19.4 ± 1.1 and 15.0 ± 2.1 with the combined treatment of enalapril or lisinopril and 2-mercaptoethanol, respectively. There was no significant difference in the infarct size of control animals and in the animals treated with HOE140 prior to the combined treatment. Infarct size reduction obtained with the combined inhibition with enalapril and 2-mercaptoethanol or lisinopril and 2-mercaptoethanol was blocked partially but significantly with the prior administration of L-NAME (Nω-nitro-L-arginine methyl ester) or aspirin, suggesting the involvement of both nitric oxide and prostaglandin pathways in the cardioprotective actions mediated by bradykinin.

Key words: ischemia, reperfusion, bradykinin, nitric oxide, cardioprotection

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