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**Stimulation of endogenous glutathione synthesis prevent postreperfusion NOS uncoupling, oxidative nitrosative stress and cardiodynamic disturbances in rats**

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Ischemia-reperfusion of isolated rat hearts is accompanied by cNOS uncoupling (index of cNOS coupling decreased 20-times), oxidative stress (generation rate of superoxide radical was increased 3-times), decrease of cNOS activity, development of contracture and myocardial contractility insufficiency. Adult Wistar rat hearts were isolated by Langendorff preparation underwent a 20-minute ischemia and a 10-minute reperfusion before biochemical examination for superoxide radical, hydroxyl radical, NOS activity and glutathione content. Pre-treatment with L-cysteine (120 mg/kg), precursor of glutathione and H2S synthesis, in combination with inhibitor of H2S synthesize enzyme CSE – PAG (11 mg/kg) increased 3-times glutathione level. GSH consumption after ischemia-reperfusion increased 4-times and its conversation to oxidized form (GSSG) increased 6-times in comparing to control reaction. It was accompanied by significantly better redox status of myocardial tissue and cardiodynamic disturbances prevention. Inhibition of GSH synthesis by BSO (buthionine sulfoximine 22 mg/kg) restored above disturbances. Thus, glutathione levels in cardiac tissue predetermine heart reaction in ischemia-reperfusion and stimulation of GSH synthesis prevents typical disturbances for this reaction.