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Multiple pre-conditioning stimuli attenuate protection against the infarct size in rabbits

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The aim of the study was to determine if the repeated periods of 5 min ischaemia and 10 min reperfusion (5 is0/10 rep) are protective against the infarct size in a collateral dependent model. Methods: Forty-one rabbits were divided into 7 groups. Group (Gp) A (n=5) was subjected to one cycle of 5 is0/10 rep, GpB (n=5) was subjected to two 5 is0/10 rep, GpC (n=7) to four 5 is0/10 rep, GpD (n=7) to six 5 is0/10 rep, GpE (n=5) to six 5 is0/10 and GpF (n=3) to eight 5 is0/10 rep while GpG (n=3) served as control. After the initial interventions all the Gps (except GpF) were subjected to 45 min regional ischaemia and 2 hours reperfusion. The elapsed time from the first preconditioning stimulus to the final 45 min ischaemic insult was 15, 30, 60, 90 and 120 mins, respectively for the Gps A, B, C, D and E. Infarct size (I) and risk zones (R) were delineated with the aid of tetrazolium staining and fluorescent particles. I/R ratio was expressed in percent (% I/R).

Reperfusion damage is mediated by a stable long-lasting cardiac metabolite which is probably not a protein

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We have previously reported that after global ischaemia (IS) of an isolated heart cardiodepressant agents are released during reperfusion (R) which induced a pronounced but reversible decrease in contractility. In a serially perfused non ischaemic second heart (H2) (double heart model), this effect was not attributable to changes in pH or concentration of electrolytes of the coronary effluent and the metabolites do probably not derive from coronary endothelium. In order to further characterize the chemical structure of these mediators we investigated whether storage of the coronary effluent for several hours or days, heating to 56°C for 30 min or protease treatment influence the negative inotropic effect.

Two isolated guinea pig hearts were perfused separately at constant flow (10 ml/min) with a modified Krebs-Henseleit-solution oxygenated with 95% O2 and 5% CO2. This was followed by serial perfusion without (group 1, n=5) and with (group 2, n=5) preceding IS (10 min) of the first heart (H1). In group 1 no significant changes of the left ventricular contractile parameters of H2 were observed during serial reperfusion. In group 2, after a global 10 min IS of H1 left ventricular (LV) systolic pressure of H2 (basal 76.3 ± 3.5 mmHg, SEM) immediately decreased by 16%, the LVdP/dtmax (1491.9 ± 112.4 mmHg/s by 22%, the LV/ITmax (1261.3 ± 56.8 mmHg/s) by 80% when R was started. Coronary perfusion pressure (71.7 ± 3.4 cmH2O) decreased by 26%. These parameters returned to baseline within 10 min. Heart rate did not change significantly. The cardiodepressant effect of the coronary effluent was not destroyed by protease treatment (chymotrypsin 0.005 U) or by heating the coronary effluent to 56°C for 30 min. Coronary effluent kept at room temperature for 24 hours before delivery to H2 also retained its activity.

These data suggest the release of a stable, long-lasting cardiodepressant agent from myocardial tissue after global IS during R. The resistance against protease treatment and heating suggest that the mediator is not a protein.