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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 ischemia and 10 min reperfusion (5 iso/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 iso/10 rep. GpB (n = 5) was subjected to two 5 iso/10 rep, GpC (n = 7) to four 5 iso/10 rep, GpD (n = 7) to six 5 iso/10 rep, GpE (n = 7) to eight 5 iso/10 rep while GpF (n = 5) served as control. After the initial interventions all the Gps (except GpF) were subjected to 45 min regional ischemia and 2 hours reperfusion. The elapsed time from the first preconditioning stimulus to the final 45 min ischemic 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).

<table>
<thead>
<tr>
<th>Groups</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>E</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>%I/R</td>
<td></td>
<td>22.2 ± 2.6*</td>
<td>19.5 ± 4.1*</td>
<td>23.3 ± 3.4*</td>
<td>41.8 ± 6.9†</td>
<td>47.1 ± 7.6*</td>
</tr>
</tbody>
</table>

*p < 0.001 vs A, f < 0.05 vs A, B, C, †p < 0.05 vs G

Conclusion: These results indicate that protection against infarct size diminishes after multiple and repetitive preconditioning stimuli in rabbits.

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 ischemia (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-ischemic 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 perfusion. 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%, +LVDP/Δtmax (1491.9 ± 112.4 mmHg/s by 22%, -LVDP/Δtmax (1261.3 ± 56.8 mmHg/s) by 30% when R was started. Coronary perfusion pressure (77.1 ± 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 (chymotrypsine 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 delivery to H2 also retained its activity.

Assessment of C-reactive protein levels after percutaneous transluminal coronary angioplasty and stent implantation

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In patients (pts) with acute coronary syndromes increased serum levels of C-reactive protein (CRP) are associated to a worse short-term outcome. The aim of this study was to establish whether coronary angioplasty (PTCA) or stent implantation (SI) have the potential to increase serum levels of CRP and whether the latter affects the short and long-term prognosis. Ninety-nine pts (mean age 58 ± 7 years, 88 men) with stable angiogram and obstructive coronary atherosclerosis were randomised to PTCA (49 pts) or SI (50 pts). There were no significant differences among the two groups with respect to baseline clinical characteristics (gender, previous myocardial infarctions, risk factors and therapy) and to angiographic characteristics before the procedures. Serum CRP levels were measured, by nephelometry, 24 hours before and 6 hours, 1 day, 2 days, 3 days and 4 days after the procedure. All pts were then followed-up for 6 months. Serum levels of CRP were obtained also at 3 and 6 months. Serum levels of CRP (mg/l) were as follows (mean ± SD values):

<table>
<thead>
<tr>
<th>CRP (mg/l)</th>
<th>Baseline</th>
<th>6-hours</th>
<th>1-day</th>
<th>2-days</th>
<th>3-days</th>
<th>3-months</th>
<th>6-months</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTCA</td>
<td>2.1 ± 0.6</td>
<td>3.9 ± 3.6</td>
<td>4.6 ± 5.2</td>
<td>4.8 ± 10*</td>
<td>4.4 ± 5.2</td>
<td>2.0 ± 0.7</td>
<td>2.7 ± 1.6</td>
</tr>
<tr>
<td>SI</td>
<td>2.0 ± 0.6</td>
<td>3.9 ± 3.5</td>
<td>4.2 ± 5.8*</td>
<td>4.2 ± 10*</td>
<td>4.0 ± 1.0</td>
<td>2.4 ± 1.3</td>
<td>2.3 ± 1.0</td>
</tr>
</tbody>
</table>

*p < 0.05 vs baseline value

The incidence of early complications and symptoms recurrence was similar after PTCA and SI (2 vs 2%, p = NS and 10 vs 16%, p = NS, respectively).

Conclusions: SI results in a much higher increase of CRP serum levels than that observed after PTCA; yet, the incidence of early complications and late outcomes is similar. Thus, a marked elevation of CRP caused by an inflammatory stimulus localized in an epicardial coronary vessel is not sufficient per se to trigger coronary instability.