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**P3092** Indirect evidence for free radical release following exercise-induced ischaemia resulting in myocardial stunning

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Direct and indirect evidence suggests that oxygen derived free radicals play a pathogenetic role in myocardial stunning. We investigated whether exercise-induced myocardial stunning was associated with free radical release in man.

Methods: 10 men (aged 26 ± 9 years) with triple vessel coronary artery disease, stable angina and normal left ventricular function performed symptom-limited treadmill exercise tests. Serial transhoracic echocardiography (ECHO) was performed before, and at regular intervals after exercise. Peripheral venous blood samples were taken prior to and immediately after exercise, and assays of lipid peroxidation were performed using HPLC as an indirect measure of free radical generation and resultant peroxidation.

Results: Heart rate, blood pressure and ST changes returned to baseline within 10 minutes of exercise in all patients. Exercise duration was 184 ± 180 sec, and maximum ST depression was 1.9 ± 1.1 mm. Quantitative ECHO data (mean ± SD) pre exercise, 30 minutes and 240 minutes post exercise are shown:

<table>
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<tr>
<th></th>
<th>Pre exercise</th>
<th>30‘ post</th>
<th>240‘ post</th>
</tr>
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<tbody>
<tr>
<td>HR (bpm)</td>
<td>99 ± 8</td>
<td>106 ± 11*</td>
<td>90 ± 8</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>40.4 ± 4.3</td>
<td>52 ± 10*</td>
<td>59 ± 7</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>3.8 ± 0.9</td>
<td>1.8 ± 0.9*</td>
<td>3.6 ± 1.1</td>
</tr>
</tbody>
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* p < 0.001 vs pre exercise

Histochrome (H)

Control

Post exercise: 1.0 ± 1.1 mm. Quantitative ECHO data (mean ± SD) post exercise are shown:

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**Histochrome (H)** — water-soluble form of 2, 3, 5, 8-pentahydroxy-7-ethyl-1, 4-naphtoquinone possess high cardioprotective activity in the animal models of coronary occlusion/reperfusion. We investigated the effect of H. treatment during thrombolytic therapy (TLT) in patients with acute myocardial infarction (AMI) on global and regional left ventricular (LV) function. 16 pts (17 men) with successful thrombolysis (intravenous- i.e. and/or intracoronary-) in a first anterior Q-wave AMI were included in the final analyses of the pilot study.

Study design: randomised, opened. There were equal number pts in each group (6 in control-C. and H. group). No statistically significant differences were noted in the mean age (53.0 ± 3.9 y in C. vs. 54.6 ± 2.8 y in H., p = NS) in the time from pain to TLT (2.51 ± 0.56 h C. vs. 3.0 ± 0.44 h, p = NS) in the time from pain to peak creatinine kinase (CK) activity (10.90 ± 1.21 h vs. 13.80 ± 1.62 h, respectively; p = NS) and adjunctive drug regimen between groups. The dosage schedule included 100 mg bolus injected i.v. just before TLT and 100 mg bolus 1 hour later. All pts underwent echocardiography on 1–2 d and 14–21 d for LV function assessment. Wall motion analysis was done within the frame of the 16-segment model. We calculated global wall motion index (WMI), regional (infarct-related) wall motion index (RWMI), end-diastolic and end-systolic volume indexes (EDVI, ESVMI/mL), ejection fraction (EF%). Serial blood samples for measuring CK plasma level were taken.

**Conclusion:**

Non-significant difference between groups were observed in peak CK (2941 ± 577 UL in H. vs. 3790 ± 770 UL in C.; p = NS).

Conclusion: H. had no significant effect on LV global and regional function after successful thrombolysis in pts with anterior Q-wave AMI in our pilot study.

**P3093** Different epicardial and endocardial activation of KATP channels might cause peaked T-waves during acute coronary occlusion

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**Purpose:** We tested a hypothesis that there is a difference in epicardial (Epi) and endocardial (Endo) activation of KATP channels, which might cause electrophysiographic ST-T changes.

**Methods:** In anaesthetized open-chest dogs, a sequence of 5-min occlusion and 30-min reperfusion of the left anterior descending coronary artery was repeated, while Epi and Endo monophasic action potentials (MAPs) were recorded from the centre of ischaemic zone(n = 14). In a separate group(n = 7), unipolar electrograms were recorded from 47 Epi sites within the ischaemic zone using a mapping system. 5-hydroxycarinate (30 mg/kg; 5-HD) or glibenclamide (0.25 mg/kg; GLB), specific blockers of KATP channels, or nicardipine (0.25 mg/kg; NCR), an opener, was administered intravenously before the third or fourth occlusion, then the data were compared with the control occlusion data. Heart rate was kept constant by atrial pacing.

**Results:** During control occlusion, shortening rate of MAP duration at 90% repolarization was greater at Epi than Endo layer (19.7 ± 1.5 vs. 13.1 ± 2.4%, mean ± SE, n = 14, p < 0.05). 5-HD shortened the shortening rate preferentially at Epi layer, and reduced the difference between the two layers (11.0 ± 3.5 vs. 11.5 ± 3.7%, n = 6, NS). In contrast, NCR augmented the shortening preferentially at Epi layer, and increased the difference (29.0 ± 2.0 vs. 9.5 ± 3.0%, n = 6, p < 0.05). Concordantly, occlusion-induced increase in the peak amplitude of T-waves was suppressed by GLB while augmented by NCR (control occlusion: 10.5 ± 0.5, GLB: 5.6 ± 0.4, NCR: 12.8 ± 0.5 mV, n = 7, p < 0.05). Collateral blood flow measured using radioactive microspheres during control occlusion was greater at Epi than Endo layer (31.3 ± 12.5 vs. 8.3 ± 3.1 mU/100 g/min, n = 8, p < 0.05).

**Conclusion:** These data suggest that epicardially dominant activation of KATP channels might cause peaked T-waves during acute coronary occlusion.