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## EVIDENCE OF HIBERNATING MYOCARDIUM BY SEQUENTIAL REST - REDISTRIBUTION THALLIUM-201-SPECT IMAGING: IMPACT ON CONTRACTILE RECOVERY.

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In an attempt to identify preserved cellular function in chronic ischemia 19 patients with severe coronary artery disease and contractile dysfunction (LVEF=30±6 %) were subjected to resting Tl-201SPECT [demarcation image] after injection of 79-112 MBq of Tl-201 and re-imaging after 6 hours; delayed cellular uptake of TL-201 was considered suggestive of viable myocardium in akinetic segments [hibernating myocardium] as identified from centerline wall motion analysis of contrast left ventricular angiograms. Prior to complete revascularization by multi-vessel PTCA [n=9] or bypass grafting [n=10] 69 of the total of 171 myocardial segments had initial resting Tl-201 defects; 48 of those 69 segments (69.6%) showed partial Tl-201 uptake on 6 hour redistribution images suggestive of cellular viability. In 38 of those 48 segments (79.1 %) wall motion improved by a significant margin ($p<0.01$), whereas no contractile improvement occurred in 31 segments, 21 of which (67.7 %) revealed no Tl-201 uptake on redistribution images.

Conclusion: Delayed Tl-201 uptake in akinetic myocardial defect areas on initial demarcation images is a sensitive marker for hibernating myocardium; after myocardial revascularization areas of delayed cellular Tl-201-uptake are likely to demonstrate recovery of segmental wall motion. Thus, delayed Tl-201 uptake in resting perfusion defects has prognostic impact by unmasking hibernating tissue with recovery potential.

## CHANGES IN REGIONAL VENTRICULAR THICKNESS AND THICKENING AFTER ANTERIOR WALL MYOCARDIAL INFARCTION

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**Previous studies from our laboratory have shown little early change in global LV mass during post-myocardial infarction (MI) remodeling, despite significant changes in cavity volumes. We postulated that thinning of the myocardium in the infarct zone during remodeling was associated with proportional increases in non-infarct zone wall thickness, accounting for no net change in global LV muscle mass. To address this, average regional LV wall thickness at end-diastole (EDTh) and systolic wall thickening (%Thi) were quantitated using ultrafast computed tomography at discharge (d/c) (7±3 days) and 6-week follow up (f/u) in 5 Pts. after a first Q-wave anterior wall MI . These data were compared with results from 11 normal Pts. Average EDTh and %Thi were normal in the septal and lateral walls at discharge and 6-weeks. However, at d/c, the EDTh was normal in the infarct region but increased above the normal range in the opposite, inferior wall. At f/u, the anterior wall thinned nonsignificantly while the inferior wall maintained its increased EDTh. Normal EDTh is .95±.02 cm and normal %Thi is 54±3 % (*p<.05 compared to normal).**

| Wall | EDTh (d/c) | %Thi | EDTh (f/u) | %Thi |
|---|---|---|---|---|
| Inferior | 1.3±.2 * | 62±7 | 1.2±.2 * | 51±12 |
| Anterior | 0.94±.1 | 21±26 * | 0.8±.2 | 12±21 * |

**Conclusions: During LV remodeling after anterior wall MI, as expected, a trend is seen towards progressive wall thinning in the infarct zone. In contrast, a significant compensatory increase in EDTh is evident in the opposite inferior wall at hospital d/c which is unchanged by 6-week follow up. These data suggest that, after Q-wave MI, changes in regional LV wall thicknesses are evident by hospital discharge and persist during early f/u with little change in regional systolic function. Regional hypertrophy/atrophy occuring proportionately between the non-infarct and infarct zones is likely accountable for preservation of global LV muscle mass during early post-MI remodeling.**

## LEFT VENTRICULAR DYSFUNCTION INDUCED BY DIPYRIDAMOLE IS PROPORTIONATE TO SEVERITY OF CORONARY ARTERY DISEASE

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Dipyridamole (D) induces left ventricular (LV) dysfunction in 73% of patients with coronary artery disease (CAD) in contrast to normal subjects. To further investigate this effect, 70 CAD patients who underwent coronary cineangiography were given 0.56 mg/kg of D over 4' i.v. and LV function was examined by nuclide ventriculography. Three groups of pts were defined. Gp I: Single vessel (N=19); Gp II: Two vessels (N=33); Gp III: Three vessels (N=18). D affected LV function significantly in Gps II and III, and much less, if at all, in Gp I.

| Gp | Control EF | D-EF | % | p | %TP | %FN |
|---|---|---|---|---|---|---|
| I | 52±9 | 49±10 | 3.6 | N.S. | 70 | 30 |
| II | 53±9 | 46±10 | 13 | 0.032 | 90 | 10 |
| III | 48±10 | 39±10 | 19 | 0.009 | 100 | 0 |

where TP (true positive responder) = decrease/no change in EF, and FN (false negative responder) = increase of EF by 5 units or more.
Conclusion: D causes more LV dysfunction in the presence of more severe CAD as judged by number of vessels obstructed. Few pts with 2- and 3-vessel disease remain unaffected by D (EF decreased in 42 of 51 such pts i.e. 82%, increased by 5 units in only 3, i.e. 6%). Nuclide ventriculography with D identifies most CAD pts with 2- and 3-vessel disease, but fails to identify as many as 30% of the patients with 1-vessel disease.

## EVALUATION OF THROMBOXANE PRODUCTION AND COMPLEMENT ACTIVATION DURING MODERATE MYOCARDIAL ISCHEMIA IN PATIENTS WITH ANGINA PECTORIS

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The complement system and arachidonic acid metabolites are involved in severe myocardial ischemia such as myocardial infarction. Furthermore, there is experimental evidence for C5a participation to thromboxane (Tx) production. To know whether both C5a anaphylatoxin and Tx are produced during moderate myocardial ischemia, 15 patients with stable angina underwent either atrial pacing or percutaneous transluminal coronary angioplasty associated with arterial and coronary sinus blood sampling. Rapid atrial pacing caused significant ST-segment depression (Δ ST=-1.9 ± 0.3 mm), decrease of % lactate extraction (from +17.3 ± 3.1% baseline to -8.5 ± 5.9% at the peak of ischemia, $p<0.05$) and increase of coronary sinus plasma $TxB_2$ levels (from 543 ± 150 baseline to 3242 ± 1105 pg/ml at the peak of ischemia, $p<0.05$) with no change of 6-keto-PGF1α levels. A similar pacing in control subjects caused neither lactate nor Tx production. C5a levels in coronary sinus plasma remained below 4 ng/ml in all patients. Coronary angioplasty caused more severe myocardial ischemia (lactate extraction decreased from +24.8 ± 2.7% baseline to -41.6 ± 22.4% at the peak of ischemia, $p<0.05$) and was not associated with the generation of C3a and C5b-9 which are accurate markers of complement activation in vivo. Both platelet and leukocyte counts were unchanged.

We conclude that Tx is produced during brief episodes of myocardial ischemia in patients with stable angina. The complement is not activated in these patients and C5a anaphylatoxin may not participate to Tx release.