

907-59 Has Thallium-SPECT a Predictive Value for the Occurrence of Cardiovascular Events in Diabetic Asymptomatic Patients?

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Diverging results have been reported about the value of Thallium (TI) for prediction of cardiovascular (CV) events in diabetic pts.

202 pts (137 males, 65 females, age 59 ± 11 yrs) were prospectively screened with stress-R1-TI-SPECT (exercise $n = 103$, dipyridamole $n = 99$). Type I diabetes mellitus was present in 48 pts and type II in 156 pts, 40 of them being under insulin therapy. 163 pts (76%) had no history of CV disease, but all pts had 2 risk factors or more; 113 pts (56%) presented a diabetic nephropathy.

They were followed for an average period of 25 months (range 4–74 months) with a systematic clinical and ECG control at the end of the follow up. Kaplan-Meier survival analysis was performed.

9 pts died from CV causes, 18 pts underwent a non fatal Q-wave myocardial infarction (MI). Other CV events (unstable angina, congestive heart failure, need for myocardial revascularization) occurred in 25 other pts.

Results:

	Normal TI (n = 108)	Abnormal TI (n = 94)	p value
CV death	4	5	NS
Q wave MI	6	12	NS
Death + MI	10	17	0.065
Other CV event	11	14	NS
All CV events	21	31	0.04

The probability of any events was 33% after an abnormal scan, and 19% after a normal scan. Results were not improved when only reversible TI-Defects were considered.

Conclusion: TI-SPECT has only little value for the prediction of future CV events in asymptomatic diabetic pts. In difference with non diabetic pts, its negative predictive value is poor.

907-60 Acute Hemodynamic and Antischematic Effects of Trandolapril in Patients With Coronary Artery Disease

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Recent data indicate that ACE-inhibition may affect the incidence of ischemic events. To evaluate the mechanisms for this effect, 14 untreated pts with CAD underwent 2 identical, incremental pacing tests, 45 min before (P1) and 15 min after (P2) 2 mg intravenous trandolapril (T, $n = 8$), or placebo ($n = 6$). Baseline variables were comparable in both groups. During P1 both groups became ischemic as evidenced by lactate production and ST-segment depression. During P2, hemodynamic, neurohormonal activation and ischemia variables were reproducible in placebo group. At baseline, before P2, T had reduced arterial and coronary venous ACE by 65% and 60%, resp., however, neither arterial, nor coronary venous angiotensin II (All) had changed. In T, maximal HR, LVSP, rate-pressure product, coronary hemodynamics and myocardial O_2 consumption were similar during both pacing tests. T clearly reduced ischemia, evidenced by less ST depression (0.09 ± 0.02 mV vs 0.14 ± 0.03 mV, P2 vs P1 resp. $p < 0.05$), and decreased LVEDP (45%, P2 vs P1). Lactate production during P1 changed to lactate consumption during P2 ($-2.1 \pm 9.0\%$ vs $8.7 \pm 8.8\%$, P1 vs P2, $p = 0.2$). Of the neurohormones, only coronary venous All was affected by T, it decreased by 37% during P2, and was 57% less, as compared to P1. Thus, ACE inhibition with trandolapril results in acute antischematic effects. These antischematic effects occurred not through interfering with overall coronary flow, nor cardiac O_2 -demand, neither by modulation of ischemia induced catecholamine activation. Rather, a selective inhibition of cardiac RAS seems involved, which, either by itself, or through a local increase of prostaglandins or bradykinin, may affect the ischemic process.

908 Cardiac Surgery Poster I

Monday, March 25, 1996, Noon–2:00 p.m.
Orange County Convention Center, Hall E
Presentation Hour: 1:00 p.m.–2:00 p.m.

908-15 Pseudo Impaired Flow Reserve in Internal Mammary Artery Grafts Early After Surgery: A Comparison to Late Postoperative Measurements in the Same Patients

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It has been frequently proposed that internal mammary artery grafts (IMA) have a decreased functional flow reserve early after surgery, recommending IMA as unsuitable for pts with unstable angina and/or left main disease. During a prospective study 15 left (LIMA) and 19 right (RIMA) grafts were investigated in 19 pts 6–8 days (I) and 1 yr (II) after surgery. Via 6F IMA catheters using a 18/1000 inch flowire[®] (12 MHz, Cardiometrics) average peak velocity (APV, cm/sec) at rest (R), after 12.5 mg papaverine (PAP) and after 0.2 mg nitroglycerine (NTG) given into IMA grafts was measured. Luminal diameter (D, mm) was measured by biplane QCA (DCI, Philips) and blood flow (Q, ml/min), blood flow for mean aortic pressure of 100 mmHg (Q100, ml/min) and coronary flow reserve (CFR) were calculated.

Results:

	D (mm)	Q (ml/min)	Q100 (ml/min)	CFR
R I	2.9 ± 0.4	41 ± 13	48 ± 18	
R II	2.8 ± 0.3	$30 \pm 13^*$	$36 \pm 16^*$	
PAP I	$3.0 \pm 0.4^*$	$89 \pm 23^*$	$116 \pm 33^*$	2.5 ± 0.5
PAP II	$2.9 \pm 0.3^*$	$86 \pm 37^*$	$102 \pm 45^*$	$3.0 \pm 0.8^*$
NTG I	$2.9 \pm 0.4^*$	$79 \pm 26^*$	$106 \pm 32^*$	2.0 ± 0.4
NTG II	3.0 ± 0.3	$78 \pm 36^*$	$95 \pm 47^*$	$2.9 \pm 0.9^*$

mean \pm SD, *P < 0.05 vs I, *P < 0.05 vs rest (R)

Conclusion: D of IMA is unchanged early and late after surgery. Flow at R is significantly lower late postoperatively. The early and late functional status of IMA grafts is comparable, if measured in the same patient. A seeming improvement of CFR late after surgery is due to a lower resting flow, but not to an increase in maximal flow.

908-16 Heparin-Bonded Circuits Decrease Myocardial Ischemic Damage

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Heparin-Bonded Cardiopulmonary Bypass circuits have been shown to reduce complement activation and minimize blood loss during cardiac surgery, but their effect on myocardial function is unknown. This study was, therefore, undertaken to determine whether Heparin-Bonded Cardiopulmonary Bypass (CPB) reduces myocardial ischemic damage during acute revascularization. In 16 pigs, the second and third diagonal vessels were occluded with snares for 90 minutes followed by 45 minutes of cardioplegic arrest and 180 minutes of reperfusion with the snares released. During the period of coronary occlusion, all animals were placed on percutaneous bypass followed by standard CPB during the periods of cardioplegic arrest and reperfusion. In 8 pigs, Heparin-Bonded circuits were used while 8 other pigs received non-bonded circuits. Ischemic damage was assessed by Wall Motion Scores with 2-D echo (4 = normal to -1 = dyskinesia); changes in myocardial tissue pH (Δ pH) with tissue pH probes; the % increase in lung H_2O using wet/dry biopsy weights and the Area of Necrosis/Area of Risk using histochemical staining. Hearts treated with Heparin-Bonded circuits had higher Wall Motion Scores ($3.5 \pm 0.3^*SE$ vs 2.3 ± 0.2), less tissue acidosis (Δ pH = $-0.31 \pm 0.2^*$ vs -0.64 ± 0.08); minimal increase in lung H_2O ($1.7 \pm 0.7^*$ vs $6.1 \pm 0.5\%$), and a decreased Area of Necrosis ($20.3 \pm 2.2^*$ vs $40.4 \pm 1.6\%$). We conclude that Heparin-Bonded circuits significantly decrease myocardial ischemic damage during acute revascularization.

*p < 0.05.