myocardial perfusion imaging agent that show redistribution phenomenon similar to that of Tl.

9:30

830-5 Safety and Feasibility of Dobutamine-Atropine Stress Myocardial Perfusion Scintigraphy. Correlation With Perfusion Abnormalities in 1076 Patients With Known or Suspected Coronary Artery Disease

A. Elhendy, R. Valkema, R.T. van Domburg, P.R. NieropJeroen, J. Bax, G.M. El-Said, M.M. Ibrahim, J.R.T.C. Roelandt. *Thoraxcenter, Rotterdam, The Netherlands*

Background: Dobutamine stress testing is increasingly used for the diagnosis and functional evaluation of coronary artery disease. However, the relation between myocardial perfusion abnormalities and complications of the test has not been studied.

Methods: We studied the hemodynamic profile, safety and feasibility of dobutamine (up to 40 μg/kg/min)- atropine (up to 1 mg) stress myocardial perfusion scintigraphy (with 201 Tl, 99m technetium MIBI or tetrofosmin SPECT) in a consecutive series of 1076 patients (age = 59 ± 11 years, 80% with previous myocardial infarction) referred for evaluation of myocardial ischemia.

Results: No infarction or death occurred during the test. The test was considered feasible (achievement of 85% of the target heart rate or an ischemic endpoint) in 1005 patients (94%). Hypotension (systolic blood pressure drop ≥40 mm Hg) occurred in 37 patients (3.4%). Independent predictors were higher baseline systolic blood pressure ($p < 0.0001$), number of ischemic segments ($p < 0.05$) and older age ($p < 0.05$). Supraventricular tachyarrhythmias occurred in 48 patients (4.4%). Independent predictors were higher infarction (fixed perfusion defect) score ($p < 0.005$) and older age ($p < 0.05$). Ventricular tachycardia occurred in 41 patients (3.8%). Independent predictors were higher infarction score ($p < 0.01$) and male gender ($p < 0.05$). All arrhythmias terminated spontaneously or after metoprolol administration.

Conclusion: Dobutamine-atropine myocardial perfusion scintigraphy is a safe and feasible method for evaluation of coronary artery disease. Patients with more severe fixed perfusion abnormalities are at a higher risk of developing tachyarrhythmias during the test.

9:45

830-6 Additive Diagnostic Values of Gating and Attenuation-Correction to Usual Rest/stress Perfusion SPECT in Coronary Artery Disease?

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Background: Either gated perfusion SPECT or attenuation corrected SPECT can be used to improve specificity in the diagnosis of coronary artery disease (CAD). We questioned if adding either modality improved diagnostic performance of rest-stress perfusion SPECT in patients having intermediate risk in clinical terms.

Methods: We performed rest Tl-201/stress gated attenuation-corrected Tc-99m-MIBI SPECT using ADAC vertex camera in 62 patients (M:32, F:30, 60 ± 10 y, coronary artery stenosis >70%, 1 vessel: 12, 2 vessel: 14, 3 vessel: 13, normal: 32). Three nuclear physicians graded the likelihood of CAD using 5 scales (1: normal to 5: diseased) for each artery territory and per patient. Sensitivity and specificity and area under ROC curves were compared between methods A) attenuation non-corrected SPECT, B) gated SPECT added to A), and C) attenuation corrected SPECT added to B), per observer.

Results: We found no difference in sensitivity (62%, 49%, 46%) and specificity (87%, 96%, 91%) between three methods for each observer. Areas under ROC curves (AUC) for diagnosis of CAD as well as for artery territories did not reveal any differences for each modality ($p > 0.05$ for each comparison).

AUC	A	B	C
obs1	0.752	0.689	0.739
obs2	0.797	0.722	0.750
obs3	0.758	0.713	0.754

Conclusion: In patients with intermediate risk of CAD, viewing gated SPECT and attenuation-corrected SPECT did not improve diagnostic performance.

TUESDAY ORAL

831 Adrenergic Receptors and Cardiac Function

Tuesday, March 31, 1998, 8:30 a.m.–10:00 a.m.
Georgia World Congress Center, Room 255W

8:30

831-1 Adrenergic Stimulation of the Remodeled Rat Infarct Heart: Differences Between Isolated Heart and Myocyte Function

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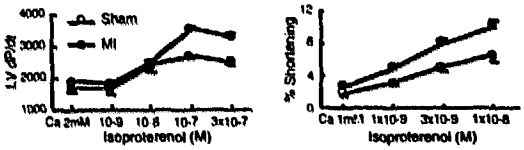
Ventricular remodeling is a key event in the evolution of congestive heart failure following a myocardial infarction (MI). We have recently shown that although remodeled hearts are dysfunctional at the global level, cardiomyocytes isolated from their remote, non-infarcted, hypocontractile segments show no contractile abnormalities both in the basal state and during inotropic stimulation with $[Ca^{2+}]o$. To investigate the effects of β-adrenergic stimulation, we studied isolated heart function in 6 infarcted hearts (6 wks Post-MI) and age-matched normal hearts (n = 6). MI hearts demonstrated significantly reduced contractile function (2 mM $[Ca^{2+}]o$, 5.5 Hz; Langendorff LV developed pressure 31 Vs 84 mmHg, LVdP/dt 933 Vs 1636 mmHg/s, $p < 0.05$) and a depressed Frank-Starling relationship ($p < 0.05$, anova). From a similar group of remodeled MI hearts (n = 8) we isolated myocytes (n = 37) and compared their contractile function (video edge detection) and $[Ca^{2+}]i$ kinetics (Fura-2) with 33 myocytes from sham operated hearts (n = 4). Isoproterenol caused a dose-dependent increase ($p < 0.001$, repeated measures anova) in both contractile (myocyte %shortening, velocity of shortening) and $[Ca^{2+}]i$ parameters (Fura-2 Ratio Amplitude, Velocity of rise in Fura-2 Ratio). The increase in these parameters was significantly more in MI than in Sham myocytes ($p < 0.05$ anova)



These data suggest a dissociation in the β-adrenergic response between the isolated heart and isolated cardiac myocytes in this model. The down-regulation of this response in the global function cannot be explained by altered β-adrenergic response in the myocytes isolated from the remodeled myocardium.

8:45

831-2 Chronic Pressure Overload is Associated With Cardiocyte Apoptosis, but Does Not Increase Vulnerability to β-Adrenergic Mediated Cardiocyte Apoptosis

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Background: The transition from left ventricular hypertrophy to heart failure may be partially mediated by apoptotic cell loss. To explore this hypothesis, we asked whether hearts hypertrophied secondary to abdominal aortic banding manifest cardiocyte apoptosis (Apo) and if they are additionally susceptible to damage by β-adrenergic stimulation.

Methods: The suprarenal abdominal aorta of rats was partially ligated. After two weeks, rats were treated with either vehicle (B–C) or Isoproterenol (Iso, 400 μ g/kg/hr, B–I by Alzet minipump) for 24 hrs. Age and weight matched rats were treated either with vehicle (C) or Iso (C–I) for 24 hrs served as controls. 5 μm sections from paraffin embedded hearts at the mid-ventricle level were analyzed by TUNEL method to assess the extent of Apo.

Results:

	C	C–I	B–C	B–I
H/B (mg/g)	2.55 ± 0.10	2.86 ± 0.11	3.12 ± 0.09*	4.31 ± 0.17*#
Apo (per area)	1.1 ± 0.5	8.5 ± 1.5*	7.9 ± 1.5*	7.5 ± 1.3*
Apo (per cells)	0.14 ± 0.07	1.43 ± 0.28*	1.08 ± 0.23*	1.24 ± 0.28*
Number of rats	5	5	5	6

(Data are mean ± SEM. H/B; heart: body weight ratio, per area; per cm^2, per cells; per 10,000 cardiocytes, *$p < 0.05$ vs. C; #$p < 0.05$ vs. B–C).

Conclusion: Both cardiac hypertrophy secondary to abdominal aorta banding and β-adrenergic stimulation induce Apo, no synergistic effect was seen when these stimuli were superimposed.