action of UFH in vitro. In light of these results, a reevaluation of UFH dosing in combination with each individual fibrin specific thrombolytic agent used for AMI is warranted in the clinical setting.

1192-34 Grafting of Three-dimensional Collagen Type 1 Scaffold on Injured Myocardium Induces Neovascularization and Improves Cardiac Function

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Grafting cells into cardiac muscle has been proposed used as a potential mechanism for augmenting cardiomyocyte numbers in the adult heart. The fate and effect of engrafting fetal cells into infarcted hearts remains unclear. One approach to overcoming this limitation is to graft a naturally modified extracellular matrix (collagen-1) scaffold onto the myocardium. We found, three-weeks after grafting, the 3-D collagen scaffold was integrated completely with the cryo-injured myocardium. The scaffold prevented LV dilation. At zero left ventricular (LV) pressure, the LV lumen diameter and the outer diameter measured at the equator were decreased from 11041±212 to 9144±135 mm, and 13469 ±187 to 11673 ±104, N=4, respectively). The scaffold improved cardiac function by shifting of the LV pressure-volume curve to the left toward control. It induced neoangiogenesis with the graft (10 neovessel per 40x field versus zero), with the new vessels connected to native coronary vasculature in the noninfarcted myocardium. In addition, this neovascularization was associated with recruitment of endothelial progenitor stem cells, $CD34^+$. When compared to the infarcted rats, hemodynamics parameters measured at 3-wks after scaffold grafting were not different. Thus, this study shows that grafting 3-D collagen-1 scaffold onto an injured myocardium integrates with the tissue, allows for cell growth and differentiation, induces mature vessel formation within the graft, and results in improved cardiac diastolic function by preventing LV dilation.

1192-35 80-Lead Body Map Detects Acute ST Elevation Myocardial Infarction Missed by Standard 12-Lead Electrocardiography

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Background: We conducted a multicenter clinical trial to determine whether an 80-lead PRIME-ECG body map system (PRIME) could detect more acute ST-segment elevation MIs (STEMI) than standard 12-lead ECGs (ECG) in chest pain patients presenting to 4 hospital EDs (Virginia Commonwealth University, Richmond, VA; Manchester Royal Infirmary, Manchester, England; Maine Medical Center, Portland, ME; Royal Victoria Hospital, Belfast, Northern Ireland).
Methods: A trained technician performed an ECG and PRIME on all consenting adult ED chest pain patients in whom MI or ischemia was considered clinically. Sensitivity and specificity of each test for detecting STEMI was calculated using 3 different MI definitions, all of which required diagnostic ST elevation on either ECG or PRIME plus: 1) elevated CKMB (CKMB-MI); 2) elevated troponin (TROP-MI); or 3) a clinical discharge diagnosis of acute MI (CLIN-MI).
Results (Table):
The MI locations most commonly missed by ECG but detected by PRIME were: 1) CKMB-MI: 1 septal, 4 posterior (1 infero-post), 1 inferior; 2) CLIN-MI: 1 septal, 8 posterior (3 infero-post), 1 inferior; and 3) TROP-MI: 2 septal, 9 posterior (3 infero-post), 1 inferior. The incidence of extensive right ventricular involvement diagnosable only by PRIME in inferior STEMI was: CKMB-MI 2/9 (22%), TROP-MI 2/9 (22%), CLIN-MI 6/18 (33%).
Conclusions:
PRIME is more sensitive for detecting STEMI than ECG, but has similar specificity. Posterior MIs are most commonly missed by ECG but detected by PRIME.

	N	ECG sensitivity	PRIME sensitivity	p	ECG specificity	PRIME specificity	p
CKMB-MI	22/365	72.7%	100%	.02	97.1%	96.5%	ns
TROP-MI	28/225	57.1%	92.9%	.008	96.5%	94.9%	ns
CLIN-MI	41/647	75.6%	90.2%	.09	98.0%	96.7%	ns

1192-36 Rest Myocardial Perfusion Imaging Results in Patients With Troponin I Elevations

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Background: Myocardial perfusion imaging (MPI) is used often to identify patients (pts) with myocardial infarction (MI). Its diagnostic accuracy in pts with smaller amounts of damage as assessed by troponin, has not been well studied. **Methods:** Pts considered low risk for MI underwent rest gated tomographic MPI and serial marker assessment (CK, CK-MB and TnI) as part of standard chest pain evaluation protocol. Pts with TnI elevations were analyzed further for this study. MI was defined as CK-MB≥8 ng/mL. MPI was considered positive if there was a perfusion defect in association with abnormal wall motion or thickening. Short axis images were divided into 17 segments and graded on a 4 point scale (0 normal, 3 high grade or absent perfusion) and a summed defect score (SDS) was derived. **Results**: A total of 214 pts had MPI and TnI elevations, of whom 93 (39%) did not meet CK-MB MI criteria. MPI was positive in 181 (75%) including 63% of TnI positive but CK-MB MI negative pts. Pts with positive MPI had a mean 2.6±1.3 segments abnormal and a mean SDS of 6.1±3.7. Pts with negative MPI had smaller MIs (peak CK-MB 18±23 vs 43±66 ng/mL, p<0.001), and were more likely to have non-significant coronary disease on angiography (37% vs 17%, p<0.05) than those with positive MPI. Higher SDS was associated with larger MIs as estimated by peak CK-MB and CK (Table). **Conclusions:** MPI and TnI offer complementary data for assessing pts with possible MI. Pts with negative MPI have small MIs and less extensive coronary disease.

SDS	0	1-3	4-6	7-9	≥ 10
% In Each Group	25	17	26	14	11
Median Peak CK-MB, ng/mL	7.2	16	20	31	43
Median Peak CK, U/L	186	263	383	383	572

1192-37 Infarct Size Limitation by Gene Transfer of Baculoviral P35 Into Rat Myocardium

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Background: Apoptosis plays a pivotal role in determining the extent of injury during myocardial infarction. Inhibition of apoptosis has been shown to protect myocardium from damage during ischemia and reperfusion. The aim of this study was to determine whether gene transfer of baculoviral protein p35, a potent inhibitor of apoptosis, into myocardium could limit infarct size. **Methods:** Male Wistar rats were directly injected into the left anterior wall with adenoviral vector encoding baculoviral p35. 72 hours later hearts were mounted on a Langendorff apparatus and perfused. The left anterior descending coronary artery was ligated for 30 minutes and reperfused for 2 hours. Area at risk and infarct size were determined by staining with bromophenolblue and triphenoltetrazoliumchloride (TTC), respectively. Apoptosis was measured by assessing the DNA-laddering, and by in-situ staining for DNA-strand breaks (TUNEL-assay) and active caspase-3. **Results:** Infarct size was significantly reduced in animals treated with p35 (59 +/- 3 % reduction, p<0.05), whereas no reduction was seen when only control vector was injected. Apoptosis was diminished significantly only in animals treated with p35. **Conclusion:** Gene transfer of the baculoviral protein p35 into the left ventricular myocardium diminishes caspase-3 activity and thereby reduces infarct size after regional ischemia and reperfusion.

POSTER SESSION

1193 Systemic Markers and Acute Coronary Syndromes

Tuesday, March 19, 2002, 3:00 p.m.-5:00 p.m.
Georgia World Congress Center, Hall G
Presentation Hour: 4:00 p.m.-5:00 p.m.

1193-27 Comparative Analyses of the Prognostic Value of Novel Cardiac Markers in Patients With Non-ST Elevation Acute Coronary Syndromes

Oscar Bazzino, José L. Navarro Estrada, Fernando Botto, María I. Sosa Liprandi, Juan J. Fuselli, José Santopinto, Rodolfo Ahuad, Roberto Nordaby, Simón Salszberg, Alfredo Hirschson Prado, José Gant López, Mario Russo, Fernando Sokn, Marcelo Pérez, Carlos Bruno, *PACS Investigators, Buenos Aires, Argentina.*

Background: Cardiac markers have different release characteristics, sensitivity and specificity. A multi-marker strategy testing for prognostic markers may improve risk stratification in patients (pts) with non-ST-elevation acute coronary syndromes (NSTACS) Methods: This prospective multicenter cohort study included 1000 consecutive pts admitted with NSTACS. Baseline clinical and electrocardiographic data were recorded. Single assays of 3rd generation troponin T(TnT), high sensitivity C-reactive protein (CRP) and myoglobin (Myo), were performed after 9 hours (median) from symptom onset. Results were kept blinded until the end of the study. The association between markers and 180 day events was examined by chi-square and logistic regression analyses. The primary endpoint was the 180-day incidence of death or myocardial infarction (D/AMI). Results: D/AMI occurred in 9.4% of pts at 180 days. Prognostic values of total CK (upper normal value x2),TnT (>0.01ng/ml), CRP (>3 mg/l)and Myo (>80 ng/ml),were:

180 day D/AMI

	n	ER(%)	OR	95%CI	S	Sp	PPV	NPV
CRP>0.3 mg/l	602	12.0	2.2	1.3-3.6	75.7	41.4	12.0	94.2
TnT>0.01 ng/ml	428	15.2	3.2	2.0-5.0	68.4	59.9	15.2	94.8
Myo>80 ng/ml	243	18.9	3.3	2.2-5.2	48.4	78.2	18.9	93.5
Total CK x 2	138	20.3	3.0	1.8-5.9	29.5	87.8	20.3	92.2

ER: Event Rate, S: sensitivity, Sp: specificity, PPV: positive predictive value, NPV: negative predictive value, OR: odds ratio

By regression analyses, total CK was not an independent predictor. The TnT-only model significantly improved by adding Myo and CRP data (delta chi-square:20.03, p=0.006). Conclusions: In a non selected population of NSTACS, a model including 4 cardiac markers showed that TnT, Myo and CRP are independent predictors of adverse outcome. Total CK has not additional prognostic value on top of TnT, Myo and CRP.A combination of these markers may assist to identify the pts at increased risk of future events.