# 388A ABSTRACTS - Myocardial Ischemia and Infarction

tiveness of monteplase administration prior to emergent PCI in AMI. METHOD: Out of 243 consecutive AMI from 1998 to 2002, we enrolled into the COMA trial 108 patients who were under 75 years of age and had been admitted within 12 hrs after the onset of AMI. Patients were randomly assigned to receive direct PCI (group P, n=57) or PCI followed by pre-treatment with intravenous monteplase (27500 IU/kg, group M, n=51). RESULTS: Primary endpoint of this trial was left ventricular function (EF) at 6 months follow-up. In the initial CAG before PCI, TIMI-3 flow was obtained in 29% of group M, but in only 7% of group P (P=0.002). There was no significant difference in the PCI success rate, major cardiac or bleeding complications in both groups. No-reflow phenomenon in group P was observed more frequently than group M (17.5% vs 4.4%, P=0.04). There were no significant differences EF in both groups. Thus, we divided the group M into subgroups according to whether or not TIMI-3 flow was observed at initial CAG. In the group M with TIMI-3 flow, LVEDVI was smaller and the EF was greater than Group P (66.0+/-1.8 vs 58.6+/-1.4, P=0.002). QCA results showed that the minimal lumen diameter was larger in the monteplase group M immediately after PCI, and the difference was even greater at 6 months. CONCLUSION: Intravenous injection of monteplase can promote rapid reperfusion and appears to maintain LV function, to suppress LV remodeling and late restenosis. We propose a combination therapy of PCI with monteplase injection in order to achieve reperfusion as early as possible.

1191-29

## Angiographic Predictors of Left Ventricular Ejection Fraction After Successful Angioplasty in Acute Myocardial Infarction: An Angiographic Risk Score for Use in the Catheterization Laboratory

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Background: We investigated the value of coronary angiographic parameters in patients with successful primary angioplasty to predict left ventricular function and 30 day mortality, using a simple angiographic risk score.

Methods: In 608 consecutive patients with successful primary angioplasty, we assessed the infarct related artery, antegrade flow before treatment, presence of distal emboilsation and myocardial blush grade on the coronary angiogram made immediately after coronary angioplasty. Endpoints were death after 30 days and left ventricular ejection fraction.

Results: A simple risk score was conceived attributing points to predictive variables. Multivariate analysis revealed that LAD related infarction (OR 8.4, CI:5.4-13.1, p<0.001; 3 points), TIMI 0-2 flow before angioplasty (OR 2.2, Cl:1.1-4.2, p<0.02; 1 point), myocardial blush 0 or 1 (OR 2.5, CI: 1.2-5.1, p=0.01; 1 point) and distal embolisation (2.2, CI:1.1-4.2, p=0.02; 1 point) were independent predictors of left ventricular ejection fraction <40% after successful angioplasty. Using this score (0 to 6 points), residual left ventricular ejection fraction and 30 day mortality can be predicted. Patients with 0 or 1 point had an ejection fraction of 49.5% ± 8.4 and a 30 day mortality of 0.8%. Patients with a score of 2 or 3 had an ejection fraction of  $44.9\% \pm 10.3$  and a 30 day mortality of 2.8%, whereas patients with a score of 4 had an ejection fraction of 38.2% ± 10.8 and a 30 day mortality of 2.7%. Patients with a score of 5 or 6 had an ejection fraction of 32.0% ± 9.4 and a 30 days mortality of 6.9% (p for trend <0.01 for mortality and <0.001 for ejection fraction). Conclusions: Successful angioplasty for acute myocardial infarction leads to a good clinical outcome. However, a simple angiographic score based on 4 angiographic parameters can further predict ejection fraction and mortality when leaving the catheterization laboratory in patients with successful reperfusion by primary angioplasty for acute myocardial infarction.

1191-30

## The Vasopeptidase Inhibitor, Omapatrilat, Increases Survival, Improves Cardiac Function, and Attenuates Ventricular Remodeling After Coronary Artery Ligation in Insulin Resistant Zucker Fatty Rats

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Background: Vasopeptidase inhibitors (VPI's) block both angiotensin converting enzyme and neutral endopeptidase. By reducing angiotensin II and enhancing endogenous vasodilators substances, VPI's may be particularly beneficial in situation where an imbalance in favour of vasoconstrictor substances occurs, such as in insulin resistance and peri and post myocardial infarction (MI).

**Methods:** MI was induced in 154 male Zucker fatty rats by ligating the left anterior coronary artery. Rats were untreated or pre-treated 7 days with omapatrilat 10 mg/kg/day. Post MI (30 days), an echocardiogram was done and at 38 days hemodynamic measurements were performed, the rats scarified, and morphologic measurements done. A large MI was defined as an infarct size  $\geq$  35 mm<sup>2</sup> and a moderate MI was defined as an infarct size  $\geq$  35 mm<sup>2</sup>.

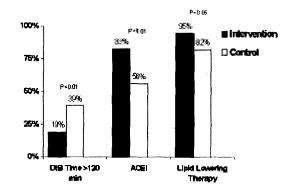
**Results:** Omapatrilat normalized blood glucose and resulted in a better early (24h) survival post MI (63% vs 41%, p=0.003) that was maintained for 38 days, the time of sacrifice (35% vs 22%, p=0.01). Omapatrilat reduced the number of rats with a large MI as compared with untreated, an effect that presumably occurred during the first few hours post MI. Long term (38 days), omapatrilat resulted in beneficial ventricular remodeling as reflected by a reduction in left ventricular (LV) diastolic (1.12cm for omapatrilat vs 1.02cm for untreated, p=0.001) and systolic (1.02cm for omapatrilat vs 0.88cm for untreated, p=0.03) circumference by echocardiography in large MI as compared with untreated. This resulted in hemodynamic improvement, and a decrease in LV and pulmonary weight indicating a decrease in reactive hypertrophy and pulmonary congestion at the time of sacrifice. **Conclusion:** This study indicates that pre treatment with the VPI omapatrilat increases early (24 hour) post-MI survival in insulin resistant rats by reducing MI size. This improvement is sustained over a 38 day follow-up and accompanied by normalization of blood glucose, improvement of LV remodeling and hemodynamic parameters.

# JACC March 19, 2003

#### Clinical Pathway Improves Acute Myocardial Infarction Quality Indicators

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Despite published guidelines, the majority of patients with acute MI still do not achieve the goals of door-to-balloon time (DtB) 90+/-30min or appropriate drug utilization. We hypothesized that a clinical pathway could increase compliance with treatment guidelines. To determine the impact of a clinical MI pathway on quality indicators, two cohorts of patients undergoing primary PTCA for acute MI were identified (control group n = 89and intervention group n = 86). The medical records of the control group (1998-99) were reviewed for DtB and ASA, beta-blocker (BB), ACE inhibitor (ACEI), and lipid lowering therapy use and compared to the prospective data of the intervention group (2000-01). The impact of the pathway was evaluated using the Wilcoxon Rank Sum Test, odds ratios and Chi Square tests. DtB was significantly lower in the intervention group (91.5 vs 108 min., p<0.01). The intervention group had fewer patients exceeding the guidelines with a DtB > 120 min (OR = 0.38, p < 0.01). The intervention group was more likely to have an ACEI (OR = 3.7, p < 0.01) or lipid lowering therapy (OR = 3.7,  $p \approx 0.02$ ) at discharge compared with the control group (figure). ASA and BB use was appropriate and was not different (ASA: 95.2% vs 96.2%; BB: 93.5% vs 92.6%). These data suggest that in the current era of published treatment guidelines, implementation of a clinical pathway can still improve MI quality indicators and further improve clinical care.



1191-46 Which Is the

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Which Is the Best Streptokinase Regimen in ST Acute Myocardial Infarction? A Prospective Study

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Objective. In a prospective study we compared the safety and efficacy of three Streptokinase regimens: 1. Infusion of 1.5 M.U./60 min (the SK1.5/60 regimen). 2. Infusion of 1.5 MU/20 min (the SK1.5/20 regimen). 3. Bolus of 0.75 M.U./10 min repeated after 50 min. only if no signs of coronary reperfusion have been detected (the SK 0.75/10 regimen) in pts. with ST acute myocardial infarction (STAMI).

Methods. A group of 849 patients admitted within the first 6 hours after the onset of the chest pain revealing STAMI) have been divided in 3 subgroups according to the thrombolytic regimen used: SK1.5/60 (306 pts.), SK1.5/20 min. (349 pts.) and SK0.75 MU/10 min. (195 pts.). All pts received heparin (1000 i.u./hour, 48-72 hours) and aspirin.. Three noninvasive reperfusion criteria have been used: 1.Rapid resolution of the chest pain. 2.Rapid decreasing of the ST segment elevations by more than 50% from the initial value. 3.Rapid increasing of the CK and CK-MB with a peak within the first 12 hrs. The incidence of haemorrhagic events, the ratio of coronary reperfusion, and the in hospital mortality were evaluated.

Results. The ratios of the coronary reperfusion were 73.3% in the SK1.5/20 and 73.8% in the SK0.75/10 sbgroup. These ratios were equal each other but significant higher that the one of 61.7% registered in the SK1.5/60 min. subgroup. (p=0.002 and 0.015, respectively). In pts. younger than 75 the 30 day mortality was 5.59% (SK1.5/20), 5.38% (SK0.75/10) and 11.4% (SK1.5/60) (1.5/20 vs 1.5/60 p= 0.008; 0.75/10 vs 1.5/20 p=0.029). One patient in the SK1.5/60 subgroup had signs of ischemic stroke. The incidence of the major haemorrhagic events were 2.94% (SK1.5/60), 0.85% (SK1.5/20) and 0.0% (SK0.75/10) (non-significant differences)

Conclusions. Two accelerated SK regimens (SK1.5/20 and 0.75/10) are safe, equally efficient and lead toward a significant higher ratio of coronary reperfusion and a significant lower mortality as compared to the classical SK1.5/60 one in pts. younger that 75.

## 1191-47 Influence of Biochemical Markers on Thrombolysis Effectiveness Early in the Course of ST-Segment Elevation Myocardial Infarction

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Background: Whilst the benefit of intravenous thrombolysis (IT) in ST-segment elevation myocardial infarction (STEMI) is unequivocal, reperfusion fails in many pts. The aim of this was to evaluate the role of plasma levels of either high sensitivity C-reactive protein (hs-CRP), cardiac troponin I (cTnl), lipoprotein (a) Lp(a), or fibrinogen (Fb) on IT