was also observed between JACC March 19, 2003 ABSTRACTS - Myocardial Ischemia and Infarction 355A

dent collateral flow may reduce microvascular damage in the setting of AMI treated with imab increases PMN apoptotic rate in patients with severe UA. Such increased blood
cling. imab. 
primary PTCA, thus favouring ContractlIe recovery and preventing left ventricular remod- clearance of circulating PMN represents a novel anb-inflammatory mechanism of Abcix-

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cytometry (Annexin V-FITC, Immunotech, Marseille) after PMN isolation from peripheral vasculature in the rat coronary altery ligation model of CHF 5 days after adenoviral-medi-

BACKGROUND: Cardiac troponln-I (cTnl) IS a more specific and sensitive biomarker (AMI) is unclear.

METHODS Seven hundred seventy-six consecutive hospital admissions for suspected acute coronarv syndrome (ACS) to an urban acute care hospital over a four month period

ble angina (UA) patients. Neutrophil apoptosis. a key mechanism to control the intensity
disease. In this study we tested the hypothesis wether reconstituted high density lipopro-

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line (ACH) as an endothellum-dependent vasodilator, nitroglycerine (NG) as direct dilator

rate with early T-wave inversion in the first 90 minutes (61.3% vs 41.7% p=O.O089). LVEF
cytokine preconditioning which favours more rapid and effective reperfusion and limits infarct

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dialysis, and (2) positron emission tomography (PET) to assess myocardial perfusion at rest and

Conclusions Can Preinfarction Angina Limit Infarct Size in Patients With a First Q-Wave Myocardial Infarction Undergoing Primary Percutaneous Coronary Angioplasty?

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BACKGROUND: In patients with acute myocardial infarction (AMI), preinfarction angina (PA) promotes protective effects in the ischaemic myocardium, reducing the necrosis extension, but its impact in patients treated with primary percutaneous coronary angioplasty (PTST) is not already defined.

METHODS: We studied 183 patients (mean age 66.4 years, 26.8% female), hospitalised in our coronary care unit for a first Q wave AMI and treated with successful primary PTST. We defined successful PTST when at least 1 of the following criteria was reached: (1) early ST-resolution >50% or (2) early T-wave inversion in the first 90 min after PTST, (3) repeat percutaneous ventricular arrhythmias, (4) CK and CK-MB time-to-peak <12 hours. Major Adverse Cardiac Events (MACES) were evaluated at hospital discharge.

RESULTS: In the study population patients with PA were 75 (mean age 70.1 years 30.7% female). No differences in age, sex or prevalence of coronary risk factors were evidenced. When PA was present, higher mean nh-m scores (2.4 v 1.51 p=0.042) and successful reperfusion (53.3% vs 36.1% p=0.0207) were reached, especially in association with early T-wave inversion in the first 90 minutes (61.3% vs 47.1% p=0.0089). LVEF on admission was similarly impaired in both PA and non-PA group (32.8% vs 43.6% p=0.0043) but incidence of early left ventricular expansion was sensibly inferior (1.4% vs 11.1% p=0.0496). At pre-discharge 2D-echo control, per cent improvement of LVFED was demodulated superior (-26.5% vs -0.2% p=0.0276), while in-hospital MACES was signiticantly limited (8.0% vs 18.5% p=0.0450).

CONCLUSIONS: Our results suggest that in patients with AMI treated with primary PTST, PA induces protective effects in the ischaemic myocardium, probably due to myocyte preconditioning which favours more rapid and effective reperfusion and limits infarct size, improving left ventricular function and in-hospital prognosis.

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Role of Endothelial Nitric Oxide Synthese in Arterial Remodeling in Heart Failure

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To examine the role of nitric oxide (NO) in systemic arterial remodeling in congeative heart failure (CHF) in vivo, we measured arteriolar morphology in the resistance hindlimb vasculature in the rat coronary artery ligation model of CHF 5 days after adenosine-mediat-

can transfer of endothelial nitric oxide synthase (eNOS). The presence of CHF was documented with increases (P<0.05) in left ventricular (LV) end-diastolic pressure, decreases LV systolic pressure, and LV dP/dt in the resistance vessels, of CHF rats,