bits. To determine whether rTFPI limits myocardial reperfusion injury, either rTFPI (100 μg/kg/min, n = 7), heparin (bolus: 150 U/kg; infusion: 50 U/kg/hr, n = 4) or arginine-phosphate vehicle (n = 7) were administered intravenously to conscious dogs beginning 15 min before and for 60 min after release from a 2 hr balloon occlusion of the proximal left anterior descending coronary artery. Reperfusion was maintained as verified with a proximal Doppler probe. Animals given rTFPI exhibited increases in PT (21 ± 7 (SD) sec, n = 7, $p < 0.01$ compared with baseline) but no increase in aPTT, whereas those given heparin exhibited marked increases in aPTT (72 ± 9 sec, n = 3, $p < 0.01$). Two days after reperfusion, the coronary artery was reoccluded, the heart was perfused with Lissamine green to delineate the area at risk, and infarcted areas were demarcated by incubation of LV sections in 2,3,5-triphenyl-tetrazolium chloride (TTC). Risk and infarcted areas were quantified by computerized planimetry. Risk area was similar among groups (28 ± 7% of LV in rTFPI, 31 ± 5% in heparin, 30 ± 3% in vehicle), but infarct area expressed as a percentage of the area at risk decreased significantly in dogs which received rTFPI (17 ± 16%) compared with either heparin (39 ± 23%, p = 0.09), or vehicle (39 ± 21%, p = 0.04). Thus, rTFPI appears to limit myocardial reperfusion injury and may do so either by more efficient anticoagulation or by a mechanism other than inhibition of prothrombin activation.

923-6 Intravenous Adenosine and Lidocaine to Limit Reperfusion Injury During Acute Myocardial Infarction: Preliminary Data

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Adenosine (ADO) and lidocaine (LDO) given prior to restoration of blood flow reduces reperfusion injury in animals. We conducted a pilot study of intravenous ADO and LDO in pts undergoing direct angioplasty for acute myocardial infarction (AMI). Pts with ≤12 hours of chest pain and electrocardiographic evidence of AMI were given LDO 1 mg/kg iv bolus and 2 mg/min iv infusion beginning at the time of recruitment, and ADO 70 mcg/kg iv infusion beginning when coronary occlusion (TIMI grade 0–1 blood flow) was confirmed angiographically. Pts with bronchospasm, blood pressure <100 mmHg, or >1° heart block were excluded. ADO and LDO were given for 1 hour after vessel patency was restored. Myocardial area at risk and final infarction area were measured with serial Tc-99m-sestamibi perfusion studies (prior to angioplasty, before hospital discharge and 6 weeks after discharge). A salvage index (SI) was constructed by correcting the change in sestamibi perfusion defect for the mass of myocardium at risk. Analysis of 25 patients completing the protocol revealed a mean (±SD) salvage of 20 ± 17% and SI = 0.55. Salvage and SI were 25 ± 18% and 0.54 for anterior infarctions, 13 ± 5% and 0.57 for inferior infarctions, respectively. These data were compared to an historical control group consisting of 50 patients undergoing direct angioplasty for AMI without adjunctive ADO/LDO. After adjustment for time to treatment and perfusion nadir, analysis of covariance revealed a similar degree of early salvage in the study and control groups ($p = 0.3$). However, at 6 weeks, the median infarct size for study pts was 0. Using logistic regression analysis, significantly more study pts had no final measureable infarction at 6 weeks than control pts at hospital discharge ($p = 0.007$). After adjusting for infarct size, location and time to treatment, this difference persisted ($p = 0.04$).

Conclusions; Adjunctive ADO and LDO during angioplasty for AMI may favorably affect late final infarction size. Randomized studies assessing 6 week final infarction size are needed.

923-7 Infarct Artery Patency does not Prevent LV Remodeling

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Prior studies suggest that an occluded infarct-related artery (IRA) is a risk factor for progressive LV remodeling following AMI. The purpose of this study was to determine whether LV dilatation also occurred in patients with patent IRAs. Serial echocardiograms at baseline and at 1 year (1 Y) were obtained in 420 SAVE patients, of whom 185 (44.0%) had a pre-randomization cardiac catheterization reviewed by a central Core laboratory. A patent IRA was present in 145 (78.4%) of these patients with both catheterization and echo data. Catheterization occurred an average of 4.4 ± 4.2 days post AMI. Patent IRA patients were 55 ± 11 years old, and 76.6% male. Ejection fraction was 31.3% ± 5.8. Echo LV size echo was expressed as end diastolic area (EDA), and end systolic area (ESA). LV dilatation occurred in the overall group (baseline EDA 69.1 ± 11.8, 1 Y 73.3 ± 13.1, $p < 0.001$; baseline ESA 48.6 ± 10.9, 1 Y 53.1 ± 14.2, $p < 0.001$). Randomization to placebo (PLA, n = 67) or captopril (CAP, n = 78) occurred 3–16 days post AMI. There was a trend for less LV dilatation in captopril-treated patients.

	PLA	CAP	P
EDA change	5.6 ± 8.1	3.1 ± 7.7	0.06
ESA change	5.6 ± 7.8	3.6 ± 9.0	0.15

Although the occluded IRA is a known risk factor for LV remodeling, this study demonstrates that LV enlargement can occur in patients with patent IRAs.

924 Acute Myocardial Infarction: Triggers and Concomitant Medical Conditions

Monday, March 20, 1995, 3:00 p.m.–5:00 p.m.
Ernest N. Morial Convention Center, Hall E
Presentation Hour: 3:00 p.m.–4:00 p.m.

924-116 Activity at the Onset of Symptoms and Outcome of Acute Myocardial Infarction

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Recent studies have confirmed that vigorous exercise can trigger myocardial infarction (MI), but it is not clear whether outcome is influenced by the activity at the onset of symptoms. This study compares clinical features and in hospital mortality for patients (pts) whose symptoms of MI began at rest, in bed, or during or after exercise.

A standard questionnaire which included information on activity at the onset of symptoms, risk factors and outcome was completed following admission for all pts admitted to the CCU from 1975 to 1993. The study population is 2468 pts with a first MI. At the onset of symptoms 40% of pts were resting, 33% were in bed and 27% were or had been exercising (>4 METS) during the preceding 30 minutes (Ex).

Pts with onset during exercise were younger (Ex 59.0 (SD10.7), Rest 60.8 (10.4), Bed 61.9 (10.6) years, $p < 0.001$) and more likely to be male (Ex 79%, Rest 67%, Bed 67%, $p < 0.001$). Cardiovascular risk factors and time delay to admission were similar for all groups. After adjusting for age and sex, pts whose symptoms began during exercise had a lower mortality (OR 0.63, 95% CI 0.44, 0.90) and had fewer in-hospital cardiac arrests (OR 0.61, CI 0.39, 0.95) than those whose symptoms began at rest. Pre-hospital cardiac arrest was not less likely (OR 1.18, CI 0.82, 1.69). Pts with onset of symptoms in bed had a higher mortality (OR 1.27, CI 1.00, 1.61) than those with onset at rest.

Outcome of myocardial infarction is influenced by the activity at the onset of symptoms. This may reflect variations in the pathophysiology of MI occurring under different conditions.

924-117 Cardiac Injury in the Critically Ill: A Surprisingly Common Finding

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Medical intensive care unit (MICU) patients often are subjected to severe cardiovascular stress and thus may be at risk for myocardial ischemia/infarction. To determine the incidence of myocardial injury in such patients, we measured daily levels of cardiac troponin I (cTnI), a long-lived, sensitive and highly specific plasma marker of cardiac injury. Two hundred twenty-seven consecutive patients (242 admissions) were considered for the study; 18 were excluded due to either suspected infarction on admission or inability to obtain the required blood samples. The MICU staff evaluated 121 of the 209 patients (58%) for possible infarction; they were unaware of the cTnI results. Overall, 32 of the 209 patients (15%) had evidence of myocardial damage based on a cTnI value ≥3.1 ng/ml. Evidence of myocardial injury by cTnI was present on admission or day 1 in 29 of the 32 patients. Only 37.5% (12/32) of these patients were diagnosed as having acute infarction by the MICU staff. An additional 9 patients had elevated values of MBCK but were not thought to have suffered cardiac damage. Five of these patients met criteria for infarction with cTnI while 4 did not. Myocardial injury occurred in 21 patients without primary cardiac diagnoses. It was present in 6/71 patients with respiratory disease, 5/54 with GI bleeding, 5/16 with sepsis, 2/13 with ketoacidosis, 1/5 with electrolyte disturbances, 1/3 with neurologic disease and 1 with traumatic hemothorax. Mortality for patients with myocardial injury, whether recognized or not, was 41% compared to 15% for those without injury ($p < 0.001$). Patients with cardiac injury also were more apt to be hypotensive (75% vs. 50%, $p = 0.007$) and require mechanical ventilation (66% vs. 27%, $p < 0.0001$). These data indicate that myocardial injury is surprisingly common in critically ill MICU patients, difficult to recognize, and associated with increased morbidity and mortality.