Conclusions: In a patient population aggressively treated to reopen the infarct related vessel, addition of an angiotensin converting enzyme inhibitor only influenced the process of infarct expansion and remodelling in patients with a severe residual stenosis in the coronary artery.

973-62 Thrombolytic Therapy Can Be Given to Half of Hospitalized Patients With Acute Myocardial Infarction

Christopher J. Ellis, John K. French, Barbara F. Williams, Susan Wyatt, June Poole, Christine Ingram, Miles G. Williams, Hamish H. Hart, Harvey D. White. *Green Lane Hospital, Auckland, New Zealand*

Despite the proven benefits of thrombolytic therapy (TT), only a minority of patients (pts) receive it (35% in the recently reported USA National Registry). We prospectively recorded the number of pts eligible to receive TT when admitted in 1993 from a 1 million catchment population (4 coronary care units, no age limits).

Eligibility was based on ECG criteria: ≥ 2 mm ST elevation in leads V1–3, ≥ 1 mm ST elevation in any other 2 contiguous leads, or new onset of left bundle branch block with symptom onset within 12 hours and no contraindications to TT. We defined 'definite' myocardial infarction (MI) as 2 of: ≥ 20 mins ischemic chest pain, peak CK > 600 u/L (× 2 normal), presence of Q waves; 'probable' MI as CK 300-600 u/L; 'aborted MI' as ST elevation given TT but with no CK rise. Over 1 year there were 1081 pts with 'definite' (n = 948), 'probable' (n = 124), or 'aborted' (n = 9) MIs.

53% (576/1081) pts were ECG criteria and time (< 12 hours) TT eligible, but 47 pts were TT ineligible because of bleeding risk (39 definite, 8 relative contraindications). Hence 49% (529/1081) of pts were actually TT eligible, of whom 470 (89%) were given TT. Overall hospital mortality for TT eligible pts was 12.7% (11.7% if TT treated, 17.0% if not TT treated).

Conclusion: With standard criteria for TT, half of hospitalized pts with a discharge diagnosis of MI are eligible for treatment at admission and should not be denied this life-saving therapy.

973-63 Stent Assisted Direct Balloon Angioplasty for Acute Myocardial Infarction, Prospective Trial

Yoshihisa Nakagawa, Hiroyoshi Yokoi, Hideyuki Nosaka, Hiroatsu Yokoi, Naoya Hamasaki, Takeshi Kimura, Masakiyo Nobuyoshi. *Kitakyushu, Kokura Memorial Hospital, Japan*

Thrombus formation is significant in the pathogenesis of AMI, and thrombus containing lesions have been considered to be contraindication for stenting. Acute reocclusion(AR) in CCU and silent closure(SC) are major problems of Direct Balloon Angioplasty (DBA). From Sep. 1994 to June 1995, AMI patients (pts) under 80 years of age were prospectively treated with new aggressive strategy of "Stent assisted DBA". DBA was attempted in all AMI pts within 12 hours of onset, except LMT infarction and cardiogenic shock. When the results of DBA was unsatisfactory and vessel size was ≥ 3.0 mm, stent implantation was performed subsequently. After stenting, 3 days Heparin, Aspirin and Coumadin were administrated as an ordinary anticoagulation regimen. Of the 111 pts (M/F = 92/19, age 62 ± 11) treated with this protocol, 85 pts(77%) received only DBA and 83 pts(97.6%) achieved successful reperfusion. In two pts, DBA was failed due to massive thrombus. The rest of 26 pts (23%) received successful stent implantation after suboptimal result of DBA. So overall successful reperfusion rate was 83 + 26/111 = 98.2%. AR in CCU occurred in 2 pts(2.3%) after DBA and subacute thrombosis of stent occurred in 2 pts(7.7%). These 4 pts received repeat angioplasty successfully. Two in-hospital death was documented(1.8%). Of the 109 pts dicharged alive, all pts received late angiography(18 ± 7 days). It revealed 1 SC after DBA and there was no SC with stent, so overall late patency rate was 108/109 = 99.1%. In conclusion, stenting for suboptimal DBA had favorable effect to restore and sustain patency and prevent AR and SC. "Stent assisted DBA" is considered to be feasible and effective strategy for AMI. Further angiographic follow up study is ongoing.

973-64 Early Behaviour of Biochemical Markers in Patients With TIMI Grade 2 Flow in the Infarct-Related Artery 90 Min After Thrombolysis for Acute Myocardial Infarction

Thierry Laperche, P. Gabriel Steg. *PERM Study Group, France*

During acute myocardial infarction (MI), Pts with TIMI grade 2 flow in the infarct vessel have a worse prognosis than Pts with TIMI 3 flow. While biochemical markers have been suggested for the non-invasive diagnosis of reperfusion early after thrombolysis, its is important to discriminate Pts with TIMI grade 2 flow from Pts with TIMI grade 3 flow.

97 Pts with acute MI ≤ 6 h, treated with thrombolytics, all underwent 90 min angiography to assess flow in the infarct-vessel (TIMI grade 0–1, TIMI 2, TIMI 3). At the onset of thrombolysis (t0) and 90 min later (t90), all had blood samples drawn to measure myoglobin (Myo), MM3/MM1 CK isoforms, Troponin T (TnT) and CK-MB. The relative increase (RI = t90 – t0/t0) over 90 minutes were calculated in each group. For all the markers studied, the mean t0 and t90 serum values were higher in TIMI 2 Pts than in TIMI 3 Pts ($p < 0.05$ for Myo). RI at t90 were consistently (but not significantly) lower in TIMI 2 Pts than in TIMI 3 Pts. In Pts treated > 3 h after the onset of pain, results of RI were:

	Myo	MM3/MM1	TnT	CK-MB
TIMI 0–1	2.25 ± 4	1.1 ± 1.5	10 ± 22	2.4 ± 2.6
TIMI 2	4.2 ± 4.5	0.9 ± 0.7	9 ± 15	2.2 ± 2.5
TIMI 3	28.5 ± 34*$	10.5 ± 17.8	110 ± 317	10 ± 16

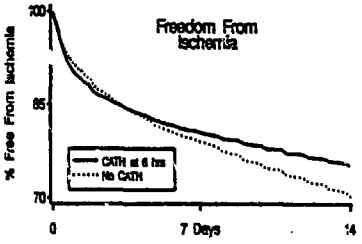
*$p < 0.05$ between TIMI 0–1 and TIMI 3, $p < 0.05$ between TIMI 2 and TIMI 3.

In conclusion, in patients treated > 3 h after onset of pain, when biochemical markers are used to estimate infarct-related artery patency 90 min after thrombolysis, indexes based on relative increase of myoglobin at 90 min tend to separate patients with TIMI 2 and 3 flow.

973-65 Ischemia After Thrombolysis Is Frequent, Somewhat Predictable, and Pre-Empted by Early Angiography

Louise Pilote, Magnus Ohman, Dave P. Miller, Robert M. Califf, Eric J. Topol. *GUSTO-I Investigators, Cleveland Clinic Foundation, Cleveland, OH*

To identify predictors of recurrent ischemia after thrombolysis and to investigate the relationship between ischemia and coronary angiography (angio), we examined the GUSTO-I database. Of the 21,772 US GUSTO-I patients, 6313 (29%) experienced ischemia before discharge. In a multiple Cox regression, women (RR: 1.25; 95% CI: 1.17–1.33), patients with high cholesterol (RR: 1.14; 95% CI: 1.07–1.22) and patients with prior angina (RR: 1.40; 95% CI: 1.32–1.49) had a higher likelihood of ischemia. Current smoking and time to thrombolysis were inversely related to ischemia (RR: 0.86; 95% CI: 0.81–0.92, RR: 0.97; 95% CI: 0.95–0.99, respectively). Patients who underwent angio had a slightly increased risk of ischemia in the 12 hours after angio (RR: 1.2; 95% CI: 1.1–1.4), but ultimately, these patients had a considerably lower risk 1 week after angio than patients without angio (RR: 0.56; 95% CI: 0.45–0.72, Figure).



In conclusion, early angio confers a net 44% reduction in ischemia at 1 week, probably because high risk patients are identified and revascularized.

973-66 A Prospective, Randomized, Controlled Study of Warfarin Plus Aspirin to Prevent Coronary Reocclusion After Thrombolysis

Giovanni Melandri, Nicoletta Candiotti, Franco Semprini, Vittorio Cervi, Letizia Bacchi Reggiani, Angelo Branzi, Bruno Magnani. *Institute of Cardiology, University of Bologna, Italy*

Aspirin (A) and warfarin (W) are poor agents in the prevention of coronary (re)occlusion after thrombolysis. Since platelets and thrombin are both important mediators of coronary (re)thrombosis we randomized 65 pts to receive either A (160 mg daily) or A (same dosage) plus W (target INR 2.5 to 3.5) after thrombolysis (front-loaded rt-PA).

Coronary angiography was performed within one week and after three months. The INR in the A + W group was 2.7 ± 0.7 at one month, 2.5 ± 0.9 at two months and 2.5 ± 0.6 at three months. 48% INR values were therapeutic, 46% subtherapeutic and 6% supratherapeutic. The two groups did not differ in age, sex, site of infarction, risk factors. In the group treated with A + W we observed a decrease in the overall % stenosis (74 ± 26 to 66 ± 29, p = 0.028) and in culprit vessel % stenosis (82 ± 14 to 74 ± 24, p = 0.07). In the A group changes were insignificant (66 ± 28 to 68 ± 29, p = NS and 77 ± 17 to 83 ± 23, p = NS, respectively). In the A + W group the culprit vessel % stenosis was improved in 43%, unchanged in 36% and worsened in 21%. In the A group the figures were 8%, 36% and 56% respectively (p = 0.0001). When all vessels were considered, again, there was a benefit in