

956-34 Effect of Time to Thrombolytic Therapy on Infarct Size Assessed by Tomographic Sestamibi Perfusion Imaging

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Previous attempts to demonstrate an association between infarct size (as assessed by left ventricular function) and the duration of chest pain prior to initiation of thrombolytic therapy have yielded conflicting results. The CORE trial is an international, randomized dose-ranging trial of poloxamer 188 which randomized 2,954 patients with acute myocardial infarction (< 12 hours' duration) between 5/4/94 and 6/25/95. A substudy used tomographic sestamibi perfusion imaging to assess infarct size in 1,180 patients. Of these patients, 1065 received acute thrombolytic therapy (streptokinase or t-PA) and had analyzable images. Infarct size as a percent of the left ventricle was measured using previously established techniques at a central laboratory. The time to thrombolytic therapy was significantly ($p = 0.014$) associated with infarct size:

Time to Thrombolysis	n	Infarct Size (% LV)
< 2 hours	309	19.5 ± 18.8
2-4 hours	396	22.5 ± 20.0
4-12 hours	360	23.9 ± 20.5

After adjustment for infarct location, prior infarct, thrombolytic and dose of poloxamer 188, time remained significant ($p = 0.027$). However, a 2 hr increase in time was associated with an increase in infarct size of only 1.1% of the LV, compared to an increase of 8.1% for prior infarction and 16.7% for anterior infarction.

Conclusion: Time to initiation of thrombolytic therapy is a significant determinant of infarct size as assessed by tomographic sestamibi perfusion imaging in the CORE trial. The magnitude of its effect is small compared to prior infarction or infarct location.

956-35 The Fate of Patients With a Previous Stroke Given Thrombolysis for Acute Myocardial Infarction

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Patients (pts) with a history of stroke (HS) have a relative contraindication for thrombolytic therapy (TT) after acute myocardial infarction (AMI). We compared the characteristics and outcome of pts with HS treated or not treated with TT among 2010 unselected pts after AMI, hospitalized in 25 CCUs in Israel.

	HS and TT (n = 29)	HS without TT (n = 87)	p Value
Men (%)	76	72	NS
Age (mean ± s.d.)	72 ± 10 yrs	67 ± 10 yrs	0.01
Diabetes (%)	24	40	NS
Hypertension (%)	59	63	NS
Recurrent MI	34	41	NS
CHF/P/Edema (%)	21	21	NS
PAF (%)	10	18	NS
Cardiogenic shock (%)	14	7	NS
Stroke (%)	3	2	NS
Coronary angiography (%)	14	14	NS
PTCA/CABG (%)	3	10	NS
30-day mortality (%)	14	23	NS

The overall incidence of stroke was similar in both groups. No cases of intracranial hemorrhage were observed among pts treated with TT. After adjustment for age, CHF, diabetes and PAF, the 30-day mortality odds ratio for those treated with TT was 0.63 (90%CI 0.18-2.15). This study suggests that selected pts with HS may benefit from TT.

956-36 Fibrinolytics vs Primary Angioplasty in Acute Myocardial Infarction (FAP): A Randomized Trial in a Community Hospital in Argentina

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Percutaneous transluminal coronary angioplasty (PTCA) has become an alternative to thrombolytics as initial approach in the treatment of acute myocardial infarction (AMI). The purpose of this study was to determine if primary PTCA: 1) was feasible in our media and 2) could improve the clinical and angiographic outcome. Patients (p) within 12 hrs of an AMI, eligible for thrombolysis, were included. Exclusion criteria were cardiogenic shock, and

LLBB. The end points were: 1) 50% reduction of ST elevation in the EKG at 120 minutes after randomization and 2) presence of TIMI 3 flow in the infarct related artery in the angiogram prior to discharge. A composite of in-hospital clinical events (death, extension of MI, development of heart failure, stroke and major bleeding) was a secondary end point. From 10/93 to 8/95, 112 p were randomized to streptokinase (SK) 1.5 K U (58 p) or primary PTCA (54 p). Baseline clinical and infarct location characteristics were similarly distributed in both groups. The mean age was 66 ± 23 years, with 71% male gender. The time from the onset of symptoms to randomization was 242 ± 138 min for PTCA and 258 ± 162 for SK ($p = NS$). The angiographic success for PTCA was 91%. The interval of time from symptoms onset to ST 50% decrease was 325 ± 144 min for PTCA and 334 ± 151 for SK ($p = NS$). Resolution of ST at 120 min was 79.6% for PTCA and 50% for SK ($p = 0.002$). In hospital mortality was 9.2% for PTCA and 10.3% for SK ($p = NS$). Angiographic follow-up was obtained in 83% of the p in a mean of 8 ± 4 days. TIMI 3 flow was found in 95% for PTCA and 63.6% for SK ($p = 0.001$). The combined end point of major events was 12.9% for PTCA and 25.8% for SK ($p = NS$). Thus event free survival was 87% and 74% respectively. **Conclusions:** 1) In this study primary PTCA was feasible and as save as SK. 2) PTCA showed a significantly: a) greater resolution in ST changes and b) greater incidence of TIMI 3 flow in the infarct related artery prior to discharge than SK. 3) In hospital major events showed a tendency to be reduced with PTCA.

957 Prognosis Following Acute Myocardial Infarction

Tuesday, March 26, 1996, Noon-2:00 p.m.
Orange County Convention Center, Hall E
Presentation Hour: Noon-1:00 p.m.

957-22 Non Q-Wave Myocardial Infarction Post Thrombolysis. What Are They Really? (How Should They Be Classified?)

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Non Q-wave MI (NQ) differ from Q-wave MI (Q) for their natural evolution. Thrombolysis (TL) has increased the number of NQ resulting from aborted transmural MI. How do they evolve and how should they be classified remains unclear. CAMI is a Canadian multicenter study where 4133 consecutive MI were prospectively recruited between 1990 and 1992. Transferred patients (1007) were excluded and 2368 accepted a complete follow-up. We compared Q and NQ with or without TL.

Variables	Q post		Q No		NQ post		NQ No		p
	TL (1)	TL (2)	TL (3)	TL (4)	TL (5)	TL (6)	TL (7)		
N (%)	1003 (32)	760 (24)	339 (11)	1024 (33)					1 vs 3 3 vs 4
21 days death*	69 (6.9)	115 (15)	24 (7.1)	98 (9.6)	NS	NS			
1 y. death*	105 (11)	162 (21)	38 (11)	182 (18)	NS	NS			
1 y. rec MI**	60 (7.6)	52 (9.3)	26 (9.6)	67 (9.0)	NS	NS			
1 y. rec Ang**	375 (47)	269 (48)	150 (55)	384 (52)	#	NS			
PTCA (1 y.)**	227 (29)	104 (19)	90 (33)	158 (21)	NS	NS			
CABG (1 y.)**	87 (11)	74 (13)	36 (13)	131 (18)	NS	NS			

*On total cohort (3126), ** with complete follow-up, #p < 0.05, &p < 0.01

All groups showed significant baseline characteristic differences. Previous MI, PTCA and CABG conferred a much higher risk to develop a NQ either with or without TL. TL was the most important prognostic factor and both groups post TL (Q or NQ) showed identical outcome (cf Table).

We conclude that NQ post TL behave like Q post TL and has a much better prognosis than NQ without TL. Therefore they should be considered either as a special category of MI or together with Q for future studies.

957-23 Nonfatal Reinfarction as an Independent Riskfactor for Subsequent Mortality in Post-Myocardial Infarction Patients

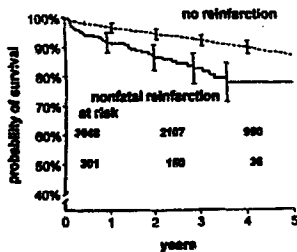
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The independent risk carried by nonfatal reinfarction for subsequent death has seldom been quantified. The prognostic significance of nonfatal reinfarction was determined from the ASPECT trial database. From 1986 till 1992 3404 post-myocardial infarction patients were assigned to long-

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term oral anticoagulant treatment (1700 patients) or placebo (1704 patients) in a double-blind, placebo controlled, multicenter trial. Mean age was 61 years and 80% were males. The trial medication was discontinued and after a median follow-up of 3 years (0.5-6 years), 359 patients had died. A first reinfarction was found in 356 patients of which 301 non-fatal (within 30 days). Subsequent mortality occurred in 42 patients. A multivariate Cox proportional hazard model was performed for history, clinical characteristics and nonfatal reinfarction as a time dependent predictor variable.

mortality after nonfatal reinfarction



	HR	95% CI
Nonfatal reMI	2.03	1.45-2.83
Age	2.30	1.84-2.88
Previous MI	1.54	1.15-2.06
History of angina	1.30	1.01-1.68
Heart failure in hospital	1.74	1.25-2.42
Thrombolytic therapy	0.71	0.51-0.98
Heart rate \geq 70bpm	1.68	1.32-2.16

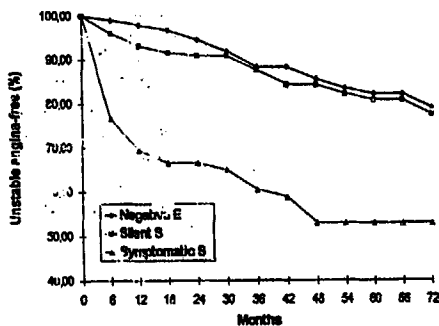
After adjusting for characteristics, as mentioned above, nonfatal reinfarction retained in the model (hazard ratio: 2.03; 1.45-2.83). Thus, nonfatal reinfarction carries a strong and independent risk for subsequent mortality in patients surviving an acute myocardial infarction.

957-24 Silent Exercise-Induced Ischemia In Stable Patients One Year After Myocardial Infarction: 6-Years Follow-Up.

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Silent exercise-induced ischemia is a common finding after myocardial infarction (AMI), to assess its prognostical relevance in stable patients, we evaluated 766 consecutive pts. (mean age: 57.2 \pm 8.6 yrs.; male: 89%) who from 5/1988 to 1/1991 underwent a Bruce Treadmill test at least 1 year after a first Q wave AMI and whose clinical and ET data were prospectively entered in our database. A cardiac follow-up (mean length: 51 \pm 20 months) was completed (Kaplan-Meier overall death at 6-yrs: 9.1%) and 3 different subgroups were retrospectively identified according to ET results: Group 1 = 156 pts. with silent exercise ischemia; Group 2 = 75 pts. with symptomatic exercise ischemia; Group 3 = 99 pts. with a negative ET. Baseline clinical variables were similar between the groups. Pts. with silent ischemia had less functional impairment and exercise ischemia than symptomatic [longer exercise duration ($p < 0.001$), higher double product ($p < 0.001$), higher ischemic threshold ($p < 0.001$), shorter time to ST normalisation ($p < 0.001$).

During the follow-up overall mortality ($p = NS$) and reinfarction ($p = NS$) were similar in the 3 groups, while event-free probability for unstable angina (p



< 0.05 , fig. 1) or PTCA/CABG ($p = 0.06$) was lower for pts. with symptomatic exercise ischemia.

Thus, silent exercise ischemia in stable pts. one year after AMI signifies ischemia of less severity when compared with symptomatic. Moreover in stable post-AMI pts., exercise ischemia, whether symptomatic or not, has a low impact on prognosis. Thus, silent exercise ischemia is not useful in identifying pts. at increased risk for subsequent coronary events.

957-25 The Predictive Value of LV Systolic Sphericity Index, a Magnification-Independent Assessment of LV Shape

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We prospectively evaluated the ability of a simple, magnification-independent measure of LV shape, *systolic sphericity index* (SSI), to predict clinical events in 700 SAVE study patients who underwent cardiac catheterization and left ventriculography within 16 days of MI. Systolic sphericity index is defined as uncorrected ellipsoidal end-systolic LV volume (X100) divided by the volume of a sphere whose diameter is the uncorrected LV long axis. Thus, the SSI of a sphere measures 100%. Patients were 57 \pm 11 years, 83% male, and 64% had sustained an anterior infarction. Average radionuclide ejection fraction was 32% \pm 7. SSI of MI patients was 29% \pm 8 (range 11-76%), which was greater than SSI of normals (20% \pm 4). SSI correlated poorly with end systolic volume, $R = 0.33$, and with EF ($R = -0.27$). Tertiles of SSI from least to most spherical predicted a progressive increase in the occurrence of severe heart failure or cardiovascular mortality over a 3.5 year followup (1:16.5%, 2:24.9%, 3:35.0% $p < 0.001$). Logistic regression analysis demonstrated that hypertension (likelihood ratio (LR) 1.61, $p < 0.001$), radionuclide EF (LR 1.09 for each 1-point decrement, $p < 0.001$), number of diseased coronaries (LR 1.75 for each additional vessel, and SSI (LR 1.04 for each 1-point increment, $p = 0.001$) were independent predictors of clinical outcome.

Conclusion: SSI is a descriptor of LV shape which does not require magnification-correction. The finding that SSI adds prognostic information independent of EF suggests that LV distortion imposes additional disadvantage beyond contractile dysfunction in MI survivors.

957-26 Prognostic Indicators of 3-Year Mortality After an Acute Myocardial Infarction

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The value of various prognostic indicators after an acute myocardial infarction (AMI) is not established. To evaluate the prognostic information of clinical and echocardiographic indexes among pts subjected to modern therapy, we followed 428 consecutive pts within the CONSENSUS II for at least 3 years. The baseline investigation was performed 3-5 days after the AMI. Death from any cause was noted in 101 pts, whereof cardiovascular death in 88 pts. Results are based on total mortality. A highly significant ($P < 0.001$) relation to mortality was found for systolic (LVVI-S) and diastolic LV volume indexes, ejection fraction, global wall motion index (WMI), age, heart rate, prior AMI, prior CHF, betablocker treatment, atrial fibrillation and Killip class. Tabulated variables (mean \pm S.D.) gave independent prognostic information in a stepwise logistic regression model.

	Alive	Dead	P-value
WMI (score)	1.37 \pm 0.3	1.65 \pm 0.4	0.0001
LVI-S (ml/m ²)	24.5 \pm 10	35.6 \pm 18.0	0.0257
Age (years)	65.1 \pm 10.9	71.0 \pm 9.9	0.0279

Mortality was not related to blood pressure, known hypertension, the Doppler E/A ratio, or sex. Thus, echocardiographic wall motion and LVVI-S together with age contain a powerful set of variables for prognostication of outcome during the 3 year period after an AMI.

957-27 Long-Term Prognosis of Patients With Acute Myocardial Infarction In the Thrombolysis Era

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The most recent intervention studies on patients with acute myocardial infarction report an in-hospital mortality of 6-8% and a 1-year mortality following discharge of 2-4%. The patients included in these studies were, however, a

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