Pre PTCA TIMI Flow	Pre PTCA CTFC	Post PTCA CTFC	Delta CTFC
TIMI 0/1	Occluded	28.5 ± 16.5, n = 70	Not applicable
TIMI 2	63.2 ± 37.0, n = 25	26.3 ± 17.8, n = 19	43.7 ± 42.8, n = 19
TIMI 3	30.6 ± 9.9, n = 19	20.5 ± 11.5, n = 18	10.3 ± 12.8, n = 18
TIMI 2 or 3	49.1 ± 32.8, n = 44	23.5 ± 15.1, n = 37	27.4 ± 35.8, n = 37

Previously we reported that normal arteries in the absence of acute MI have a CTFC of 21.0 ± 3.1 (n = 78). PTCA of arteries with TIMI 3 Flow restored a normal CTFC (20.5 vs 21.0, p = NS). Adjunctive PTCA of arteries with TIMI 2 flow improved flow by 43.7 frames (p = 0.0006) but did not restore a normal CTFC (26.3 vs 21.0 p = 0.01) and approximates that reported for non-culprit arteries in acute MI (25.5 frames). Likewise, rescue PTCA of TIMI 0/1 flow did not restore a normal CTFC (28.5 vs 21.0, p = 0.0001). Slower Pre PTCA CTFCs were correlated with greater improvements in CTFC (r = 0.91, p < 0.0001). *Conclusions:* PTCA of the residual stenosis in arteries with TIMI 3 flow after thrombolysis results in normal flow (CTFC = 21). Although PTCA of the residual stenosis in arteries with TIMI 2 flow greatly improves flow, the flow remains persistently slowed by 5 frames (20% > normal). This 5 frame delay is similar to that observed in non-culprit arteries in acute MI, & may reflect a persistent abnormality in microvascular resistance. Whether PTCA of arteries with TIMI 2 flow improves clinical outcomes remains to be determined.

946-20 Clinical Usefulness of Dual SPECT Imaging of Tc-99m Sestamibi and ^{123}I-IPPA for Predicting Myocardial Viability After Thrombolysed Myocardial Infarction

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There is controversy on the value of Tc-99m Sestamibi SPECT (MIBI) for detection of myocardial viability. This study was performed to investigate the ability of MIBI in conjunction with ^{123}I-Phenylpentadecanoic acid (IPPA) in identifying dysfunctional but viable myocardium after early (<4 hours) thrombolytic treatment for myocardial infarction (MI). We compared, in corresponding myocardial segments, perfusion and metabolic patterns in 22 patients with first uncomplicated MI (16 males, age 36 to 64 yrs) after 4 ± 1.5 days from symptoms onset. Myocardial viability was defined by the discrepancy between perfusion defect on MIBI and fatty acid metabolism on IPPA SPECT: dual SPECT image acquisition in 3 projections (anterior, 45º and 70º LAO) was carried out at 60 min from MIBI and 30 min from IPPA injections. The left ventricle was divided into 9 segments (apex, 4 distal and 4 basal segments) and analyzed using a quantitative uptake score as normal = 1, mild reduction = 2, severe reduction = 3, defect = 4. To assess functional recovery of asynergic areas, all patients underwent 2D echocardiography evaluating wall motion score index (WMSI) and ejection fraction (EF) on the same day of radionuclide imaging and 6 weeks after: improvement was defined as decrease of WMSI ≥ 1. WMSI at baseline well correlated with IPPA score (r = 0.76, p < 0.01) while did not with MIBI score (r = 0.61, p = ns). The IPPA score was significantly larger than the MIBI score (6.7 ± 3.1 vs 3.4 ± 2.1, p < 0.01) The discrepancy between IPPA and MIBI was significantly correlated with the extent of the improvement of WMSI (r = 0.86, p < 0.01) and of EF (r = 0.80, p < 0.01) at 6 weeks. In conclusion, the identification by dual MIBI and IPPA SPECT of segmental metabolism-perfusion mismatch (possibly due to stunning phenomenon after thrombolysed myocardial infarction) predicts myocardial viability and later recovery of left ventricular contractile function.

946-21 Persistent Myoglobin Elevation is Associated with Slower Flow in Patent Culprit Arteries Following Successful Thrombolysis

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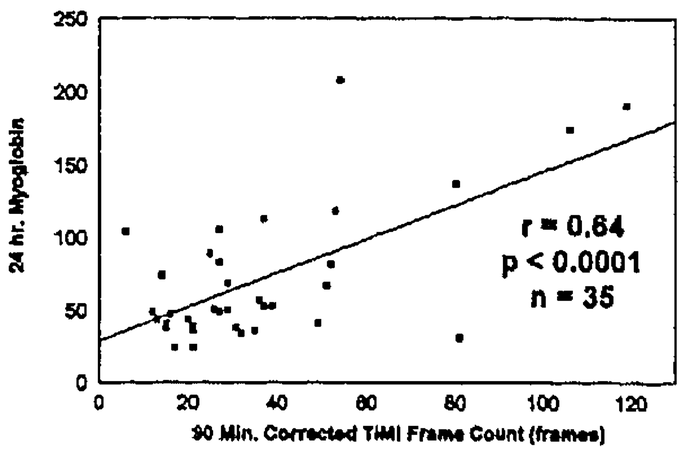
This study examined the relationship between the release of myoglobin and blood flow in patent culprit arteries at 90 min. following thrombolysis with TNK in TIMI 10A. The frames required for dye to reach standard landmarks were counted to arrive at the Corrected TIMI Frame Count (CTFC), a previously described index of coronary blood flow. Slower 90 min. flow (i.e. higher CTFCs) was correlated with higher 12 hour (r = 0.66, p = 0.0001, n = 34), & higher 24 hour myoglobin levels (r = 0.65, p = 0.0001, n = 35) (24 hour data shown in figure) and tended to be higher at 6 hours (r = 0.32, p = 0.058). The 24 hr. mean myoglobin of pts. with slow flow (CTFC ≥ 40) (116.5 ± 65.5, n = 9) did not differ from that of pts. with a closed artery (137.8 ± 156.9, n = 5, p = NS) but was higher than pts. with more rapid 90 min. flow (CTFC < 40) (55.5 ± 4.7, n = 26, p = 0.001).



Conclusions: Slower flow in patent culprit arteries at 90 min. following thrombolysis is associated with a persistent elevation of myoglobin at 12 and 24 hrs which approximates that observed in occluded arteries, possibly as a result of delayed washout.

946-22 Does TIMI Frame Count Reflect Myocardial Blood Flow?

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TIMI frame count (TFC), presented as a simple, reproducible quantitative index of coronary blood flow, is the number of frames needed for the appearance of the front of the contrast bolus to reach predefined distal landmarks. From the indicator dilution theory follows, however, that appearance time has only a weak relation with flow. Mean transit time (MTT), on the other hand, is fundamentally related to myocardial flow. The MTT-method has been successfully tested against direct flow measurements in dogs. Therefore, we considered a comparison between the simply applicable TFC and laborlous MTT useful.

We studied 109 coronary arteriograms of stable patients, previously included into cholesterol lowering trials, from whom the MTT's were already assessed. TFC was determined by 2 independent angiographers, blinded to the MTT data. If this resulted in a difference of > 5 frames, consensus was reached with a third observer. MTT, from injection site to a myocardial area of interest, was assessed by digital subtraction angiography with videodensitometric analyses of the time course of the contrast.

Linear regression analysis between TFC (frames) and MTT (sec) on all arterial and on LAD, LCX and RCA territories separately showed correlation coefficients of 0.12, 0.20, 0.26 and 0.31, respectively. These poor results did not change substantially by excluding those observations, in which there was a difference of > 5 frames between observers (about 20% of all cases).

Conclusion: As expected, TFC does not seem to reflect myocardial flow, as measured by MTT, in stable coronary patients. TFC, therefore, is probably of limited value in the quantitation of myocardial blood flow restoration in acute myocardial infarction.

946-23 Transluminal Extraction Atherectomy vs. Balloon angioplasty in Acute Ischemic Syndromes (TOPIT): Hospital outcome and six-month status

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The TOPIT Trial Is a randomized, multicenter study comparing the use of transluminal extraction atherectomy (TEC) versus balloon angioplasty (PTCA) in native vessels for clinical situations which are associated with intracoronary thrombus. We hypothesize that pretreatment with TEC enhances outcome during percutaneous revascularization for high risk patients with acute ischemic syndromes. *115* patients (mean age *60.0*) were randomized to TEC while *135* patients (mean age *58.7* year) to PTCA. Clinical Indications included primary reperfusion for acute myocardial infarction (*31.61%*), unstable angina (*30.8%*), post-Infarction angina (*24.8%*), and thrombolytic failure (*10%*).

Outcomes were:

	TEC 115	PTCA 135	p value
Death in hospital	1 (0.87%)	0	NS
Emergent CABG	1 (0.87%)	1 (0.87%)	NS
Emergent PTCA	5 (4.35%)	5 (3.70%)	NS
Initial % stenosis	75.2 ± 16.7	78.6 ± 17.5	NS
Final % stenosis	28.2 ± 18.4	27.8 ± 16.9	NS
Final % thrombosis	8%	3%	NS
3× increase CPK	1.63%	5.69%	0.082