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Assessment of reperfusion of the infarct zone after acute myocardial infarction by serial cardiac troponin T measurements in serum

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Abstract

***Background*—The purpose of this study was to derive indices of reperfusion and non-reperfusion after acute myocardial infarction (AMI) from changes in serum concentrations of cardiac troponin T and to test the predictive value of these indices.**

***Methods*—The indices were derived from a retrospective analysis of changes in serum troponin T concentration in 71 patients given thrombolytic treatment who had immediate and late angiography (group 1). These troponin T indices were first tested in a blinded and prospective study of 53 consecutive patients eligible for thrombolytic therapy (group 2). They were then used for the non-invasive assessment of reperfusion of AMI in 48 patients (group 3).**

***Results*—In group 1 troponin T serum concentration curves were biphasic in patients who had reperfusion $\leqslant 5{\cdot}8$ h after the onset of symptoms. Release of the cytosolic troponin T pool resulted in a peak at 14 h and ended at 38 h. The probability of reperfusion was >95% when the ratio of peak cytosolic troponin T concentration to concentration at 38 h (PV1/38) exceeded 1·42 or the ratio of troponin T concentration at 14 h to that at 38 hours (14/38) exceeded 1·09. The probability of the presence of non-reperfused AMI was <5% when troponin T PV1/38 and 14/38 ratios were <0·99 and <0·84 respectively. These discriminatory values of troponin T indices correctly classified (efficiency 96%) 48 of the 53 group 2 patients in whom immediate and late angiography were performed. When troponin T indices were used to classify 48 group 3 patients who were not studied by immediate angiography, thrombolytic therapy was deemed to have been successful in 82% of the treated patients, with spontaneous recanalisation in 11% and 23% of the non-treated patients assessed by PV1/38 and 14/38 respectively.**

***Conclusion*—The PV1/38 or 14/38 ratios of serum troponin T concentration indicated the effectiveness of thrombolytic therapy in achieving reperfusion of AMI.**

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Cardiac troponin T (cTNT) measurements are known to be useful in patients with suspected acute myocardial infarction (AMI),[1-5] but their role in patients with confirmed AMI is less clear. Retrospective analyses of the kinetics of cTnT release in patients with reperfused and non-reperfused AMI showed considerable differences on day 1 and 2 after the onset of symptoms.[6] These data indicated that cTnT measurements may be useful in the non-invasive prediction of reperfusion of the infarct zone. A retrospective description of the altered kinetics of cTnT in serum is not sufficient to define the predictive power of indices derived from cTnT concentration changes. Therefore, in the present study we derived cTnT indices from cTnT serum concentration curves in a group of patients who had immediate and late angiography after thrombolytic therapy. The accuracy of these indices was then tested blinded and prospectively in a second group of patients who were followed up angiographically. Finally, the indices were used in the non-invasive assessment of reperfusion of the infarct zone in a third group of patients not studied by immediate angiography.

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Patients and methods

GROUP 1

We studied 71 patients in group 1 (table 1). In five additional patients an occlusion of the infarct-related artery was found on late angiography. These patients were excluded from the study. Group 1 patients were admitted with AMI to the Heidelberg University Hospital between October 1988 and February 1989 and were eligible for thrombolytic treatment. All patients complied with the following inclusion criteria: (*a*) persistent anginal pain for at least 30 minutes and lasting no longer than 5 h; (*b*) ST segment elevations of at least 0·5 mV in at least two leads of the standard 12 lead electrocardiogram; (*c*) no contraindications for thrombolytic treatment; (*d*) no evidence of valvar heart disease (except trivial mitral regurgitation), cardiomyopathy, left bundle branch block, or previous AMI in the same location; (*e*) age less than 75 years; (*f*) willingness to undergo coronary angiography.

Recanalisation of the infarct-related artery was evaluated by coronary angiography (transfemoral approach) performed immediately after the start of intravenous thrombolytic therapy. Injections of contrast material were repeated every 10 minutes until recanalisation was achieved or persistent occlusion of the infarct vessel was confirmed. Persistent recanalisation was confirmed by a last injection of contrast material 10 minutes after the first successful recanalisation. Angioplasty was

Table 1 Clinical characteristics of group 1 patients

Recanal (h)	No	Age (yr)	M/F	Infarct vessel			Recanal therapy			TIMI acute				TIMI 4 wk			
				LAD	LCX	RCA	L	P	L+P	T0	T1	T2	T3	T0	T1	T2	T3
Successful recanalisation group																	
3·8 (1·2)	53	58	44/9	21	12	20	11	24	18	0	0	5	48	4	0	0	49
2–3	11	56	11/0	4	3	4	4	4	3	0	0	1	10	1	0	0	10
3–4	18	59	15/3	6	6	6	4	8	6	0	0	2	16	1	0	0	17
4–5	13	58	10/3	5	1	7	1	5	7	0	0	1	12	1	0	0	12
5–6	8	55	6/2	2	1	3	2	4	2	0	0	1	7	1	0	0	7
6–7	2	52	1/1	1	1	0	0	2	0	0	0	0	2	0	0	0	2
7–8	1	72	1/0	1	0	0	0	1	0	0	0	0	1	0	0	0	1
Permanent occlusion group																	
—	18	63	14/4	7	2	9	5	10	7	10	6	2	0	10	1	1	1

Age, mean age; L, thrombolytic agents; LAD, left anterior descending coronary artery; LCX, left circumflex coronary artery; L + P, thrombolytic agents followed by acute PTCA; P, PTCA; RCA, right coronary artery; Recanal therapy, recanalisation therapy performed acutely; TIMI, patency status of the infarct-related artery as proposed by the Thrombolysis in Myocardial Infarction study group; T0, T1, T2, T3, TIMI perfusion grades; Recanal, time from onset of symptoms to first documentation of TIMI grade > 1 flow.

Table 2 Clinical characteristics of group 2 patients

Recanal (h)	No	Age (yr)	M/F	Infarct vessel			Recanal therapy			TIMI acute				TIMI 4 wk			
				LAD	LCX	RCA	L	P	L+P	T0	T1	T2	T3	T0	T1	T2	T3
Successful recanalisation group																	
3·5 (1·3)	45	59	35/10	21	6	18	22	0	23	0	0	5	40	4	0	1	40
1–2	3	52	3/0	2	1	0	2	0	1	0	0	1	2	0	0	0	3
2–3	7	58	6/1	4	0	3	1	0	6	0	0	1	6	1	0	0	6
3–4	17	57	14/3	9	3	5	9	0	8	0	0	1	16	2	0	1	14
4–5	7	65	4/3	2	1	4	3	0	4	0	0	0	7	0	0	0	7
5–6	9	64	6/3	4	1	4	6	0	3	0	0	2	7	1	0	0	8
6–7	2	59	2/0	0	0	2	1	0	1	0	0	0	2	0	0	0	2
Permanent occlusion group																	
—	8	60	8/0	3	2	3	1	2	5	8	0	0	0	5	0	0	3

See footnote to table 1 for abbreviations.

performed during the initial angiographic evaluation, when the flow of contrast material in the infarct-related artery was TIMI grade $\leqslant 2^7$ 1 h after the start of intravenous thrombolytic treatment or when residual diameter narrowing of the infarct-related artery was >90%. The invasive investigation was usually repeated on day 19 after onset of AMI. The success of recanalisation and the intensity of coronary artery opacification by contrast material were assessed from cineangiograms by two experienced cardiologists who were unaware of the serological test results. A second catheterisation was performed in five of the 18 patients in whom recanalisation of the occluded coronary artery failed. All but two patients with early recanalisation developed Q wave infarction.

GROUP 2

The second group of patients (table 2) was studied prospectively between May 1990 and February 1991 at the University Hospitals of Heidelberg and Hamburg to test the accuracy and predictive power of the cTnT indices determined in group 1 patients. We studied 63 consecutive patients admitted to the coronary care units with AMI who complied with the inclusion criteria outlined above. These patients were participating in an investigation on the thrombolytic effectiveness of different r-tPA (alteplase) dosages. Ten patients were excluded from final analysis because of death <32 h after onset of pain in 1 patient; uncertain time of onset of pain in five patients; prolonged resuscitation in two patients, and incomplete blood sampling in two patients. Thus data from 53 patients were analysed. In these 53 patients angiography was attempted no more than 90 minutes after the start of thrombolytic therapy and repeated a mean of 20 days afterwards. Percutaneous transluminal coronary angioplasty was performed only when patients reported angina during AMI or had signs of ischaemia on exercise testing before hospital discharge.

GROUP 3

The third group of patients (table 3) was studied non-invasively by selected cTnT indices to

Table 3 Clinical characteristics of group 3 patients

Therapy (h)	No	Age (yr)	M/F	AMI location		TIMI 4 wk				PTCA	No angio
				Ant	Inf	T0	T1	T2	T3		
Thrombolytic therapy											
3·0(1·4)	28	63	20/8	13	15	0	1	1	14	8	12
1–2	4	65	3/1	2	2	0	0	0	3	1	1
2–3	12	65	6/6	8	4	0	1	0	6	2	5
3–4	6	57	5/1	2	5	0	0	0	2	1	4
4–5	3	59	3/0	0	3	0	0	0	1	0	2
5–6	1	63	1/0	1	0	0	0	1	0	1	0
6–7	1	62	1/0	0	0	0	0	0	1	0	0
7–8	1	56	1/0	0	1	0	0	0	1	0	0
Medical therapy											
—	20	71	13/7	15	5	6	0	0	0	0	14

Age, mean age; AMI, acute myocardial infarction; Ant, anterior wall; Inf, inferior wall; No angio, coronary angiography not performed; TIMI 4 wk, patency status of the infarct related artery at 4 weeks after onset of symptoms as proposed by the Thrombolysis in Myocardial Infarction study group; T0 T1 T2 T3, TIMI perfusion grades; Therapy, time from onset of symptoms to initiation of intravenous thrombolytic therapy.

asses the success of reperfusion therapy. We studied 55 consecutive patients with Q wave AMI and onset of anginal pain <10 h before admission to hospital between March 1991 and October 1991. Seven patients were excluded: one patient died <32 h after onset of pain, in two the time of onset of pain was uncertain, and in four patients blood sampling was incomplete. Twenty eight patients satisfied the inclusion criteria for thrombolytic therapy as outlined above and were treated by streptokinase infusion. The remaining 20 patients underwent routine medical therapy with intravenous heparin, β blockade, nitrate, and oral asprin. Angiography was performed late in this group and only if clinically indicated, because of persistent anginal pain during AMI, a positive exercise test before hospital discharge, or young age.

METHODS

In all patients a 12 lead electrocardiogram was recorded twice on day 1 and once on days 2, 3, 4, 7, 15, and before discharge. If new anginal pain occurred a 12 lead electrocardiogram was immediately recorded. The clinical data of all patients were analysed without knowledge of the troponin T test results.

All patients gave written informed consent after thorough explanation of the study protocol and of the possible harmful and beneficial effects of the planned therapeutic and diagnostic procedures. The study was approved by the ethics committee of the universities.

MYOCARDIAL MARKER PROTEINS

Blood samples were obtained on admission, every 8 h on the first 2 days, once a day on the 3 subsequent days, and finally every second day until day 10 after admission. Care was taken to obtain additional blood samples between 12 and 16 h (14 h sample), and between 36 and 40 h (38 h sample). To allow clotting the samples were kept at room temperature for 15 minutes then centrifuged, and stored as serum aliquots at minus 20°C.

We measured concentrations of cTnT in duplicate by an enzyme immunoassay developed by our group.[8] This test is now commercially available from Boehringer Mannheim, Germany and can be performed with a Zymun test system ES 33 analyser. With the present test kit cTnT measurements take 90 minutes.

The total serum enzyme activity of creatine kinase (CK:EC 2·7·3·2·) was measured colorimetrically by a Chem 1 analyser (Technicon, Terrytown, USA) with the reagents provided by the manufacturer. The upper limit of normal was 80 IU/l at 25°C. CK-MB serum enzyme activity was measured colorimetrically using a kit from Boehringer Mannheim (CK-MB-NAC). The upper limit of normal was 10 IU/l at 25°C.

DATA ANALYSIS

A distinct cTnT peak on day 1 or 2 after onset of AMI was defined as an increase in concentration in at least two consecutive cTnT measurements that exceeded the previous and subsequent cTnT values by >10%. The predictive power of the cTnT indices was assessed in the retrospectively analysed group 1 by linear logistic regression models by the method of maximum likelihood.[9] The power of the derived cTnT indices to predict reperfusion was tested in the prospectively analysed group 2 in terms of sensitivity (number of true positive test results in all patients with reperfusion), specificity (number of true negative test results in all patients without reperfusion), positive predictive value (number of true positive test results in all positive troponin T test results observed), and negative predictive value (number of true negative test results of all negative test results observed). Significance was defined at the 5% probability level.

Results

DERIVATION OF CTNT INDICES (GROUP 1 PATIENTS)

Figure 1A shows the median serum concentrations of cTnT in 50 patients who had recanalisation of the infarct-related artery $\leqslant 5{\cdot}8$ h after onset of symptoms and in 18 patients with a permanently occluded coronary artery. Subtraction of the median serum concentrations of cTnT in both AMI groups gave the differential cTnT washout in reperfused and non-reperfused AMI (fig 1B). The reperfusion dependent early increase in serum cTnT concentration was detectable until 38 hours after onset of pain and on average reached a peak value at 14 hours. Two criteria

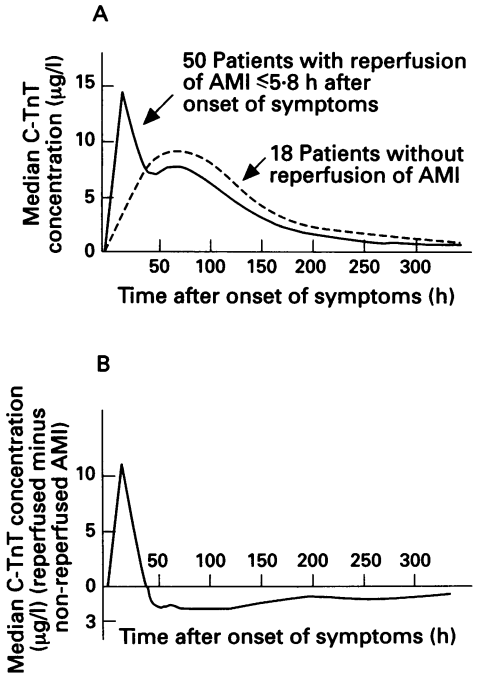

Figure 1 (A) Median serum concentration of cardiac troponin T in 50 patients with recanalised infarct-related arteries $\leqslant 5{\cdot}8$ h after onset of symptoms and in 18 patients with permanent occlusion of the infarct-related artery. (B) Differential troponin T release obtained by subtraction of the median serum concentration curves of the patients with non-reperfused and reperfused myocardial infarction. Increased troponin T washout in reperfused myocardial infarction produces a positive difference. AMI, acute myocardial infarction; c-TnT, cardiac troponin T.

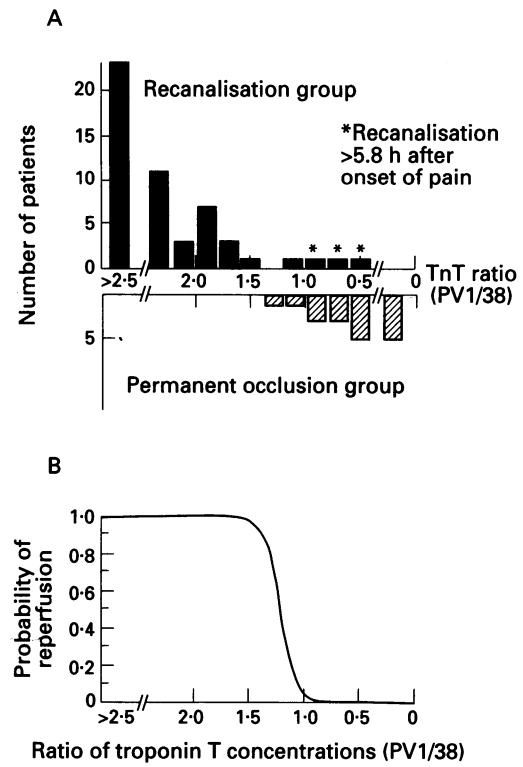

Figure 2 (A) Frequency distribution of troponin T ratio values (PV1/38) in patients with recanalisation ⩽5·8 h and >5·8 h after onset of symptoms (asterisks) and in patients with a permanently occluded infarct-related artery (hatched columns). (B) Univariate analysis of correct classification of infarct reperfusion according to the value of the troponin T ratio. Y axis: probability of correct classification of reperfusion at ⩽5·8 h versus at >5·8 h or non-reperfused acute myocardial infarction. X axis: probability of correct classification as reperfused or non-reperfused acute myocardial infarction. TnT, troponin T.

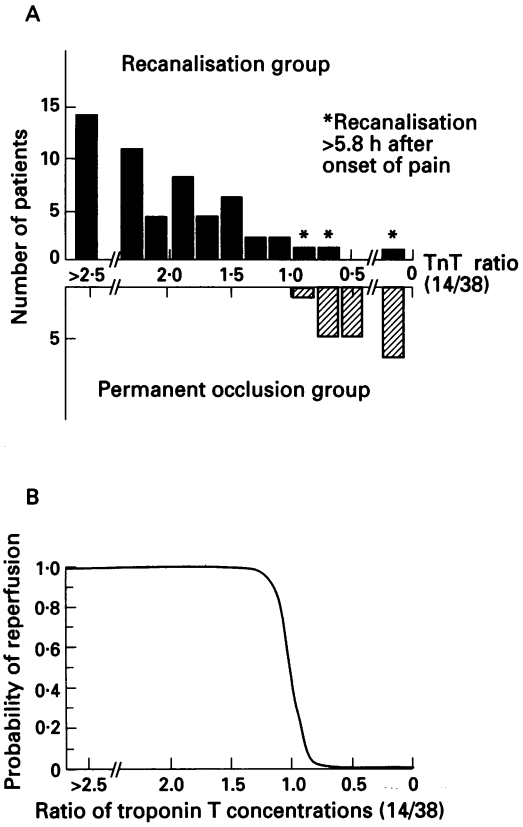

Figure 3 (A) Frequency distribution of troponin T ratio values (14/38) in patients with recanalisation ⩽5·8 h and >5·8 h after onset of symptoms (asterisks) and in patients with permanently occluded infarct-related artery (hatched columns). (B) Univariate analysis of correct classification of infarct reperfusion according to the value of the troponin T ratio. Y axis: probability of correct classification of reperfusion at ⩽5·8 h versus at > 5·8 h or non-reperfused acute myocardial infarction. X axis: probability of correct classification as reperfused or non-reperfused acute myocardial infarction. TnT, troponin T.

were selected as possible indicators of early reperfusion of AMI that were independent of AMI size. These were the ratio of the cTnT value of peak 1 divided by the 38 hours value (PV 1/38) and the ratio of the cTnT value of 14 hours divided by the 38 hours value (14/38).

DEFINITION AND EVALUATION OF DISCRIMINATOR VALUES OF CTNT INDICES (FIGS 2 AND 3, GROUP 1 PATIENTS)

The frequency distributions of PV1/38 and 14/38 cTnT ratios in patients with reperfused and non-reperfused AMI are shown as histograms in figs 2A and 3A respectively. The cTnT ratios of three patients with recanalisation at 6·5, 6·8, and 7 h after onset of symptoms and in whom no early serum cTnT peak was observed are indicated by asterisks. The cTnT ratios of these three patients overlap with those found in patients with permanent occlusion of the infarct-related arteries. cTnT ratios clearly distinguished between the remaining 50 patients with successful recanalisation and the patients in the group with permanent occlusion.

Figures 2B and 3B show use of logistic regression analysis to define the probability of correct classification by selected cTnT indices of patients with infarct reperfusion ⩽5·8 h after onset of symptoms and of non-reperfused AMI. The probability of recanalisation at ⩽5·8 h was >95% when cTnT ratios PV1/38 and 14/38 exceeded 1·42 and 1·09 respectively. The probability of the presence of non-reperfused AMI was >95% when PV1/38 or 14/38 cTnT ratios were <0·99 and <0·84 respectively.

Table 4 shows the discriminatory power of the selected cTnT indices in group 1 patients. The cTnT PV1/38 and 14/38 ratios of five (7%) and three (4%) patients were in the 95%-5% probability range for the presence of reperfused or non-reperfused AMI. Therefore, these patients could not be classified by selected cTnT indices. All remaining patients who had recanalisation ⩽5·8 h after onset of symptoms and all patients with non-reperfused AMI were correctly classified. When the three patients with recanalisation >5·8 h are included as patients with reperfused AMI the sensitivities and negative predictive values of the selected indices are reduced to 94%/93% and 83%/84% for the PV1/38 and 14/38 cTnT ratios respectively.

Table 4 Discriminatory power of selected troponin T indices in group 1 patients

TnT index PV1/38			*TnT index 14/38*		
TnT ratio	*Class*	*Patients*	*TnT Ratio*	*Class*	*Patients*
>1·42	Reperfusion	48/71 (68%)	>1·09	Reperfusion	49/71 (69%)
1·42–0·99	Undefined	5/71 (7%)	1·09–0·84	Undefined	3/71 (4%)
<0·99	No reperfusion	18/71 (25%)	<0·84	No reperfusion	19/71 (27%)
	Early Rep/N-Rep	*Rep/N-Rep*		*Early Rep/N-Rep*	*Rep/N-Rep*
Sensitivity	100%	94%	Sensitivity	100%	93%
Specificity	100%	100%	Specificity	100%	100%
PV pos	100%	100%	PV pos	100%	100%
PV neg	100%	83%	PV neg	100%	84%
Efficiency	100%	95%	Efficiency	100%	94%

Early Rep, patients with recanalisation <5·8 h after onset of symptoms; N-Rep, patients with permanent occlusion of the infarct related artery; PV neg, negative predictive value; PV pos, positive predictive value; Reperfusion, patients with successful recanalisation; TnT, troponin T; PV 1/38, ratio of troponin T concentrations at peak value 1 to those at 38 h after onset of symptoms; 14/38, ratio of troponin T concentrations at 14 to those at 38 after onset of symptoms.

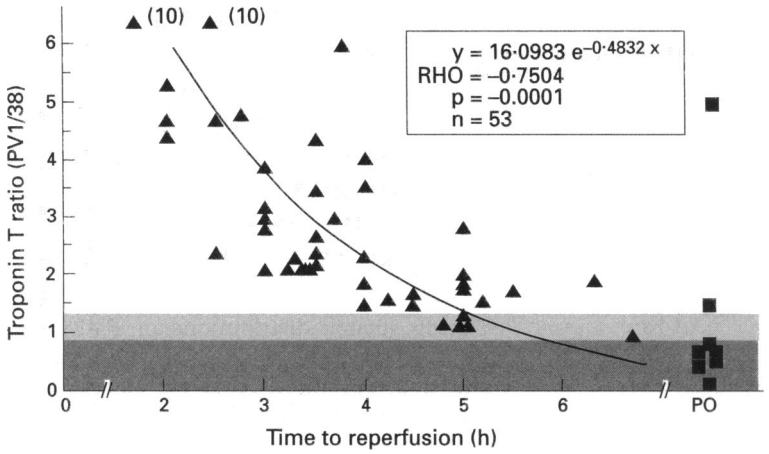

Figure 4 Relation between values of troponin T ratios (PV1/38) and duration of ischaemia before recanalisation in 45 group 2 patients (triangles). Troponin T ratios of the remaining eight patients with a permanent occlusion of the infarct-related artery are shown on the right (squares). The lightly shaded area indicates the range of troponin T ratios that cannot be used to classify patients correctly. The darkly shaded area indicates ratios with <5% probability of reperfusion. PO, permanent occlusion.

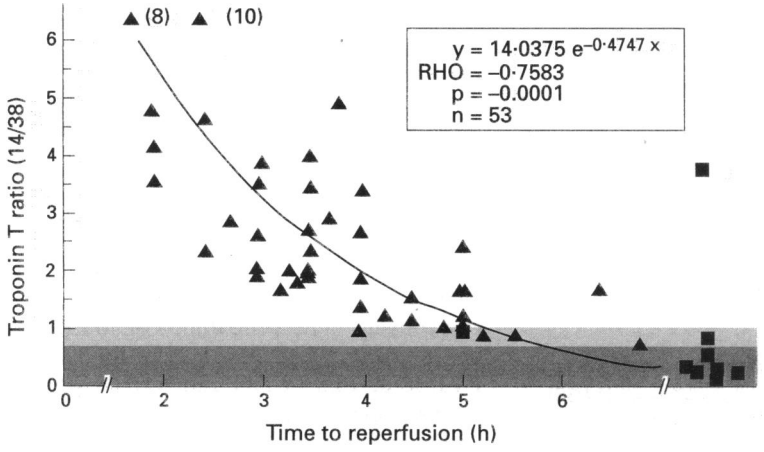

Figure 5 Relation between values of troponin T ratios (14/38) and duration of ischaemia before recanalisation in 45 group 2 patients (triangles). Troponin T ratios of the remaining eight patients with a permanent occlusion of the infarct-related artery are shown on the right (squares). The lightly shaded area indicates the range of troponin T ratios that cannot be used to classify patients correctly. The darkly shaded area indicates ratios with <5% probability of reperfusion. PO, permanent occlusion.

EVALUATION OF THE CTNT INDICES IN THE PROSPECTIVE ANALYSIS OF GROUP 2 PATIENTS

Figures 4 and 5 show the PV1/38 and 14/38 cTnT ratios in relation to the duration of ischaemia until successful recanalisation in 53 group 2 patients with immediate and late angiography. The range of cTnT ratios between the discriminator values of 95% and 5% probability of reperfusion is indicated by a lightly shaded area, whereas the range of cTnT ratios with <5% probability of reperfusion (or >95% probability of presence of non-reperfused AMI) is indicated by the darker area.

The values of the cTnT ratios increased exponentially with decreasing duration of ischaemia before recanalisation. Therefore, all patients with recanalisation ⩽4 h after onset of symptoms were correctly classified. In one (PV1/38) and two (14/38) of the eight patients with permanent occlusion of the infarct related artery cTnT ratios exceeded the discriminator values. In one of these two patients the times to peak CK and CK-MB activity of 7·5 h and 7·0 h indicated reperfused AMI. In the other patient with borderline cTnT ratios the times to peak CK and CK-MB activity were 17 h and 16 h respectively.

The discriminatory power of selected cTnT indices was tested in group 2 patients (table 5). cTnT ratios in five (9%) and four (8%) patients were in the 95%-5% probability range for PV1/38 and 14/38 cTnT ratios respectively. These patients could not be classified. All the remaining patients with successful recanalisation were correctly classified (sensitivity 100%). The specificity of the PV1/38 and 14/38 cTnT indices was reduced to 75% and 86% by the two patients discussed in detail above. All patients with cTnT ratios below the 5% probability level of reperfused AMI showed occluded coronary arteries on angiography (negative predictive power = 100%).

NON-INVASIVE CLASSIFICATION OF GROUP 3 PATIENTS BY CTNT INDICES (FIGS 6 AND 7)

In four (14%) of the 28 patients treated with thrombolytic agents cTnT criteria indicated non-reperfused AMI. In one of these four patients with an open coronary artery at 3 weeks thrombolytic treatment was started 5 h after the onset of pain, whereas in the remaining three patients treatment was started < 3·5 h after the onset of symptoms. In the 20 patients not treated with thrombolytic agents, three (11%) (fig 6) and five (23%) (fig 7) patients had cTnT ratios indicating spontaneous reperfusion of the AMI zone < 5·8 h after the onset of symptoms. In this group the highest cTnT ratios were found in two patients in whom ventricular fibrillation developed before thrombolytic therapy was started. All patients with an occluded coronary artery on late angiography had cTnT

Table 5 Discriminatory power of selected troponin T indices in group 2 patients

TnT index PV1/38			*TnT index 14/38*		
TnT Ratio	*Class*	*Patients*	*TnT Ratio*	*Class*	*Patients*
> 1·42	Reperfusion	42/53(79%)	> 1·09	Reperfusion	43/53(61%)
1·42–0·99	Undefined	5/53(9%)	1·09–0·84	Undefined	4/53(8%)
< 0·99	No reperfusion	6/53(11%)	< 0·84	No reperfusion	6/53(11%)
	Rep/N-Rep			*Rep/N-Rep*	
Sensitivity	100%		Sensitivity	100%	
Specificity	75%		Specificity	86%	
PV pos	95%		PV pos	98%	
PV neg	100%		PV neg	100%	
Efficiency	96%		Efficiency	96%	

See footnote to table 4 for abbreviations.

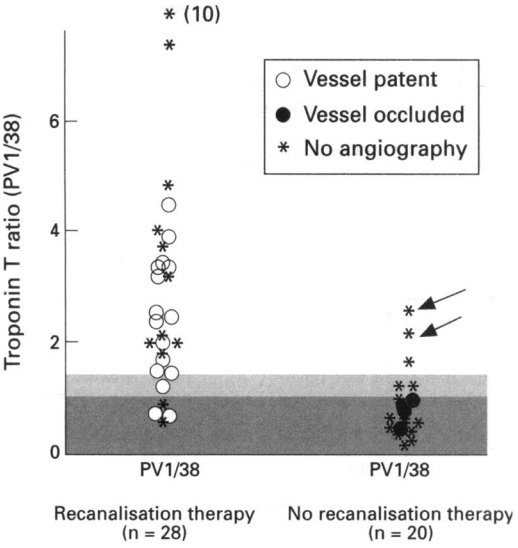

Figure 6 Troponin T ratios (PV1/38) of 28 group 3 patients treated with thrombolytic agents are shown on the left and results for 20 group 3 patients not treated by thrombolytic agents are shown on the right. Arrows indicate patients with repetitive ventricular fibrillation after admission. The lightly shaded area is the range of troponin T ratios that cannot be used to classify patients correctly. The darkly shaded area indicates ratios with <5% probability of reperfusion.

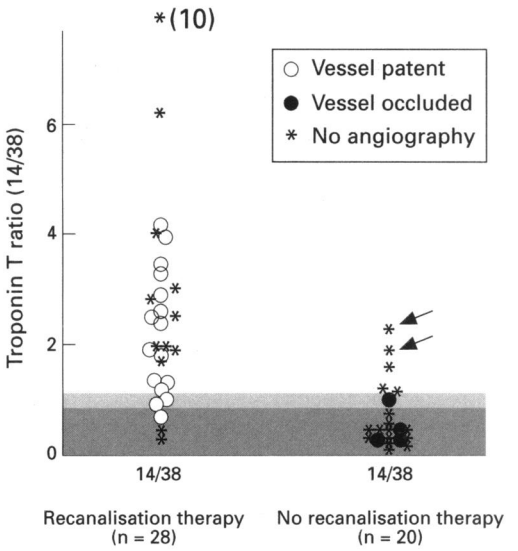

Figure 7 Troponin T ratios (14/38) of 28 group 3 patients treated with thrombolytic agents are shown on the left and results for 20 group 3 patients not treated by thrombolytic agents are shown on the right. Arrows indicate patients with repetitive ventricular fibrillation after admission. The lightly shaded area is the range of troponin T ratios that cannot be used to classify patients correctly. The darkly shaded area indicates ratios with <5% probability of reperfusion.

ratios below the discriminator values for non-reperfused AMI.

Discussion

This study shows that cTnT measurements during the first two days after onset of AMI can be used to assess the early success of thrombolytic therapy. After reperfusion of the infarct zone a functionally unbound fraction of cTnT is released which shows kinetics in serum similar to cytosolic CK. By relating the changes in serum concentration of this rapidly appearing cTnT pool to the cTnT concentrations resulting from degradation of myofibrils, we derived indices that strongly depended on the success of thrombolytic therapy and on the duration of ischaemia before recanalisation was achieved. All patients with recanalisation $\leqslant$ 4 h after onset of symptoms were correctly classified by the discriminator values of cTnT indices that we selected (PV1/38 > 1·42, 14/38 > 1·09). Furthermore, 15 (75%) and 14 (70%) of the 20 patients with recanalisation >4 h after the onset of symptoms were also correctly identified as successfully treated patients. However, the discriminatory power of the selected indices decreased with increasing duration of ischaemia before recanalisation. Thus patients with recanalisation >5·8 h after onset of pain could not reliably be classified by cTnT criteria.

The discrepancy between biochemical and angiographic indices of reperfusion was greater in patients classified as having non-reperfused AMI according to angiography. Two of the eight group 2 patients with non-reperfused AMI clearly had a successful reperfusion according to biochemical indices. Whereas in one patient it seems likely that the wrong vessel was classified as the infarct-related artery, the discrepant findings were not immediately obvious in the second patient. The most likely explanation in this patient is that periods of intermittent recanalisation not detected by angiography may have increased cTnT, CK, and CK-MB washout from the infarcting myocardium. This indicates that reocclusion occurring after short periods of reperfusion may not be detectable. In six of the nine patients with TIMI grade II flow the cTnT ratios indicated successful reperfusion whereas in three patients no increased washout of cytosolic cTnT was seen.

Thus though biochemical and angiographic indices yield similar results in most patients, both methods of evaluation have specific limitations. The disadvantage of angiography is that the snap-shot visual estimation of flow of contrast material is not a true measure of reperfusion.[10]

LIMITATIONS OF THE STUDY

In the present study most patients developed Q wave AMI. Therefore, the indices derived in this study should be applied only to a similar group of patients. In patients with non-Q wave AMI a subtotal obstruction of the infarct-related artery or significant collateral blood flow to the infarcting myocardium are frequent findings.[11 12] Both circumstances may result in an increased wash-out of cytosolic marker molecules and therefore may interfere with biochemical analysis of the success of reperfusion therapy.

CLINICAL SIGNIFICANCE

CTnT indices not only allow the non-invasive prediction of reperfusion but also seem to reflect the efficiency of reperfusion. cTnT ratios were highest in patients with brief periods of ischaemia and TIMI grade III flow after recanalisation. Thus high cTnT ratios indicate early and very efficient reperfusion. In contrast, borderline cTnT ratios correspond to either a poor or a late reperfusion of AMI or both. Therefore the analysis of troponin T ratios can be used to confirm the angiographic evaluation of the effects of thrombolytic therapy on reperfusion of AMI.

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