CARDIOVASCULAR MEDICINE

Relation between different methods for analysing ST segment deviation and infarct size as assessed by positron emission tomography

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Objective: To study the relation between resolution of ST segment deviation and infarct size using positron emission tomography.

Methods: 45 patients with ST segment elevation acute myocardial infarction treated with thrombolysis or percutaneous coronary intervention were studied prospectively. An ECG was taken before and at (mean (SD)) 100 (45) min after reperfusion therapy. ECGs were analysed by three methods. Residual ST segment deviation, obtained from the ECG immediately after completion of reperfusion therapy, was defined by summation for each of the three methods. Relative resolution of ST segment deviation was defined as the absolute resolution divided by the ST segment deviation score at baseline $\times 100$ (%). After 29 (14) hours, myocardial blood flow was measured with $^{13}\text{NH}_3$. For each patient, the regions with a myocardial blood flow $< 80\%$ of normally perfused myocardium (=hypoperfusion) and $< 50\%$ (=no reflow) were automatically delineated.

Results: Substantial differences were found between different ECG analysis methods. There were moderate correlations between the area with myocardial hypoperfusion and ST segment deviation scores at baseline and after reperfusion therapy. After reperfusion therapy, residual ST segment deviation in the single lead with maximum ST segment deviation was as good at discriminating between tertiles of myocardial damage as summed ST segment elevation. Relative ST segment resolution did not discriminate between different degrees of myocardial damage.

Conclusions: In the individual patient, residual ST segment deviation after reperfusion in the single lead with maximum ST segment deviation is at least as good as summed ST elevation in predicting final myocardial damage.

In ST segment elevation acute myocardial infarction (STEMI), the amount of resolution of ST segment elevation after reperfusion has an important predictive value for recovery of left ventricular function and long term prognosis.^{1–10} In addition, a correlation has been found between resolution of ST segment elevation and microvascular reperfusion as assessed by myocardial contrast echocardiography.¹¹

Using positron emission tomography (PET), more than one third of patients have been shown to have impaired myocardial tissue perfusion despite the presence of persistent TIMI grade 3 flow in the infarct related epicardial coronary artery.¹² Microvascular obstruction as assessed by magnetic resonance imaging (MRI) also predicts more frequent cardiovascular complications.¹³

Taken together, these observations have led to the widely held hypothesis that ST segment changes during the course of an evolving STEMI reflect myocardial tissue reperfusion rather than epicardial coronary artery patency, and therefore are better predictors of clinical outcome.

Our aim in the present study was to investigate the relation between ST segment deviation, resolution of ST segment deviation, and myocardial tissue flow early after reperfusion therapy as assessed by PET.

METHODS

Patient population

Patients who had a first STEMI of less than six hours' duration and who had a TIMI (thrombolysis in myocardial infarction) flow grade 2 or 3 at early angiography were

included in the study prospectively and consecutively. All patients showed ST segment elevation of ≥ 0.1 mV in two or more limb leads or ≥ 0.2 mV in two or more contiguous precordial leads and had 12 lead ECGs available on admission and after reperfusion therapy. Patients received either thrombolytic treatment or primary percutaneous coronary intervention (PCI). The fibrinolytic regimen consisted of recombinant staphylokinase or accelerated recombinant tissue plasminogen activator (rt-PA), both combined with aspirin and intravenous heparin. All patients underwent coronary angiography at 90 minutes after the initiation of fibrinolytic treatment. Two independent experienced angiographers read the angiograms. The patency of the infarct related vessel was scored according to the TIMI criteria of reperfusion.¹ In all patients, PET nitrogen-13 labelled ammonia ($^{13}\text{NH}_3$) measurements were done within 48 hours after initiation of reperfusion therapy.

The study was granted ethics approval by the human subject review committee of the University of Leuven, and written informed consent was obtained in all cases.

Electrocardiographic analysis

Two ECGs (on admission and after reperfusion therapy) were analysed quantitatively in the ECG Core Laboratory of the University of Leuven. Measurements were computer assisted using a custom made in-house program. Values were

Abbreviations: PET, positron emission tomography; PCI, percutaneous coronary intervention; STD, ST deviation; STE, ST elevation; STEMI, ST segment elevation acute myocardial infarction; TIMI, thrombolysis in myocardial infarction

rounded to 0.05 mV. Two investigators undertook the measurements independently and were blinded to the angiographic and clinical findings. ST segment deviation was measured 20 ms after the end of the QRS complex. The reference baseline was the line connecting the preceding and following PR segment. Anterior STEMI was defined as ST segment elevation in two of the following leads: V1–V6, I, and aVL. Non-anterior STEMI was defined as ST segment elevation in two of the following leads: II, III, and aVF. If ST segment elevation was confined to leads I, aVL, V5, and/or V6, the location was designated anterior; if the elevation was also present in leads II, III, and aVF, or if ST segment depression was present in leads V1–V4, the location was designated non-anterior.

ST segment deviation was calculated using three different methods. All of these have been employed in recent studies on the prognostic value of the resolution of ST segment deviation in large scale clinical trials. The three methods were as follows.

Summed ST elevation (sum STE)—The criteria used were described by Schröder and colleagues in 1994²: for anterior STEMI the sum of ST segment elevation was measured from leads I, aVL, and V1–V6, and for non-anterior STEMI, in leads II, III, aVF, V5, and V6. ST segment depression was not measured.

Summed ST deviation (sum STD)—The criteria used were described by Schröder and colleagues in 1995³: for anterior STEMI, the sum of ST segment elevations in leads V1–V6, I, and aVL was added to the sum of ST segment depression in leads II, III, and aVF. For non-anterior STEMI, the sum of ST segment elevation in leads II, III, aVF (and I, aVL, V5, and V6, if present) was added to the sum of ST segment depression in leads V1–V4. For both infarct locations, reciprocal ST segment depression was used only in leads with ≥ 0.1 mV of ST segment depression at baseline.

Maximum ST elevation (max STE)—For this method only the single ECG lead with maximum ST segment deviation at that time point was taken into account.¹⁴ In anterior STEMI, the greatest ST segment elevation in a single lead was measured. In non-anterior STEMI, the greatest ST segment deviation was measured, either as ST segment elevation in one inferior lead or as ST segment depression in a precordial lead V1–V4, whichever was greater at the given time point of evaluation.

The following definitions were applied:

- **ST segment score at baseline**—the summed score for each of the three methods, obtained from the ECG before initiation of reperfusion therapy and expressed in mV.
- **ST segment score after reperfusion therapy**—the summed score for each of the three methods, obtained from the ECG immediately after completion of reperfusion therapy and expressed in mV.
- **Absolute ST segment deviation resolution**—ST segment score at baseline minus ST segment score after reperfusion therapy, expressed in mV.
- **Relative ST segment deviation resolution**—absolute ST segment deviation resolution divided by ST segment score at baseline $\times 100$, expressed as per cent. Categorisation of relative resolution of ST segment deviation was as follows: complete ($> 70\%$), partial (30–70%), and none ($< 30\%$), as proposed by Schröder and colleagues.²

Positron emission tomography

The NH_3 studies were undertaken with a whole body positron emission tomograph (Ecat Exact HR+; CTI Siemens, Knoxville, Tennessee, USA). An emission scan was acquired after injection of 740 MBq of $^{13}\text{NH}_3$. A summed frame of the perfusion study was constructed. The myocardial image was

resampled into 16 radial slices, delineated using an algorithm developed in our department.¹⁵ Each polar map was divided into 33 regions—four rings of eight regions and one region for the apex. A region of normal tracer uptake was automatically defined on the $^{13}\text{NH}_3$ polar map using a previously established technique.¹² The mean values of that region were used as the reference value (100% $^{13}\text{NH}_3$ uptake), and the entire polar map was normalised to this value. In each patient, the infarct related region was manually designed on the polar map by grouping some of the 33 regions, depending on the coronary anatomy as seen on the angiogram.¹⁶ The left ventricular extent of a perfusion defect was computed by summing the myocardial wall volumes of the pixels in the area concerned, and expressed as a percentage of total left ventricular volume.

A three compartment model was applied to calculate absolute blood flow values.¹⁷

The following definitions were applied:

- **Area with myocardial hypoperfusion**—extent of the myocardial region in which the relative uptake of $^{13}\text{NH}_3$ was less than 80% of the reference value, expressed as percentage of the left ventricle (fig 1).
- **Area of no reflow**—extent of the myocardial region in which the relative uptake of $^{13}\text{NH}_3$ was less than 50% of the reference value, obtained by automatic delineation, expressed as percentage of the left ventricle. A relative uptake of $^{13}\text{NH}_3 < 50\%$ is generally regarded as corresponding to transmural infarction (fig 1).¹²
- **Mean flow index in the area of no reflow**—mean value of the relative uptake of $^{13}\text{NH}_3$ in the area of no reflow, expressed as percentage of the left ventricle.
- **Myocardial damage severity index**—[50 minus mean flow index in the area of no reflow] divided by $50 \times$ extent of the area of no reflow. This index takes into account the level as well as the extent of severe myocardial hypoperfusion and thus provides a measure of global myocardial damage.¹⁸

Statistical analysis

Results are given as mean (SD). Differences between groups were investigated using Student *t* tests for unpaired data. For evaluating the relation between flow indices and ST segment deviation, we used linear regression analysis. To compare mean values from tertiles of the myocardial damage severity index, we applied univariate analysis of variance. The Bonferroni correction was used to adjust for the multiplicity of tests. Significance was indicated at $p < 0.05$.

RESULTS

Clinical characteristics

Forty five patients were included in the study protocol (38 men and seven women, mean (SD) age, 58 (9) years). The infarct related vessel was the left anterior descending coronary artery in 21 patients, the right coronary artery in 13, and the left circumflex coronary artery in 11. Reperfusion therapy consisted of primary PCI in 14 patients, administration of fibrinolytics as sole treatment in 28, and fibrinolytic treatment followed by rescue PCI in three. Restoration of patency of the infarct related artery was achieved in all study patients (TIMI grade 3 flow in 36; TIMI grade 2 flow in nine). Pre-treatment ischaemic time was 222 (187) minutes. The second ECG was recorded at 100 (45) minutes on average after reperfusion therapy.

PET imaging was done 29 (14) hours after the initiation of reperfusion treatment.

Figure 1 Examples of ECGs before and after reperfusion therapy in a patient with a large anterior (A) and a small inferior (B) ST segment elevation acute myocardial infarct (STEMI). The area with myocardial hypoperfusion (light and dark grey in panel A) and the area of no reflow (dark grey in panel A) were automatically defined. The patient in panel B had only a limited area of myocardial hypoperfusion (light grey in panel B), but no region with no reflow.

ECG

The incidence of the different categories of relative resolution of ST segment deviation showed substantial variation depending on the method of ECG analysis used. Complete resolution of ST segment deviation was present in 18 (40%), 14 (31%), and 10 (22%) patients according to sum STE, sum STD, and max STE, respectively (table 1). The three methods of ECG analysis were concordant in only nine patients (fig 2).

Likewise, partial resolution of ST segment deviation was observed in 14 (31%), 17 (38%), and 22 (49%) patients. Finally, 13 (29%), 14 (31%), and 13 (29%) patients had either no resolution or showed an increased ST segment deviation score after reperfusion therapy.

Figure 2 Schematic representation of the numbers of patients showing complete (> 70%) resolution of ST segment deviation according to the three different ECG analysis methods. STD, ST deviation; STE, ST elevation.

Positron emission tomography

The mean flow index was 60.5 (11.0)% in the areas of myocardial hypoperfusion and 40.2 (6.5)% in the no reflow areas. The mean extent of the area of myocardial hypoperfusion was 24.7 (12.7)% of the left ventricle. The mean extent of the areas of no reflow was 8.3 (10.6)%.

Correlation of ECG and PET data

ST segment deviation scores according to the three different ECG analysis methods were correlated with several PET indices of myocardial tissue flow (table 2). A moderate but very significant linear correlation was found between the extent of the area with myocardial hypoperfusion and the ST segment deviation scores obtained with the three methods, both at baseline and after reperfusion therapy. Somewhat less striking but still significant correlations were found between the myocardial damage severity index and the ST segment deviation scores at baseline and after reperfusion therapy according to sum STE and max STE, although the data points are relatively widely scattered (fig 3).

Table 1 Relative ST segment deviation resolution as a categorical variable according to the three different ECG analysis methods			
Resolution	Sum STE	Sum STD	Max STE
Complete	18 (40)	14 (31)	10 (22)
Partial	14 (31)	17 (38)	22 (49)
None	13 (29)	14 (31)	13 (29)

Total number of pairs of ECGs analysed was 45 in each group. Data are expressed as n (%).
"Complete" indicates >70% resolution; "partial", 30–70% resolution; "none", <30% resolution.
Max, maximum; STD, ST deviation; STE, ST elevation. See Methods for a description of the three methods.

Figure 3 (A) Scatterplot for ST segment deviation after reperfusion therapy (mV). (B) Scatterplot for relative ST segment deviation resolution (mV). Method 1, summed ST elevation; method 3, maximum ST elevation.

Absolute ST segment deviation resolution as assessed by all three ECG methods also showed a moderate but significant correlation with the area of myocardial hypoperfusion.

Correlations between absolute ST segment deviation resolution and myocardial damage severity index were not significant.

No correlation was found between the relative ST segment deviation resolution and any of the PET indices of myocardial damage.

Infarct size

The myocardial damage severity PET index combines the level as well as the extent of severe myocardial hypoperfusion. It should provide a good estimate of the loss of myocardial tissue. In the present patient population, the myocardial damage severity index ranged from 0–19%. Patients were divided into tertiles according to their myocardial damage severity index.

Fifteen of the 45 patients had a myocardial damage severity index of zero—that is, they had no myocardial segments in which the relative uptake of $^{13}\text{NH}_3$ was less than 50% of the reference value. In the second tertile, all patients had a myocardial damage severity index greater than zero but below 1.25%, whereas all values in the third tertile were above 1.25% (table 3).

On average, the time period elapsed between the onset of chest pain and the initiation of reperfusion therapy tended to be longer in the third ν the second ν the first tertile (267 ν 213 ν 186 minutes).

Patients with more pronounced myocardial damage had significantly more ST segment deviation at baseline, regardless of the ECG analysis method used. ST segment deviation at baseline according to sum STE was most predictive of final myocardial damage (1.20 ν 0.89 ν 0.82 mV in tertile III ν II ν I, $p = 0.0008$). The residual ST segment deviation after reperfusion therapy was significantly higher with increasing myocardial damage severity index. After reperfusion, residual ST segment deviation according to max STE showed the greatest significance in discriminating between tertiles of myocardial damage (0.25 ν 0.17 ν 0.09 mV in tertile III ν II ν I, $p = 0.002$).

Absolute resolution of ST segment deviation was highest in the tertile with the most extensive final myocardial damage, the intertertile differences only reaching significance when analysed according to sum STE.

The greatest absolute resolution of ST segment deviation was observed in the group with the most extensive myocardial damage (tertile III), while intermediate values were found in the group with the least pronounced myocardial damage (tertile I). The smallest absolute resolution of ST segment deviation was seen in the group with

Table 2 Linear correlations between various ST segment deviation scores on ECG and PET derived indices of myocardial damage

	Area with myocardial hypoperfusion		Myocardial damage severity index	
	R	p Value	R	p Value
ST segment deviation score at baseline				
Sum STE	0.64	<0.0001	0.40	0.02
Sum STD	0.56	0.0003	0.32	0.12
Max STE	0.56	0.0002	0.36	0.04
ST segment deviation score after reperfusion therapy				
Sum STE	0.44	0.01	0.39	0.04
Sum STD	0.36	0.04	0.34	0.08
Max STE	0.48	0.004	0.42	0.02
Absolute ST segment deviation resolution				
Sum STE	0.49	0.004	0.24	0.4
Sum STD	0.39	0.03	0.14	NS
Max STE	0.41	0.02	0.21	0.7
Relative ST segment deviation resolution				
Sum STE	0.08	NS	0.07	NS
Sum STD	0.06	NS	−0.0004	NS
Max STE	0.12	NS	0.11	NS

R, linear correlation coefficient. Correlations are significant at $p < 0.05$.

Table 3 Clinical and ECG characteristics of the patients, divided into tertiles according to the myocardial damage severity index

Variable	Tertile I (n = 15)	Tertile II (n = 15)	Tertile III (n = 15)	p Value
Age (years)	57.3	57.7	61	0.5
Time between onset of pain and initiation of reperfusion therapy (min)	186	213	267	0.5
TIMI flow grade	2.9	2.8	2.7	0.2
Random peak creatine kinase (U/l)	1754	1538	5708	0.01
ST segment deviation at baseline (mV)				
Sum STE	0.82	0.89	1.20	0.0008
Sum STD	1.12	1.14	2.04	0.01
Max STE	0.29	0.26	0.54	0.006
ST segment deviation after reperfusion therapy (mV)				
Sum STE	0.22	0.58	0.79	0.004
Sum STD	0.28	0.69	0.90	0.006
Max STE	0.09	0.17	0.25	0.002
Absolute ST segment deviation resolution (mV)				
Sum STE	0.61	0.30	1.11	0.01
Sum STD	0.85	0.45	1.13	0.11
Max STE	0.19	0.10	0.29	0.06
Relative ST segment deviation resolution (%)				
Sum STE	57.4	26.3	47.2	0.06
Sum STD	61.2	31.1	51.1	0.08
Max STE	50.6	28.6	50.2	0.09

Data are given as mean values per tertile.

Differences are significant at $p < 0.5$.

Max, maximum; STD, ST deviation; STE, ST elevation. See Methods for a description of the three methods.

intermediate myocardial damage (tertile II). A similar trend was observed for relative resolution, but the differences did not reach significance.

DISCUSSION

In large scale clinical trials of fibrinolytic treatment in STEMI, the amount of resolution of ST segment elevation has been shown to correlate with recovery of left ventricular function and to predict long term prognosis.²⁻¹⁰ These observations have led to the hypothesis that ST segment changes during the course of STEMI are better at predicting clinical outcome than patency rates of the infarct related epicardial coronary vessel because they reflect myocardial tissue perfusion. A less consolidated belief of many investigators is that the so called “complete” resolution of ST segment deviation corresponds to a small myocardial infarct with limited myocardial necrosis. In this study, we have explored the correlation between ST segment changes and myocardial tissue perfusion.

A first important finding was that categorisation of resolution of ST segment deviation showed substantial differences depending on which method of ECG analysis was used, the percentage of patients categorised as having complete resolution varying between 22–40%. It can therefore be expected that the different ECG analysis methods differ in their ability to predict infarct size and prognosis. These differences can at least in part explain the discrepancies between the results obtained from different studies and seem to preclude pooling of these data.

We found a significant relation between the ST segment changes on the ECG and the extent of myocardial hypoperfusion as assessed by PET, thus confirming the hypothesis that ST segment changes in the course of STEMI reflect myocardial tissue perfusion.

Importantly, no significant correlation was found between the relative resolution of ST segment deviation and any of the PET indices of myocardial damage. This implies that relative resolution does not allow any firm conclusions to be drawn regarding infarct size in the individual patient. This contrasts with the findings in large patient populations in which the average relative resolution of ST segment deviation shows significant prognostic power.²⁻¹⁰ To explain this paradox, one

needs to know the exact relation between relative resolution of ST segment elevation and the percentage of jeopardised myocardium saved by reperfusion therapy. Presuming that this relation exists, we hypothesise that the explanation for the paradox is as follows: if in a patient with a large myocardial infarct half the jeopardised myocardium is saved by reperfusion, this patient is still left with a rather large infarct. When, in contrast, half of a small area of jeopardised myocardium is saved, that patient will end up with a very small infarct. In both patients, the relative resolution of ST segment deviation may have been the same, but final infarct sizes differ greatly. When large patient populations are considered, however, pooling many patients with different amounts of jeopardised myocardium, the patients in whom a greater percentage of the jeopardised myocardium is saved will, on average, have smaller infarcts and consequently a better prognosis. This is illustrated by the figures obtained by Dong and colleagues,¹⁹ who studied the correlation between ST segment resolution and myocardial salvage assessed by Tc99m sestamibi scintigraphy in a large number of patients ($n = 243$) after reperfusion therapy for acute myocardial infarction. They found that ST segment resolution correlated with myocardial salvage, albeit with a very wide distribution of the scintigraphy results—for example, the final infarct size was 12.5 (12.0)% of the left ventricle in the group with complete ST segment resolution, 20.0 (13.9)% in the group with partial ST segment resolution, and 22.7 (19.4)% in the group with no resolution ($p < 0.001$; values are mean (SD)). As with the results we have obtained, such a wide distribution would preclude any prediction of final infarct size in the individual patient.

When patients were subdivided into tertiles according to their myocardial damage severity index, summed ST segment elevation at baseline emerged as the most powerful predictor of final infarct size. After reperfusion therapy, residual ST segment deviation in the single lead with maximum ST segment deviation was found to predict infarct size at least as well as summed ST segment deviation. This finding is in agreement with a recently published study in which residual ST segment elevation in the lead with maximum ST segment deviation after reperfusion therapy emerged as the best predictor of 180 day cardiac mortality.¹⁴ It provides the

clinician with a simple tool that is readily and universally accessible to estimate the final result of reperfusion therapy.

Study limitations

The major limitation of our study was the absence of PET examinations preceding reperfusion therapy. However, in view of the well known time dependency of the benefit of coronary reperfusion therapy, postponing this treatment by performing PET scintigraphy was deemed unethical. Likewise, no $^{13}\text{NH}_3$ PET re-studies after 4–6 weeks were done, which could have improved the diagnostic value for viability.

The size of the index infarct was not homogeneous in the study population. This may have reduced the possibility of finding a correlation between resolution of ST segment deviation and infarct size. Furthermore, it should be acknowledged that reperfusion is a dynamic phenomenon, and a single ECG recording may have missed possible ischaemic events in the hours following reperfusion.

Conclusions

Methods of ECG analysis differ in their ability to predict the absence of myocardial tissue reflow. Final infarct size as assessed by PET is significantly, but only moderately, correlated with the ST segment deviation score obtained before and after reperfusion therapy, but not with relative ST segment deviation resolution.

Residual ST segment deviation in the single lead with maximum ST segment deviation after reperfusion therapy seems to be equally as good as summed ST segment deviation as a predictor of the extent and severity of myocardial damage.

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