Electrocardiographic changes after myocardial infarction as indicators of deranged regional left ventricular wall motion

A serial M mode echocardiographic mapping study

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SUMMARY Electrocardiographic and echocardiographic findings were compared in 44 patients with a first transmural infarction. Each patient was investigated on days 1, 2, 10, and 360. The electrocardiogram was classified according to QRS and ST segment changes. Local left ventricular function was determined from mean systolic wall velocity measurements by an M mode echocardiographic mapping technique in 10 of 16 segments suitable also for electrocardiographic evaluation. Mean systolic wall velocity was corrected for differences in anterior and inferior wall motion.

Wall motion was normal in segments without QRS or ST changes throughout the study. All segments with QRS or ST changes showed significantly lower corrected systolic wall velocity values during the acute stage. Segments with ST depression, alone or in combination with a minor Q wave, had corrected mean systolic wall velocity values similar to those of normal segments after one year. Segments with major Q waves and all segments with ST elevation showed reduced corrected mean systolic wall velocity values throughout the study. Segments with ST elevation, irrespective of Q waves, showed the most severely reduced wall motion with significantly lower corrected mean values than segments with minor or major Q waves without ST elevation on days 10 and 360.

Thus when electrocardiograms are used for defining local left ventricular function, consideration must be given to the phase of illness, QRS morphology, and presence of ST segment elevation.

Transmural myocardial infarction is characterised by changes in the QRS complex, which are preceded by transient ST segment elevation indicating ischaemia.¹ Persistent ST elevation, on the other hand, may indicate left ventricular dyskinesia.^{2,3} By contrast, subendocardial necrosis is accompanied by non-specific ST segment changes and generally not by QRS changes.^{1,4} Knowledge of wall motion disturbances in these cases is incomplete.

Myocardial necrosis causes wall motion disturbances which can be visualised with left ventricular cineangiography^{5,6} and echocardiography.⁷⁻⁹ During an ischaemic attack left ventricular dysfunction similar to that found during myocardial infarction occurs transiently at the time of pain and changes on the electrocardiogram.¹⁰⁻¹²

Previous studies on echocardiographic and electrocardiographic findings in acute myocardial infarction

are mostly limited to single comparisons in the later stages of the illness. In our study repeated echocardiographic analyses in different stages of the disease were performed to consider the relation between certain QRS and ST segment changes and segmental wall motion dysfunction. We have also investigated whether these electrocardiographic changes, indicative of either transmural or subendocardial infarction, carry different information about regional wall motion in relation to time.

Patients and methods

Consecutive patients with electrocardiographic signs suggesting a transmural myocardial infarction on admission to our coronary care unit were investigated on the day of admission (day 1) and on days 2, 10, and 360. The following features excluded patients from inclusion in the study: a delay longer than 48 h; a history or electrocardiographic signs of previous

Table 1 Pertinent data from the study group

	Day of investigation			
	1	2	10	360
No of patients*	44	42	39	27
Reasons for withdrawal				
Complete heart block	—	1	1	1
Left bundle branch block	—	—	1	1
Reinfarction	—	—	—	2
Died	—	1	3	11
Not available for investigation	—	—	—	2
Treatment				
Digitalis	9	11	13	11
Diuretics	31	28	22	14
Beta blocking agents	6	6	6	7
None of above drugs	8	9	11	7

*33 men, 11 women; mean age 62 ± 11 years.

myocardial infarction; valvular heart disease; left bundle branch block; complete heart block; or pacemaker treatment. Patients who required pacemaker treatment, who developed left bundle branch block, or had a reinfarction were excluded from further analyses. This left 47 patients for investigation, but in 3 of these unacceptable echocardiograms from more than six of 16 left ventricular segments were obtained and they were therefore excluded. Thirty three men and 11 women with a mean age of 62 (range 31–82) years were therefore studied, and Table 1 gives the data including reasons for withdrawal and their treatment.

ECHOCARDIOGRAPHY

Echocardiograms were obtained with an Organon-Technica echocardiograph and recorded on a Honeywell 1856 fibreoptic line scan recorder. Two 2.25 MHz collimated transducers (D=13 mm) focused on either 7.5 or 4 cm were used.

The M mode investigation was guided by a preceding cross sectional survey of left ventricular anatomy. The M mode transducer was positioned on the anterior chest wall in electrocardiographic positions V2, V4, and V5 as well as one subxiphoidal and two positions on the high anterolateral chest wall. With this multiple positioning 16 separate left ventricular segments could be studied (Fig. 1).^{9 11–13} Segmental mean systolic wall velocity from at least two beats was calculated as shown in Fig. 2.

To obtain comparable values of systolic wall velocity from anterior and inferior segments a correction was made. Data from previously investigated normal subjects, and from other reports, show that values from inferior wall segments are higher than those from the anterior wall.^{9 14 15} This difference may reflect cardiac pendulum motion rather than true differences in contractile behaviour.¹⁶ The different correction values for each segment were obtained from

Fig. 1 Illustration of the four recording planes by schematic sections of the left ventricle and the segment model including eight basal (1a to 8a) and 8 mid-ventricular (1b to 8b) segments. Wall motion from segments marked with thick lines were used for comparison with electrocardiography. B, basal; M, mid-ventricular; A, apex; Ant, anterior; Lat, lateral; Post, posterior; Inf, inferior; Sept, septal.

our previous investigation of normal subjects.⁹ Mean values of systolic wall velocity from anatomically opposite segments were measured and the difference for each pair halved. By adding the halved difference, the correction value, to the anterior segment and subtracting it from the opposite inferior segment the different segments show comparable corrected values. The numerical values of the obtained differences have been used in the present study. The corrected values given are the measured mean values plus or minus the correction value for each segment. Table 2 shows the data from normal subjects with measured values, correction values, and corrected values.

Fig. 2 M mode recording from a parasternal recording position illustrating measurements of mean systolic wall velocity (V_{mean} mm/s) in the septal (S) and the posterior wall (PW) segments 1a and 5a. RV, right ventricle; LV, left ventricle.

ELECTROCARDIOGRAPHY

Conventional 12 lead electrocardiograms were recorded on Siemens-Elcoma Mingograph immediately before each echocardiographic examination. According to a previous study based on postmortem findings the conventional electrocardiogram allows for information on 10 of the 16 segments; namely, 1a, 2b, 3b, 4a, 4b, 5a, 5b, 6b, 8a, and 8b¹⁷ (Fig. 1). Reciprocal electrocardiographic signs of posterior or septal infarctions were not evaluated. The QRS and ST changes were graded by two independent observers without knowing the echocardiographic data. We considered ST segment depression to be present if the downward displacement exceeded 0.1 mV and elevation to be present if the upward displacement exceeded 0.2 mV. Major Q waves were considered to be present if the Q wave was more than 0.2 mV deep and was larger than the R wave or QS complexes. Minor Q waves were considered to be present if the Q wave was more than 0.2 mV deep and 0.04 s wide or if

the R wave amplitude was reduced by 0.2 mV or less than 50% of the normal value for age (Table 3) or if the peak to peak amplitude

was less than 0.5 mV (leads aVL, aVF, I, II, and III), Q wave being less than R wave.

The procedure used was based on studies by Askenazi *et al.*¹⁸ and the Minnesota code for myocardial infarction.^{19 20} Each recording position was marked with ink in order to secure constant recording sites during hospital admission.

STATISTICS

Conventional statistical methods were used. Results are expressed as mean $\pm$ 1 SD. Student's *t* test was used to evaluate differences between groups, and the 5% level was used for determining significance.

Table 2 Left ventricular systolic mean wall velocity (V_{mean}) without and with correction (V_{mean_c}) in healthy controls ($n=37$) of similar age and sex to the study group

	Anterior segments							
	8a	8b	1a	1b	2a	2b	3a	3b
V_{mean} (mm/s)	16	18	17	21	19	19	17	17
Correction	+12	+11.5	+12	+8.5	+13.5	+10.5	+14.5	+11
V_{mean_c}	28	28.5	29	29.5	32.5	29.5	31.5	28
	Posterior segments							
	4a	4b	5a	5b	6a	6b	7a	7b
V_{mean} (mm/s)	40	39	41	38	46	40	46	39
Correction	-12	-11.5	-12	-8.5	-13.5	-10.5	-14.5	-11
V_{mean_c}	28	28.5	29	29.5	32.5	29.5	31.5	28

Table 3 Normal R wave amplitudes according to age

	Age (years)		
	40-49	50-59	60
V2	3.4	4.0	7.0
V4	17.0	12.9	20.9
V5	13.4	12.5	15.4

Results

Echocardiograms were adequate at the first examination in 89% of segments in the 44 patients. The corresponding figures for the examination 24 h later and on days 10 and 360 were 90% in 42, 83% in 39, and 87% in 27 respectively. The reproducibility of two blind observations from the same recordings and between two recordings was analysed in 15 patients. Significant correlations ($r=0.90-0.95$) were obtained for all positions, with SEE between 4.90 parasternally and 8.1 in V5. The highest deviation between two measurements was in the range 10-44 mm/s.

ELECTROCARDIOGRAPHICALLY NORMAL SEGMENTS

Segments without QRS and ST changes showed corrected mean systolic wall velocities within the normal range at the first examination. Similar values were also recorded on days 2, 10, and 360 (Table 4, Fig. 3 and 4).

ELECTROCARDIOGRAPHICALLY ABNORMAL SEGMENTS

Segments without ST elevation v normal (Table 4; Fig. 3)—Segments with isolated ST depression had significantly lower ($p<0.001$) corrected mean systolic

Fig. 3 Corrected mean systolic wall velocity in segments grouped according to electrocardiography into normal, ST depression, minor or major Q wave changes. a=significantly lower than electrocardiographically normal segments on the same day; b=significantly higher than on day 1; c=significantly lower than segments with isolated ST depression; d=significantly lower than segments with minor Q wave changes.

wall velocities on days 1 and 2. This was followed by an improvement with significantly ($p<0.01$) higher values after day 1. Segments with minor Q waves had reduced ($p<0.01$) corrected mean systolic wall velocities on days 1, 2, and 10. Contractile performance improved with higher values ($p<0.01$) on day 360 compared with the first and second measurements.

The lowest corrected mean systolic wall velocity values were seen in segments with major Q waves,

Table 4 Corrected mean systolic wall velocity (mm/s) in electrocardiographically classified segments

	Days after acute myocardial infarction			
	1	2	10	360
Normal	28.4±16.4 (155)	28.9±17.7 (138)	30.6±17.8 (124)	27.0±15.7 (135)
ST depression	12.4±17.4 (27)	18.2±15.0 (49)	26.4±17.8 (54)	24.1±15.0 (17)
ST elevation	15.0±13.2 (49)	12.1±17.6 (25)	10.5±13.9 (6)	—
Minor Q wave	13.9±12.1 (14)	13.9±21.4 (19)	20.0±13.1 (29)	23.2±17.8 (33)
Minor Q wave +ST elevation	9.4±11.0 (41)	6.4±15.1 (40)	6.1±17.1 (26)	7.9±16.4 (12)
Major Q wave	5.7±9.5 (7)	3.5±14.1 (6)	7.0±12.9 (14)	11.4±11.8 (15)
Major Q wave +ST elevation	8.0±11.3 (46)	3.1±12.8 (61)	5.9±13.4 (51)	2.6±14.6 (25)
All with ST elevation	10.8±11.8 (136)	6.3±15.0 (126)	7.1±14.7 (83)	4.3±15.2 (37)
All without ST elevation	11.2±11.2 (21)	11.4±19.1 (25)	15.7±13.0 (43)	19.5±15.8 (48)

Values given as mean ±1 SD.

Figures in parentheses = No of evaluated segments.

Electrocardiographic changes after myocardial infarction

which remained significantly ($p<0.01$) depressed throughout the year. Segments with major Q waves also differed ($p<0.01$) from segments with isolated ST depression on days 2, 10, and 360 and from segments with minor Q waves on days 10 and 360. Values for segments with major Q waves did not change significantly over the study year.

The possible role of digitalis as a cause of the ST depression was investigated in the nine patients taking this drug (144 segments). These segments showed higher corrected mean systolic wall velocities (15.1 ± 20.3 mm/s) on day 1 than the remainder with ST depression (10.0 ± 19.9 mm/s) although this difference was not significant. This slight difference was even smaller on days 2, 10, and 360.

Segments with ST elevation v normal (Table 4 Fig. 4) Segments with isolated ST elevation showed lower ($p<0.001$) corrected mean systolic wall velocities on days 1, 2, and 10. ST elevation without QRS changes was not seen on day 360.

Segments with ST elevation and minor Q waves were associated with slightly lower corrected mean systolic wall velocities throughout the study compared with normal segments and with segments with isolated ST elevation on day 1 ($p<0.05$). This minor difference was no longer evident on day 2 and similar values were seen subsequently.

The greatest reduction in corrected mean systolic wall velocities was seen in segments with ST elevation with major Q waves which also showed lower values

Fig. 4 Corrected mean systolic wall velocity in segments grouped according to electrocardiography into normal, ST elevation, minor or major Q wave changes with ST elevation. a = significantly lower than normal segments on corresponding day; c = significantly lower than segments with isolated ST elevation.

Fig. 5 Corrected mean systolic wall velocity in segments with Q waves without ST elevation and in all segments with ST elevation regardless of QRS. b = significantly different from day 1; c = significantly lower than segments without ST elevation.

than those with isolated ST elevation on days 1 and 2 ($p<0.001$ and $p<0.01$ respectively). There was no significant difference, however, when these were compared with segments with ST elevation with minor Q waves, and there was no significant change over the year.

Segments with Q waves without ST elevation v all segments with ST elevation (Table 4, Fig. 5)

Corrected mean systolic wall velocities on day 1 were reduced by the same amount in both groups. Values for segments with Q waves without ST elevation were higher on subsequent days with significantly ($p<0.05$) higher values on day 360 compared with days 1 and 2. In contrast, all segments with ST elevation showed lower values on day 2, 10 ($p<0.05$), and 360 ($p<0.01$) when compared with day 1. Of all segments with ST elevation 12% showed dyskinesia on admission, and 24, 22, and 39% on days 2, 10, and 360 respectively. This difference in wall motion between segments with Q waves without ST elevation and those with ST elevation was significant ($p<0.01$) from day 10 with a further increase in difference at the one year follow up.

Discussion

The role of electrocardiography in diagnosing, localising, and sizing myocardial infarction has been evaluated in both animal studies and postmortem examinations.¹⁹⁻²¹ The diagnostic value of a pathological Q wave as an indicator of left ventricular dysfunction is uncertain: some workers have found strong correlations between pathological Q waves and segmental asynergy,^{5 22 23} while others have not.^{20 24} We therefore considered that a study of wall motion properties

after a myocardial infarction measured by serial echocardiography with different levels of deranged QRS and ST segments was indicated.

Any comparison between echocardiography, which gives information about local wall motion, and electrocardiography, which reflects electrical activity, must be made with caution. They not only measure different qualities, but also information is obtained from different sample volumes. The electrocardiogram reflects a much larger portion of the ventricular wall than the narrow segment covered by the echocardiographic beam. In the present study the three anterior chest wall positions were the same for echocardiography and electrocardiography. But matching of echocardiographic and electrocardiographic information is more difficult for the inferior wall. We have based our comparisons on a previous echocardiographic study where systolic wall velocity and QRS changes were related to postmortem findings in patients with myocardial infarction: this study showed that conventional electrocardiography reflected 10 out of the 16 left ventricular segments with sufficient sensitivity and specificity.¹³ We therefore limited this investigation to these 10 segments, which include three of four antero-septal segments, four of six anterolateral segments, and three of four inferoposterior segments. The middle third of the septum (two segments) and the basal anterolateral wall, as well as one posterior and one mid-ventricular antero-septal segment were not evaluated.

When making M mode measurements recording errors must be considered. In order to minimise spatial problems internal landmarks are used—that is, posterior and anterior edges of the right ventricle, papillary muscles, and leaflets, defined for each view.^{13 25} Certain limitations inherent in the echocardiographic method remain and require comment. When comparing wall motion in the anterior and inferior left ventricular walls, using a fixed external probe location, we, like others, have found higher mean systolic wall velocity values in inferoposterior wall segments (39–46 mm/s) compared with anterior (16–21 mm/s) wall segments in healthy controls.⁹ It is unlikely that the normal left ventricle displays an inhomogeneous contraction pattern of this magnitude,¹⁴ and a more likely explanation is cardiac rotational and pendulum motions, which cannot be separated from ventricular contraction motion by M mode echocardiography. Rotational motion is fortunately relatively small. Instead, most of the differences seen in anterior and inferior wall motion are explained by the anterior pendulum motion of the left ventricle during systolic contraction.^{15 16} Corrections for these movements of the heart are often made in wall motion studies with cross sectional echocardiography,^{21 26} but this has not previously been done in M mode

recordings. We have attempted to correct for cardiac motion by using data from a healthy population of similar age and sex.⁹ The corrected mean systolic wall velocity obtained by this method is 28–32 mm/s for all segments.

Different QRS scoring systems have been used to estimate left ventricular damage in myocardial infarction.^{17 27 28} Strong correlations between electrocardiographic scores and overall ventricular function as well as enzymatically estimated infarction size have been found by several groups.^{17 29 30} We have used a simplified scoring system with three QRS classes: normal, minor, and major Q waves subgrouped according to changes in the ST segment.

The non-specific electrocardiographic finding of ST depression is often seen in subendocardial infarction.¹ Nevertheless, it was a good indicator of left ventricular dysfunction in the acute stage of the myocardial infarction and was almost as good as Q waves or ST elevation. During the later stages, however, the relation between ST depression and wall motion disturbances weakened. Several other mechanisms, including drugs such as digitalis and beta blockers, also produce ST segment changes.^{31 32} Beta blocking drugs were used infrequently but digitalis, which was prescribed to an increasing percentage, did not appear to be important. The overall effect of drugs on the ST segment, however, was impossible to evaluate.

In contrast to ST depression, ST segment elevation is influenced less by parameters not associated with the ischaemic process. Acute ischaemia causing ST elevation leads to a reduction in wall motion,^{33 34} and our findings from the acute phase confirm this. Wall motion in segments with ST elevation even without the appearance of Q waves was a third of normal. Segments with persistent ST elevation have been studied extensively and found to show poor ventricular function.^{2 3} This is also supported by our findings as segments with ST elevation also showed the poorest wall motion, while segments with only Q waves showed less pronounced derangement with time. Indeed, ST elevation on admission suggested dyskinesia in 12% of the segments while the same electrocardiographic finding during late convalescence was an even stronger (39% of segments) indicator of dyskinesia.

Pathological Q waves in myocardial infarction are usually attributed to transmural necrosis. Our findings show that large Q waves indicate poor segmental function compatible with a concept of transmural necrosis. In contrast, minor Q waves recorded during the first two days corresponded to poor contractile function. This electrocardiographic sign, however, does not indicate reduced left ventricular wall motion at later stages of the disease; contrary wall motion is similar to that of segments without QRS

changes.

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