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Electrocardiographic and Ventriculographic Recovery Patterns in Q Wave Myocardial Infarction

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To further define the capacity for recovery after acute phase electrical and mechanical injury in patients with Q wave myocardial infarction who were treated with standard measures, 120 lead body surface potential maps and radionuclide angiograms were recorded at day 5 before discharge and month 6 after infarction in 23 patients with a first infarction (12 anterior and 11 inferior by standard 12 lead electrocardiographic criteria). In addition to assessment of spatial changes in electrocardiographic and wall motion patterns, five quantitative variables were evaluated: minimal Q zone integral, ΣQ wave integral, maximal ST integral, left ventricular ejection fraction and left ventricular wall motion abnormality score. From day 5 to month 6 after infarction, the only change in the inferior infarction group was a gain in ΣQ wave (-91 ± 40 μV·s × 10^2 to -68 ± 24 μV·s × 10^2; $p < 0.05$). In contrast, all variables improved over the same time period in the anterior infarction group: Q zone minimum, -34 ± 20 to -24 ± 13 μV·s ($p < 0.05$); ΣQ wave, -160 ± 122 × 10^2 to -120 ± 90 μV·s × 10^2 ($p < 0.05$); ST maximum, 44 ± 19 to 18 ± 9 μV·s ($p < 0.01$); ejection fraction, 54 ± 7 to 63 ± 17% ($p < 0.05$); and wall motion score, 6 ± 3 to 3 ± 3 ($p < 0.01$). The major change in ST maximal integral occurred early (day 5 to discharge) and the improvement in ejection fraction occurred late (discharge to month 6 after infarction); change of the other variables was only apparent between day 5 and month 6.

In the anterior infarction group there were significant correlations between the temporal changes in both depolarization variables and ST maximal integral ($r = 0.58$) and between change in ejection fraction and wall motion abnormality score ($r = 0.82$). In the inferior infarction group the only intratechnique correlation was between temporal change in maximal Q zone and ΣQ wave ($r = 0.71$). There were no significant intertechnique correlations in either group. Thus, Q wave infarction is not an all or none phenomenon. Rather, acute phase indexes of electrical and mechanical function represent a balance of reversible and irreversible ischemic damage. Recovery appears to be gradual, independent, electrocardiographic versus ventriculographic and quantitatively disparate between clinical groups. The effects of post infarction therapeutic interventions on such indexes are, therefore, probably best assessed in specific groups, over relatively long periods and with randomized controls.

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We have found spatial and quantitative body surface potential mapping techniques to be useful tools in the study of the pathophysiology and natural history of myocardial infarction by optimizing the acquisition of surface-available electrocardiographic data (1–5). Recently, some longitudinal infarct mapping studies (5), as well as therapeutic intervention studies (6,7), have supported the view that the recovery phase of myocardial infarction is biologically dynamic and potentially modifiable by therapeutic interventions.

The purpose of this study of patients with anterior or inferior Q wave infarction was to gain insight into the balance of irreversible and potentially reversible damage that is present during the acute infarction phase in each clinical group. To accomplish this, electrical (body surface potential maps) and wall motion indexes (radionuclide ventriculo-

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grams) of myocardial injury were compared during the acute, early and late recovery phases of patients with a first Q wave infarction.

Methods

Study patients. In a group of 29 consecutive patients with a first Q wave myocardial infarction (2) initially studied on the 5th day after the onset of symptoms, we performed clinical follow-up and 120 lead body surface electrocardiographic mapping and radionuclide left ventriculographic studies on 23 of the subjects before discharge, and at 6 months after infarction (Table 1). The criteria for initial diagnosis of infarction were the presence of a compatible history of prolonged ischemic-type cardiac pain, diagnostic serum cardiac enzyme elevations (peak creatine kinase > twice upper limits of normal) and diagnostic 12 lead electrocardiographic changes of acute Q wave infarction (2). Patients were classified as having an anterior Q wave infarction if they had infarct-associated Q waves (≥0.04 second) in leads I, aVL and, V_1 to V_6 or R waves of less than 0.2 mV in leads V_1 and V_2 of the standard 12 lead electrocardiogram. Patients were designated as having an inferior Q wave infarction if they had infarct-associated Q waves in lead II or aVF of the standard 12 lead electrocardiogram. Excluded at the time of initial study were patients with a previous

Table 1. Clinical, Body Surface Map and Radionuclide Angiographic Data in 23 Patients During Acute and Recovery Phases of Myocardial Infarction

Case	Age (yr) & Sex	Beta-Blocker			Q Zone Integral Minimum (μV·s)			ΣQ Wave Integral (μV·s × 10^2)			ST Integral Maximum (μV·s)			LVEF (%)			Total WMA Score		
		T_1	T_2	T_3	T_1	T_2	T_3	T_1	T_2	T_3	T_1	T_2	T_3	T_1	T_2	T_3	T_1	T_2	T_3
Group 1 (anterior infarction)																			
1	50M	+	+	+	−48	−37	−36	−129	−120	−66	26	22	20	58	64	73	1	2	0
2	57M	0	0	0	−52	−40	−29	−395	−300	−238	33	23	18	50	59	77	8	2	0
3	36M	+	0	0	−63	−50	−33	−335	−249	−165	34	24	37	57	58	62	1	0	1
4	67M	0	+	0	−16	−25	−24	−63	−62	−38	84	48	21	59	45	58	9	9	5
5	56M	+	+	+	−56	−58	−46	−297	−328	−333	44	28	13	62	57	96	7	4	0
6	45M	+	+	+	−19	−21	−6	−79	−79	−37	27	14	11	57	67	72	4	4	0
7	75F	0	0	0	−38	−22	−23	−27	−41	−40	75	45	30	37	24	38	10	12	8
8	81F	+	+	+	−7	−11	−6	−40	−51	−67	30	5	5	51	41	61	4	1	0
9	68F	+	+	+	−18	−13	−12	−163	−124	−126	51	28	21	51	50	34	7	3	9
10	67F	+	+	+	−17	−19	−10	−69	−99	−83	30	13	4	61	56	70	6	7	2
11	41M	0	0	0	−18	−21	−39	−190	−142	−136	49	25	13	55	58	59	8	5	6
12	61M	+	+	+	−61	−42	−29	−136	−176	−112	48	23	21	52	54	57	6	4	5
Mean					−34	−30	−24	−160	−148	−120	44	25	18	54	53	63	6	4	3
± SD					±20	±15	±13	±122	±97	±90	±19	±12	±9	±7	±12	±17	±3	±4	±3
p Value					NS	NS	0.05	NS	NS	0.05	0.01	NS	0.01	NS	0.05	0.05	NS	NS	0.01
Group 2 (interior infarction)																			
13	57M	0	0	0	−9	−9	−9	−60	−47	−44	9	14	15	48	37	41	5	4	7
14	55M	0	+	+	−16	−14	−12	−136	−147	−105	17	67	33	71	57	59	1	2	1
15	56M	0	+	+	−17	−9	−16	−108	−120	−84	10	30	23	76	60	62	3	3	3
16	61M	0	0	0	−11	−12	−10	−98	−87	−59	8	6	21	55	48	54	3	3	5
17	42M	0	0	+	−8	−9	−9	−41	−31	−55	21	48	51	57	54	59	3	3	6
18	68M	0	0	0	−15	−13	−12	−154	−116	−113	12	7	5	45	60	63	2	6	3
19	45M	0	0	+	−14	−11	−11	−103	−79	−50	14	11	21	55	63	55	2	1	4
20	70M	+	+	+	−24	−10	−10	−90	−52	−40	17	7	23	43	35	42	10	10	10
21	68M	+	+	+	−9	−7	−9	−57	−55	−62	5	23	12	72	67	64	3	4	3
22	48M	0	0	+	−6	−6	−9	−29	−31	−62	7	31	17	83	92	71	6	4	3
23	40M	0	+	+	−15	−10	−10	−126	−103	−77	14	26	23	66	67	67	3	1	1
Mean					−13	−10	−11	−91	−79	−68	12	25	22	61	58	58	4	4	4
+ SD					±5	±2	±2	±40	±39	±24	±5	±19	±12	±13	±16	±10	±3	±3	±3
p Value					0.05	NS	NS	NS	NS	0.05	0.05	NS	NS	NS	NS	NS	NS	NS	NS

F = female; LVEF = left ventricular ejection fraction; M = male; NS = not significant; T_1 = day 5 before discharge; T_2 = day before discharge (≃ day 16); T_3 = 6 months after infarction; WMA = wall motion abnormality. + = present; 0 = absent.

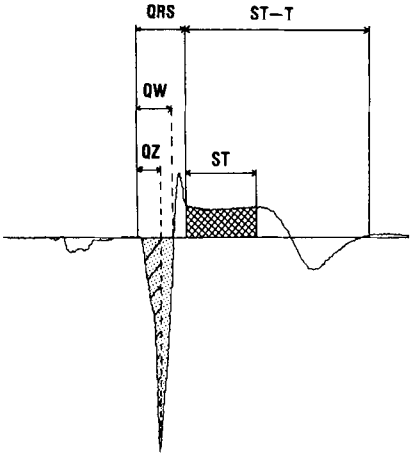
history of myocardial infarction, congenital or valvular cardiac disease, previous cardiac surgery, third degree atrioventricular block or left bundle branch block and patients in Killip functional class IV (2). The reasons for incomplete follow-up studies in 6 of the original 29 subjects were death (4 subjects), patient refusal (1 subject) and computer malfunction (1 subject). Of the four patients who died, all had initial anterior infarction and all had day 5 left ventricular ejection fraction values of less than 30% (range 21 to 29%).

The protocol for this study was approved by the Research Review Committee of the Victoria General Hospital and all patients gave informed consent before entry into the study. All usual coronary care unit diagnostic and treatment procedures were followed. Subsequent patient management was by individual physicians, with no specific or controlled therapy consequent to participation in the study. All protocol tests were done independently of any therapy; in particular, medications were not withdrawn or held on any test day. Beta-adrenergic blocking agents were the most widely used medication; their incidence was equal in both study groups (Table 1). Six patients, three in each group (Patients 6,7,12 to 14 and 20), took long-acting nitroglycerin preparations throughout the study period. Nine other patients (Patients 1, 2, 8, 10, 15, 17, 18, 21, and 23) received long-acting nitroglycerin intermittently during the study (six of the nine only during the in-hospital period). No other vasodilators (hydralazine, prazosin or captopril) were used. Three patients (Patients 7, 11, and 16) took digoxin at some point during the recovery phase and one patient (Patient 2) had coronary artery bypass graft surgery between hospital discharge and 6 months after infarction. Two patients (Patients 4 and 13) had auscultatory evidence of mitral insufficiency that was considered hemodynamically mild; in Patient 4 this was apparent only on the day 5 examination, whereas in Patient 13 it was present before discharge and at 6 months after infarction.

Nuclear imaging. Radionuclide left ventriculograms were performed according to previously described techniques using an Ohio Nuclear Series 420 Gamma Camera interfaced to a commercially available (Digital Equipment Corporation) Gamma II computer system (2,4). Briefly, multigated blood pool images were recorded in both the anterior and the left anterior oblique positions, and data were analyzed using commercially available software (2,4). Global left ventricular ejection fraction was calculated from the left anterior oblique view and regional wall motion abnormality score was assessed from both the anterior and the left anterior oblique views using a nine segment, 4 point scoring system where, in each segment; 0 = no abnormality; 1 = hypokinesia; 2 = akinesia and 3 = dyskinesia (2,4). The anterior view was divided into five equal segments designated clockwise as anterobasal, anterolateral, apical, inferior and inferobasal (2,4). Four segments were utilized in the left anterior oblique view, identified clockwise as posterolateral, inferoapical, apical septum and basal septum (2,4). A total wall motion abnormality score was calculated for each patient from the sum of the segmental scores (2,4). Previous validation studies have confirmed that these radionuclide wall motion analysis techniques correlate very highly ($r \simeq 0.85$) with biplane contrast ventriculographic analysis (8).

Body surface mapping. Body surface potential mapping data were obtained on the same days as the radionuclide angiographic data, using previously described techniques (1–5). Briefly, digitized signals from 117 torso and 3 limb electrocardiographic leads were simultaneously recorded with Wilson's central terminal as reference potential at 500 samples/channel for 15 seconds. Each channel amplifier had a gain of 2,000 with 10 bit samples; resolution was 10 μV for the least significant bit in the dynamic range $\pm$ 5 mV. The maps were all recorded with the patient in the supine position. Processing was done offline with selective averaging of all P-QRS-T complexes at each lead site. The computer program rejected artifact and ectopic activity and placed the averaged complex with the PR segment as baseline. Editing for incorrect waveform and baseline drift was performed and all leads judged invalid were deleted and then interpolated from surrounding leads. In this study of myocardial infarction we concentrated on three time integrals: Q zone, Q wave and ST segment (Fig. 1). Map display format was isointegral (Fig. 2), constructed for each individual, as mean maps for both infarct groups and with respective temporal difference (subtraction) maps (1,5). All

Figure 1. Signal-averaged torso lead illustrating the electrocardiographic time integrals evaluated in this study. The Q zone (QZ) integral was calculated as the area under the first half of the QRS curve (**diagonal lines**). The ST segment integral (**cross-hatched area**) represented the area under the first 3/8 portion of the ST-T curve; both of these integrals were calculated in every lead. The Q wave (QW) integral represented the area under the QRS curve from the initial negative deflection to the return of the waveform to baseline (**stippled area**); it was only calculated in leads with a Q wave (2).



JACC Vol. 8, No. 3
September 1986:521–8

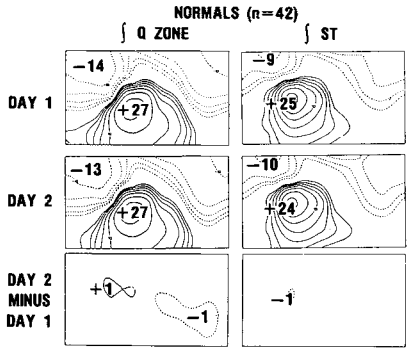


Figure 2. Group mean isointegral Q zone and ST segment maps of 42 normal adults at two separate times and mean subtraction maps illustrating the map display format. Each (**rectangle**) represents the body surface, unfolded, with the left half representing the anterior torso and the right half representing the back. The lines are isocontour lines joining time integrals of equal value. In the recorded maps (Day 1; Day 2) the **solid lines** represent positive values and the **dotted lines** represent negative values. In the temporal difference (subtraction) maps (Day 2 minus Day 1) the **solid lines** represent an increase in values at the later (Day 2), relative to the prior (Day 1) time; the **dotted lines** represent decreased values with time. The maximal and minimal values are numerically identified. The mean maps of normal subjects were constructed using the group mean value of the respective integrals at each lead site (5). In addition to map format, they illustrate the very small temporal differences to be expected, in normal circumstances, in depolarization and repolarization integrals (5).

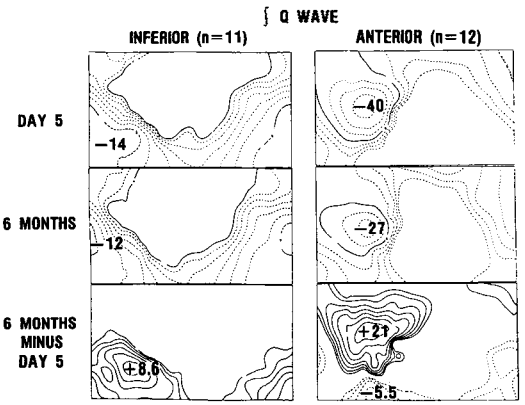


Figure 3. Mean Q wave integral maps at day 5 and month 6 after infarction and temporal difference maps for the two patient groups. The anterior infarction group was characterized by infarct-related Q wave distributions largely over the anterior torso at day 5 and month 6. Similarly, the inferior infarction group revealed the primary infarct-related Q wave distributions over the interior torso at both study times. The difference maps indicate a substantial positive change (**solid lines**) in Q wave values over the primary infarct-reflecting torso areas (anteriorly in the anterior group, inferiorly in the inferior group). This means that, with time, in these areas Q waves became less negative in potential or shorter in duration.

time-integral values were expressed as microvolt·seconds (μV·s).

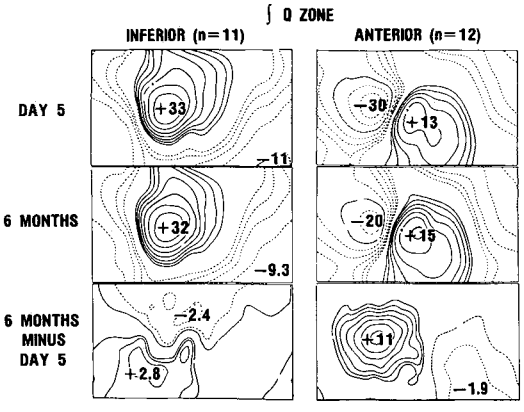
Data analysis. Five quantitative variables (two radionuclide and three body surface map) were selected for analysis. They were: left ventricular ejection fraction, left ventricular total wall motion abnormality score, Q zone integral minimum that is, the least (most negative) Q zone value on the body surface, ΣQ wave integral, that is, the sum of the area under *all* torso Q waves, and, ST segment maximum, that is the greatest (most positive) ST value on the torso. These two radionuclide and three map variables were selected for analysis because they were representative reflectors of global and segmental left ventricular systolic function and ventricular depolarization and repolarization events, respectively. All had also been previously found to be significant discriminators in a multivariate analysis of anterior and inferior Q wave infarction groups (2).

Statistics. All grouped data were expressed as mean ± 1 SD. In both anterior and inferior infarction groups, repeated measures analysis of variance were used to test the null hypotheses that there were no temporal differences in the five variables over the course of the three study times. When these hypotheses were rejected, Tukey's multiple comparison test was used to determine where the differences occurred. For the anterior and inferior infarction groups the null hypotheses that there were no bivariate correlations of long-term (day 5 to month 6) temporal change in any variables were tested by the Pearsonian correlation coefficient.

Results

Body surface mapping (Fig. 3 to 5). The spatial distributions of Q zone and Q wave integrals over the torso reflected the characteristic patterns of infarction in the re-

Figure 4. Mean Q zone integral maps at day 5 and month 6 after infarction and temporal difference maps for the two patient groups. Note that in the difference maps the gain (**solid lines**) in Q zone values over the anterior torso in the group with anterior infarction appears substantially larger than the gain (**solid lines**) in values over the inferior torso in the group with inferior infarction. Both patient groups showed small reciprocal changes (**dotted lines**) (posteriorly in the anterior group, superiorly in the inferior group).



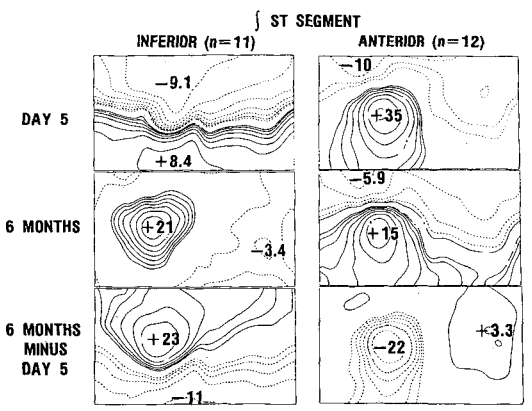


Figure 5. Mean ST integral maps at day 5 and month 6 after infarction and temporal difference maps for the two patient groups. The primary temporal changes in the repolarization pattern were decreases in ST values over the anterior torso in the group with anterior infarction and over the inferior surface in the group with inferior infarction. Reciprocal temporal changes are represented in the difference maps of both groups by the **solid lines** (positive change, or increase in ST values).

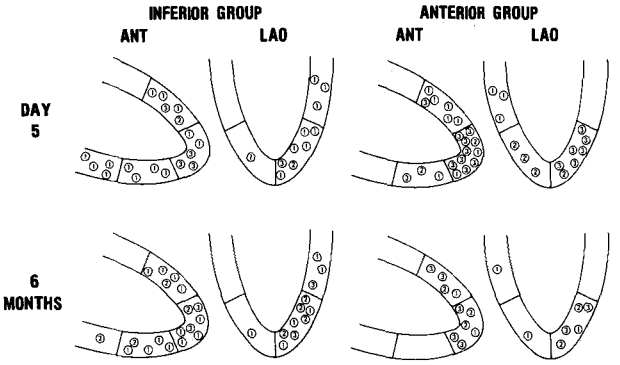


Figure 6. Distribution of radionuclide angiographic wall motion abnormalities in the two study groups at day 5 and month 6 after infarction. ANT = anterior view; LAO = left anterior oblique view. The numbers within the circles represent the degree of wall motion abnormality: 1 = hypokinesia; 2 = akinesia; 3 = dyskinesia.

spective patient groups at all study times (Fig. 3 and 4). In the anterior infarction group the primary infarct pattern was a markedly negative depolarization pattern over the anterior torso; in the inferior infarction group, the primary pattern of negative Q zone and Q wave distributions was located over the inferior torso. Group-mean difference maps (month 6 − day 5) revealed, however, that there were quantitative changes in Q zone and Q wave values in both groups from the acute phase to 6 months after infarction. Patients with anterior infarction had a net gain in both depolarization integrals over the anterior chest; those with inferior infarction had a gain in values over the inferior torso, both anteriorly and posteriorly. Only very small reciprocal decreases in torso Q zone or Q wave distributions were apparent in either group.

ST integral distributions demonstrated marked spatial changes in both groups (Fig. 5). Difference maps confirmed the changes in ST values. These included not only the primary decrease in ST values over the anterior and inferior torso in the anterior and the inferior infarction group, respectively, but also a very substantial reciprocal increase in ST values over the superior torso in the inferior infarction group (Fig. 5). Both the primary and the reciprocal changes in the inferior infarction group were very similar to those previously described (5) in another group of 32 patients with inferior infarction followed up for a mean of 8 months. Long-term postinfarction body surface map ST patterns of patients with anterior infarction have not previously been reported.

Nuclear imaging (Fig 6). The spatial distributions of the left ventricular wall motion abnormalities in both study groups during the acute and recovery phases are summarized in Figure 6. Among the 17 patients with anterior infarction there were 33 wall motion abnormalities on day 5. The incidence decreased, however, to 17 abnormalities at 6 months after infarction, with the greatest decrease occurring in the apical segment. In the group with inferior infarction, the incidence of wall motion abnormalities remained temporally unchanged: 29 at day 5; 30 at 6 months.

Quantitative analysis (Tables 1 and 2). Comparison of temporal changes in the five selected electrocardiographic and radionuclide variables for both infarction groups is illustrated in Table 1. In the inferior infarction group the only significant change in any variable between day 5 and month 6 after infarction was a modest gain of ΣQ wave integral values. In contrast, the anterior infarction group displayed significant changes between day 5 and month 6 in all variables. Most of the changes among the patients with anterior infarction represented an apparently gradual, continuous improvement in electrical and mechanical function with time. The reduction in maximal ST segment occurred, however, before discharge and remained unchanged at 6 months whereas ejection fraction showed a biphasic temporal change, with a slight decrease in the mean predischarge value, relative to day 5 and a subsequent significant increase by month 6. It seems reasonable to speculate that the biphasic change in global left ventricular systolic performance may have reflected relative hypercontractility of noninjured segments at day 5, with removal of the hypercontractile stimulus by discharge, and subsequent improvement in initially dysfunctional segments by month 6. The significant inverse correlation of temporal gain in ejection fraction with reduction of wall motion abnormality score (Table 2) supports at least the last part of this supposition.

The inter- and intratechnique correlations of all long-term (day 5 to month 6 after infarction) temporal changes in the body surface map and radionuclide variables for the two patient groups are illustrated in Table 2. The highest

Table 2. Matrices of Bivariate Correlation Coefficients of Change (day 5 to month 6) in Body Surface Map and Radionuclide Variables in 23 Patients With a First Q Wave Infarction

	ΔQ Wave	ΔST	ΔEF	ΔWMA
Group 1 (Anterior infarction) (n = 12)				
ΔQ zone	0.38	0.58	0.22	0.05
ΔQ wave	—	0.58	0.04	0.10
ΔST	—	—	0.32	0.12
ΔEF	—	—	—	−0.82
Group 2 (Inferior infarction) (n = 11)				
ΔQ zone	0.71	−0.28	0.24	−0.06
ΔQ wave	—	−0.41	0.41	0.20
ΔST	—	—	−0.41	0.23
ΔEF	—	—	—	0.35

Δ = difference between day 5 and month 6 after infarction; EF = left ventricular ejection fraction; Q wave = ΣQ wave integral values; Q zone = minimal Q zone integral; ST = maximal ST integral; WMA = total wall motion abnormality score.

correlation (r = −0.82) was the inverse relation of changes in ejection fraction and wall motion abnormality score within the anterior infarction group. Although the data indicate a significant relation between the postinfarction changes in global and segmental left ventricular systolic performance in this group, they also indicate that only about 67% of the variation (r^2 = 0.67) in one variable was due to its dependence on the other variable; 33% of the variation of this best intratechnique correlation was undetermined or random. Other significant intratechnique correlations were found between changes in each of the depolarization variables and change in maximal ST segment (r = 0.58) in patients with anterior infarction and between changes in the depolarization variables (r = 0.71) for patients with inferior infarction. Significant intertechnique (body surface map versus radionuclide) correlations were not found in either group. Overall, these data suggest that postinfarction temporal changes in electrocardiographic and wall motion variables occur independently of each other. This heterogeneity of temporal change is not surprising in light of the widely variable correlations of multiple indirect quantitative indicators of myocardial injury that are found during the acute phase of myocardial infarction (4).

Discussion

This study has three major findings. The data provide direct evidence that some patient groups have significant positive change, or recovery, in electrical and mechanical measures of myocardial injury in the postacute phase of infarction. 2) The data suggest that recovery is largely gradual, perhaps even over a several month interval, with relatively few changes occurring during the in-hospital period. 3) The data demonstrate that recovery patterns are heterogeneous among different clinical groups and measurement variables.

Comparison with previous studies. Pathophysiologic studies in the postinfarction period utilizing multiple variable appear to be relatively uncommon, although studies examining electrocardiographic (3,5) or wall motion patterns (9–15) have been reported. Quantitative sequential electrocardiographic mapping studies (3) of the early, evolutionary phase of infarction revealed very small changes in depolarization and repolarization patterns and none if the patients were admitted more than 8 hours after the onset of symptoms. Sequential mapping studies (5) limited to patients with inferior Q wave infarction revealed no significant changes in depolarization (Q zone) patterns during a 6 month recovery period, but relatively large temporal changes in repolarization (ST segment) patterns. Radionuclide angiographic infarct follow-up studies have been more common (9–14). Overall, these studies have shown heterogeneous changes in postinfarction left ventricular ejection fraction and, with one exception (13), no change in group mean values. In a radionuclide study of 45 patients before hospital discharge and at 6 to 14 months after infarction, Borer et al (13) found a significant temporal increase in mean ejection fraction in a subgroup of 17 patients with a predischarge exercise ejection fraction value greater than 40%. They concluded from their results that "the capacity for improvement in myocardial function does exist during the year after acute infarction."

The data from our study are not inconsistent with any of the previously reported data (3,5,9–15). We also found a great deal of intergroup and intragroup variability in postinfarction electrocardiographic and wall motion recovery patterns. The major differences between this study and those mentioned were 1) the sampling of multiple variables, 2) the prior definition of specific clinical groups, and 3) the broad temporal spectrum of the sampling period. We believe that the latter two factors allowed the detection of gradual

and significant group mean changes in electrocardiographic and ventriculographic infarct recovery patterns, despite intragroup temporal variability.

Limitations. Confounding factors in this study were the use of postinfarction beta-blockers and other medical (and in one case, surgical) therapy. When the study began, the pattern of postinfarction management was in flux after publication of data from large secondary prevention trials (6,7), but at the time follow-up was completed, the majority of patients in the groups with inferior or anterior wall infarction were taking beta-blocker medication (Table 1). Although there were no statistically significant differences in the prevalence of beta-blocker use in the two patient groups, it is not possible to conclude that the greater degree of recovery in the group, with anterior infarction would have occurred in the absence of beta-blocker therapy. Rather, it is possible that beta-blocker therapy in the postinfarction setting may have had a permissive, or even active, effect in expediting, or making more efficacious, the recovery process. This hypothetical beneficial effect on recovery might be entirely separate from other hypotheses on the pathophysiology of postinfarction beta-blocker benefit, namely, anti-ischemic or antiarrhythmic effects (16). The role of each of these possible beneficial mechanisms of beta-blocker therapy remains speculative.

We also performed retrospective analysis of temporal changes in the electrocardiographic and ventriculographic variables between day 5 and month 6, with the patients grouped according to whether they received no (n = 8), some (n = 15) or continuous (n = 6) nitroglycerin therapy. We found that there were significant gains in ΣQ wave and Q zone minimal values in patients from both groups who were taking nitroglycerin, although there were no significant changes in ST maximum or either of the ventriculographic variables. In the patients not receiving nitroglycerin therapy there were no temporal changes in any variable. These findings are provocative but they must be interpreted with caution. They suggest that nitroglycerin might be at least partially responsible for the observed biologically positive temporal changes. Moreover, the dichotomy of the electrocardiographic, but not ventriculographic, recovery in the patients receiving nitroglycerin suggests that, if caused by nitroglycerin, the recovery was *not* due to the hemodynamic effects of nitroglycerin.

Implications. The greater recovery seen in the group with anterior infarction (Table 1) suggests that the reversible component of acute Q wave infarction may be greater when the acute phase electrocardiographic and ventriculographic indexes of damage are relatively large as they appear to be in anterior, as compared with inferior, infarction (2). It would appear, however, that the relation between potential for recovery and acute phase indexes of infarct "size" is valid only up to a certain degree of acute injury. If acute phase injury is very great, such as with an ejection fraction of less than 30%, then recovery may be severely prejudiced. In this study, four of four patients with an acute phase ejection fraction of less than 30% died during the follow-up interval. Thus, there appear to be three possible patterns to the recovery phase of Q wave infarction. If acute phase injury is relatively small, as exemplified by our patients with inferior infarction (Table 1), little significant recovery in indirect measures of damage is seen. In contrast, if acute phase injury is moderate, as in our patients with anterior infarction who survived, significant recovery can be anticipated. Finally, if acute phase injury is very severe, recovery may be greatly prejudiced and further deterioration and death are highly likely.

The concept of prolonged ischemic-induced myocardial dysfunction short of myocardial cell necrosis ("stunned myocardium") has been previously recognized and the assessment of intervention efficacy in modification of myocardial damage after coronary occlusion has, consequently, been recommended to include evaluations for at least 2 weeks after the intervention (17). A major implication of this study is that recovery from infarct-related injury or "stunning," or both, may require a much longer period, possibly up to 6 months after infarction. Assessments of modification of infarction injury should, therefore, probably be equally extended. A related implication is that acute intervention trials should ideally be performed as randomized control trials because the absence of a positive long-term temporal improvement in the intervention group may represent not only the lack of a positive treatment effect but also, at least in some patient groups, a "negative" effect of removal of naturally occurring recovery processes. In addition, the heterogeneous recovery pattern of different clinical groups suggests that different groups may benefit from temporally specific or agent-specific interventions. For example, patients with inferior Q wave infarction may require very early interventions if modification of injury indexes is to be obtained, whereas patients with anterior Q wave infarction may still have the capability for reversibility of damage even if the early therapeutic window is missed. This remains to be tested.

Finally, the data suggest that postinfarction changes in electrocardiographic patterns are largely independent of changes in left ventricular wall motion patterns. Because of the wide correlative variability of indirect measurements of acute infarct injury (4) and their apparently independent temporal patterns (Table 2), we believe that the various measurements should probably be viewed as complementary, rather than as substitutionary, data.

Conclusions. Our findings support and extend the concept that acute myocardial infarction is not an all or none phenomenon. The determinants and mechanisms of reversibility of acute phase electrical and mechanical injury, and

their relation to prognosis, require further investigation if intervention therapy is to be utilized most effectively.

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References

1. Montague TJ, Smith ER, Spencer CA, et al. Body surface electrocardiographic mapping in for myocardial infarction: manifestation of left and right ventricular involvement. Circulation 1983;67:665–73.
2. McPherson DD, Spencer CA, Johnstone DE, et al. Acute myocardial infarction: comparison of body surface map and nuclear angiographic patterns (abstr). Clin Invest Med 1983;6(suppl I):23.
3. Smith ER, Montague TJ, Cameron DA, Gewirtz H, Rautaharju PM, Horacek BM. Electrocardiographic evolution of acute myocardial infarction: analysis by body surface potential mapping. (abstr). Circulation 1979;60(suppl II): II-71.
4. McPherson DD, Horacek BM, Spencer CA, et al. Indirect measurement of infarct size: correlative variability of enzyme, radionuclear angiographic and body-surface-map variables in 34 patients during the acute phase of first myocardial infarction. Chest 1985;88:841–8.
5. Montague TJ, Smith ER, Johnstone DE, et al. Temporal evolution of body surface map patterns following acute inferior myocardial infarction. J Electrocardiol 1984;17:319–28.
6. The Norwegian Multicenter Study Group. Timolol-induced reduction in mortality and re-infarction in patients surviving acute myocardial infarction. N Engl J Med 1981;304:801–7.
7. Beta-blocker Heart Attack Trial Research Group. A randomized trial of propranolol in patients with acute myocardial infarctions. I. Mortality results. JAMA 1982;247:1707–14.
8. Miller RM, Bessoudo RM, Fraser DB, Martin RH, Johnstone DE. Factors influencing the accuracy of segmental wall motion analysis by multigated blood pool imaging (abstr). Proc Radiol Soc North Am. The 66th Scientific Assembly, Dallas, Texas, November, 1980:217.
9. Kostuk WJ, Ehsani AA, Karliner JS, et al. Left ventricular performance after myocardial infarction assessed by radioisotope angiocardiography. Circulation 1973;47:242–9.
10. Rigo P, Murray M, Strauss HW, et al. Left ventricular function in acute myocardial infarction evaluated by gated scintiphotography. Circulation 1974;50:678–84.
11. Reduto LA, Berger HJ, Cohen LS, Gottschalk A, Zaret BL. Sequential radionuclide assessment of left and right ventricular performance after acute transmural myocardial infarction. Ann Intern Med 1978;89:441–7.
12. Mangschau A, Karlsen RL, Lippestad CT, Nerdrum HJ. Systolic time intervals and ejection fraction in assessing left ventricular performance following acute myocardial infarction. Acta Med Scand 1984;215:341–7.
13. Borer JS, Rosing DR, Miller RH, et al. Natural history of left ventricular function during 1 year after acute myocardial infarction: comparison with clinical, electrocardiographic and biochemical determinations. Am J Cardiol 1980;46:1–12.
14. Nemerovski M, Shah PK, Pichler M, Berman DS, Shellock F, Swan HJC. Radionuclide assessment of sequential changes in left and right ventricular function following first acute transmural myocardial infarction. Am Heart J 1982;104:709–17.
15. Hirsowitz GS, Lalsier JB, Marks DS, Lee TG, Goldberg AD, Goldstein S. Sequential radionuclide angiographic assessment of left and right ventricular performance and quantitative thallium-201 scintigraphy following acute myocardial infarction. Am Heart J 1984;107:934–9.
16. Bigger JT, Coromilas J. How do beta-blockers protect after myocardial infarctions? Ann Intern Med 1984;101:256–7.
17. Braunwald E, Kloner RA. The stunned myocardium: prolonged, postischemic ventricular dysfunction. Circulation 1982;66:1146–9.