Precordial ST-Segment Mapping

1. Clinical Studies in the Coronary Care Unit

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SUMMARY

Precordial ST-segment mapping was applied serially in the coronary care unit for the study of 46 patients with myocardial infarction (MI), using a 49-lead system. Data from the maps were compared with clinical status of patients, conventional ECGs obtained simultaneously, and serum enzyme levels. Stability of the maps over a one hour period was noted in the early phase of admission. However, a drop of 32% of the sum of ST-segment elevations (+ Σ ST) was detected in eight patients with uncomplicated anterior MI over the first 24 hours after admission. Extension of infarction was associated with abrupt rise of + Σ ST, and was diagnosed in two cases from maps in the presence of unchanged standard ECGs. The course of ST elevations was followed more accurately by the map than the standard ECG in eight patients. Pericarditis invalidated the technique completely, due to persistent + Σ ST. The standard ECG was superior to the map in following patients with inferior MI. A case of true posterior MI was more accurately delineated by maps of the posterior thorax than by the standard ECG. Intraventricular conduction defects and pacemaking interfered with the maps. Early repolarization produced stable maps; however, mapping showed no advantages over the standard ECG. Preinfarction angina can probably be followed by serial mapping of ST-segment depression.

Due to major developments of the past few years in the management of arrhythmias in patients with acute myocardial infarction (MI), current morbidity and mortality in the coronary care unit (CCU) have been almost exclusively associated with power failure. Since such failure appears to be related to the degree of damage sustained by the left ventricle, it becomes crucial to assess the magnitude of ischemic injury as early as possible in the clinical course. In experimental studies epicardial and precordial maps have been used to detect and quantitate ischemic damage and to assess the effect of interventions on the degree of ischemic injury. These means of limiting the amount of necrosis have also been applied to patients with MI. Such interventions require knowledge of natural course of ST deviations in patients. In a few studies ST maps were found sensitive in detecting extension or in following evolution of infarcts. However, others have expressed doubt as to the usefulness of the technique. The present study reports on the results of serial precordial mapping applied in patients treated in the CCU and discusses the advantages of this modality over the conventional ECG.

Material and Methods

Forty-six patients (33 males and 13 females) age 55.3 ± 1.7 (SEM, range 31 to 79) years who were admitted to the CCU of Boston City Hospital with the presumptive clinical diagnosis of MI were studied. All patients had ST elevation in their 12-lead ECG and chest pain of over one hour’s duration within the 24 hours before admission, except for one patient who was studied 48 hours after onset of pain. Excluded were patients with left and right bundle branch block (LBBB, RBBB), and patients who had a pacemaker implanted. All ECGs were recorded using a Hewlett-Packard 1511A electrocardiograph. A self-retaining ECG electrode (HP Part No. 9301-0122) with a contact diameter of 15 mm, attached to the “V” lead was used for precordial mapping. Studies were not done during an episode of chest pain, save for the initial ones on admission. Paper speed was 25 mm/sec and the standardization was 0.1 mV/mm. Patients were studied in the supine position following marking of a grid on the anterior chest wall, consisting of 49 recording sites, arranged in seven horizontal rows, each including seven marks (fig. 1). The seven horizontal rows were designated by letters A to G and the vertical columns by numbers 1 to 7. The top right mark (A7) was made at the second intercostal space to the right of the sternum, A6 was made at the second intercostal space to the left of the sternum, A5 was placed at the same horizontal level on the anterior axillary line, A4 and A3 marks were spaced evenly between A3 and A5, and A2 and A1 were made at the same horizontal level on the mid- and posterior ax-
illar lines. Using the first row as a guideline, six more rows were placed. Horizontal rows below the rib cage were spaced the same distance apart as the second and the third intercostal spaces. The last row of marks (fig. 1) was approximately six to eight cm below the xiphisternum. Interelectrode spacing both horizontally and vertically depended on the size of the patient’s chest. The marks were made with a skin pencil and assured daily exact repositioning of the “V” electrode.

Time of the ECG recordings, clinical status of the patient, and blood pressure were recorded. A 12-lead ECG was made just before the 49-lead map. Patients had daily ECGs and maps while in the CCU. A final set of studies was done just before discharge from the hospital. Care was taken to obtain tracings from each site with stable baseline (no more than 1.0 mm baseline deviation in five successive beats). Five beats or more from such stable tracings were analyzed and averaged together for each data point. ST-segment deviations were measured in mm at 0.06 sec after the nadir of the S wave to the nearest 0.5 mm. The T-P or the P-R (when the T-P was difficult to delineate because of tachycardia) intervals were used as a baseline.\(^{11, 12, 16}\)

The sum of ST elevations of all 49 leads (+ \(\Sigma\) ST), as used by other workers,\(^{11-15, 18}\) was employed as an index of the magnitude of myocardial injury in patients with an anterior MI. For patients with inferior MI the sum of ST depressions (– \(\Sigma\) ST) recorded in the high anterior chest was measured, in addition to the + \(\Sigma\) ST measured in the lower chest and upper epigastrium.\(^{11, 13, 17}\) The ST elevation (+ST) and ST depression (–ST) values were charted, and their distribution was taken as an expression of the location and extent of the ischemic damage.\(^{12, 13, 18}\)

In one patient with both lateral and true posterior MI three left parascapular and three left paracapular leads were recorded at the third, fourth, and fifth intercostal spaces for verification of the suspected true posterior component of the infarct.\(^{14}\) In all patients daily determinations of creatine phosphokinase (CPK), with normal values up to 50 I.U./L;\(^{18}\) serum glutamic oxalacetic transaminase (SGOT), with normal values up to 20 I.U./L;\(^{19}\) and lactic dehydrogenase (LDH), with normal values up to 110 I.U./L,\(^{20}\) were carried out for as long as the patients remained in the CCU. No intramuscular injections were administered during the total CCU course of the patients.\(^{21}\) Daily variations of + \(\Sigma\) ST and – \(\Sigma\) ST were correlated with clinical events, enzyme values, and standard ECGs. The routine management of the patients was not altered by the study protocol, and diuretics, digitalis, and antiarrhythmic drugs were administered as clinically indicated.\(^{1-22}\)

Patients with MI were classified according to Killip’s clinical classification system (Class I, no signs of cardiac decompensation; Class II, mild to moderate heart failure with rales over an area 50% or less of lungfield; Class III, pulmonary edema with rales over an area more than 50% of lungfields; Class IV, cardiogenic shock with hypotension and signs of poor peripheral perfusion).\(^{23}\) The results of the statistical analyses are reported as mean ± standard error of the mean (SEM).

Results

A total of 263 precordial ST maps was analyzed.

Reproducibility of the ST Precordial Maps

Fourteen consecutive patients with acute anterior MI had two maps recorded 59 ± 4 (range 35 to 78) min apart (from the beginning of the first map to the end of the second map, time required for mapping was 12 to 15 min). The first map was done 6 ± 1 (range 1.3 to 12) hours following onset of pain. Thirteen patients were clinically stable during the study period; nine patients had chest pain, unchanged in character between these two recordings, and the remaining four patients were free of pain. Stability of + \(\Sigma\) ST, blood pressures, and heart rates over this time interval is shown in table 1. Range of differences of + \(\Sigma\) ST from first to second maps was –5.5 to +3.5 mm. None of the differences was significant (paired differences). One patient, who was not included in the results shown in table 1, showed a marked change of + \(\Sigma\) ST between the two maps (49 to 74), but severe chest pain developed in the interim. The patient’s blood pressure and heart rate remained stable.

The importance of varying the recording site around the individual components of the grid was assessed in seven patients; by applying the “V” electrode one cm above, below, to the right and left of several of the 49 marks of the map, the degree of +ST in a given lead might vary by as much as 1.5 mm.

Anterior Myocardial Infarction

A total of 25 patients with anterior transmural MI were studied. Eleven had an anteroseptal MI and 14...
had extensive anterior damage with varying degrees of lateral wall involvement. The initial maps were recorded in 20 patients within 6.7 ± 0.8 (range 1–12) hours, in two patients within 14 hours, and in three patients within 24 hours following the onset of chest pain. Eight patients had had a previous inferior transmural MI and one had had a nontransmural MI. A total of 7.0 ± 0.4 (range 5–14) maps per patient were recorded. Seven patients were in Class I, 12 in Class II, and three patients in each of classes III and IV. Classification was based on the highest class reached. Eleven patients received digitalis at some point of their hospitalization.

Six patients died, three of cardiogenic shock and one each from hypoxic encephalopathy following ventricular fibrillation, cerebral embolism and cardiac arrest (the latter after transfer from the CCU, without documentation of the exact cause). Maximum +Σ ST (+ Σ STmax) of these patients was 101.2 ± 13.9 (range 50.0 to 148.5) mm. Comparison of this value with the +Σ STmax of the rest of the patients, which was 81.4 ± 10.2 (range 30.5 to 195.0) mm, did not reveal significant differences (unpaired t-test). Two of these six patients also had pericarditis. From the patients who died of cardiogenic shock, one had a +Σ STmax of 50 mm and a history of two previous MIs; in the other two patients +Σ STmax values were 148.5 and 89 mm.

Recorded +ST from individual leads ranged from 0 to 10 mm. Occasionally small −ST (0.5 to 1.0 mm) was recorded in leads of rows F and G or columns 6 and 7 (fig. 1). The location of the MI was judged from the leads showing maximal +ST on both the standard ECG and the map; these showed close agreement. On the map, the central maximum area was concentrically surrounded by progressively lesser degrees of +ST as the distance of electrodes from the central areas increased.

In anteroseptal MI column number one to three or four displayed +ST. In extensive anterior or anterolateral MIs all columns frequently showed +ST (fig. 2). Since pericarditis complicating MI affects the ST segment in an unpredictable way, the 25 patients were analyzed separately with regard to the study of certain variables, depending on the presence or absence of pericarditis. There was no correlation between +Σ STmax and peak values of CPK, SGOT, and LDH in the 15 patients without pericarditis (r = 0.406, 0.477, 0.372 respectively, NS). The relation between +Σ STmax and clinical class in all 25 patients is shown in figure 3.

Of the 25 patients, 17 suffered either an extension of MI or complicating pericarditis. The remaining eight patients constitute a group in whom natural evolution of +ST could be studied. Data obtained during mapping on these patients the initial four days and after 17.5 ± 1.5 (range 10 to 23) days are shown in table 2. A drop of +Σ ST of 32.1 ± 5.4 (range 12.6 to 56.9)% was measured in these patients within the first 24 hours. Evolution was characterized by decrease of magnitude of +ST displayed by the leads showing the maximal +ST and by return to the isoelectric line of ST leads showing initially smaller degrees of +ST.

Table 1

<table>
<thead>
<tr>
<th>+Σ ST (mm)</th>
<th>HR (beats/min)</th>
<th>BP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>First map</td>
<td>79.0 ± 11.0 (25.5–155.5)</td>
<td>92 ± 3 (75–113)</td>
</tr>
<tr>
<td>Second map</td>
<td>78.0 ± 10.0 (25.5–152.0)</td>
<td>90 ± 3 (71–110)</td>
</tr>
</tbody>
</table>

Ranges are shown in parentheses.

* = systolic blood pressure.
† = diastolic blood pressure.

Abbreviations: +ΣST = sum of ST elevations; HR = heart rate; BP = blood pressure.

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Selected leads from precordial map of a 60-year-old male patient with extensive anterior myocardial infarction. Depicted tracings were obtained from horizontal rows A, C, and E and vertical columns 2, 3, 4, and 5. C3 recording was made from the conventional V3 position. Localization of recording sites can be facilitated by comparison with figure 1.
patients were omitted from this analysis because a pericardial friction rub occurred in close proximity to extension (coincident in one and preceding by one day in the other). As a result change of + Σ ST could not be attributed solely to extension. No correlation was found between increase in + Σ ST and increase in CPK \( (r = -0.010, \text{NS}) \), SGOT \( (r = 0.406, \text{NS}) \), and LDH \( (r = -0.234, \text{NS}) \). Initial + Σ ST of these 12 patients \( (78.3 \pm 14.7; \text{range 5.5 to 155.5 mm}) \) and the similar value of the patients without extension \( (68.4 \pm 9.3; \text{range 30.5 to 132.5 mm}) \) were not statistically different (unpaired t-test). Extension of MI occurred in the CCU 2.4 ± 0.3 (range 1 to 5) days after hospitalization in these 12 patients. Four patients had two and one had three extensions, which were diagnosed as above, occurred from the third to tenth day after admission and were associated with further increase of + Σ ST, of smaller magnitude than the initial one \( (15.0 \pm 3.5, \text{range 4.5 to 29}) \). Good correlation was found between + Σ ST\text{max} and the final + Σ ST \( (P < 0.001, \text{paired t-test}) \). Only the six patients who died were excluded from this analysis. A good correlation also was found between + Σ ST\text{max} and final + Σ ST of patients without pericarditis (with or without extension) \( (P < 0.01, \text{paired t-test}) \).

Of the ten patients with pericarditis, seven developed a friction rub: six patients \( 3.5 \pm 0.6 \) (range 2 to 6) days after admission to the CCU and one the twelfth day after admission, in the ward. One patient had recurrent episodes of pericarditis and eventually developed Dressler's syndrome, three weeks after admission. Friction rub was heard by at least three observers (one of the authors always included) on at least three occasions over the course of one day (four patients), two days (one patient), three days (one patient), and several days (in the patient with Dressler's syndrome). (A cardiologist examined the patients twice daily and the house staff rotating in CCU, repeatedly. Patients in the ward were examined by the house staff repeatedly and once a day by the attending physician.) Three other patients were diagnosed as having pericarditis despite the absence of friction rub, on the basis of recurrent episodes of pleuritic pain, pain intensified by coughing and lying in the left lateral position, and partially relieved by sit-

### Table 2

**Electrocardiographic Data from Serial Mapping of Patients with Anterior Myocardial Infarction without Extension or Pericarditis \( (N = 8) \)**

<table>
<thead>
<tr>
<th></th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Final map</th>
</tr>
</thead>
<tbody>
<tr>
<td>+ΣST (mm)</td>
<td>66.3 ± 14.2</td>
<td>41.8 ± 8.8</td>
<td>37.3 ± 9.5</td>
<td>30.6 ± 8.0</td>
<td>11.8 ± 4.0</td>
</tr>
<tr>
<td></td>
<td>(30.5 - 132.5)</td>
<td>(12.0 - 79.0)</td>
<td>(6.5 - 78.5)</td>
<td>(6.5 - 72.0)</td>
<td>(3.0 - 33.0)</td>
</tr>
</tbody>
</table>

Ranges are shown in parentheses.
+ΣST = sum of ST elevations.
ting up, persisting fever (101.5–102.0°F) with negative results of complete fever work-up and clear-cut response to salicylates.

Data from maps of patients with pericarditis are shown in table 3. The final map was obtained 21.5 ± 2.8 (range 13 to 40) days after admission. Details on patients with pericarditis but without extension of MI are shown in table 4. The final records were obtained after 20.4 ± 2.3 (range 15 to 29) days of hospitalization. Initial + Σ STs (60.1 ± 10.2, range 5.5 to 132.5) of patients without pericarditis (seven with a subsequent extension) and of those without pericarditis (five with subsequent extension) (92.7 ± 12.6; range 59 to 155.5, table 3) were not statistically different (unpaired t-test). Initial + Σ STs of patients with pericarditis but no extension (71.7 ± 10.3, table 4) and of those with uncomplicated MI were not statistically different (66.3 ± 14.2, table 2, unpaired t-test). Final + Σ ST (tables 2 and 4) of these groups were also not statistically different (unpaired t-test).

Comparison of all recorded maps with the + Σ ST of the six standard precordial leads revealed some association (r = 0.631). Plots of daily + Σ ST of the map and + Σ ST of the six precordial leads were in good agreement in 15 of 25 patients (fig. 4). The final + Σ ST of one patient (last set of plots in fig. 4) was found to be higher than the + Σ ST of last map done in the CCU (36.5 vs 11.5). No chest pain occurred in the interim (13 days). Rare premature ventricular beats had been observed three days prior to the last map. No enzyme values were available for that period. The patient had a clinically diagnosed ventricular aneurysm, later proven by fluoroscopy. ECGs three months later showed persistent +ST. In eight patients there was some disagreement between the map and the standard precordial leads. The discrepancy was due to changes of +ST occurring outside the area covered by the six precordial leads (figs. 1 and 5). In two patients extension of MI clearly depicted by a rise in the + Σ ST of the map was unaccompanied by changes in + Σ ST of the precordial standard leads (fig. 6).

Inferior Myocardial Infarction

Ten patients with inferior transmural MI were studied. The first map was recorded 7.7 ± 1.5 (range 3 to 14) hours from onset of chest pain (eight patients) and 19 and 48 hours in the other two patients. Four patients had in addition a lateral MI (+ST in leads V₅ and V₆ and the left lateral portion of the map). One patient had a previous anterior MI. Five to six studies per patient were recorded. Two patients had only two maps recorded because of early death. Four patients were receiving digitalis at the time of recording of some of the maps. Three subjects were in Class I, five in Class II, and one each in Classes III and IV. Maximum +ST was recorded in horizontal lines F and G and maximum −ST in A and B. Regression of −Σ ST was noted to occur by reduction of the area displaying − ST and lowering of magnitude of − ST of upper transverse rows.

In five patients an uncomplicated MI evolved. The mean −Σ ST and +Σ ST on the first four days and at the final recordings done after 17.4 ± 0.9 (range 15–20) days of hospitalization are shown in table 5. Evolution of −Σ ST and +Σ ST was most rapid in the first 24 hours (table 5).

Table 3

| Electrocardiographic Data from Serial Mapping of Patients with Anterior Myocardial Infarction and Pericarditis (N = 10) |
|---|---|---|---|---|---|
| | Day 1 | Day 2 | Day 3 | Day 4 | Day 5 |
| +ΣST (mm) | 92.7 ± 12.6 | 93.2 ± 14.3 | 94.6 ± 10.1 | 89.0 ± 9.7 | 75.5 ± 7.9 |
| (39.0–155.5) | (33.5–195.0) | (38.0–136.0) | (33.0–150.0) | (32.0–106.0) | (11.0–50.5) |

Ranges are shown in parentheses.

Table 4

| Electrocardiographic Data from Serial Mapping of Patients with Anterior Myocardial Infarction and Pericarditis but without Extension (N = 5) |
|---|---|---|---|---|---|
| | Day 1 | Day 2 | Day 3 | Day 4 | Day 5 |
| +ΣST (mm) | 71.7 ± 13.9 | 71.9 ± 13.9 | 80.3 ± 13.9 | 75.6 ± 12.7 | 67.9 ± 12.0 |
| (45.0–92.5) | (35.5–96.0) | (38.0–117.0) | (33.0–108.0) | (32.0–104.5) | (15.5–38.5) |

Ranges are shown in parentheses.

+ΣST = sum of ST elevations.
Five patients suffered an extension of MI, which was defined by occurrence of chest pain, worsening of the clinical status (arrhythmias, ativoventricular blocks, or congestive heart failure), and re-elevation of enzymes (mean change of CPK: 166.6 ± 46.5 (range 43.0-278.0 I.U./L). Increases of +Σ ST by 5.4 ± 2.5 (range 4.5 to 13.0) mm and of −Σ ST by 12.1 ± 4.8 (range 4.0 to 23.5) mm were also noted. In only one patient were changes in both the + Σ ST and − Σ ST recorded. No correlation was found between changes in enzymes and changes in + Σ ST or − Σ ST. The initial − Σ ST of this group was 24.2 ± 7.6 (range 3.0-47.0) mm; the initial + Σ ST was 10.9 ± 5.4 (range 0-31.5) mm. Extension occurred the second day after admission to the CCU in three patients and the third day in two patients.

No correlation was found between − Σ STμax or + Σ STμax of the ten patients and clinical class or peak CPK, SGOT, and LDH. There was strong correlation between − Σ ST of maps and sum of −ST from six precordial leads (r = 0.927). Similarly the correlation between + Σ ST of the maps and the sum of +ST derived from leads II, III, aVF and V₅, V₆ (in the cases of lateral wall involvement) was found to be good (r = 0.874). The above correlations between maps and standard ECG included all the recorded material. Recorded +ST in leads II, III, and aVF was always higher than that seen in horizontal rows F and G.

Plots of daily − Σ ST and the sum of −ST by the precordial standard leads were in agreement in all patients. Similarly plots of daily + Σ ST and sum of +ST of leads II, III, aVF and occasionally V₅ and V₆ demonstrated agreement in five patients. In four patients the recorded + Σ ST was of very small magnitude and in one patient there was no +ST recorded in the map despite marked +ST by the conventional inferior leads. One patient developed
pericarditis (three-component friction rub, fever, and pleuritic pain) on the fifth hospital day and two days following a lateral wall extension of MI. Maps and 12-lead ECGs did not reveal any changes.

Other Types of Myocardial Infarction

Two patients, one with lateral and the other with both lateral and true posterior MIs, were studied. Maps in the first patient depicted the extent of MI which was otherwise diagnosed by V6 and V4. In the other patient the map revealed extensive high anterolateral MI; smaller +ST was seen in leads I and aVL, and no +ST in the standard precordial leads. Mapping of the posterior thorax revealed extension and evolution of the true posterior component of the infarct; the 12-lead ECG did not satisfy in all the tracings accepted criteria for the diagnosis of true posterior MI (R/S > 1 in V1 and/or V2 and R of 0.04 sec duration in V1 and/or V2).

Intraventricular Conduction Blocks — Pacemaking

Four patients who developed periods of transient RBBB were mapped. Since daily maps were done during both normal and abnormal intraventricular conduction, these patients were separately analyzed.

Three had an anterior MI and one had an inferior MI. In all patients with anterior MI, RBBB was recorded intermittently during mapping (fig. 7). Two patients had a temporary pacemaker inserted, one for sinus arrest and the other for RBBB and periods of sinus arrest. The effects of pacing or RBBB on the ST current of injury are shown in figure 7. Patients with intermittent LBBB occurring during mapping were not encountered.

Early Repolarization

Five patients with mean age of 38.4 ± 3.3 (range 31 to 49) years were diagnosed as having early repolarization. Four were black. + Σ ST on the first day was 54.5 ± 5.7 (range 43.5 to 71.5) mm. Twenty-four hours later + Σ ST was 54.9 ± 5.7 (range 45.5 to 74.5) mm. A last ST map done five to 23 days following admission revealed a + Σ ST of 52.9 ± 6.1 (range 36.5 to 68.0) mm (NS, paired t-test). No Q waves or enzyne alterations were noted. Musculoskeletal and gastrointestinal pathology were implicated for the chest pain in four patients. Maximal +ST ranged from 2.5 to 5 mm and was noted primarily in V2 to V3 of the standard ECG and columns 2 to 5 of maps (fig. 1). Peaked tall T waves were often but not invariably noted. Three patients had previous ECGs showing +ST; one had a normal coronary arteriogram. Exercise transiently abolished +ST in these patients. Daily

<table>
<thead>
<tr>
<th>Day 1</th>
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<th>Final map</th>
</tr>
</thead>
<tbody>
<tr>
<td>−ΣST</td>
<td>28.6 ± 3.7</td>
<td>19.9 ± 5.0</td>
<td>15.3 ± 3.4</td>
<td>15.7 ± 2.3</td>
</tr>
<tr>
<td>(14.0 – 34.5)</td>
<td>(2.0 – 32.0)</td>
<td>(2.0 – 22.0)</td>
<td>(8.0 – 21.5)</td>
<td>(0 – 14.0)</td>
</tr>
<tr>
<td>+ΣST</td>
<td>18.2 ± 7.3</td>
<td>11.5 ± 5.1</td>
<td>11.4 ± 5.0</td>
<td>8.4 ± 4.2</td>
</tr>
<tr>
<td>(2.5 – 44.0)</td>
<td>(1.5 – 26.5)</td>
<td>(1.5 – 26.0)</td>
<td>(1.5 – 24.5)</td>
<td>(0 – 13.5)</td>
</tr>
</tbody>
</table>

Ranges are shown in parentheses.
+ΣST = sum of ST elevations; −ΣST = sum of ST depressions.

The illustration on top (a) depicts the "subtraction effect" of complete right bundle branch block (CRBBB) vector on the current of injury in a patient with intermittent CRBBB and anterior myocardial infarction. Note that epen beats with CRBBB show some ST elevation instead of the expected ST depression secondary to the block. Lead C1 is obtained in V1 standard position. The illustration in the middle (b) shows the "addition effect" of LBBB-like vector resulting from right endocardial pacing on the current of injury. ST elevation of paced beats (first and last) is greater than the ST elevation due to the infarct (minimal in this lead) of normally conducted beats. The illustration on the bottom depicts the effects of paced beats (PB) and CRBBB on the current of injury in a patient with an anterior myocardial infarction. A fusion beat (F) is also shown. C2 is obtained in the V3 standard position.
plots of $\pm \Sigma ST$ of maps and the $\pm \Sigma ST$ derived from the six precordial standard leads were in agreement.

**Subendocardial Ischemia**

One patient with recurrent chest pain and $-ST$ was studied serially. Measured $-\Sigma ST$ was 30, 27, and 68.5 mm the initial three days, the last recording associated with severe, prolonged chest pain, and unchanged enzymes. Eventually this patient developed an anterior transmural MI and died of cardiogenic shock. There was good correlation of the initial two recordings (30 and 27 mm) and the occurrence of moderate chest pain as well as of the severe and prolonged chest pain and the final $-\Sigma ST$ of 68.5 mm. The 12-lead ECG depicted the extent of subendocardial ischemia in this case by a parallel rise of $-\Sigma ST$ of the six precordial leads: 5, 4.5, and 13 mm corresponding to 30, 27, and 68.5 mm of the map.

**Discussion**

**General Considerations**

Experimental studies and pathological examination of the hearts of patients who have died of an acute MI have proved that there is no clear-cut demarcation between necrotic and normal tissue and that an area of injured tissue surrounding the devitalized portion of the MI remains for several hours in a delicate balance between survival and death. It has also been shown that it is possible to modify (increase or decrease) the ischemic damage by applying hemo-
dynamic and pharmacologic interventions. Although the experimental situation in the animal is considerably different from that in the human, in whom frequently obstruction in the three main vessels is present, interest in applying techniques for reduction of ischemic injury in the clinical setting has been developed.

Epicardial and precordial mapping have been proposed as methods for monitoring ischemic damage. Rakita et al. have used epicardial electrodes to delineate the margins of areas injured by ischemia in animal experiments. Maroko and Braunwald and their coworkers have elaborated on the factors affecting the size of the infarct using epicardial $+ST$ mapping. There is considerable evidence that epicardial $+ST$ mapping reflects the ischemic injury. Good correlation has been found between the height of $+ST$ resulting from ischemia and the change of myocardial membrane action potentials, the reduction of coronary blood flow, the drop in local intramyocardial $pO_2$, the degree of anaerobic metabolism, the amount of CPK depletion, and histological, ultramicroscopic, and histochemical evidence of necrosis. Precordial $ST$ mapping expresses faithfully the underlying electrophysiology. Wilson et al. concluded that precordial leads were the best substitute for direct epicardial leads from the anterior surface of the ventricles.

It is important to confirm the stability of precordial $ST$ maps. Continuously maintained coronary occlusion produced unchanged $+\Sigma ST$ for three hours. Two maps done 15 minutes apart in a clinical study resulted in only a 5% difference in the $+\Sigma ST$. Stability of the maps in the early phase of MI has been shown in our study. This is of great clinical importance, since therapeutic interventions are applied early in the CCU course. Since the ischemic injury, as assessed by intramyocardial electrodes, changed directly in relation to heart rate, and changes in blood pressure have been shown to alter $+\Sigma ST$, these two parameters should be frequently recorded when serial ST mapping is carried out.

The ST segment is rarely horizontal so the methodology in quantitation of the ST shifts must be rigid. Bruce et al. have shown that an area 0.04 to 0.06 sec from the nadir of the S wave is the portion of the ST segment which is least affected by the QRS complex, T wave, and the Ta. The same workers maintain that the T-P segment is the appropriate baseline. It is fair to ask the question whether the $+ST$ of precordial maps represents accurately the magnitude and extent of myocardial injury. In recent studies of simultaneously recorded epicardial and precordial leads in dogs with experimental MI, the latter produced much smaller $+ST$ than the former. Although there is a definite "muffling effect" of thoracic wall, both systems moved in parallel fashion during interventions. With regard to the extent of the infarct, Rakita and Kjekshus and their coworkers have shown that the subendocardial area of ischemic damage is broader than the epicardial one. Damage of subendocardial layers does not result in $+ST$ in epicardial recordings, although Durrer et al., who have carried out extensive plunge electrode studies, have not found such an electrically silent zone. ST maps (even epicardial) therefore are adequate to show directional changes when interventions are applied, but do not reflect absolute "size" of the infarct. In addition, since injury boundaries are not as predictable and discrete as in experimental MI, the projection of damaged areas on the chest wall may be quite variable. Cancellation of ST shifts often take place in the precordial leads when adjacent epicardial areas generate opposite ST changes, as seen in the boundaries of ischemic injury.

**Anterior Myocardial Infarction**

Although death has been linked to high $+ST$ of the standard ECG, mortality did not correlate well with $+\Sigma ST_{max}$ in our study. Some of the patients who
died had pericarditis, previous MIs, or succumbed to an arrhythmia, thus forming an inhomogeneous group. A larger group of patients with a first MI who died with pump failure might be required to study the relationship between mortality and $\Sigma ST^{\text{max}}$.

ST segment elevation in our patients assumed patterns previously described.$^{6,12,13,18}$ Some patients areas of $-ST$ were noted. The nature of these mild $-ST$s is unclear; when $-ST$s are located opposite an injured area they are thought of being reciprocal changes.$^{3}$ Using midmural leads Rakita et al.$^{8}$ could not find $+ST$ directly beneath areas displaying $-ST$, and CPK depletion has not been detected in areas showing $-ST$.$^{6}$

Maps are no longer useful for following patients with MI when pericarditis occurs; patients then maintain stable high $+\Sigma ST$ for a number of days (tables 3 and 4) despite clinical and biochemical evidence of recovery. Since pericarditis cannot be differentiated by ECG from extension of injury,$^{28}$ serial enzymes and frequent auscultation are helpful in the differential diagnosis. Pericarditis is recognized far less frequently than it occurs.$^{44}$ Since friction rubs are transient, other clinical parameters may be used for its identification.$^{1}$ Initial $+\Sigma ST$ of our patients with subsequent pericarditis was not found to be higher than that of the patients without this complication, as observed in a study using the standard ECG.$^{38}$

We found no correlation of $+\Sigma ST^{\text{max}}$ with peak enzyme values. Daily sampling does not necessarily identify the real peak enzyme value, which, even if it were available, might not accurately reflect the extent of cell death. High peak values or lower but sustained ones can be produced by similar over-all muscle necrosis.$^{39}$ In addition, enzyme levels depend to some extent upon the subendocardial component of the MI, while $+\Sigma ST^{\text{max}}$ reflects only epicardial involvement, further attenuated at the precordium.$^{39}$

In uncomplicated infarct a 32% drop of $+\Sigma ST$ was noted in the first 24 hours.$^{14}$ Due to the wide range of drop of $+\Sigma ST$, effect of interventions cannot be assessed by the change of the map within the first 24 hours in the individual patient. Studies are needed to elucidate the pattern of evolution of ST maps during this acute phase of MI. Variability of resolution was noted in our patients. Reid et al.$^{15}$ speculated as to its association with undetected extension of MI. The finding in one of our patients who showed elevation of $+\Sigma ST$ at discharge from the hospital (fig. 4) underlines the possibility of undetected extension after transfer from the CCU as noted by Reid et al.$^{15}$ Undetected pericarditis$^1$ and aneurysm are other mechanisms which could contribute to persisting high $+ST$.

The good correlation of initial $+\Sigma ST$ and final $+\Sigma ST$ in uncomplicated MI (table 2) needs further explanation. The high values in discharge tracings may have eventually resolved to low $+ST$ levels. In addition, high $+ST$ may be a function not only of degree of ischemic damage but also of chest wall morphology and thickness.$^{11,12}$ It is interesting that the minor $+ST$ noted by Reid et al. in normal volunteers were higher in men than in women, a difference which could be attributed to the differences in chest characteristics.$^{15}$

It is possible that more extension of infarction would have been detected in our series had serial mapping been continued after transfer from the CCU.$^{13}$ As reported by others,$^{16}$ maps in this study detected extension not diagnosed by the standard ECG. Extension occurring several days after the initial ischemic insult supports the notion that the injured area remains in a stage of active evolution for several days.$^{12}$ Some of our patients suffered more than one extension.$^{13}$ A corollary of continued changes in infarction is that interventions should be made for several days, rather than for a few hours following admission. It is very important to remember, however, that re-elevation of $+\Sigma ST$$^{3}$ is suggestive of extension of MI only when the change in the map is associated with clinical and enzymatic evidence of new injury.

**Inferior Myocardial Infarction**

The significance of precordial "reciprocal" changes, i.e., $-ST$, is unknown. Rakita et al., exploring the heart with an epicardial electrode,$^{8}$ found no reciprocal relationship between the $+ST$ and $-ST$ in experimental MI. Reciprocal $-ST$ was also found to be less stable than $+ST$.$^{8}$ There are also theoretical problems in examining $+ST$ over the lower precordium in inferior MI. According to Wilson's concept, epicardial leads have a "semidirect" function, recording primarily local underlying potentials, although they are affected by potentials of the whole heart. For anterior MI, precordial mapping may provide a reasonable approximation of closely adjacent epicardial events, but for inferior MI not only is the distance greater, but the infarcted myocardial surface is not parallel to the precordium, thus distorting the recording of injury potentials.

Despite these problems, maps have been used before for the study of patients with inferior MI, although no attempt has been made to characterize quantitatively a group of such patients.$^{12,17}$ In our patients with uncomplicated inferior MI, maps showed the same early rapid evolution seen in patients with anterior MI (tables 2 and 5). In 50% of our patients extension was detected. Comparable further enzyme elevations to the ones found with anterior MI.
were also noted although the changes of $\pm \Sigma ST$ and especially $+ \Sigma ST$ were much smaller. In only one patient of five with extension, changes in both $- \Sigma ST$ and $+ \Sigma ST$ were noted; this may represent a clinical confirmation of the poor relationship shown by Rakita et al. between $+ST$ and $-ST$.\(^5\) The absence of correlation of the $- \Sigma ST^{max}$ or $+ \Sigma ST^{max}$ and enzyme peak values is not surprising, since the same was found in patients with anterior MI, in whom maps are more representative of degree of injury.\(^5\) This also explains the absence of correlation between the maps and the clinical class. Digitalis might have increased $- \Sigma ST$ in some patients, although the effect of the cardiac glycoside upon $-ST$ could not be isolated.\(^3\)

Disagreement in half of our patients between the $+ \Sigma ST$ of the map and the $+ \Sigma ST$ from the standard inferior leads, and failure of the former to record existing injury, renders the map unreliable for the individual patient, despite the good correlation of the two systems when all the recorded material was taken into consideration. In addition, agreement in all our patients between the $- \Sigma ST$ by the map and the standard ECG shows that the former provides no advantage over the latter.

Other Types of Myocardial Infarction

The diagnostic sensitivity of the ST maps, as compared with the 12-lead ECG, was not assessed in this study, since selection of patients was based on the standard ECG. It has been shown that only one fourth of patients with subsequent diagnosis of MI present with a classic ECG picture.\(^7\) It is possible that serial maps could detect areas of injury which are outside the six standard precordial leads. Reid et al.\(^12\) found transient $+ST$ in the lateral portion of the map of a patient with a normal standard ECG. In one of our cases the map depicted the high anterolateral component of the infarct in the absence of $+ST$s of the standard precordial leads. Multilead maps have been used for the diagnosis of true posterior, high anterior, and high lateral MI.\(^16, 38, 90\) When the clinical picture is not fully explained by changes on the 12-lead ECG, patients should have their chests "scanned" with precordial maps and additional leads from the back. In patients with a large chest, the "electrode blanket" used by other workers\(^18\) cannot cover all chest areas. Also, since recordings from the "electrode blanket," which contains leads set at fixed distances, derive from noncomparable areas of the chest of patients of varying size, comparison of maps from different patients may not be valid.

Intraventricular Conduction Blocks — Pacemaking

Massie and Walsh\(^40\) have shown that ST and T vectors are parallel in patients with intraventricular conduction blocks. When this is not the case, some other factor contributing to the ST shift is operating. Most of the time this alteration is due to a current of injury vector (fig. 7). The superimposed $+ST$ either nullifies or reverses the expected shift of the ST segment secondary to the block.\(^40\)

The effect of LBBB on the current of injury can be simulated by right ventricular endocardial pacing which produces LBBB-like QRS complexes (fig. 7). These observations may often be helpful in recognizing acute MI in the presence of bundle branch blocks or pacemakers.

Early Repolarization

Maps remained unchanged in our patients with early repolarization. Since there is no specific configuration of $+ST$ for this normal variant and peaked T waves are often seen in the early phase of MI, serial ECGs and enzymes are required to differentiate early repolarization from MI or pericarditis.\(^41\) Previous ECG tracings showing $+ST$ and effects of exercise are also helpful in the diagnosis.\(^41\) The high prevalence of this ECG aberration in young individuals, particularly of the black race, was again confirmed.\(^41\) There was no advantage of the $+ST$ map over the standard ECG in following patients with early repolarization.

Subendocardial Ischemia

Experimental studies have shown that $-ST$ is associated with milder ischemia than that producing $+ST$.\(^45\) Although the standard ECG was adequate for diagnosis in our case, maps could be used to better characterize the magnitude and extent of subendocardial ischemia; this could have had prognostic implications, since the location of ischemia often heralds the locus of subsequent injury.\(^45\) Maps of $-ST$ have also been utilized to follow patients with nontransmural MI.\(^19\) Since there are no data correlating magnitude of subendocardial injury as assessed by mapping with subsequent necrosis, the validity of using maps for following patients with this type of MI is open to question. The natural course of $- \Sigma ST$ in patients with nontransmural MI and preinfarction angina needs further study.

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