Diagnostic Electrocardiographic Sequences in Acute Pericarditis

Significance of PR Segment and PR Vector Changes

By David H. Spodick, M.D.

SUMMARY

Diagnostic electrocardiographic patterns were analyzed in 50 consecutive patients who had unequivocal clinical evidence of acute pericarditis. Distributions of P and QRS, and of P-R segment, ST-segment and T wave changes were plotted by lead and by mean frontal vector (A). Transient gross deviations of P-R segments, mainly in Stages 1 and/or 2, occurred in 41 patients (82%) and could produce an optical illusion of ST elevations when the J-points were actually on the baseline. A P-R was close to 180° opposite to A P and was not related to P wave or heart rate changes. In Stage 1, A ST tended to be concordant with A QRS and A T. T wave inversions in Stage 3 produced an A T which was distributed over an arc of 210° with no range of predilection.

Transient increase in magnitude of a normally oriented PR vector was consistent with the subepicardial atrial injury of acute pericarditis. It is the analogue of the classic Stage 1 ST-segment abnormalities of subepicardial ventricular injury and was equally as widespread in the electrocardiogram and almost as prevalent. P-R segment deviations were always depressions for leads of "epicardial" patterns. ST-segment deviations departed from the classic elevation pattern in 10 patients: 7 patients in whom ST was depressed in Lead III, 5 of whom had a horizontal QRS axis; and 3 patients in whom ST was depressed in aVL, two of whom had a vertical QRS axis. In Stage 3, the much wider range for A T as compared with the relatively concentrated early injury vectors, A P-R and A ST, is ascribed to greater inhomogeneity of post-injury ventricular repolarization.

Additional Indexing Words:
ST segment
ST vector
T vector

In acute pericarditis the electrocardiogram (ECG) alone can be diagnostic if it evolves in any of several patterns, designated "typical," which are variants of a basic four-stage ECG evolution seen in a majority of cases. In the remainder, recognition of pericarditis may not be aided or may even be confused by various "atypical" ECG patterns. Such patients, in whom diagnosis of pericarditis depends entirely on evidence other than the ECG, have been discussed elsewhere.

The four-stage ECG sequence is based on ST-segment and subsequent T wave changes and their return to or toward their previous configuration. Stage 1 is most characteristic, and quasi-diagnostic, with ST-segment elevation in almost all leads of "epicardial pattern" plus corresponding ST depression in "cavity pattern" leads like aVR. Stage 2 is an unstable continuum in which the ST (J) junctions return to the baseline and T wave amplitude begins to decrease either pari passu with the J-point return or, more characteristically, after the J-point becomes isoelectric. Sometimes one or more leads are "out of phase" for this change, leading or lagging the others. By this time, P-R segment deviations usually have appeared, frequently giving a false impression of continued ST elevation (Fig. 1). In Stage 3 the T wave polarity changes, resulting in shallow to full T wave reversal with respect to the prepericarditis ECG. Stage 4 represents electrocardiographic resolution—usually complete—of the process. This stage may be recorded following Stage 1 or 2 as well as Stage 3. (When a Stage 4 appears to follow Stage 1, this is equivalent to arrest.

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of ECG evolution at Stage 2, in which the J-points become isoelectric without further T wave changes. More commonly, there is orderly progression through all stages *seriatim*, which can be considered both typical and pathognomonic of acute pericarditis.

One or more stages may be absent if the pericarditic process resolves quickly or evolves too

Figure 1

*(Top) Electrocardiograms (in patient in whom Stage 3 was not detected by daily recording). Note P-R segment deviations in Stage 2 (middle trace). P-R segment isoelectric in V1 and in aVL (A P-R = −120°). In Stage 1 (top trace) ST-segments depressed in aVR and V1, but elevated in leads of "epicardial" pattern excepting isoelectric in Lead III (A ST = +30°). (Bottom) ECG in Stage 1 of acute pericarditis showing concomitant P-R segment depression and ST-elevation.*
rapidly in relation to the frequency of ECG monitoring. The ECG may yet be virtually diagnostic of acute pericarditis if it progresses either from Stage 1 to 2 to 4 (omitting 3), directly from Stage 1 to 4, or from Stage 2 to 3 and/or 4 (omitting Stage 1, as may occur in patients first examined long after onset); these are the "typical variant"1 evolutionary patterns.

This study was designed to clarify and characterize, in quantitative terms, the range of electrocardiographic variability in cases of acute pericarditis with typical or "typical variant" evolutionary ECG patterns. In the course of the investigation it became apparent that P-R segment deviations (previously described1) are almost as ubiquitous and equally as characteristic in acute pericarditis as are ST-segment deviations.

**Methods and Material**

Twelve-lead electrocardiograms were analyzed in 50 consecutive patients without evidence of heart disease, who had unequivocal clinical evidence of acute pericarditis, on the basis of a pericardial rub5 (44 patients), pericardial effusion6 (2), or both (4) but occurring in the presence of diagnostic or quasi-diagnostic ECG evolution by Stages,1 i.e. either: (a) Stages 1 to 2 to 3 to 4, (b) Stage 1 to 2 (or 4), or (c) Stage 2 to Stage 3 and/or 4. It was recognized that one or more stages might be missing solely because of late arrival of the patient (Stage 1) or rapid evolution (Stages 2 and/or 3) or discharge of the patient (Stage 4). Hence, the numbers of patients displaying each stage forms the statistical base, while the numerical partition of patients by particular stage-to-stage evolutionary patterns could not be precise and is not part of the study.

**Qualitative Observations.** Directions of changes and absence of changes in P-R segments, ST-segments and T waves were noted for each lead.

**Measurements and Calculations.** On the hexaxial scale, instantaneous mean frontal vectors (Å), expressed to the nearest 10 degrees, were plotted in the usual manner for P waves, P-R segments in Stages 1, 2 and/or 3, QRS complexes, ST-segments in Stage 1 and T waves in both Stages 1 and 3. The T-P interval was the ECG baseline. The differences in degrees between corresponding mean vectors were calculated between Å P-R and Å P; Å P-R and Å ST; Å ST and Å QRS; Å ST and Å T in Stages 1 and 3; Å T in Stage 1 and Å T in Stage 3; and Å T in Stage 3 and Å QRS. Percentage distributions were calculated and expressed to the nearest whole number percent.

**Results**

**Stages Identified**

Stage 1 was recorded in 43 patients, Stage 2 in 23, Stage 3 in 32 and Stage 4 in 37. Stage 2 resulted in return of all J points to the baseline before significant T wave change in 12 cases. In 11 cases various leads were "out of phase" for this change. This unstable stage was separately analyzed only for P-R segment deviation.

**P-R Segment**

P-R segment changes (figs. 1-3, 5; table 1) occurred in 41 patients (82%) in both limb and precordial leads. These were always depressions except in lead aVR, and in four patients in lead V1. They occurred mainly in Stage 1 (5) or Stage 2 (18) or both (17); one patient had P-R changes in stage 3. In two patients first recorded in Stage 2, P-R segment deviations were the principal initial evidence for acute pericarditis, with configurations superficially resembling ST deviations (fig. 5).

**Limb Leads**

The P-R segment vector was always negative, with 38 of 41 (93%) oriented from −120° to −150° (fig. 2). The greatest number could be easily plotted at −120° and at −150° because the P-R segment was most often isoelectric in leads aVL (17 cases) and III (15 cases) respectively. It was always negative in II and aVF and always positive in aVR. Mean P-R vectors (Å P-R) were plotted against corresponding Å P and the differences calculated (fig. 3). Thirty-two P-R vectors were within 180 ± 10 degrees of the corresponding Å P—i.e., almost directly opposite.

**Precordial leads (table 1).** The P-R segment was negative or isoelectric in leads V2-V6, and in 37 of 41 patients in V1; in four subjects V1 showed P-R segment elevation. From V5-V6, P-R was almost always depressed.

**Relation to heart rate.** For the 40 patients in whom P-R deviations occurred in Stage 1, Stage 2 or both, the heart rate change from Stage 1 to 2 was measured in relation to the occurrence of P-R changes by Stages. Rate changes averaged -2.8 beats/min with no apparent relationship to appearance and disappearance of P-R segment shifts from Stages 1 to 2.

**Table 1**

<table>
<thead>
<tr>
<th>P-R</th>
<th>V1</th>
<th>V2</th>
<th>V3</th>
<th>V4</th>
<th>V5</th>
<th>V6</th>
</tr>
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<tr>
<td>−</td>
<td>11</td>
<td>30</td>
<td>38</td>
<td>39</td>
<td>40</td>
<td>30</td>
</tr>
<tr>
<td>0</td>
<td>26</td>
<td>11</td>
<td>3</td>
<td>2</td>
<td>1</td>
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<td>+</td>
<td>4</td>
<td>0</td>
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<td>0</td>
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</table>

Abbreviations: − = depressed; 0 = not deviated; + = elevated.

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CASES

Figure 2

Mean P-R segment vector (Å P-R) distribution in 41 patients. (Bottom) Patterns of scalar P-R segment displacements for each limb lead. 0 = isoelectric; - = depressed; + = elevated.

ST-Segments

Stage 1, which is defined by acute ST deviations, was documented in 48 cases, the initial ECGs in the remaining two patients showing Stage 2.

Limb leads. Å ST in 38 cases (79%) was in the sextant +30° and +60°. ST-segments were always depressed in aVR and elevated in Lead II. Isoelectric ST occurred in Lead III in 19 patients (40%), in aVL in 16 (34%), in aVF in three (6%) and in Lead I in a single patient; ST-segment depressions were recorded in Lead III in seven patients (14%) in whom Å ST was oriented from 0° to +20°, and in aVL in three patients in whom Å ST was oriented from +70° to +90°. Five of the seven patients with ST depressions in Lead III had net QRS negativity in the same lead (Å QRS between +20° and -20°). Two of the three patients with depressed ST in aVL had vertical Å QRS (+60° and +70°).

Å ST was plotted against Å QRS and Å T in Stage 1. Figure 4 shows the differences in degrees between Å ST and Å QRS and Å ST and Å T. There is a general correspondence in both cases, Å ST in the great majority falling within 30 degrees of both Å QRS and Å ST. With respect to Å QRS, Å ST was oriented identically (16 patients) or equally either to the right (15 patients) or left (17 patients). By contrast, there was a much greater tendency for Å ST to be oriented to the left of Å T (25 cases).

Precordial leads. Precordial ST-deviations occurred in 47 of 48 patients in Stage 1 (table 2). In V5 and V6 the ST junction was virtually always elevated (46 cases) or isoelectric (one case). ST-depressions occurred only in leads V1 (14 cases) and V2 (two cases). In lead V1, ST was usually either isoelectric (25) or depressed (14); it was elevated in only eight cases (17%). However, V2

Figure 3

Deviation of mean P-R vector (Å P-R) from mean P wave vectors (Å P). Differences cluster around 180°.

Figure 4

Deviations of mean ST-vector (Å ST) from Å QRS and Å T in Stage 1. Å ST is usually within 20° of either Å QRS or Å T.
Table 2

<table>
<thead>
<tr>
<th>ST</th>
<th>V1</th>
<th>V2</th>
<th>V3</th>
<th>V4</th>
<th>V5</th>
<th>V6</th>
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<tr>
<td>+</td>
<td>8</td>
<td>24</td>
<td>34</td>
<td>41</td>
<td>46</td>
<td>46</td>
</tr>
<tr>
<td>0</td>
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<td>21</td>
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<td>−</td>
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showed ST-elevations in the majority (24/41; 60%). When subgrouped according to frontal A QRS, the precordial ST patterns showed no statistical differences in distribution (X² test).

T Waves

Stage 3 T wave inversions were recorded in 32 patients in both limb and precordial leads. In the remaining 18 patients, Stage 3 either was missed owing to rapid evolution or reverted directly to Stage 4 from Stage 1 or 2. Stage 4 was documented in all but three of the 32 cases in Stage 3.

Limb leads. There was no range of predilection although A T usually (25/32 = 80%) was leftward oriented and half of these were between −120° and −150°. Because of the large scatter, the abnormal A T of Stage 3 was plotted against A QRS, A T in Stage 1 and A ST and their differences in degrees plotted on a 360°—not hexaxial—scale. In each case there was a wide range of differences, with approximately equal predilection for nearly opposite (180° ± 30°) vectors, i.e. for A T in Stage 3 vs A QRS (17 out of 32 cases); vs A T in Stage 1 (16 out of 32); and vs A ST (19 out of 32).

Precordial leads. Table 3 shows the precordial distribution of T wave configurations in Stage 3.

Table 3

<table>
<thead>
<tr>
<th>Precordial T Wave Morphology in Stage 3 (N = 32)</th>
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<tr>
<td>V1</td>
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<tr>
<td>−</td>
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<tr>
<td>(+)</td>
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<td>0 or (−+)</td>
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<td>+</td>
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Inverted (−) T waves increased in frequency from V₁ to V₆. Terminally inverted (+−), flat (0) or proximally inverted (−+) T waves occurred in all 32 cases in V₃ and V₆. Thirty of these (94%) were (−) or (+−). Upright (+) T waves were always absent in V₃ and V₆. In nine cases (28%) T was inverted in all leads from V₁ to V₆.

Discussion

Measurable P-R segment shift is clearly a characteristic feature of acute pericarditis. It occurred in 82% (41/50) of patients and was unrelated to heart rate. The mean P-R segment vector (A P-R) was always “left” oriented and tended to be close to 180° from A P (fig. 3). The cluster of cases with A P-R at −120° and −150° occurred because P-R segments were so frequently not detectably displaced in leads aVL and III respectively. There was more variability in precordial leads (table 1), but from lead V₂ through V₆ the P-R segments were either negative or isoelectric and in V₄-V₆ virtually always negative. Most cases showed a P-R vector in Stage 2 (35 cases); half (22 cases) showed P-R deviations in Stage 1; in 17 of

Figure 5

Acute pericarditis. Initial ECG in patient showing advanced Stage 2 with marked P-R segment deviations giving illusion of ST-segment elevations. J point isoelectric with T-P intervals.
these patients $\hat{A}$ P-R was seen in both these stages.

Since none of the patients had abnormalities of P wave size or configuration, the P-R segment shifts probably represent sub-epicardial atrial injury owing to pericardial inflammation. This is supported by the strong tendency for P-R segment vectors to be oriented exactly opposite to mean P wave vectors, i.e. the difference between $\hat{A}$ P and $\hat{A}$ P-R or "spatial atrial gradient" was $180 \pm 10$ degrees in 32 out of 41 cases (fig. 3). As shown by Brody this conforms to the normal directions of atrial activation and recovery vectors. Thus, acute pericarditis is characterized by a transitory increase in magnitude, but not of direction, of $\hat{A}$ P-R which is independent of heart rate or P wave changes. Since the preponderance of atrial tissue is anatomically posterior and to the right, the orientation of such a generalized atrial injury vector ($\hat{A}$ P-R) would indeed be expected to be negative with respect to most body surface electrodes, analogous to the appearance of the vector of generalized subepicardial ventricular injury ($\hat{A}$ ST) which was oriented toward the preponderant ventricular tissue. The absence of P wave changes is consistent with the absence of more serious atrial injury. The fact that the P-R segment deviations were not localized and the absence in appropriate leads of P-R segment elevations excludes electrocardiographic inferences of atrial infarction.

Of practical significance is that a patient whose ECG is recorded first in Stage 2 may only show P-R depressions and these may give an optical illusion of ST-segment elevations if the T-P interval is not consciously utilized as the baseline (fig. 5).

The preponderant left-inferior orientation of the frontal plane ST-vectors was generally in accordance with expectations from previous experience, i.e. $\hat{A}$ ST was principally concordant with the normal range $\hat{A}$ QRS. In "horizontal hearts" ($\hat{A}$ QRS $+20^\circ$ to $-20^\circ$), $\hat{A}$ ST tended to be most parallel to the lead $-aVR$ axis and in "vertical hearts" ($\hat{A}$ QRS $+60^\circ$ to $+90^\circ$) most parallel to the Lead II axis. (The statement of Surawicz and Lasseter that $\hat{A}$ ST parallels Lead II in horizontal hearts and Lead III in vertical hearts was made without a plot of $\hat{A}$ QRS and may be a misprint.)

Precordial ST-deviations (table 2) showed the mean horizontal plane ST-vector orientation to be leftward and anterior. (ST-depressions in 14 patients in $V_1$ may be considered equivalent to those in aVR since the solid angles subtended by these electrode positions involve much of the same atrial and "endocardial" tissues, such that $V_1$ and aVR in many ECGs are morphologically similar or identical.) However, the horizontal plane ST-vector distribution was not influenced by the general orientation of the frontal plane QRS vector. Taken together, horizontal and frontal $\hat{A}$ ST yield a net spatial ST-vector orientation which is: left-inferior-anterior.

By contrast with the fairly concentrated distribution of $\hat{A}$ P-R and $\hat{A}$ ST, the patterns of measurable T wave inversions yielded a very wide range for $\hat{A}$ T in Stage 3. This probably reflects distributional inhomogeneity of the injury and recovery process in the myocardium.

References
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