Diagnostic Electrocardiographic Sequences in Acute Pericarditis

Significance of PR Segment and PR Vector Changes

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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...
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.

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
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”11 evolutionary patterns.

This study was designed to clarify and character-
ize, in quantitative terms, the range of electrocar-
diographic variability in cases of acute pericarditis
with typical or “typical variant”11 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 (A),
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 A
P-R and A P; A P-R and A ST; A ST and A QRS;
A ST and A T in Stages 1 and 3; A T in Stage 1 and
A T in Stage 3; and A T in Stage 3 and A QRS. Per-
centage 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 (A P-R) were plotted
against corresponding A P and the differences
calculated (fig. 3). Thirty-two P-R vectors were
within 180 ± 10 degrees of the corresponding A
P—i.e., almost directly opposite.

Preccordial leads (table 1). The P-R segment was
equielectric 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
ever 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 appear-
ance and disappearance of P-R segment shifts from
Stages 1 to 2.

Table 1

P-R Segment Deviations in Precordial Leads (N = 41)

<table>
<thead>
<tr>
<th>P-R</th>
<th>V1</th>
<th>V2</th>
<th>V3</th>
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<th>V5</th>
<th>V6</th>
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<tr>
<td>−</td>
<td>11</td>
<td>30</td>
<td>38</td>
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<tr>
<td>0</td>
<td>26</td>
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Abbreviations: − = depressed; 0 = not deviated; + = elevated.

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Mean P-R segment vector (A-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. A ST in 38 cases (79%) was in the sextant between +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 A ST was oriented from 0° to +20°, and in aVL in three patients in whom A 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 (A QRS between +20° and -20°). Two of the three patients with depressed ST in aVL had vertical A QRS (+60° and +70°).

A ST was plotted against A QRS and A T in Stage 1. Figure 4 shows the differences in degrees between A ST and A QRS and A ST and A T. There is a general correspondence in both cases, A ST in the great majority falling within 30 degrees of both A QRS and A ST. With respect to A QRS, A 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 A ST to be oriented to the left of A 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
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 ($X^2$ 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 $-120^\circ$ and $-150^\circ$. 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 ($180^\circ \pm 30^\circ$) 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>V1</th>
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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 V5 and V6. Thirty of these (94%) were (-) or (+-). Upright (+) T waves were always absent in V5 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 $180^\circ$ from A P (fig. 3). The cluster of cases with A P-R at $-120^\circ$ and $-150^\circ$ 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 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](http://circ.ahajournals.org/). 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.

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these patients Å 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 Å P and Å P-R or "spatial atrial gradient" was 180 ± 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 transmural increase in magnitude, but not of direction, of Å 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 (Å 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 (Å 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., Å ST was principally concordant with the normal range Å QRS. In "horizontal hearts" (Å QRS +20° to -30°), Å ST tended to be most parallel to the lead -aVR axis and in "vertical hearts" (Å QRS +60° to +90°) most parallel to the Lead II axis. (The statement of Surawicz and Lasseter that Å ST parallels Lead II in horizontal hearts and Lead III in vertical hearts was made without a plot of Å 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 V1 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 V1 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 Å ST yield a net spatial ST-vector orientation which is: left-inferior-anterior.

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

References

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