The Clinical Significance of Postextrasystolic T-Wave Changes

By Harold D. Levine, M.D., Bernard Lown, M.D., and Richard B. Streeter, M.D.

Examination of the T wave of the beat following the pause occurring in many conditions, notably premature systoles, may show variations from the regular T waves in the same lead without concomitant change in the QRS complex. This phenomenon, when pronounced, is generally associated with evidence of myocardial impairment and is correlated with abnormal reaction to stress tests for coronary insufficiency. Inquiry is here made into the factors affecting this phenomenon, its mechanism, topography, persistency and clinical significance.

The QRS complex and the T wave do not ordinarily vary in form after an extrasystole. Occasionally, however, one notices that the T wave immediately after a compensatory pause undergoes alteration either in size, shape or direction without modification of the antecedent QRS complex. This change is exemplified in figure 1 recorded in a man 58 years old with an old anteroseptal infarction. In lead V_2 particularly, the T waves, from being upright in the regular complexes, have become diphasic in the beat after the compensatory pause, with a pronounced terminal inversion. In terms of Wilson's formulation such changes must be regarded as "primary" since they are not predicated upon alterations in the QRS complex. They are indicative of a change in the electrical recovery or repolarization of the heart.

Though this phenomenon has received some attention in the experimental and clinical literature to date, no systematic study is available which sets forth criteria for evaluating the significance of such changes, establishes its incidence or assesses its clinical import. The object of the present report is threefold: first, to determine whether a disturbance in repolarization which is limited to the postextrasystolic beat is associated with clinical evidence of myocardial damage; second, to observe whether the absence of T-wave changes in the beat immediately following the compensatory pause reflects the lack of demonstrable heart disease; and third, to correlate the presence or absence of such T-wave changes with the result of the Master two-step test for coronary insufficiency.

METHODS AND MATERIAL

The T wave of the beat following a compensatory pause (that is, the postextrasystolic T wave) was compared with the T wave of the regular ventricular complexes in the same lead and characterized as either "positive" or "negative." A T-wave change to be considered "positive" had to fulfill at least one of the following criteria: (1) a definite total change in the direction of the T wave; (2) a change to a diphasic form with the major portion having a direction opposite that of the original T wave; (3) a change in T wave voltage of 2 mm. or more; or (4) a change in amplitude of less than 2 mm. when associated with a prolongation of the Q-T interval by 0.05 second or more. These arbitrary criteria define an alteration which is striking and clearly present at first sight. Before judgment was passed on a postextrasystolic T-wave change, premature beats had to be frequent and present in extremity and precordial leads. These criteria of positivity had to be fulfilled without alteration of the immediately preceding QRS complex. The "secondary" T-wave changes not uncommonly seen after interpolated ventricular premature beats were not characterized as "positive." Patients with persistent bigeminal rhythm were excluded from consideration since in this disturbance of rhythm every other T wave is postextrasystolic, the alternate T waves belong to the premature beats, and no normal T waves exist as a basis for comparison. All individuals in this survey had multiple leads including three conventional and three unipolar limb leads and six unipolar chest leads.

Three principal categories of patients were studied: a group of 62 patients exhibiting positive postextrasystolic T-wave changes; a group of 77
individuals with negative postextrasystolic T-wave changes; and 26 individuals with frequent premature beats upon whom a Master two-step test was performed. In each instance the electrocardiogram was studied and a judgment whether the T-wave change was positive or negative was made prior to a perusal of the case history or the result of the Master test.

The population constituting the positive postextrasystolic T-wave group consisted of 30 observed in the routine reading of electrocardiograms at the Peter Bent Brigham Hospital. Twenty patients exhibiting this change were found in a concurrent study of the relation of auriculoventricular conduction time to paroxysmal rapid action of the heart1 and the remaining 12 were discovered in a group of 50 individuals with frequent premature beats examined in the cardiovascular survey of the population of Framingham, Massachusetts, now being conducted by the National Heart Institute of the United States Public Health Service.

The group with negative postextrasystolic T-wave changes serving as a control population was derived in a similar fashion. Thirty-nine were found in the routine reading of cardiograms and 38 were detected in the above-mentioned mass population survey for heart disease. As a further check on the clinical significance of absence of T-wave changes in the postextrasystolic beat an additional group was studied. Instead of using the electrocardiogram as the starting point in selection, patients were chosen on the basis of the diagnosis of functional heart disease. From a group of private patients diagnosed by Dr. S. A. Levine as having functional heart disease 62 were selected because they had frequent premature beats.

The 26 patients who had the two-step stress test had a so-called “single” Master test with the standard number of climbs defined for their respective sex, weight and age. This group was selected on the basis of frequent extrasystoles from among 300 individuals who had such a stress test.

Results

A. Incidence and Significance of Positive Postextrasystolic T-Wave Changes.

Of 2143 consecutive electrocardiograms taken at the Peter Bent Brigham Hospital 161 tracings showed premature beats. Twenty-two of these exhibited positive postextrasystolic T-wave changes. This indicates a rough incidence of one positive change in 100 routine electrocardiograms, or one in seven tracings showing premature beats.

The etiologic background of 62 patients with positive postextrasystolic T-wave changes is represented in table 1. Thirty-six of the patients (or 58 per cent) had coronary artery disease. Adding the other 16 patients with established cardiovascular disease, the incidence of heart disease among patients exhibiting a positive postextrasystolic T-wave change is 84 per cent. The only patient in this group diagnosed as having functional heart disease was an 82 year old man with generalized arteriosclerosis. At the time of recording these changes in the T wave three of the patients were having gastrointestinal hemorrhages; in two of these the

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<tr>
<th>TABLE 1.—Clinical Features in 62 Patients With Positive Postextrasystolic T-Wave Changes</th>
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<tr>
<td>Etiologic background</td>
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<tr>
<td>Recent or old myocardial infarction</td>
</tr>
<tr>
<td>Definite angina pectoris</td>
</tr>
<tr>
<td>without infarction</td>
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<tr>
<td>Probable angina pectoris</td>
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<tr>
<td>Diffuse myocardial fibrosis at postmortem</td>
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<tr>
<td>Hypertensive heart disease without angina pectoris or infarction</td>
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<tr>
<td>Syphilitic heart disease</td>
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<tr>
<td>Rheumatic heart disease</td>
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<tr>
<td>with congestive heart failure</td>
</tr>
<tr>
<td>Gastrointestinal hemorrhage with normal hearts</td>
</tr>
<tr>
<td>Functional heart disease</td>
</tr>
<tr>
<td>Ruptured aneurysm of circle of Willis</td>
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<tr>
<td>Questionable heart disease</td>
</tr>
<tr>
<td>Total</td>
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<tr>
<td>Age range</td>
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<td>Mean age</td>
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<td>Sex distribution</td>
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POSTEXTRASYSTOLIC T-WAVE CHANGES

Fig. 1. Clear-cut "coronary" type postextrasystolic T-wave changes in a 58 year old man with an old anteroseptal infarct. In this case the changes were pronounced in leads V2 and V4, less striking in the other precordial leads, and not apparent in the extremity leads. The late "coronary" type of T-wave inversion here suggests predominantly subepicardial changes.

Fig. 2. Postextrasystolic T-wave changes of "subendocardial" type present in all extremity and chest leads studied. Such widespread changes are unusual. The patient was a 50 year old man with severe hypertensive heart disease and angina pectoris. MASTER test positive. The premature beats were auricular in origin.

Asthma. In the majority of cases a positive postextrasystolic change is therefore correlated with convincing evidence of heart disease. The great preponderance of the male sex and the mean age of 62 years further reflect the predominant background of coronary artery disease in this population.

In 25 of the 62 patients premature beats were frequent enough to be present in all or most of the 12 leads recorded. This permits a
statement of the topographic distribution of
the positive postextrasystolic T-wave changes.
In 16 of the 25 the changes were predominantly

or exclusively recorded in the precordial leads
(generally leads V3 and V4), in four in the limb
leads and in five cases both in the limb and

chest leads. This tendency to specific spatial
orientation around position V3 or V4 suggests
a disturbance in the myocardial zone supplied
through the descending branch of the left
coronary artery. Another aspect of the topog-
rophy of this T-wave change is its distribution
with reference to the subepicardial and sub-
endocardial laminae of the ventricular wall.
More than two thirds of the patients showing
positive postextrasystolic change had a "sub-
epicardial" type of change in the sense of a
decreased height or terminal or total inversion
of the T wave. The association of these changes
with prolongation of the Q-T interval fre-
quently produced the characteristic appearance
that has been designated as the "coronary
contour" (fig. 1). The remaining third of the
cases showed a "subendocardial" type of change,
and in the second beat following the pause
(fig. 5). At times T-wave changes were present

in a series of postextrasystolic beats following
single or repetitive premature beats and
gradually or abruptly resumed a normal ap-
pearance. This phenomenon is somewhat remi-
niscent of the transient T-wave changes which
may continue for hours, days or weeks, follow-
ing a paroxysm of tachycardia (the so-called
post-tachycardial syndrome).

In two instances positive postextrasystolic
T-wave changes were associated with left
bundle branch block. Generally the existence
of left bundle branch block obscures evidence
of concomitant myocardial damage since these

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**Fig. 3.** Postextrasystolic T-wave changes following
auricular premature beats recorded in same individual
over a four-year period. (A) Lead II recorded January
8, 1946, showing an auricular premature beat with
ventricular aberration; T-wave inversion in postex-
trasystolic beat. (B) Lead V4 recorded April 7, 1950,
showing paired auricular premature beats. Note pro-
found T-wave deepening in the two beats after the
pause. The patient was a 51 year-old artist with hyper-
tensive heart disease and chronic alcoholism.

**Fig. 4.** Postextrasystolic changes following paired or interpolated premature beats. T-wave inversion
in two beats following paired ventricular premature beats. The patient was a 68 year old man with
angina pectoris. Master test positive.

**Fig. 5.** T-wave changes in second beat following
paired ventricular premature beats. A short pause pre-
cedes first postextrasystolic beat, long pause precedes
second postextrasystolic beat.
changes are lost in the electrocardiographic changes produced by the left bundle branch

![Image of electrocardiograms](http://circ.ahajournals.org/)

**Fig. 6. U-wave changes in beats following long pauses.** (A) Lead V4 immediately before exercise. Note appearance of broadening of T wave (arrow) in postextrasystolic beat due to incorporation of U wave in latter half of T wave. The premature ventricular beat arises at the apex of a U wave. (B) Lead V1 two and a half minutes after exercise. Note that in the third and fourth beats, which follow long pauses (the third beat is an escape beat), the U wave rides up on the descending limb of the T wave. In the beats following shorter pauses the U wave arises almost at the isoelectric line. (C) Lead V4 three minutes after exercise. Premature beat has onset at apex of U wave. The U wave of the succeeding postextrasystolic beat again arises on the descending limb of the T wave.

**TABLE 2.—Clinical Features in 77 Patients with Negative Postextrasystolic T-Wave Changes**

<table>
<thead>
<tr>
<th>Etiologic background</th>
<th>Number</th>
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<tbody>
<tr>
<td>No cardiovascular disease</td>
<td>35</td>
</tr>
<tr>
<td>Functional heart disease</td>
<td>11</td>
</tr>
<tr>
<td>Questionable heart disease</td>
<td>10</td>
</tr>
<tr>
<td>Hypertension</td>
<td>5</td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
<td>11</td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
<td>1</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>77</td>
</tr>
<tr>
<td>Age range</td>
<td>14–87 years</td>
</tr>
<tr>
<td>Mean age</td>
<td>47</td>
</tr>
<tr>
<td>Sex distribution</td>
<td>Males 42 Females 35</td>
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</table>

block itself. The presence of positive postextrasystolic T-wave alterations may, in these instances, offer otherwise unobtainable evidence of myocardial impairment.

In a small number of cases changes in the postextrasystolic T wave were associated with changes in the U wave of the same ventricular complex. While the T wave of the postextrasystolic beat in some cases diminished in amplitude, the U wave became taller, producing in effect a broad, notched T wave. The electrocardiograms reproduced in figure 6 were recorded in a 57 year old woman undergoing a Master test. The upper strip (fig. 6A), taken before exercise, shows an apparent increase in the T-wave area after a premature beat. This results from the incorporation of a U wave in the T-wave area. The escape beat following a sinus pause in the next strip recorded two and one-half minutes after exercise (fig. 6B) shows no apparent change in the T wave but the U wave “rides up” on the downstroke of the T wave. In the third strip (fig. 6C) a similar U wave was in the process of being inscribed on the downstroke of a T wave following sinoauricular block, but this was interrupted at its apex by the beginning of a ventricular premature beat. Similar incorporation of a U wave in the T wave has been observed in another patient following a short bout of paroxysmal tachycardia.

**B. Incidence and Significance of Negative Postextrasystolic T-Wave Changes**

The over-all results in this group of 77 patients are shown in table 2. A negative postextrasystolic T-wave change was observed in four patients who had survived myocardial infarctions. In 12 other patients with cardiovascular disease the postextrasystolic T waves showed no changes. Thus 16 (or 20.8 per cent) of the 77 patients in this group had definite heart disease. This group without postextrasystolic changes differs from the first group in that the mean age was 13 years younger and in that there was no significant male sex predominance. This may be a reflection of the relative absence of coronary artery disease in the group with negative postextrasystolic T-wave changes.

To explore further the possible value of the postextrasystolic T-wave alterations, a group
of patients was investigated whose cardiac disturbance was regarded as functional. Such patients frequently tax the acumen of the clinician; numerous tests may be necessary before a final diagnosis can be established. If the T wave after a compensatory pause offers, in a positive or negative way, evidence of the status of the myocardium, it might be of particular value in this group of patients. Table 3 shows the over-all results for the 62 patients believed to have functional heart disease. By the criteria enumerated above only two patients (or 3 per cent) had positive changes. One of these patients was an 82 year old man. The other was a 62 year old woman whose chest pain at times came on with effort; early angina discomfort could not be excluded. It would seem then that a positive postextrasystolic change is rare in the absence of heart disease. In its mean age and sex distribution this group of patients with functional heart disease was similar to the group without T-wave changes following premature beats (that is, negative postextrasystolic changes).

C. Relation of Postextrasystolic T-Wave Changes to Results of Master Two-step Test

The results of the relation of postextrasystolic T-wave changes to the Master test are shown in table 4. In 92 per cent of the 26 patients studied the two technics coincided, that is, a positive postextrasystolic T-wave change coincided with a “positive” Master test (fig. 7) and a negative postextrasystolic change corresponded to a “negative” Master test. The only patient with a positive postextrasystolic change and a negative Master test had typical angina pectoris of effort; the two patients with positive Master tests and negative postextrasystolic T-wave changes also had the characteristic symptoms of angina pectoris. This experience, limited though it is, none the less suggests that an extrasystole, or the pause following it, may induce a derangement in repolarization in an already injured myocardium which is comparable in its significance with the electrical alterations induced in the same heart by exercise.

**DISCUSSION**

These studies point up a high degree of correlation between the presence of positive postextrasystolic T-wave changes and clinical evidence of significant heart disease and, to a lesser extent, the absence of such changes when clinical evidence of heart disease is not demonstrable. The criteria of “positivity” here employed are arbitrary. The value of the standards evolved is that they provide an index for the existence of underlying heart disease comparable to that obtained from the currently employed two-step stress test. In an era of multiple, complex, expensive and,

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<th>Table 3.—Incidence of Positive and Negative Postextrasystolic T-Wave Changes in 62 Patients with Functional Heart Disease and Frequent Premature Beats</th>
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<tr>
<td>Negative P.E.S.T.</td>
</tr>
<tr>
<td>Positive P.E.S.T.</td>
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<tr>
<td>Minor changes</td>
</tr>
<tr>
<td>Total</td>
</tr>
<tr>
<td>Age range</td>
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<td>Mean age</td>
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<th>Table 4.—Relation of T-Wave Alterations in Postextrasystolic Beat to Two-Step Master Test</th>
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<tr>
<td>Positive P.E.S.T.</td>
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<td>Negative P.E.S.T.</td>
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at times, even hazardous, laboratory procedures, it is worthy of emphasis that the interpretation of the postextrasystolic T-wave change is based on information already available in the routine electrocardiogram.

A conception of the mechanism of the changes in ventricular repolarization following premature beats must take the following possible factors into consideration: (1) changes in repolarization related to cycle length; (2) the effect of the particular occasion for the pause; (3) hemodynamic factors in the postextrasystolic beat; and (4) mechanical factors.
1. Changes in Repolarization Related to Cycle Length. It has been suggested by Ashman that the T wave of the normal human electrocardiogram has the same direction as the QRS complex because of local differences in the duration of the excited state, specifically because the rate of repolarization is slower in the subendocardial than in the subepicardial laminae of the ventricles. This is a physiologic phenomenon. Where there are such local differences in the rate of repolarization, giving rise to T waves having the same direction as the QRS complexes, experimental acceleration of the heart at more and more rapid rates eliminates these local differences in the rate of repolarization so that eventually the T wave has a direction opposite the QRS complex and an equal magnitude. It seems probable that somehow shortening the cycle length produces a greater shortening of repolarization in those parts of the ventricle where this process was originally longest. Having thus inverted the T wave by accelerating the heart, the preexistent local differences may now manifest themselves if the heart is slowed; the T wave then becomes upright again. Conversely, if the T wave is originally inverted, acceleration of the heart apparently produces a greater relative shortening of the excited state subepicardially, thus making the T wave upright, and slowing the heart produces reinversion of the T wave. A clear demonstration of this idea was published recently by Segers and co-workers. A patient showed inverted T waves during the so-called "post-tachycardiac syndrome"; at this time postextrasystolic T waves were upright. Following recovery, when the regular T waves were upright, postextrasystolic T waves were inverted. Although, as will become clear, this conception is open to some objections, it does seem probable from experimental evidence that induced (for example, by carotid sinus stimulation) or spontaneous (for example, in the pause following extrasystoles) lengthening of the cardiac cycle may under certain circumstances unmask T-wave changes.

In those cases in which extrasystoles were recorded in varying degrees of prematurity, the postextrasystolic T-wave changes were more pronounced the greater the degree of prematurity. A number of cases were observed in the present study in which striking changes were present when the premature beats were early, but absent or equivocal when late. These observations accord with Scherf's view that it need not be an extrasystole, nor whether the beat is an auricular, nodal or ventricular premature beat, but the length of the pause that in large part determines the change in the T wave. Scherf has recorded similar T-wave changes clinically in the beat following the long pause in blocked auricular premature beats and in partial auriculoventricular heart block while Scherf as well as Grant and coworkers detected such changes after the long pauses in auricular fibrillation.

2. The Effect of the Particular Occasion for the Pause. Although an explanation for these T-wave changes has been presented predicated upon changes in heart rate, and therefore of cycle length, this need not be the only explanation for such changes. In certain cases T-wave changes were found to be quite independent of heart rate. In a 66 year old man with angina pectoris, for example, inverted T waves were recorded in lead V4 before exercise (fig. 7); the postextrasystolic beat in this strip showed a more deeply inverted T wave and a longer Q-T interval than the regular beats in this same lead. Yet in this same lead the T waves became upright immediately after exercise though the cycle length was very close to, or identical with, that before exercise. In this same patient moreover no changes were recorded in the T waves during the long cycles produced by carotid sinus stimulation or following a spontaneous sinus pause of duration comparable to a compensatory pause which, in the same lead, had been followed by T-wave inversion. Alzamora found that by the alternative tests of exercise and of carotid sinus stimulation one can, in effect, "dissect" the T wave. Whereas exercise may cause inverted T waves to become upright, carotid sinus stimulation (or the postextrasystolic beat) tends to produce inversion of atypical (notched, flat, isoelectric or diphasic) T waves or deeper inversion of originally inverted T waves. In this way he was able to unmask occult T-wave changes in leads that originally showed atypi-
cal but not necessarily pathologic T waves. The present results are in general accord with those of Alzamora, but the fact that in about one third of the cases here reported the T wave following the pause became upright rather than inverted (figs. 2, 8A) suggests that the effect of the long spontaneous pause, no matter what its cause, is not necessarily identical premature beats, found none in the postextrasystolic beats of five patients with auricular premature beats. They inferred from this that it must be an eccentric electrical activation of the ventricle that makes manifest a latent disorder in its repolarization. In view of the

![Image](http://circ.ahajournals.org/)

**Fig. 8.** T-wave changes following pause with blocked or conducted auricular premature beats or auricular fibrillation. (A) Change from an inverted T wave in the regular beats to a diphasic T wave in the beat following a pause resulting from blocked auricular premature beats (leads II and III). The patient was a 45 year old hypertensive brewer with possible cirrhosis of the liver. (B) Total inversion of T wave in beat following a conducted auricular premature beat recorded (lead V6) in a 55 year old man with central nervous system and cardiovascular syphilis. No angina pectoris. (C) Change from diphasic to large upright T wave in beat following long pause in auricular fibrillation.

with that induced by carotid sinus stimulation. Isolated observations in the present study suggest, as suspected by Scherf,3 that the length of the pause is only one factor and that the occasion of that pause may also be a factor in producing these changes.

Meyer and Schmidt,9 who found T-wave changes in the complexes after ventricular

![Image](http://circ.ahajournals.org/)

**Fig. 7.** Postextrasystolic T-wave changes correlated with a positive Master test. Note that the T wave of the first beat following the premature beat in the upper strip (lead V1) is 2 mm. deeper and the Q-T interval 0.06 second longer than that of the regular beats in this lead. Immediately following standard exercise the T wave in lead V1 showed a total reversal in direction from inverted to upright, then over the course of the next few minutes gradually returned to its original direction. The patient was a 60 year old man with angina pectoris.

previous3,9 and present (figs. 2, 3, 8B) demonstration of T-wave changes following auricular premature beats, this view must be regarded as untenable.

3. **Hemodynamic Factors in the Postextrasystolic Beat.** In many communications upon
this subject there has been a tendency to relate these changes to alterations in cardiac dynamics attendant upon the greater cardiac filling associated with prolonged diastole. This cannot in itself be an entirely adequate explanation else all individuals with extrasystoles might be expected to show this type of change. A possible extension of this principle may, however, explain why this change is more prone to occur in association with myocardial impairment. Johnston and DiPalma have shown that there exists normally, at the height of mechanical systole, a difference of pressure in the various laminae of the ventricular wall, the pressure being greater in the deeper than in the more superficial layers. This pressure difference has been adduced to explain, in whole or in part, the normal difference between the deeper and more superficial layers in the duration of the excited state. Though no direct evidence upon this point is available, it is suggested that an exaggeration of these mechanical differences, due to the greater intracavitary pressure developing during prolonged diastole, produces, reflexly or directly, further restriction of coronary blood flow to the inner layers of the myocardium when there is already some actual or latent inadequacy of the flow. The electrical phenomenon of repolarization might thus be affected by the long pause. It should be noted that one must invoke a reverse explanation for those cases in which the direction of the T wave suggests exclusive or predominant impairment in the subepicardial laminae of the ventricle.

4. Mechanical Factors. Alternatively Grant, Estes and Doyle ascribe T-wave inversion after long pauses in a case of auricular fibrillation to mechanical impact of the heart against the chest wall contingent upon the prolonged diastole of the filling period. Figure 8C in the present study, on the other hand, shows a taller T wave after such a pause; a mechanical subepicardial injury cannot explain this change.

Since the T wave may become inverted in a perfectly normal heart as the rate increases, and again become upright as the rate slows, it is theoretically reasonable that changes in myocardial repolarization, though empirically associated generally with evidence of myocardial impairment, need not necessarily indicate the latter. The difference may be quantitative rather than qualitative. It is now well known, for example, that nervous influences such as sympathetic-parasympathetic imbalance may, in certain cases of neurocirculatory asthenia, be associated with T-wave changes which at one time were considered to be evidence of disease of the myocardium. In the 62 cases of "functional heart disease" here presented, two showed distinct T-wave changes in the postextrasystolic beats and 16 showed minor changes in T-wave voltage and/or the Q-T interval duration which we have arbitrarily considered as normal variations. Of further interest in this connection were the postextrasystolic T-wave changes recorded in the woman described above in whom postmortem examination showed a ruptured aneurysm of the circle of Willis but no myocardial infarct or coronary artery disease. The evidence at hand suggests, therefore, that postextrasystolic T-wave changes may in rare instances occur in the absence of disease in the myocardium proper.

Von Fernbach reported postextrasystolic T-wave changes as a temporary phenomenon in two patients with myocarditis following tonsillitis. Although in the present study transient changes have been observed relatively infrequently in chronic rheumatic heart disease, it might be expected to be more frequent during acute rheumatic fever, at a time when, presumably, myocardial factors are of greater moment than mechanical valvular factors. The observation that angina pectoris may disappear following an acute myocardial infarct and that the anoxemia test may be positive more frequently in angina pectoris before than after an episode of myocardial infarction suggests that the response to this test may vary with time; this has been explained as indicating that the patient may have regained an adequate collateral circulation. In a limited number of observations in the present study in which tracings were taken over a number of years, the tendency has been rather for a persistence of these changes. Figure 3 is an example of T-wave changes
following auricular premature beats on two occasions separated by over four years. It would seem possible, however, that similar transitory changes might occur in the postextrasystolic T waves of patients with coronary artery disease.

There is considerable confusion in the literature concerning the significance of simple changes in the duration of the Q-T interval of the postextrasystolic beat. It is not clear, for example, which of various criteria suggested might serve as a yardstick enabling one to differentiate pathologic from physiologic prolongation of the Q-T interval. Indeed present experience coincides with that of Ashman that there is a lag rather than a prompt adjustment of the Q-T interval to changes in cycle length, a fact which he has advanced to explain the absence of easily measurable changes in the T wave of the postextrasystolic beat in normal persons.

The U wave, whose significance is still unsettled, is commonly held to be related intimately to the process of myocardial repolarization and to coincide with the supernormal phase of myocardial excitability. The present evidence suggests that in certain cases alterations in the U wave may constitute an integral part of the postextrasystolic changes here described.

**Summary and Conclusions**

Pronounced “primary” changes may occasionally be detected in the voltage or direction of the T wave or in the duration of the Q-T interval of the beat following ventricular premature beats, following conducted or blocked auricular premature beats or following long pauses in auricular fibrillation. These changes are generally associated with coronary artery disease but may also occur in association with a wide variety of conditions producing profound permanent or transient myocardial impairment. It is possible that in rare instances they may occur in individuals with no detectable heart disease. Changes in the U wave may at times constitute an integral part of these postextrasystolic changes. The mechanism of these changes is not clear. Variations in cycle length are probably contributory but do not explain all the phenomena observed. The mechanical effect of overdistension of the ventricular wall in vulnerable hearts with selective local restriction of blood flow may be another factor.

It is concluded that the ventricular complex following a fortuitous lengthening of the cardiac cycle, however produced, is a convenient one to examine for T-wave changes. These alterations, when striking, suggest myocardial “ischemia” or “ischemia-like changes.”

**Acknowledgment**

This study was stimulated by a lecture in theoretic electrocardiography delivered by Dr. Richard Ashman on November 18, 1949.

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The Clinical Significance of Postextrasystolic T-Wave Changes
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