Postextrasystolic T wave changes and angiographic coronary disease

TOBY R. ENGEL, STEVEN G. MEISTER, AND WILLIAM S. FRANKL

From the Division of Cardiology, The Medical College of Pennsylvania, Philadelphia, Pennsylvania, U.S.A.

The significance of postextrasystolic T wave changes in beats following induced extrasystoles was assessed by angiography in 55 patients. These T wave changes were found in 81 per cent of coronary artery disease patients but also in 68 per cent of patients with normal coronary arteries (PNS). All patients with normal baseline electrocardiograms and normal coronary arteries showed postextrasystolic T wave changes. In electrocardiographic leads corresponding to the distribution of major coronary arteries, T wave changes occurred just as frequently when the artery was normal (54%) as when the artery was stenosed (55%). Left ventricular asynergy was not associated with an increased frequency of postextrasystolic T wave changes and in fact ejection fraction was greater and end-diastolic pressure lower in patients with T wave changes. Thus, postextrasystolic T wave changes appear not to be useful in diagnosing or localising coronary artery disease.

Postextrasystolic T wave changes (T changes) have been ascribed to cardiac, or specifically to coronary artery disease (von Kapff, 1932; von Fernbach, 1934; Scherf, 1944; Ashman *et al.*, 1945; Levine *et al.*, 1952; Mann and Burchell, 1954; Childers, 1966; McLachlan, 1962; Edmands and Bailey, 1971). However, surveys before the advent of cardiac angiography have not achieved a consensus as to the diagnostic significance of T changes (Fagin and Guidot, 1958; Robitaille and Phillips, 1965). Accordingly, patients with T changes following induced extrasystoles were evaluated by angiography for associated coronary artery disease and ventricular dysfunction.

Subjects and methods

Fifty-five patients were studied in the postabsorptive state during cardiac catheterisation. Thirty-seven patients had baseline ST segment or T wave abnormalities, including 2 because of right and one because of left bundle-branch block.

Electrocardiograms were recorded at 0.1 to 20 Hz with a paper speed of 25 mm/s. Before angiography, a catheter in the left ventricle was manipulated to induce extrasystoles. At least two single ventricular extrasystoles with compensatory pause were recorded in each of the 12 standard electrocardiographic leads.

Left ventriculography and coronary arteriography

Received for publication 8 September 1976

were performed in all patients. Asynergy was diagnosed if there was a segmental abnormality of systolic wall motion in the right anterior oblique projection. Mitral prolapse was diagnosed if there was protrusion of a portion of the posterior mitral leaflet beyond the mitral annulus during systole. Significant coronary artery disease was defined as ≥ 70 per cent luminal narrowing of one or more major vessels.

Postextrasystolic beats were analysed for T wave changes without knowledge of the angiographic diagnosis. T change was considered present if there was an alteration in vector, amplitude, or contour as compared with sinus beats. Only postextrasystolic beats with unchanged QRS were considered, in order to obviate consideration of T changes secondary to aberrancy. Fig. 1 to 3 illustrate the spectrum of T changes observed.

T changes were classified according to lead groups thought to correspond to the right coronary artery (II, III, aVF) and left anterior descending artery (I, aVL, V1-6). The circumflex artery was not analysed because of uncertainty as to which leads were appropriate to examine (Williams *et al.*, 1973). Recognising that the right praecordial leads best represent the septum and that septal wall motion was not visualised in the projection used, lead groups were analysed for corresponding asynergy as follows: inferior wall from mitral annulus to papillary muscle insertion (II, III, aVF); anterior wall, and apex (I, aVL, V1-6).

Fig. 1 Representative changes in the T wave vector of postextrasystolic beats. The examples in this figure, as well as Fig. 2 and 3, were carefully chosen as typical of the postextrasystolic T wave changes seen, rather than the best illustrative traces. Both panels are from patients with normal baseline T waves, normal coronary arteries, and mitral prolapse.

Fig. 2 Representative changes in the T wave contour of postextrasystolic beats. Panel A is from a patient with normal coronary arteries. Panels B and C are from patients with coronary artery disease.

Statistical comparisons were made with a χ^2 test. Left ventricular end-diastolic pressures and ejection fractions were compared using an unpaired t test.

Results

Thirty-six of the patients studied had significant coronary artery disease and the remaining 19 had normal coronary arteries. T changes were found in 29 (81%) patients with abnormal coronary arteries but were found as well in 13 (68%) patients with normal coronary arteries (Table). In each group, T changes were just as frequent if the resting electrocardiogram was normal or if there were baseline ST-T abnormalities. There were only 5 patients with normal baseline electrocardiograms and normal coronary arteries, but all had T changes.

T changes, when present, were most often seen in the precordial leads (Table). T changes in leads corresponding to the distribution of major coronary arteries occurred just as frequently when the artery was normal as when the artery was significantly

Fig. 3 Representative changes in the T wave amplitude of postextrasystolic beats. Panels A and B are from patients with normal coronary arteries. Panel C is from a patient with coronary artery disease.

stenosed. In total, 47 of the arteries examined were stenosed, and 26 (55%) were associated with T changes in corresponding leads; 34 of 63 (54%) normal arteries were associated with corresponding T changes. Again, when lead groups were analysed for baseline ST-T abnormalities, no significant difference in the prevalence of T changes was observed.

Seven patients with normal coronary arteries had mitral prolapse. The presence of prolapse was not associated with a significantly increased prevalence of T changes (5 of 7 patients with prolapse had T changes while 8 of 12 normal subjects without prolapse had T changes).

Anteroapical asynergy was associated with T changes in leads I, aVL, V1 to V6 in 12 of 15 patients (80%). But even when anteroapical wall motion was normal, 75 per cent had T changes in those leads. T changes in leads II, III, and aVF were found in 5 of 21 (24%) with inferior asynergy and in 38 per cent of patients with normal inferior wall motion. Ejection fraction was in fact significantly greater ($P < 0.05$) in patients with T changes (0.68 ± 0.02 SEM) than in patients without such changes (0.58 ± 0.05). Left ventricular end-diastolic pressure as well was significantly lower ($P < 0.01$) in patients with T changes ($14.0 \text{ mmHg} \pm 0.8$) than in patients without T changes (20.0 ± 3.0).

Discussion

Postextrasystolic T wave changes (T changes) were first described in patients with coronary artery disease (White, 1915). Such T changes were subsequently associated with disease; with coronary obstruction (von Kapff, 1932; Levine *et al.*, 1952; Childers, 1966), with cardiac dysfunction (von Fernbach, 1934; Scherf, 1944; Ashman *et al.*, 1945; Mann and Burchell, 1954), and with ischaemia analogous to T wave changes in the stress

electrocardiogram (Levine *et al.*, 1952; McLachlan, 1962). Bias was inherent in previous reports because electrocardiograms tended to be recorded more often in patients with heart disease. Moreover, only electrocardiograms of patients with spontaneous extrasystoles were used. Clinical correlations were made before the advent of angiography, and thus could not allow for precise or regional characterisation of coronary artery disease.

Dissenting reports failed to implicate T changes as diagnostic of heart disease (Fagin and Guidot, 1958; Robitaille and Phillips, 1965). Other correlates of T changes were identified, such as the inotropic state of the postextrasystolic contraction (Edmands and Bailey, 1971). T changes relate to the coupling interval of the extrasystole or the duration of the compensatory pause (Scherf, 1944; Levine *et al.*, 1952; Edmands and Bailey, 1971). However, in the absence of accurate clinical correlates, as can be obtained only by angiography, the actual significance of T changes has remained uncertain.

In this study we induced extrasystoles and therefore did not select patients on the basis of ectopy or other electrocardiographic abnormalities. T changes were found to be more frequent than previously reported, perhaps because we recorded extrasystoles in every lead. In fact, T changes were present in the majority of patients, regardless of diagnosis. The prevalence of T changes was similar if the baseline electrocardiogram was normal or if there were baseline ST-T abnormalities (either in the leads concerned or elsewhere). Since such T changes have been described as well after supraventricular extrasystoles (Scherf, 1944), and since many normal subjects have extrasystoles, T changes may, in fact, represent a frequent event.

Mann and Burchell (1954) have discussed only frank inversion of previously positive T waves. Such changes were not typical of those seen in this

Table Prevalence of post-extrasystolic T-changes

Coronary arteries	N	Electrocardiogram	% Post-extrasystolic T wave changes		All patients
			Baseline electrocardiogram ST-T abnormal	ST-T normal	
LAD stenosed	22	I, aVL, V1-6	77	89	82
LAD normal	33		71	75	73
RCA stenosed	25		36	29	32
RCA normal	30	II, III, aVF	43	25	33
CAD	36		83	77	81
Normal coronaries	19	All leads	57	100	68

Abbreviations: LAD, left anterior descending; RCA, right coronary artery.

study. In addition, Scherf (1944) incriminated only T wave lowering or inversion in leads I and II. These criteria for T changes were not assessed in this study. Rather, we considered all changes in the vector, amplitude, or contour of T waves, which we felt to be more inclusive.

In this study, patients with normal coronary arteries were just as likely to have T changes (68%) as patients with angiographically shown coronary artery disease (81%). Regardless of disease elsewhere, electrocardiographic leads representing the distribution of normal coronary arteries were just as likely to show T changes (54%) as leads associated with stenotic coronary arteries (55%). Changes were most frequently seen in the praecordial leads. Levine *et al.* (1952) suggested that this occurred because of involvement of the left anterior descending artery but the results of this study show that T changes are just as frequent when the left anterior descending is normal. Therefore, T changes did not accurately assess coronary artery disease in this study.

T changes did not seem to indicate regional left ventricular asynergy. Additionally, left ventricular end-diastolic pressures and ejection fractions were similar in patients with or without T changes. Thus, myocardial dysfunction or ischaemia could not be implicated as a cause of T changes. The results of this study suggest that T changes are a manifestation of the extrasystole *per se* or the following compensatory pause and probably do not reflect cardiac status. The T changes described in this investigation (Fig. 1 to 3) cannot be used to diagnose or localise coronary artery disease.

References

- Ashman, R., Ferguson, F. P., Gremillion, A. I., and Byer, E. (1945). The effect of cycle-length changes upon the form and amplitude of the T deflection of the electrocardiogram. *American Journal of Physiology*, **143**, 453-461.
- Childers, R. W. (1966). Usefulness of extrasystoles in cardiac diagnosis and prognosis. *Medical Clinics of North America*, **50**, 51-71.
- Edmunds, R. E., and Bailey, J. C. (1971). The postextrasystolic T wave change. *American Journal of Cardiology*, **28**, 536-540.
- Fagin, I. D., and Guidot, J. M. (1958). Post-extrasystolic T wave changes. *American Journal of Cardiology*, **1**, 597-600.
- Levine, H. D., Lown, B., and Streeper, R. B. (1952). The clinical significance of post extrasystolic T-wave changes. *Circulation*, **6**, 538-548.
- McLachlan, E. M. (1962). Post-extrasystolic T wave changes and myocardial ischaemia. *New Zealand Medical Journal*, **61**, 397-401.
- Mann, R. H., and Burchell, H. B. (1954). The significance of T-wave inversion in sinus beats following ventricular extrasystoles. *American Heart Journal*, **47**, 504-513.
- Robitaille, G. A., and Phillips, J. H., Jr. (1965). A study of the post extrasystolic T wave changes associated with interpolated premature ventricular beats. *American Journal of the Medical Sciences*, **250**, 315-322.
- Scherf, D. (1944). Alterations in the form of the T waves with changes in heart rate. *American Heart Journal*, **28**, 332-347.
- Von Fernbach, J. (1934). Die Veranderungen des Electrocardiogramms nach Kammerextrasystolen. *Deutsches Archiv für klinische Medizin*, **177**, 59-64.
- Von Kapff, W. (1932). Über post extrasystolische Änderung der T-Zacke. *Zeitschrift für Kreislaufforschung*, **24**, 273-277.
- White, P. D. (1915). Alternation of the pulse: a common clinical condition. *American Journal of the Medical Sciences*, **150**, 82-97.
- Williams, R. A., Cohn, P. F., Vokonas, P. S., Young, E., Herman, M. V., and Gorlin, R. (1973). Electrocardiographic, arteriographic and ventriculographic correlations in transmural myocardial infarction. *American Journal of Cardiology*, **31**, 595-599.

Requests for reprints to Dr. Toby R. Engel, Division of Cardiology, The Medical College of Pennsylvania, 3300 Henry Avenue, Philadelphia, Pennsylvania 19129, U.S.A.