

**SELECTION OF APPLICANTS FOR INSURANCE  
WITH ISOLATED T-WAVE ABNORMALITIES  
IN THE ELECTROCARDIOGRAM**

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**I**SOLATED T-wave abnormalities (that is, abnormal T waves in an otherwise normal electrocardiogram) are a difficult finding to evaluate and one on which there is no unanimity, either on their significance or on what constitutes an abnormal T wave. The difficulty arises from their inherent characteristics.

Abnormal T waves may be present in all types of heart disease—in acute conditions (such as pericarditis) and in chronic conditions (such as congenital, rheumatic, hypertensive, and arteriosclerotic heart disease). They can be produced in a normal heart, too, by a variety of extracardiac conditions (such as biochemical changes). As a result, T-wave abnormalities are not in themselves diagnostic. In the absence of other electrocardiographic abnormalities, their significance can be determined, if at all, only by correlation with the clinical background.

The most difficult cases to underwrite are those with T-wave abnormalities only and with no physical signs on examination. In these the diagnosis, if one can be made, rests upon the history and symptoms. When such a case presents itself for insurance, the uppermost question is: Are the T-wave abnormalities due to coronary disease?

Coronary disease may be present without diagnostic evidence in the electrocardiogram. A coronary occlusion sometimes leaves no residual evidence in the electrocardiogram except abnormal T waves, either because the occlusion produced T-wave changes only, or because the Q waves, in the course of time, have lost their coronary contours; this has occurred in approximately one-fourth of our coronary occlusions. There may even be no history; in 5 per cent of our cases, the occlusion occurred without symptoms or with symptoms so mild and atypical that the individual did not consider them of any importance and did not seek medical aid; in an additional 20 per cent the symptoms were so atypical that an occlusion was not suspected until the electrocardiogram was made (as a precautionary measure). The diagnosis of angina pectoris often rests entirely on the history and symptoms; it can be confirmed by an electrocardiogram made after exercise that shows S-T segment changes characteristic of coronary insufficiency, but it cannot be made from the resting

record, with or without abnormal T waves, and is not ruled out by a post-exercise electrocardiogram that does not show the characteristic changes. The cases in which coronary disease is present but has not yet become manifest clinically, either as angina pectoris or coronary occlusion, may have no symptoms at all or may have symptoms ranging from those so atypical that they are presumed noncardiac in origin to those that would be classed as suspicious of coronary disease. In evaluating symptoms, one must always bear in mind that they are often atypical.

To compound the confusion further, the behavior of abnormal T waves is quite unpredictable. T waves may become abnormal and remain abnormal. Abnormal T waves may become normal and remain normal. A T wave that is merely low may have been inverted a short time ago and may be inverted again a short time (or a long time) hence. Abnormal T waves, either low or inverted, may fluctuate between normal and abnormal for years. It is my observation that these changes may occur whether or not there is underlying coronary disease, either covert or manifest.

Such is the elusive finding with which we are dealing.

#### MATERIAL

The material used in this study comprised all males in the Prudential's Home Office employee file aged 40-69 whose electrocardiograms showed abnormal T waves but were otherwise normal, whose physical findings were normal on clinical and laboratory examinations, and who had no ratable history—except for the history of coronary disease in Groups 4 and 5 below. In every respect, all of them were physically equivalent to standard applicants for insurance, except for the impairment by which they became entrants to this study.

They were subdivided into five groups according to whether they had (1) no symptoms (288 cases); (2) symptoms thought to be noncardiac in origin (102 cases); (3) symptoms considered suspicious of coronary disease (26 cases); (4) the classical symptoms of angina pectoris (28 cases); or (5) a definite history of coronary occlusion survived by at least sixty days (27 cases). The electrocardiograms of those who had had a coronary occlusion showed T-wave abnormalities only and could not be distinguished from any other electrocardiogram showing isolated T-wave abnormalities. There were 89 deaths among these 471 individuals.

The T-wave abnormalities were classified as minor or major. In general, T waves were considered to show minor abnormality when they were low and major abnormality when they were diphasic or inverted, with some allowances made in Lead 1 and  $aV_L$  for a vertical heart and in Lead 2 and  $aV_F$  for a horizontal heart. Low, diphasic, or inverted T waves

are not necessarily abnormal in Lead 3 or in the right precordial leads, and T waves in  $V_R$  must be inverted to be normal. Only cases with T waves that were thought to be abnormal were included in the study.

If a minor T-wave abnormality later became a major T-wave abnormality, or if the history and symptoms progressed from the original classification to a higher classification, the case was entered again in the appropriate group (provided no other impairment had developed meanwhile), so that many cases were included in two or more groups.

The observation period was September 1, 1933, to June 30, 1964, with no new entrants after December 31, 1963. Expected deaths were computed on the Male 1955-1960 Basic Table, Select, and its 1957-1960 Ultimate section.

Our male normal control group, physically equivalent to the T-wave groups except that their electrocardiograms were normal and there was no history or suspicion of coronary disease, had a mortality ratio of 92.5 per cent (235 deaths among 1,952 individuals) by these tables for the same age range and observation period. There were 87 deaths due to coronary disease, 41 due to other cardiovascular diseases, and 107 due to unrelated causes. The observation period averaged 12.4 years. The expected deaths on the T-wave groups were adjusted to 92.5 per cent of those computed by the Basic Tables and are so shown in Tables 1-4; the mortality ratios, then, relate to the mortality on our male normal control group as the measure of standard.

#### HOW LOW IS A LOW T WAVE?

We, and most others, have long since discarded the old rule that a T wave measuring less than 1 millimeter is low, and a T wave measuring 1 millimeter or more is normal. We consider a T wave low when it is lower than we would expect it to be in comparison with the QRS complex—the size of the QRS complex being the area above the isoelectric line less the area below it. Thus, a T wave measuring less than 1 millimeter might be considered normal if the QRS complex is small, and a T wave exceeding 1 millimeter might be considered low if the QRS complex is large. Our conception of what constitutes adequate T-wave voltage is a subjective standard acquired by having seen thousands of electrocardiograms. It is, unfortunately, impossible to define with precision.

It is obvious, therefore, that there would be differences of opinion as to what constitutes an abnormal T wave. Accordingly, three separate classifications of the T waves were made, in each of which the T waves were more definitely abnormal than in the preceding one.

The original selection, made by the author, included T waves considered borderline in amplitude.

The first revision, also made by the author, excluded T waves that were considered borderline and moved a few cases from the major to the minor group, where the T-wave negativity was slight and the QRS complex small in  $aV_L$  or  $aV_F$ . Those excluded by this requirement would probably not be considered abnormal by most observers.

The second revision was made independently by one of my colleagues in the medical profession. Working with the cases retained in the first revision and looking only at the electrocardiograms, he selected what he considered the worst of the T-wave abnormalities to see if he could increase the mortality. In so doing, he excluded many T waves that he thought abnormal but not so abnormal as those he retained. In general, he required that the T waves be lower in comparison with the QRS complexes than I did in my first revision; he moved some cases from the major to the minor group, where the diphasic or inverted T waves were very shallow in the standard and precordial leads; and he was especially lenient with low T waves in  $aV_F$  alone, as they are often seen in apparently normal young adults (although our subjects were not young).

The classification of symptoms and history was agreed upon between us before the T-wave revisions were started and was not changed.

The results of the successive T-wave classifications are shown in Tables 1 and 2. After preliminary testing, the five subdivisions based on symptoms and history were reduced to three. The group reporting no symptoms and the group reporting symptoms thought to be noncardiac showed no consistent difference in mortality and were combined. The angina pectoris and coronary occlusion groups were combined, as their relation to each other is less important for this study than the comparison of the coronary cases as a whole with the supposedly noncoronary cases.

From Table 1 it will be noted that, in cases with minor T-wave abnormalities and no clinically manifest coronary disease or suspicious symptoms at entry, the first revision increased the mortality ratio only 4 points; the second revision did not increase the mortality at all.

In Tables 1A and 2A is shown the coronary disease that developed during the observation period. The figures under "Manifest Coronary Disease" represent the number of cases in which coronary occlusion (with or without angina pectoris) or angina pectoris (without coronary occlusion) became clinically manifest among the cases showing no clinical evidence of coronary disease at entry. For those with clinically evident coronary disease at entry, the figures represent an extension during the observation period, that is, a first occlusion among those who had angina

TABLE 1

## MORTALITY EXPERIENCE ON MINOR T-WAVE ABNORMALITIES

HISTORY AT ENTRY	NO. OF CASES	ACTUAL DEATHS	EXPECTED DEATHS	MORTALITY RATIOS	CAUSE OF DEATH		
					Coro-nary	Other C-V	Unre-lated
Original Selection							
Noncardiac symptoms or none.....	372	53	37.08605	143%	23	8	22
Suspicious symptoms.....	27	4	2.22089	180%	2	0	2
Manifest coronary disease.	64	25	6.17849	405%	19	4	2
First Revision							
Noncardiac symptoms or none.....	295	44	29.91725	147%	19	7	18
Suspicious symptoms.....	23	5	1.94070	258%	3	0	2
Manifest coronary disease.	61	24	5.53864	432%	18	4	2
Second Revision							
Noncardiac symptoms or none.....	218	36	24.56510	147%	16	7	13
Suspicious symptoms.....	16	3	1.17500	255%	3	0	0
Manifest coronary disease.	59	23	5.64033	408%	17	4	2

TABLE 1A

CORONARY DISEASE BECOMING MANIFEST DURING OBSERVATION PERIOD  
AMONG CASES WITH MINOR T-WAVE ABNORMALITIES AT ENTRY  
(Original Selection)

HISTORY AT ENTRY	NO. OF CASES	AVERAGE OBSERVATION PERIOD IN YEARS	MANIFEST CORONARY DISEASE			AVERAGE NO. CORONARIES PER YEAR PER 100 CASES
			Coronary Occlu-sion	Angina Pec-toris	Total	
Noncardiac symptoms or none.....	372	10.3	48	19	67	1.75
Suspicious symptoms.....	27	8.8	3	1	4	1.68
Manifest coronary disease	64	8.2	25	.....	25	4.76

pectoris at entry or a second occlusion among those who had had an occlusion before this observation began. The coronary disease becoming manifest during the observation period consists of coronary deaths, coronaries dying of other causes, and coronaries still living. Since very few of the coronaries died of other causes, the total manifest coronary disease less the coronary deaths represents, very nearly, the coronaries still living in the two groups that were supposedly noncoronary at entry, a potential source of further increased mortality. By comparison, our male normal control group had 212 coronaries becoming clinically manifest during an average exposure of 12.4 years among 1,952 entrants, a rate of 0.88 per year per 100.

In Tables 3 and 3A the cases with minor T-wave abnormalities that were excluded from the experience in successive revisions (ignoring the cases that were reclassified but not excluded) have been studied separately, as it is here that the temptation to issue standard insurance is greatest. The borderline T waves in supposedly noncoronary cases excluded in the first revision show a mortality ratio of 126 per cent, which is within standard limits but is 26 points higher than that of the normal control group; and the rate at which clinically manifest coronary disease developed, 1.27 cases per year per 100 entrants, is half again as high as the rate of 0.88 in the normal control group. The "best" of the abnormal T waves, excluded from the same clinical group in the second revision, had a mortality ratio of 154 per cent, outside standard limits and a little higher than the 147 per cent on those that were retained as the "worst" of the group.

#### RELIANCE ON HISTORY

It has already been said that the evaluation of abnormal T waves must be based on correlation with the clinical background. Since the cases in this study are all without physical signs, the clinical background is the history. And in all except coronary occlusion the history consists of symptoms, which the applicant can withhold if he is so minded. A history of coronary occlusion is not so readily concealed because, if it is recognized clinically, a considerable period of confinement ensues. It has been pointed out, however, that some coronary occlusions do go unrecognized. Moreover, some histories, even though fully recounted, are so atypical that they may be misinterpreted. As a result, some unfavorable histories are almost certain to be accepted unwittingly.

We believe that the histories on our cases are more dependable than those obtained, on the whole, from insurance applicants. It is quite possible, therefore, that the experience on applicants for insurance would be somewhat higher than that shown in the foregoing tables. In addition,

**TABLE 2**  
**MORTALITY EXPERIENCE ON MAJOR T-WAVE ABNORMALITIES**

HISTORY AT ENTRY	NO. OF CASES	ACTUAL DEATHS	EXPECTED DEATHS	MORTALITY RATIOS	CAUSE OF DEATH		
					Coro-nary	Other C-V	Unre-lated
Original Selection							
Noncardiac symptoms or none.....	61	12	7.60182	158%	9	0	3
Suspicious symptoms.....	12	2	.83869	238%	2	0	0
Manifest coronary disease.	40	15	2.32606	645%	15	0	0
First Revision							
Noncardiac symptoms or none.....	55	12	6.69545	179%	9	0	3
Suspicious symptoms.....	8	1	.26770	374%	1	0	0
Manifest coronary disease.	37	14	2.19938	637%	14	0	0
Second Revision							
Noncardiac symptoms or none.....	24	7	3.76183	186%	6	0	1
Suspicious symptoms.....	5	0	.22898	.....	0	0	0
Manifest coronary disease.	21	9	1.26288	713%	9	0	0

**TABLE 2A**  
**CORONARY DISEASE BECOMING MANIFEST DURING OBSERVATION PERIOD**  
**AMONG CASES WITH MAJOR T-WAVE ABNORMALITIES AT ENTRY**  
**(Original Selection)**

HISTORY AT ENTRY	NO. OF CASES	AVERAGE OBSERVATION PERIOD IN YEARS	MANIFEST CORONARY DISEASE			AVERAGE NO. CORONARIES PER YEAR PER 100 CASES
			Coronary Occlu-sion	Angina Pec-toris	Total	
Noncardiac symptoms or none.....	61	10.7	15	2	17	2.60
Suspicious symptoms.....	12	8.8	2	0	2	1.89
Manifest coronary disease	40	7.0	19	.....	19	6.79

TABLE 3

## MORTALITY EXPERIENCE ON MINOR T-WAVE ABNORMALITIES EXCLUDED

HISTORY AT ENTRY	NO. OF CASES	ACTUAL DEATHS	EXPECTED DEATHS	MORTALITY RATIOS	CAUSE OF DEATH		
					Coro-nary	Other C-V	Unre-lated
Excluded in First Revision							
Noncardiac symptoms or none.....	79	9	7.12709	126%	4	1	4
Suspicious symptoms.....	5	0	.37722	.....	0	0	0
Manifest coronary disease.	5	2	.26850	745%	2	0	0
Excluded in Second Revision							
Noncardiac symptoms or none.....	100	13	8.46019	154%	6	0	7
Suspicious symptoms.....	9	3	.80358	373%	1	0	2
Manifest coronary disease.	14	4	.85476	468%	4	0	0

TABLE 3A

CORONARY DISEASE BECOMING MANIFEST DURING OBSERVATION PERIOD  
AMONG MINOR T-WAVE ABNORMALITIES EXCLUDED

HISTORY AT ENTRY	NO. OF CASES	AVERAGE OBSERVATION PERIOD IN YEARS	MANIFEST CORONARY DISEASE			AVERAGE NO. CORONARIES PER YEAR PER 100 CASES
			Coro-nary Occlu-sion	Angina Pec-toris	Total	
Excluded in First Revision						
Noncardiac symptoms or none.....	79	9.0	7	2	9	1.27
Suspicious symptoms.....	5	8.4	0	0	0	.....
Manifest coronary disease	5	8.6	2	.....	2	4.65
Excluded in Second Revision						
Noncardiac symptoms or none.....	100	9.5	14	5	19	2.00
Suspicious symptoms.....	9	7.9	1	0	0	1.41
Manifest coronary disease	14	8.0	6	.....	6	5.36



it should be emphasized that the mortality ratios for suspicious history are averages: the rating applied to an individual case would depend on how suspicious the underwriter feels. It might be little more than that required for a supposedly noncardiac case, or it might equal the rating for coronary occlusion.

#### WHAT OF ABNORMAL T WAVES THAT HAVE RETURNED TO NORMAL?

When T-wave abnormalities disappear, they cannot be ignored, for, as previously stated, their disappearance does not prove that the abnormalities were of no significance.

TABLE 4  
ELECTROCARDIOGRAM NORMAL, T WAVES PREVIOUSLY MINOR

HISTORY AT ENTRY	NO. OF CASES	ACTUAL DEATHS	EXPECTED DEATHS	MORTALITY RATIOS	CAUSE OF DEATH			MANIFEST CORONARY DISEASE		AVERAGE OBSERVATION PERIOD IN YEARS
					Coro-nary	Other C-V	Unre-lated	Coro-nary Oc-cclusion	Angi-na Pec-toris	
Original Selection										
Noncardiac symptoms or none .....	142	20	10.21909	196%	9	1	10	17	3	7.9
Suspicious symptoms .....	10	1	.66397	151%	1	0	0	1	1	7.0
Manifest coronary disease..	18	6	1.59931	375%	5	1	0	5	.....	9.7
First Revision										
Noncardiac symptoms or none .....	110	16	8.31791	192%	5	1	10	13	2	8.1
Suspicious symptoms .....	10	1	.65758	152%	1	0	0	1	1	6.9
Manifest coronary disease..	19	7	1.68878	415%	6	1	0	7	.....	9.9
Second Revision										
Noncardiac symptoms or none .....	67	11	5.38855	204%	2	1	8	5	1	7.9
Suspicious symptoms .....	8	1	.58365	171%	1	0	0	1	1	7.1
Manifest coronary disease..	18	6	1.55923	385%	5	1	0	6	.....	9.0

In Table 4 is the mortality experience on the minor T-wave abnormalities from Table 1 that returned to normal (they did not necessarily remain normal), observed from the date of the first normal electrocardiogram following the abnormal one. The clinical classification is that appropriate at the time they entered Table 4. Individuals who had meanwhile developed hypertension or some other ratable impairment were disqualified for entry. The mortality ratios are higher, on the whole, than those in Table 1 when the T waves were abnormal on current examination.

The percentage of entrants dying and the percentage developing manifest coronary disease did, in fact, show a slight decrease, but the average length of exposure was less, so that there was, actually, very little difference. The expected deaths showed a somewhat greater percentage decrease than did the actual deaths, partly because of the shorter duration and partly because the mortality rates were set back to select duration one. As a result, the mortality ratios are somewhat higher, but the proportionate number dying is about the same.

#### CONCLUSIONS

The results of this study may be summarized as follows:

Minor T-wave abnormalities in an otherwise normal electrocardiogram require a small rating, and major T-wave abnormalities a somewhat higher rating.

Low T waves cannot be divided into better or worse on the basis of their amplitude.

Correlation with the clinical background is a necessity, and some additional debit should be imposed for histories considered suspicious.

The return of abnormal T waves to normal, according to this experience, certainly merits no credit.

Isolated T-wave abnormalities without underlying heart disease are relatively rare. In a file of some 15,000 individuals with about 40,000 electrocardiograms there were found only 381 individuals who had minor T-wave abnormalities without clinically manifest heart disease on entering this study, and some of these did, in fact, have coronary disease that became manifest during the observation period. Major T-wave abnormalities with a negative clinical background are much rarer, and cases so reported should be looked upon with considerable skepticism.

## DISCUSSION OF PRECEDING PAPER

WILLIAM A. KELTIE:

From a study of 433 otherwise healthy home-office employees with abnormal electrocardiograms, the author has concluded that applicants for insurance presenting the same E.K.G. variants require a modest mortality rating in the order of 40-80 per cent above the standard insurance level.

Evidence to support this underwriting conclusion is also provided in a report just published in the *Canadian Medical Journal*. It describes a study of cardiovascular disease sponsored by the University of Manitoba Medical Department.

Four thousand healthy North American males, 91 per cent under age 40, were subjected to periodic E.K.G.'s and medical examinations over a fifteen-year period beginning in 1948. The health of all but 5 of the survivors was known in 1963. During the investigation period, 213 died, and 143 developed coronary heart disease.

Some 274 exhibited T-wave changes in their electrocardiogram without cardiac symptoms similar to the group described by Miss Lyle. Among those with T-wave variants, 24 died, where only 13 were expected to. Some 28 developed coronary heart disease, where 9 were expected.

The report concludes, that for subjects exhibiting T-wave changes in the electrocardiogram, the chance of acquiring coronary heart disease was three times normal and the mortality rate was twice the normal level.