

THE MEANING OF MINOR T WAVE ABNORMALITIES IN INSURANCE MEDICINE

M. IRENÉ FERRER, MD

Consultant in Cardiology

Metropolitan Life Insurance Company

Professor Emeritus of Clinical Medicine,

College of Physicians and Surgeons, Columbia University

Consultant Electrocardiographer,

Presbyterian Hospital, Columbia Presbyterian

Medical Center, New York, NY

Although the impact of an abnormal electrocardiogram upon risk evaluation in insurance applicants continues to be of great importance, some electrocardiographic abnormalities carry more and some less weight. Probably one of the most difficult areas of risk evaluation is the analysis of T wave abnormalities. The T wave of the electrocardiogram is the most labile, unstable and non-specific component of the electrocardiogram. Hence, abnormalities of this wave require very careful inspection and overall investigation, lest over-interpretation, and thus over-rating the risk, occur. It should at once be emphasized that T abnormalities are separate from ST abnormalities and that it is unwise to use a blanket term "ST-T abnormalities" to describe electrocardiographic changes; the deviations should be analyzed separately.

Incidence

The presence of isolated T wave abnormalities in various asymptomatic populations is known to be a common finding. Although it is stated¹ that "non-diagnostic T changes are present in 2.4 percent and 4.5 percent of all routine cardiograms", the figures from an insurance population are somewhat more pertinent in insurance risk evaluation. In a recent study of 19,734 electrocardiograms in insurance applicants², 6,201, or 32% of the total were abnormal. Of these 6,201 abnormal tracings, 1,601 showed only T wave and ST abnormalities — an incidence of 8% of the total 19,734 tracings reviewed. There were 1,015 cases with only T abnormalities (without any associated ST abnormalities) — an incidence of 0.05% of the total 19,734 tracings. Of these solitary T abnormalities the breakdown was striking — 732 (72%) were low T waves (no diphasic or negative contour) and only 283 (or 28% of all the T abnormalities) were diphasic or negative.

It is thus quite clear that low T waves represent the majority of the changes one encounters in insurance electrocardiograms.

Definition

The definition of minor T wave abnormalities (T waves lower than 1.0 mm in height) is limited to low T or flat waves in more than one lead. Diphasic contours are not part of the definition. Diphasic T waves are uncommon and infer a changing electrocardiogram. If present, a repeat tracing is needed to determine whether the T wave which is changing become inverted or simply becomes low. Inversions of T waves have been captioned as major T wave abnormalities in the terminology of the insurance industry and carry a much more serious implication and risk than the minor T abnormality.

It is of primary importance in reviewing minor T wave abnor-

malities to note the extent and location of these low T waves. If the low T waves exist in all 12 leads — a diffuse etiology is suggested (see below). If the low T waves are localized to certain anatomically oriented leads (e.g., inferior, lateral, anterior) disease is more likely. It would also be most useful to know the duration — i.e., how long such minor T wave changes have been present — but often in risk evaluation it is not possible to obtain numerous or serial electrocardiograms on the applicant, especially if he or she is asymptomatic and had the tracing performed only for insurance purposes. If the minor T abnormalities have been present and unchanged for some time, it reduces their significance somewhat.³

Etiology or Low T Waves (Minor T Wave Changes)

Once inspection of the electrocardiogram has ruled out any accompanying ST abnormality or any negative T waves in addition to low T waves, a number of cardiac diagnoses can be ruled out. There are numerous non-cardiac causes for low T waves and these include hypothermia, fever, hyperventilation, anxiety, food intake, tachycardia, neurological diseases, digitalis drugs, anti-arrhythmic drugs, cocaine, psychotropic drugs (phenothiazines, tricyclics, lithium), electrolyte abnormalities, strokes, shock, anemia, allergic responses and certain endocrine diseases. Low serum and presumably, therefore, low myocardial potassium is a very frequent cause and if prominent U waves accompany the low T waves the diagnosis of this electrolyte imbalance is clear. There are some more benign causes for low T waves, such as for example, chilling of the myocardium by swallowing of a cold drink (cooling the esophagus which lies over the cardiac musculature) taken just before the electrocardiogram is recorded. The ingestion of a meal will also lower T waves, probably by producing a transitory potassium flux out of the serum. Hyperventilation, anxiety and moderate sinus tachycardia can also induce T wave lowering operating through the catecholamine systems. Although gastrointestinal diseases per se do not alter T waves, if vomiting or diarrhea occurs the resulting lowering of potassium can lower T waves. These low T waves due to non-cardiac causes are usually diffuse i.e., are present in all 12 leads.

The list of non-cardiac causes of low T waves is long, varied and challenging as to solution. The list of cardiac causes is less extensive, more obvious and usually allows for a clinical definition. The list includes primary myopathies, myocarditis (including bacterial, AIDS, and other viral diseases such as coxsackie infections) diffuse connective tissue or neuromuscular disorders, metastatic infiltration of the myocardium from distant tumors, and the rare diseases such as amyloido-

sis, hemochromatosis, primary cardiac neoplasms or sarcoidosis. Coronary artery disease (CAD) is rarely expressed only as low T waves. Rather in CAD there are negative T waves, abnormal ST segments or abnormal QRS complexes (Q waves, poor R progression). However, it is often the practice in insurance medicine to request an exercise test to uncover myocardial ischemia due to CAD when minor T wave abnormalities are present. However, as Brackenridge notes⁴ "if the exercise tracing is negative for ischemia, the rating for minor T wave changes in the electrocardiogram at rest can usually be reduced to zero." When there is more clinical latitude available in the analysis of low T waves in the resting electrocardiogram it is useful (where possible) to note if anxiety, hyperventilation or tachycardia existed when the record was taken. A very common contribution to low T waves is the ingestion of a meal 2-3 hours before the electrocardiogram was taken — the optimal time for increased serum glucose and potassium shifts — and, hence, it is best that the electrocardiogram taken for insurance evaluation be done fasting. Similarly a cold or hot drink just before the test can produce factitious lowering of T waves. Hence, inquiry as to such pre-test imbibitions is wise.

When there is stable and bona fide lowering of the T waves, it is sometimes assumed that the degree of lowering is of prognostic use. Actually there is no prognostic relationship between slightly diminished T waves and totally flat T waves. Indeed the implication that the degree of lowering of T, or, in the case of negative T waves, the depth of the negativity measured in millimeters, is related to the seriousness of the condition is quite incorrect. Since overall electrocardiographic interpretation relies heavily upon numerical criteria it has become a habit for electrocardiographers to present numerical structures as signs of the degree of abnormality. This concept, of course, does apply to many ECG features — e.g., width of abnormally prolonged QRS complexes where very marked prolongation of the QRS (i.e., beyond 0.15 sec.) implies serious and diffuse disease in the myocardial fibers themselves as opposed to localized bundle branch lesions when QRS is shorter, or actual numerical measurements of axis deviation. However numerical evaluation of abnormal T wave does not provide any basic information as to the extent or seriousness of the underlying condition.

Shifts in bodily position can easily alter T wave contours. For this reason it is imperative to record electrocardiograms in the standard position, i.e., supine, and not in the sitting position. The record should indicate the bodily position of the applicant. It is sometime the practice — especially by the paramedic technicians — to allow the subject to sit in a chair if there is no examining table, couch or bed available where the electrocardiogram is being secured. This can lead to errors not only of axis deviation (due to compression and elevation of the diaphragm in the sitting position with consequent rotation of the cardiac mass into an unusual plane) but can change the T waves as well, especially in those leads affected by diaphrag-

matic shifts (II, III, AVF and sometimes V6).

The instability of T wave contour is well illustrated in the responses of this wave during exercise. A normal resting tracing may develop low or even negative T waves during exertion. For this reason the T wave behavior is never used as a criterion to evaluate an electrocardiogram for myocardial ischemia. This decision is deduced only from the behavior of the ST segment. The explanation for such alterations in the shape and direction of the T wave during exercise may, in some measure, be related to the fact that the T wave is the recovery wave of the ventricles electrically speaking — i.e., after electrical excitation (which of course triggers the mechanical contraction) the myocardial cells must return to a resting phase in order to be ready for the next electrical excitation process. However, the recovery rate of the ventricular myocardium is not uniform throughout as the endocardial surface of this myocardium has a recovery rate which is slower than that of the epicardial surface. During exercise the endocardial surface has greater mechanical stress due to the increased ventricular myocardial shortening during contraction and subsequent ejection. It is likely, therefore, that this endocardial stress delays electrical recovery to a greater extent than occurs at the epicardial surface, causing an imbalance between endocardial and epicardial recovery and, hence, altering the form of the T wave.

It is also of interest to note that there are constant changes in T waves during early cardiac development. There are marked changes in T direction or polarity with growth. In the first 24 hours of life the T waves may be almost flat in contour in all leads and then become larger in a few days. The T wave in the right V leads may be upright in the first 24 hours but after the first few days, or occasionally even as late as 3 weeks after birth, the T waves in the right V leads normally become quite negative and remain so up to approximately one year of age. Thus after age 3 weeks and up to one year the normal T wave is negative in the right, and positive in the left, precordial leads. It is usual for the negative T waves in V1-V3 to become upright in the early years of life, but the change may not take place until the late teens or early twenties, and this feature has been called the persistence of the "juvenile" pattern. The details and cause of this developmental shift are unknown.

Conclusions

It is quite clear that the finding of solitary (or isolated) low T waves in the electrocardiogram of insurance applicants carries little prognostic implication per se. Rather it can be a flag or signal alerting the examiner to investigate further before automatically applying a rating based simply on the finding described as minor T wave abnormalities.

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