Tall Upright T Waves in the Precordial Leads


SUMMARY

One hundred ten cases of T waves more than 10 mm in height in the precordial leads were studied. The T waves were wide in ischemic heart disease and thin and peaked in uremia. Marked changes in the ventricular gradient were observed in ischemic heart disease and in uremia with hyperkalemia. In other conditions, the changes in the ventricular gradient were of a lesser degree. In cases of left ventricular hypertrophy, the T waves were tall from V₁ to V₄ in systolic (pressure) overloading, from V₄ to V₆ in diastolic (volume) overloading, and from V₂ to V₆ in combined types of overloading.

The appearance of tall T waves was the earliest electrocardiographic sign encountered in clinical and experimental cases of acute myocardial infarction. In anterior myocardial infarction they disappeared within 24 hours indicating initial transient ischemia, while in angina pectoris and posterior myocardial infarction they lasted for a longer time probably due to persistent subendocardial ischemia.

Additional Indexing Words:

Ventricular gradient
Ventricular hypertrophy
Uremia
Angina pectoris
Hyperkalemia
Myocardial infarction

The voltage of the T wave is increased in unipolar precordial leads in a small percentage of normal individuals. It is also increased in anterior and posterior myocardial infarction, angina pectoris, hyperkalemia, anemia, left ventricular hypertrophy, and a variety of other conditions.

In the present report, the morphology of the QRS complex and the T wave has been studied with a view to comparing the features in various conditions and to establishing diagnostic criteria that might elucidate the etiological basis of the tall T wave.

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Methods

One hundred ten electrocardiograms with T waves measuring more than 10 mm in height in the precordial leads, which were taken at the K.E.M. Hospital, were studied. The patients were of both sexes and their ages ranged from 20 to 80 years.

There were 35 cases of ischemic heart disease: among these were four cases of acute anterior myocardial infarction, seven of acute posterior myocardial infarction, and the remaining 24 were cases of angina pectoris. Eleven of these 24 were cases of postinfarction angina. There were also 10 cases of uremia, eight of neurocirculatory asthenia, 22 of hypertension, and 10 of rheumatic valvular disease. In the remaining 25, diverse etiologies were present: there were six cases of cardiomypathy, four of congenital heart disease, four thyroid disease, four of anemia, and seven were from apparently normal persons.

The 14-lead electrocardiograms were analyzed as follows: (1) Height of T was measured as the distance from the summit of T to the isoelectric line (fig. 1). (2) Width was measured from the beginning of T to the end of T (fig. 1). (3) The QTc (corrected) interval was measured from the beginning of Q to the end of T and was corrected for the rate. (4) The axes of the QRS and
of the T wave were measured in the horizontal plane by measuring their respective areas in microvolt seconds in V1 and V6. These values were plotted on V1 and V6, and perpendiculars were dropped from these points to the lead axis; the point of intersection of the two perpendiculars was joined to the center, and these vectors represented the direction and magnitude of the axis of the QRS and of the T wave (fig. 1). The ventricular gradient was then obtained by vectorial summation of AQRS and AT by the parallelogram method (fig. 1). We are aware of the limitations in the calculation of the ventricular gradient in the horizontal plane. V1 and V6 are unipolar leads and do not exclusively represent anterior and posterior and right and left electrical potentials, respectively. However, in the absence of any superior method, we calculated the ventricular gradient using unipolar precordial leads V1 and V6 as stated.

Hemoglobin was estimated in all cases, and in 65 cases serum potassium was estimated by means of a flame photometer. Master's two-step exercise test was performed in 45 cases.

A similar analysis was made of 100 cardiograms of normal persons studied in an epidemiological survey conducted in our department.

Experimental myocardial infarction was produced in 10 mongrel dogs by ligating the anterior interventricular branch of the left coronary artery using the closed chest technique, and electrocardiograms were recorded 2 minutes, 4 hours, 8 hours, and 24 hours later.

Results and Discussion

Morphological Features and Duration of Tall T Waves (Table 1)

The average height of the T wave in various conditions varied from 11.2 mm to 13.1 mm, but these alterations did not permit an electrocardiographic differentiation.

The T wave was significantly thin and peaked in cases of uremia, with an average width of 0.16 sec. On the other hand, it was broad in cases of myocardial infarction and hypertensive heart disease in which the average width was 0.24 sec and 0.26 sec, respectively.

The QTc interval was prolonged in 60% of the cases of uremia with hyperkalemia although the T waves were narrow. The reason...
is that the QRS interval increases *pari passu* with the hyperkalemia. Moreover, many of these patients might have had associated hypocalcemia which would have contributed to the prolongation of the QT interval.

In conclusion the only morphological features of diagnostic significance in our studies were the narrowness and tenting of the T wave which were consistently present in our cases of uremia with hyperkalemia. The other measurements failed to reveal any striking differences.

Tall T waves of more than 10 mm in height in the precordial leads were the earliest changes recorded in both acute anterior and posterior myocardial infarction (fig. 2). However in anterior infarction the height of the T wave decreased on the second day and was followed by ST-segment changes or T-wave inversions. In posterior infarction the T waves remained tall for many days and in some cases for as long as 4 years.

The hemoglobin levels were normal in all cases except those of anemia in which they

**Table 1**

**Morphological Features of the Tall T Waves**

<table>
<thead>
<tr>
<th>Morphological features</th>
<th>Ischemic heart disease</th>
<th>Left ventricular hypertrophy</th>
<th>Neurocirculatory asthenia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
<td>Angina pectoris</td>
<td>Anterior</td>
</tr>
<tr>
<td>Average height of T wave (mm)</td>
<td>5.6</td>
<td>11.6</td>
<td>11.2</td>
</tr>
<tr>
<td>Average width of T wave (sec)</td>
<td>0.20</td>
<td>0.22</td>
<td>0.23</td>
</tr>
<tr>
<td>Prolonged QTc interval (% of cases)</td>
<td>0</td>
<td>41</td>
<td>40</td>
</tr>
</tbody>
</table>

The sequences of ST-segment and T-wave changes in a 45-year-old man admitted for acute chest pain on December 10, 1965 (10-12-65). The level of serum glutamic oxalacetic transaminase was less than 35 units and that of serum potassium was 4.5 mEq/L. The tall T waves persisted less than 24 hours.

**Table 2**

**Effect of Exercise on Tall T Waves**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of cases</th>
<th>T-wave amplitude</th>
<th>Positive Master's test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>10</td>
<td>Increase 4</td>
<td>Decrease 1</td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>15</td>
<td>Increase 2</td>
<td>Decrease 6</td>
</tr>
<tr>
<td>Hypertension</td>
<td>10</td>
<td>Increase 2</td>
<td>Decrease 4</td>
</tr>
<tr>
<td>Neurocirculatory asthenia</td>
<td>8</td>
<td>Increase 2</td>
<td>Decrease 3</td>
</tr>
<tr>
<td>Aortic incompetence</td>
<td>2</td>
<td>Nil 1</td>
<td>1</td>
</tr>
</tbody>
</table>

*Master's two-step exercise test was performed in all cases.*
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The effect of exercise on the electrocardiogram of a
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were less than 8 g/100 ml. Thus, the levels of serum potassium and hemoglobin were not responsible for the increased voltage of the T wave in cases other than those with anemia or hyperkalemia.

Effect of Exercise (Table 2; Fig. 3)
The height of the T wave increased in four normal persons and six persons with abnormal conditions and decreased in one normal and 14 abnormal persons after exercise. There were no changes in five normals and 15 ab normals. It is interesting to note that in seven cases of angina pectoris the T wave showed no changes in magnitude although Master's two-step exercise test was considered positive in every case. Transient changes in the magnitude of the T waves which occurred on exercise in the remaining cases of angina may represent transient changes in the electrolyte concentrations and may bear no relation to myocardial ischemia. The associated changes in the S-T segment make this test of diagnostic importance.

Ventricular Gradient
The average ventricular gradients in the horizontal plane in different conditions are given in table 3.
The T wave measures the changes in potential during repolarization, but all the forces

Table 3
Average Ventricular Gradient in the Horizontal Plane in Different Conditions

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Average ventricular gradient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>+23°</td>
</tr>
<tr>
<td>Ischemic heart disease:</td>
<td></td>
</tr>
<tr>
<td>Angina pectoris</td>
<td>+40°</td>
</tr>
<tr>
<td>Acute anterior myocardial infarction</td>
<td>+38°</td>
</tr>
<tr>
<td>Acute posterior myocardial infarction</td>
<td>+56°</td>
</tr>
<tr>
<td>Left ventricular hypertrophy:</td>
<td></td>
</tr>
<tr>
<td>Aortic incompetence or stenosis</td>
<td>+15.5°</td>
</tr>
<tr>
<td>Hypertension</td>
<td>+19°</td>
</tr>
<tr>
<td>Uremia</td>
<td>- 1.3°</td>
</tr>
<tr>
<td>Neurocirculatory asthenia</td>
<td>+17°</td>
</tr>
</tbody>
</table>

hypertensive man, aged 50 years. Tall T waves have become taller and narrower on exercise. The ST segment shows no change.
that contribute to its formation are not completely understood. If the ventricular myocardium were absolutely homogeneous with respect to its metabolic processes and the generation of electromotive forces and if the processes of depolarization and repolarization were equal and opposite, the algebraic sum of the areas of the QRS complex and of the T wave would be zero. However, in the human heart, repolarization is slower than depolarization. Moreover, there are differences in the duration of depolarization in various parts of the ventricles so that repolarization occurs in some parts while depolarization is not yet completed in other parts. These net differences in ventricular electrical activity are termed the "ventricular gradient" (fig. 4). Determination of the ventricular gradient reveals whether T wave alterations in the electrocardiogram are primary (gradient abnormal) or secondary (gradient normal).

**Figure 4**

The average ventricular gradient (V̂G) is markedly deviated in uremia and in ischemic heart disease indicating primary T-wave changes. A.I./A.S. = aortic incompetence or aortic stenosis; N.C.A. = neurocirculatory asthenia; H.H.D. = hypertensive heart disease; Ant. Myo. Inf. = anterior myocardial infarction; A.P. = Angina pectoris; and Post. Myo. Inf. = posterior myocardial infarction.

**Figure 5**

The features of tall T waves in a normal individual. The ventricular gradient and QTc are normal. The heart was normal clinically, and the x-rays of the chest were normal.
TALL UPRIGHT T WAVES

were associated with characteristic elevation of ST segment indicating that the forces of repolarization occurred early and were increased in magnitude. This may be considered a variant of the normal phenomena.

**Left Ventricular Hypertrophy**

In our series, minor variations (less than 8°) in the average ventricular gradient as compared to the average normal value also occurred in cases of left ventricular hypertrophy due to hypertension, aortic valve disease, and congenital heart disease.

The distribution of tall T waves in precordial leads in these cases was a matter of interest. In cases of aortic stenosis and hypertension, conditions which are associated with systolic overload of the left ventricle, the T waves were tall from V₁ to V₄. The T wave in V₆ was either normal, flat, or even inverted. Some authors have suggested that in aortic stenosis the tall T waves in V₁ to V₄ may be the result of myocardial ischemia.³ Our results indicate that the explanation may not be so simple, as Master’s two-step exercise test performed in 10 cases of hypertension, was negative. Similar inconclusive results after exercise have been reported by Datey and Misra.⁸

In cases of aortic incompetence, patent ductus arteriosus, and ventricular septal defect, conditions which are associated with diastolic overload of the left ventricle, the T waves were tall from V₁ to V₆. In two cases of aortic incompetence, the voltage of T in V₆ was more than 10 mm. In cases with combined systolic and diastolic overload such as aortic stenosis with incompetence, the T waves were tall from V₂ to V₆ (fig. 6).

**Figure 6**

*The spread of tall T waves in left ventricular hypertrophy caused by systolic and diastolic overload of the left ventricle. A.S. = aortic stenosis; A.I. = aortic incompetence; M.S. = mitral stenosis; Syst. = systolic; and Diast. = diastolic.*

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Uremia with Hyperkalemia

In uremia with hyperkalemia, the ventricular gradient was markedly altered, the average value being \(-1.3^\circ\). In all cases the T waves were narrow and peaked. Thus in a given electrocardiogram, narrow, tented, tall T waves, associated with marked deviation of the ventricular gradient from normal, point to a diagnosis of uremia with hyperkalemia.

Ischemic Heart Disease

The ventricular gradient was significantly altered in each and every case of ischemic heart disease. The average value in cases of angina pectoris was \(+40^\circ\). It ranged from \(+5^\circ\) to \(+50^\circ\) in anterior myocardial infarction and from \(-22^\circ\) to \(+74^\circ\) in posterior myocardial infarction, the average values being \(+38^\circ\) and \(+56^\circ\), respectively. In contrast to uremia with hyperkalemia, the T waves in ischemic heart disease were wide, and hence a differentiation could be made.

The classical electrocardiographic changes commonly encountered in the angina of effort are ST-segment depressions and T-wave inversions. Notched T waves and tall upright T waves are uncommon in angina pectoris but have been reported by some authors.\(^3,5,7,9\)

We encountered tall T waves in 13 cases of angina of effort without any previous history of myocardial infarction. Sodi-Pallares and Calder\(^7\) have stated the opinion that as a result of subepicardial ischemia the recovery process follows the same path as the activation process and produces T-wave inversion commonly seen in angina pectoris. On the other hand in cases of subendocardial ischemia, the T waves become upright and show a great increase in the voltage. Thus, it appears that the pattern of T-wave changes in angina depends on the presence of subepicardial or subendocardial ischemia.

In myocardial infarction, destruction of some of the myocardial fibers eliminates a number of vectors which usually participate in the production of a normal T vector. The normal equilibrium of repolarization is thus disturbed and vectors which are normally not manifest now appear resulting in primary T-wave changes.\(^10\) This would result in marked changes in the ventricular gradient as observed in our cases (figs. 7 and 8).

When the diaphragmatic and posterior portions of the left ventricle are destroyed by myocardial infarction, the predominant T waves are inverted or flat (fig. 7).

Figure 7

Persistence of tall T waves in a case of posterior myocardial infarction. The patient, a 48-year-old man, was admitted on November 23, 1963, for acute chest pain.
Figure 8

The sequence of T-wave changes in a man aged 55 years. In April 1965 he complained of angina of effort; on November 1, 1965, he had an acute attack of chest pain. Note the evolution of the tall T waves in this case of anterior myocardial infarction.

Vectors are initially directed anteriorly and slightly superiorly. The RS-T depression would thus be demonstrated only in posterior and inferior leads which are not routinely recorded in the electrocardiogram. Now leads V₂ to V₅ are the mirror image to these theoretical leads and the tall T waves recorded therein are reciprocal reflections of T-wave inversions, and according to Wachtel and Teich⁴ represent ischemia of posterior and diaphragmatic regions.

We agree that this mechanism will satisfactorily explain the occurrence of tall T waves in leads V₂ to V₅ in those cases of posterior and diaphragmatic infarction which show evolution of changes with time (fig. 8). However, in all our cases of posterior infarction the tall T waves in leads V₂ to V₅ have persisted for many months and in some cases for more than 4 years without showing changes of evolution. We suggest that the tall T waves in these cases may represent persistent ischemia.

Experimental Myocardial Infarction

In experimental anterior wall myocardial infarction produced by ligaturing the interventricular artery near its origin from the left coronary artery in a closed chest animal, the first change recorded was an increase in voltage and width of the T wave from V₁ to V₅ within 2 minutes of ligation. These changes persisted for 8 hours and T-wave inversions occurred after 24 hours. The sequence of changes encountered in animal experiments resembled those obtained in clinical cases of acute myocardial infarction in our series. In
four cases of acute anterior myocardial infarction, electrocardiograms recorded within the first 4 hours of the attack had tall T waves in right precordial leads. These changes occurred before any other electrocardiographic changes of myocardial infarction. However, in all cases, by the next day the T waves lost their height and became inverted.

Concluding Comment

It has been suggested that initial reactive hyperemia which occurs in the damaged area of the myocardium may alter the course of repolarization. However, this does not explain persistent tall T waves in angina pectoris and we believe it is more likely that tall T waves in precordial leads are the result of subendocardial ischemia. In cases of anterior myocardial infarction, the tall T wave persists for a short time only, as the changes of evolution cause it to regress and become inverted. Hence it will be recorded only in those cases in which the electrocardiogram is taken within a few hours of the onset of chest pain. On the other hand the tall T waves in angina pectoris and posterior myocardial infarction denote persistent ischemia and may persist for a long time.

References
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