THE Q-T INTERVAL IN ACUTE RHEUMATIC CARDITIS

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In the initial stages of rheumatic fever, carditis can usually be diagnosed with ease, since the significant murmurs are almost always to be heard. In the presence of polyarthritis, pyrexia, and raised sedimentation rate, it can be safely assumed that this carditis is active. It may, however, be extremely difficult to determine how long such activity persists: the temperature and pulse rate may be normal, the electrocardiogram may show no gross changes, and such laboratory tests as are performed may show no abnormality, and yet, in some of these cases, it seems probable that carditis is still active. This study is an attempt to use electrocardiographic data to supply criteria of such sub-clinical activity.

Donders (1868) originally studied the duration of systole on the radial plethysmograph. Two years later Garrod (1870), using the same technique, evolved a formula for correcting the first, or systolic portion of the plethysmograph, for heart rate, and suggested that alterations in the duration of systole might be useful in the diagnosis of heart disease.

Berliner in 1931 first noted that prolongation of the Q-T interval of the electrocardiogram occurred in rheumatic valvular disease. Drawe et al. (1937) measured the Q-T interval in 100 rheumatic and 100 normal children. They showed that 25 per cent of the former and 4 per cent of the latter were above the upper limit of normal as judged by Ashman and Hull's (1937) criteria. They did not state, however, whether acute carditis was present when this measurement was taken. Taran and Szilagyi (1947) found that the duration of electrical systole, both absolute and relative to diastole, was significantly lengthened in all cases of acute rheumatic carditis. They further stated that this prolongation was not a function of the cardiac rate, but rather of the severity of the disease; that this prolongation preceded all other laboratory criteria of rheumatic activity, and that it did not return to normal until long after all other diagnostic signs had reverted to normal.

In view of the importance of this statement an investigation was undertaken to see whether prolongation of the Q-T interval was a reliable index of active carditis and whether it could prove of prognostic significance.

MATERIAL

In all, 134 cases were studied. The patients were under treatment in the special unit for juvenile rheumatism at the Canadian Red Cross Memorial Hospital, Taplow; no special selection of cases was made. Some were local patients, admitted in the initial stages of the rheumatic attack, but the majority were transferred from other institutions, provincial and metropolitan, throughout Great Britain, where they had already been under treatment for varying periods of time. The majority of the patients were children (see Table I).

**TABLE I**

<table>
<thead>
<tr>
<th>Age Distribution</th>
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<tbody>
<tr>
<td>0-5 years</td>
<td>6 cases</td>
</tr>
<tr>
<td>6-10 years</td>
<td>56 cases</td>
</tr>
<tr>
<td>11-15 years</td>
<td>50 cases</td>
</tr>
<tr>
<td>16-20 years</td>
<td>13 cases</td>
</tr>
<tr>
<td>Over 20 years</td>
<td>9 cases</td>
</tr>
</tbody>
</table>

On admission to hospital the cases were divided clinically into one of three main groups, those presenting evidence of active carditis, those presenting evidence of inactive carditis, and those cases in which no clinical carditis was detectable. Carditis was diagnosed clinically by the presence of one or more of the following: a diastolic murmur, cardiac enlargement, the presence of a pericardial friction rub, tachycardia out of proportion to elevation of temperature, and grosser electrocardiographic changes such as prolongation of P-R interval. Activity was recognized by pyrexia, tachycardia, and raised sedimentation rate.

The group of cases of active carditis was further subdivided into those patients who showed a steady
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unreadable recovery and those who showed evidence of prolonged rheumatic activity.

TABLE II
CLASSIFICATION ACCORDING TO DEGREE OF CARDITIS OR ABSENCE OF CARDITIS

1. Carditis
   (i) Active carditis
      (a) Uninterrupted recovery . . . . 55 cases
      (b) Prolonged activity . . . . 45 cases
      (c) Recovery . . . . 100 cases
   (ii) Inactive carditis . . . . 12 cases
2. No carditis . . . . 22 cases

METHODS
All electrocardiograms were taken on an American Cambridge continuous film electrocardiograph. The time marker was accurately checked against an oscillator of known frequency. All recordings were taken with the patient semi-recumbent at an angle of thirty degrees to the horizontal, Lombard and Cope (1919) having shown that systole varied with posture, being longer in the standing position.

The absolute Q–T interval varies slightly from complex to complex, as does the cycle length, but Katz (1921) showed that, while cycle length and the length of systole may vary phasically, these variations are not synchronous nor of like degree. To obviate distortion of cycle length by sinus arrhythmia, which in some cases was marked, the heart rate was calculated by counting the complexes over the entire length of tracing taken, covering at least two-thirds of a minute and usually one minute. The average cycle length was then calculated from the heart rate.

The Q–T interval was measured from the beginning of the Q wave until the end of the T wave in the standard lead in which the T wave was highest. This was usually lead two. At least six complexes were measured with calipers under a magnifying lens and the average length of the Q–T interval was taken.

The absolute duration of Q–T depends upon the heart rate, and thus measurements must be corrected for heart rate before they can be compared. Numerous formulae exist for this correction (Lombard and Cope, 1919; Fridericia, 1920; Ashman and Hull, 1937; Schlamowitz, 1946). All these formulae have been criticized, but the square root formula devised by Bazett (1920) is generally agreed to be one of the most reliable, and, because of its simplicity, has been used in this study. Taran and Szilagyi (1947) used Bazett’s formula but expressed it as

\[ K = \frac{Q-T}{\sqrt{C}} \]  

where \( C \) = cycle length. They called \( K \) the corrected Q–T or Q–Tc for short. This method and nomenclature have been adopted here.

Various values for the upper limit of Q–Tc have been laid down by different workers, Bazett (1920), Hegelin and Holzman (1937), Ashman and Hull (1937), and Taran and Szilagyi (1947). For this study electrocardiograms were taken on a short series of normal subjects; the upper limit of normal for Q–Tc was found to conform to Ashman and Hull’s criteria which, accordingly, were adopted. Therefore the upper limit of the normal for Q–Tc has been taken as 0.422 second for men and children and 0.432 second for women.

RESULTS

(1) Q–Tc and heart rate. It is imperative that any formula which is employed to correct Q–T for cycle length must not be distorted by extremes of heart rate. Bazett’s formula was tested as shown in Fig. 1, in which 426 measurements of Q–Tc from

![HEART RATE](http://heart.bmj.com/)  

FIG. 1.—Q–Tc correlated with heart rate.

80 patients, both with and without carditis, are plotted against the heart rate.

The longest values for Q–Tc occurred with neither the slowest nor the most rapid heart rates, but did in fact appear at heart rates between 80 and 120. In the presence of active carditis it is natural that this degree of tachycardia should obtain.
(2) Q-Tc correlated with carditis. Table III shows that the Q-Tc was prolonged in ninety of a hundred cases of active carditis. In the remaining ten cases the Q-Tc was within normal limits. Two of these patients had suffered previous pericarditis which was shown by Tung (1941) to shorten Q-Tc. In the remaining eight cases no factor was present that is known to shorten the Q-Tc. In this series the upper limit of normal was taken as 0.422 second for children as opposed to 0.405 second used by Taran and Szilagyi (1947). This may explain why only 90 per cent of these cases of active carditis showed a prolonged Q-Tc against 100 per cent in Taran and Szilagyi's series. Out of twelve cases with rheumatic heart disease, which were considered inactive on admission, five showed a prolonged Q-Tc. There was no other evidence of active carditis, but data given later in this paper tend to prove that this did exist. Of twenty-two cases considered to show no evidence of a heart lesion clinically, eleven cases showed a prolonged Q-Tc. Four of these cases were shown to have suffered carditis by the subsequent appearance of significant murmurs. It therefore seems probable that some of the remaining seven patients in this group suffered minimal cardiac damage, unrevealed by any of the criteria upon which a clinical diagnosis of carditis was made.

(3) Q-Tc variation during the course of rheumatic fever. The behaviour of Q-Tc was studied, both in patients making a rapid recovery from the rheumatic attack (Table II, Group (a)) and those showing prolonged rheumatic activity (Group (b)). The behaviour of Q-Tc in six cases belonging to Group (a) is shown in Fig. 2 and demonstrates the return of Q-Tc to normal with recovery.

Fig. 3 shows the behaviour of a typical Group (a)
case. This patient was admitted in the initial stages of her third attack of rheumatic fever. Mitral stenosis, due to previous rheumatic carditis, was present as witnessed by a presystolic murmur. On admission the sedimentation rate was raised and the Q-Tc was grossly prolonged, which was taken as evidence of acute carditis in this attack. With recovery her Q-Tc reverted to normal.

Fig. 4 and 5 demonstrate the behaviour of Q-Tc in two patients of Group (b) showing evidence of prolonged rheumatic activity, or what may be called chronic rheumatic carditis. Fig. 4 shows that although the sedimentation rate came down to normal occasionally, the Q-Tc was prolonged for the whole of the 240 days covered by the graph. Prolongation of Q-Tc may occur in chronic rheumatic heart disease without active carditis. This will be discussed later.

Fig. 5 illustrates another case in Group (b), in this instance complicated by congestive failure. Two points should be noted: the steady fall in sedimentation rate with the onset of congestive failure; and the rapid shortening of Q-Tc on two occasions when digitalis was exhibited. This effect was noted by Cheer and Dieuiade (1931). This latter point is also illustrated by Fig. 6 which shows the shortening of Q-Tc in two normal patients when digitalis was exhibited. In neither of these patients was the direction of the T wave seen to change in the electrocardiograms subsequent to the administration of digitalis.

Fig. 7 illustrates the effect on Q-Tc of an exacerbation of rheumatic carditis. When admitted, this patient was judged to be quiescent, following a second attack of rheumatic fever. The antistreptolysin titre was 150 units, the sedimentation rate was normal, and the Q-Tc was not prolonged. Established heart disease was present and rheumatic nodules were noted. He then developed scarlet fever; the Q-Tc immediately rose and at the same time the P-R interval lengthened from 0:16 to 0:27 sec. The antistreptolysin titre rose to 400 and later to 833 units. The sedimentation rate also rose and he suffered a severe exacerbation of carditis. The P-R interval first came back to normal; this was followed by a return to normal of the sedimentation rate. The Q-Tc, however, remained prolonged. In the present series the P-R interval was always found to be normal when the Q-Tc was within normal limits.

(4) Correlation of Q-Tc and sedimentation rate. The sedimentation rate still remains one of the most

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**Fig. 4.**—Behaviour of Q-Tc in chronic rheumatic carditis: Group 1 (b). A.S.T. = Antistreptolysin “O” titre.
important signs of rheumatic activity; accordingly, the duration of Q-Tc was compared with the sedimentation rate in patients with, and without, carditis. The results are illustrated in Fig. 8.

In Fig. 8, 293 readings of Q-Tc from sixty patients with active carditis are plotted against the sedimentation rate on a semi-logarithmic scale, and it will be seen that, in general, the length of Q-Tc varies directly as the sedimentation rate. It will be noted, however, that some patients with active carditis show a short Q-Tc in spite of a high sedimentation rate; some of these patients were suffering from pericarditis, and others were receiving digitalis, the effect of this drug on systole being well known. It will also be observed that the Q-Tc was frequently prolonged when the sedimentation rate was within normal limits. Such observations were made towards the end of the patients' stay in hospital, and illustrate the persistence of a long Q-Tc at a time when other evidence of activity, such as the sedimentation rate, had subsided.

Fig. 9 shows 36 readings of 11 patients with
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acute rheumatic fever, but no clinical evidence of carditis; the Q-Tc is below the upper limit of normal in every case, and bears no relationship to sedimentation rate.

(5) Q-Tc and relapses. These findings confirm Taran's claim that prolongation of Q-Tc is a reliable index of active carditis. It follows therefore that increase in physical activity in the presence of a prolonged Q-Tc might theoretically be expected to precipitate a relapse. The relapses that occurred were analysed accordingly. Fig. 10 illustrates this point.

This child was admitted in the middle of a severe attack of rheumatic carditis. The Q-Tc was grossly prolonged, the sedimentation rate was raised, erythema marginatum was noted, and a crop of nodules appeared. After 90 days in hospital the Q-Tc and the sedimentation rate were within normal limits. At this point she suffered another β-hemolytic streptococcal infection, which was followed by a severe exacerbation of carditis; the Q-Tc rose and a slower rise in sedimentation rate occurred. Rash and nodules re-appeared. Two hundred and sixty days after admission the sedimentation rate, temperature, and pulse rate had returned to normal. She was then considered clinically inactive and was allowed up, in spite of a prolonged Q-Tc. An immediate relapse followed, accompanied by a further rise in Q-Tc and sedimentation.

Fig. 8.—Correlation of Q-Tc with ESR in patients with acute rheumatic fever without carditis.

Fig. 9.—Correlation of Q-Tc with ESR in patients with active carditis. Compare Fig. 9.
rate, necessitating return to bed. The anti-streptolysin titre of 200 throughout this episode was very strong evidence against an occult streptococcal re-infection and subsequent rheumatic recurrence.

The criteria for judging an exacerbation of rheumatic carditis to be a relapse, rather than a recurrence, must be specified and adhered to rigidly. In much of the present series essential data were not obtained and definite conclusions could not be drawn. However, of twenty patients who apparently relapsed, fifteen were noted to have a long Q–Tc at the time that physical activity was increased. There was no other evidence of active carditis in any of these patients. This and other material is now under careful analysis to find out whether prolongation of Q–Tc at the time that physical activity is increased is a significant factor in causing relapses.

**Discussion**

These results prove that prolongation of Q–Tc occurs in active rheumatic carditis, but throw no light on the mechanism of its production. It is extremely doubtful if alterations in the diastolic filling pressure, or in the diastolic volume of the cardiac chambers, can be responsible, as in the majority of cases the venous pressure is not raised clinically. Prolongation of systole due to biochemical causes may be similarly discounted. In the patients who make an uninterrupted recovery from an attack of rheumatic carditis, it would appear that this prolongation depends upon actual involvement of the myocardium by the rheumatic process.

In chronic cases where the heart suffers prolonged myocardial and valvular damage, other factors are involved. In this group, actual muscular hypertrophy of the heart, consequent on valvular
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Further work on this aspect of the problem may be of great help in assessing the degree of physical activity that may safely be permitted in the individual case.

SUMMARY AND CONCLUSIONS

Prolongation of Q–Tc is a valuable index of active carditis in rheumatic fever, and active carditis may be detected by the presence of a prolonged Q–Tc long after all other clinical and laboratory criteria of activity have gone.

Prolongation of Q–Tc may be the only evidence of cardiac involvement in acute rheumatic fever.

In chronic rheumatic carditis, prolongation of Q–Tc may be due to causes other than active carditis.

Measurement of Q–Tc may be of help in assessing the degree of physical activity that may be allowed in the individual case.

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REFERENCES


Tung, C. L. (1941). Ibid., 22, 35.