The Q-T Interval in Rheumatic Fever

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In 143 rheumatic children the Q-T interval was found to be normal in those with quiescent rheumatic heart disease. In 29 with fatal pancarditis the average Q-T was above the average normal, though not beyond the upper limit of normal. Although parallel changes in the Q-T interval and the clinical manifestations of rheumatic fever occurred in about 66 to 75 per cent of cases, there were significant number of instances where changes in the Q-T and in the clinical course of the disease were in opposite directions.

The difficulties encountered in the diagnosis of rheumatic fever are well known. Frequently the history offers only questionable evidence of arthritis, chorea, or cardiac involvement, and physical examination may fail to demonstrate signs of active rheumatic fever. If the presence of carditis can be established, the diagnosis is immediately placed on a more certain foundation, and, at the same time, one is provided with information of some prognostic value. In recent years, therefore, almost every feature of the electrocardiogram has been explored for evidence of cardiac involvement in rheumatic fever. The demonstration of delayed A-V conduction, S-T and T-wave changes or arrhythmia has often been of value in this connection. More recently, attention has been directed toward the Q-T interval in rheumatic fever.

The duration of electrical systole as measured by the Q-T interval is known to be prolonged in a variety of pathologic states. In certain instances a definite lengthening of the Q-T interval has been seen in rheumatic carditis. In an analysis by Drawe and associates of the electrocardiograms of 100 children with rheumatic heart disease, of whom 25 had acute rheumatic carditis, prolongation of the Q-T interval was demonstrated in 25. No statement was made as to whether the patients with prolongation of the Q-T interval were the same as those with evidence of carditis. In a control group of 100 children, only 4 exceeded the upper limit of normal as defined by Ashman and Hull.

Recently, further claims for the usefulness of the Q-T interval in separating quiescent rheumatic patients from those with carditis have been made by Taran. He has studied a group of 100 rheumatic children, in half of whom the disease was inactive. The remaining patients presented evidence of active rheumatic fever and carditis. The duration of the Q-T interval normally is dependent upon the heart rate. Hence, Taran made use of Bazett's observation that the relation of the Q-T to the R-R interval can be expressed by the formula,

$$\frac{Q-T}{\sqrt{R-R}} = K.$$

For purposes of comparing the Q-T intervals of various individuals, normal and abnormal, at different heart rates, Taran has redefined the ratio of Q-T:R-R as the corrected Q-T interval, namely, Q-Tc. Thus,

$$Q-T_c = \frac{Q-T}{\sqrt{R-R}}.$$

There was a distinct separation in Taran's cases of the inactive from the active group. When the Q-Tc of each patient was recorded as a point on a graph in which the upper limit of normal for Q-Tc was indicated by a horizontal line, all of the points for the patients in the active group fell above this line, whereas all except one of the points for the inactive group fell below the line. Taran has chosen the figure 0.405 as the upper limit of normal for Q-Tc. For the source of this figure, he cites Ashman and Hull. The latter authors, however, have used this figure not in the Bazett formula but in Ashman's formula,

$$Q-T = K \log(10(R-R) + 1).$$
That the value of $K$ or Q-Tc is not interchangeable in the two formulas has been previously demonstrated.\textsuperscript{13}

Furthermore, Taran points out that the average duration of Q-T for his quiescent cases is very close to the average Q-T of 0.325 second found by Hafkesbring and associates\textsuperscript{14} in normal children. The latter authors also found the average corresponding heart rate for their normal children to be 92.7, a figure equivalent to an R-R interval of 0.647 second. If one applies the average Q-T of 0.325 and the average R-R of 0.647 to Taran's formula, the average Q-Tc is found to be 0.404. Hence, it seems improbable that the upper limit of normal for Q-Tc would be only 0.405.

**Clinical Material and Method**

The present study was undertaken in order to determine the usefulness of the measurement of the Q-T interval in rheumatic fever. It has been recommended by Ashman\textsuperscript{15} that along with any observation of the Q-T in rheumatic children a comparable group of normals be studied. The normals should afford controls of suitable age and environment. Our normal group has been previously reported.\textsuperscript{13}

The rheumatic fever subjects of this study were ward patients at the House of the Good Samaritan. They include 63 boys and 80 girls in the age group of 7 to 14 years. The patients fell into two main groups:

(A) Patients with quiescent rheumatic heart disease. This group is composed of 102 patients who at the time of discharge from the hospital showed definite evidence of rheumatic heart disease with or without appreciable cardiac enlargement but with no clinical or laboratory evidence of rheumatic activity.

(B) Patients with active rheumatic fever. This group includes 29 patients with fulminating pancarditis resulting in death and 29 others with varying lesser degrees of active rheumatic disease. Among the latter were 17 patients also studied during a quiescent stage of their illness, and who, therefore, were also included in Group A above.

Only those electrocardiograms which showed sharply defined T waves were used. Usually measurements were made on standard Lead II, using average figures for Q-T and R-R intervals from a sequence of 8 to 12 cardiac cycles to minimize the

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![Fig. 1.—Q-Tc distribution curves of fatal and quiescent rheumatic patients compared with the K distribution curve for normal children.](http://circ.ahajournals.org/)

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\[ \text{BAZETT } K \ (\text{or } QTc) = \frac{Q-T}{VR-R} \]

**NORMAL CHILDREN**

**PATIENTS WITH PANCAIRDITIS**

**QUIESCENT RHEUMATIC HEART DISEASE**

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*Fig. 1.—Q-Tc distribution curves of fatal and quiescent rheumatic patients compared with the K distribution curve for normal children.*
effects of sinus arrhythmia. A special reflectoscope was used to magnify the tracings and facilitate accurate measurement.

**Results**

**A. Patients with Quiescent Rheumatic Heart Disease**

This group is composed of 102 children who at the time of their discharge from the hospital presented no clinical or laboratory evidence of rheumatic activity, but all of whom, on physical examination, showed definite signs of rheumatic heart disease. Repeated electrocardiograms were taken during their hospitalization, which varied from one to fourteen months, and the final tracing, taken just prior to their discharge, was used in this study. The Q-T, for this group varied from 0.353 to 0.442 and averaged 0.395. The latter is slightly below the figure 0.404, the average value for our control group. Figure 1 shows the distribution of the values for Q-T of these quiescent rheumatic individuals superimposed on the curve of normal children of all ages. The shape of the curves is similar. The actual values for Q-T as measured—not corrected by any formula—are shown by the solid dots in figure 2. The individual measurements fall well within normal limits.

**B. Patients with Active Rheumatic Fever**

For the purposes of analysis the patients composing this group have been divided into three subgroups; namely, a fatal group, a group that was observed during the subsidence of rheumatic activity, and a group that was studied during rheumatic recurrence. There is, however, some overlapping between the fatal and recurrent subgroups in that some of the patients who developed fatal recurrence have been included in both subgroups.

1. **Patients with Fatal Rheumatic Heart Dis-**
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A group of 29 fatal cases of rheumatic fever was reviewed. In 21 of these the presence of acute pancarditis was demonstrated at autopsy. In the other 8 the fulminating clinical course and physical findings gave evidence of a similar process. Patients receiving digitalis at the time of the electrocardiographic study or during the previous three weeks were eliminated owing to the shortening effect of this drug on the Q-T interval. Five patients receiving small doses of xanthines or salicylates were not excluded. The tracings used in this analysis were taken during the final overwhelming phase of rheumatic infection. The individual Q-T measurements are shown by the open circles in figure 2. It is evident that the patients with pancarditis in general had faster heart rates than those who were quiescent. The Q-T intervals of the patients with pancarditis, although somewhat longer on the average than those of the quiescent group, fell nevertheless case.

Fig. 3.—Q-Tc of 20 patients at the time of hospital admission (A) and at discharge from the hospital (D), after complete disappearance of rheumatic activity.

Fig. 4. A.—Relation of changes in corrected Q-T interval (Q-Tc) to the clinical course of two patients who developed fatal recurrence of rheumatic fever following streptococcal pharyngitis (See fig. 4, B)
within normal limits. An analysis of the same data in terms of Q-Tc is shown in figure 1. Here the distribution of the Q-Tc of the pancarditis group is superimposed on the distribution curve of normal children of all ages. There is considerable overlapping of the two curves, but it is apparent that the fatal cases tend to have a somewhat greater Q-Tc than the normals. The average Q-Tc of 39 electrocardiograms of this fatal group is 0.419, which is statistically different from our average normal of 0.404.13

2. Patients with Subsiding Rheumatic Fever. The patients with active rheumatic fever included a group of 20 individuals who improved appreciably while under observation. These patients presented definite evidence of active rheumatic fever1 on admission to the hospital, but on discharge, after an average of six months of hospitalization, they showed no clinical signs of rheumatic activity, and their sedimentation rate, hematocrit, and P-R interval were well within normal limits. At the time of discharge from the hospital, 10 of these patients still showed signs of rheumatic heart disease, and 10 did not.

The Q-Tc of the electrocardiograms taken at the time of admission, when there was unequivocal evidence of active rheumatic fever, were compared with those of the tracings taken just prior to discharge when all clinical and labora-

![Fig. 4, B.—The dotted area represents the time when patient was symptom-free and outside the hospital, and no electrocardiographic data available then.](image-url)
A group of 15 patients who suffered an exacerbation of rheumatic fever while under observation was also studied. In 6 of these patients, recurrent rheumatic fever resulted in death and they were therefore also included in the study of the fatal group. In the remaining 9, clinical evidence of moderately severe rheumatic activity appeared. Most of these patients were convalescent on the hospital wards during a streptococcal epidemic in 1939. In them it was possible to observe a recurrence of rheumatic fever two or three weeks following streptococcal pharyngitis or tonsillitis. The Q-T interval was measured prior to the streptococcal infection, during it, and, two or three weeks later, at the height of the rheumatic process. An attempt was made to grade the activity of rheumatic fever in these cases in order to determine whether there was any correlation between it and the duration of the Q-T interval. Activity was graded from I to IV as follows: (I) mild (fever, arthritis, chorea, nosebleeds, rashes, nodules, elevated sedimentation rate); (II) moderately severe (changing murmurs, prolonged P-R interval, enlarging heart); (III) severe (congestive failure, evidence of pancarditis); (IV) fatal.

The clinical course of every patient was plotted according to the above criteria, indicating, roughly, his condition at the time of the electrocardiographic measurements, which were also plotted. In 3 cases out of the 15, a fairly accurate correlation could be observed between the patient’s course and the fluctuations in his Q-T. An example of this type is shown in figure 4, A. Here, despite the fact that the patient was already suffering from the severe recurrence which led to his death, the Q-T did not reach an abnormal level at any time. In 8 other cases the correlation between the Q-T, and the patient’s clinical condition was not so close, but there was a tendency for the two to move in the same direction. As the patient’s condition deteriorated, a slight lengthening of the Q-T, occurred, and vice versa.

In 4 of the cases of recurrent rheumatic fever, on the other hand, the duration of the Q-T, moved in a direction opposite to the patient’s clinical course. An example of this type is seen in figure 4, B, where a fulminating carditis was accompanied by a shortening of the Q-T.

**Discussion**

Our results indicate that the Q-T interval in individuals with quiescent rheumatic heart disease is similar to that of normal individuals. In this respect, our findings agree with those of Taran and Pokress and Goldberger.

An analysis of the Q-T interval in rheumatic fever by the latter authors has been reported since our own studies were completed. Their results indicate that prolongation of the Q-T interval may occur in active rheumatic fever but does not invariably do so. They did not observe the very close relationship between the length of the Q-T interval and the presence of rheumatic activity that had been previously found by Taran. From the data of Pokress and Goldberger, the Q-T of 50 active rheumatic patients can be determined. The average Q-T, for this group is 0.409, a figure which is similar to our own average of 0.412.

Pokress and Goldberger found only 14 out of 50 patients with active rheumatic fever to have Q-T intervals which exceeded the normal upper limit accepted by these authors. If our criteria for normal subjects are used, the number of their active rheumatic patients with prolongation of the Q-T interval is reduced to 10, since the upper limit for our normal controls is slightly higher than for theirs.

Our observations show a tendency for active cases to have a longer Q-T interval, on the whole, than normal subjects and quiescent cases. Fluctuations in the clinical conditions of the patient were often associated with alterations in the Q-T interval. Even in severe carditis, however, the Q-T interval did not exceed the upper limit of normal in a single case. Our studies, therefore, do not support the claims of Taran and Szilagyi for the value of the Q-T interval as an accurate index of rheumatic carditis.

**Summary and Conclusions**

The Q-T interval of 143 rheumatic children between the ages 7 and 14 years was measured.

One hundred and two patients with quiescent rheumatic heart disease had Q-T intervals similar to those of normal children.

Twenty-nine patients with fatal pancarditis had Q-T intervals within the normal range,
but their average Q-T interval was slightly longer than that of normal control subjects.

In a group of patients with active rheumatic fever, changes in the Q-T interval occurred parallel with changes in the clinical condition in about two-thirds to three-fourths of the cases, but the opposite was also noted in a significant proportion.

Duration of the Q-T interval may be determined in the study of rheumatic patients as a part of an over-all estimate of activity of the disease. Its usefulness is minimized by technical difficulties in measurement and by the infrequency with which it is abnormal.

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