The Q-T Interval in the Electrocardiogram of Children with Tuberculosis

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It is stated that prolongation of the Q-T interval is a reliable criterion of active carditis. A control investigation of a group of tuberculous children was undertaken with particular reference to this point. It was observed that prolongation of the Q-T interval is a rather frequent finding in childhood tuberculosis, but it is not an expression of the cardiac status of the patient. The value of the Q-T interval as an indicator of active carditis is therefore questioned.

It is a well known fact that many cases of active rheumatic heart disease do not show the known criteria for "activity," namely, elevated temperature, elevated sedimentation rate, anemia, rapid ventricular rate, or fluctuating electrocardiographic findings. Every effort to establish objective criteria to include all cases of rheumatic activity is justifiable. Extreme caution, however, should be exercised in the selection of these criteria, so that persons without rheumatic activity are not branded with the mark of disability. The recent awakening of interest in the value of the Q-T interval in patients with acute carditis commands a series of control studies. If prolongation of the electrical systole is shown to occur non-specifically, then one can hardly be justified in dignifying this phenomenon with the quality of a selective criterion.

For the purpose of checking the significance of the Q-T interval in cases of carditis a group of 59 children in the age group of 7 to 14 years was studied electrocardiographically and the findings correlated with the clinical status of each patient. None had clinical or x-ray evidence of heart disease with the exception of one child who, in addition to pulmonary tuberculosis, had rheumatic heart disease. All of the children had tuberculosis; some had active and some, inactive lesions. A number of the children with inactive cases had upper respiratory infections at the time the tracings were recorded. This was thought to account for the transient rise in temperature or sedimentation rate.

As the age group of the patients selected for the study was the same as that of the patients reported in the paper by Taran and Szilagyi,1 the same method of measuring the Q-T interval in the electrocardiogram was adopted in order to make our results comparable to theirs. In patients with sinus arrhythmia a sequence of several beats was averaged in the calculation of the Q-T time. At all times an average of the Q-T interval in the various leads was taken, although actually the longest Q-T interval in any of the leads is the correct one.

The Q-T, corrected in relation to the heart rate, was calculated according to Bazette's formula: \( QT_c = \frac{QT}{\sqrt{RR}} \), where QT is the Q-T interval in seconds, and RR is the cardiac cycle in seconds.2 At the suggestion of Dr. Walter Modell,3 the calculations were made with a slide rule as illustrated in figure 1. The measured QT is found on scale D. The slide is then moved to the point indicating the RR interval on scale B. The QTc is read on scale D, where the index I on C is in opposition with D. This calculation is rendered even more simple by the nomogram recently devised by Kissin, Schwarzschild, and Bakst.4 Difficulties were encountered in measuring the Q-T interval in some cases. The high T waves present a well-defined end point, but the low, diphasic or inverted T waves have an end point not easily distinguishable. It appeared generally that

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complexes with a high T wave had large Q-T intervals, and it thus naturally suggests itself that the length of the Q-T interval in complexes with low-amplitude T waves may not be the correct one, as a portion of this interval may be submerged in the isoelectric phase. In such cases the complexes with the clearly discernible end points were considered. The measurements were made with the aid of adequate illumination, a strong magnifying glass, and a pair of draftsman's steel calipers. The pulmonary and 3 in the extrapulmonary group. The abnormal readings were as follows: 0.408, 0.415, 0.427, 0.426, 0.406, 0.408, 0.406.

In the "inactive" group there were 35 cases. Of these, 12 children had abnormal Q-T intervals (34 per cent). The readings were as follows: 0.419, 0.410, 0.455, 0.405, 0.460, 0.419, 0.410, 0.428.

In the "active" group there was one child with associated rheumatic fever; the Q-T interval in this child was only 0.4.

**Fig. 1.**—The slide-rule and the method of calculation of QT-c. (See text.)

readings were made against the time lines in the very sections where the graph was measured.

**Results**

Of the 59 children studied, 24 had active tuberculosis: 14 children had pulmonary disease and 10 had extrapulmonary lesions. In this "active" group, Q-T intervals longer than 0.405 seconds, considered by Taran and Szilagyi as upper limit of normal, were observed in 7 children (29 per cent). Four children were in the

In the "inactive" group there was one child with a prolonged P-R interval but no confirming evidences of rheumatic fever. The Q-T interval in this child was 0.428.

The relationship of the QTc to temperature and sedimentation rate is indicated in table 1. It appears that in the group with abnormal QTc's, there was a predominance of patients with elevated sedimentation rates and elevated temperatures. However, the fact that in the group with normal QTc's the number of patients with elevated sedimentation rates with
or without elevated temperatures is about equal to the number of patients with normal temperatures and sedimentation rates, suggests the conclusion that temperature and the sedimentation rate are not important factors in determining the value of the Q-T interval. In 6 cases, records were secured under varying clinical circumstances. In 3 of these, previously normal QTc's became abnormal with a rise in sedimentation rate and/or temperature. In 2 cases, normal QTc's remained normal after sedimentation rate became elevated. In one case a normal QTc diminished in duration with a rise in the sedimentation rate.

As noted in table 1, 26 patients had normal temperatures and normal sedimentation rates at the time the tracings were secured; in 11 children, intercurrent upper respiratory infections were thought to be responsible for the

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slightly elevated temperature and equally slightly elevated sedimentation rate. One child had measles, and one had coexistent rheumatic heart disease.

As to the prognostic significance of the abnormally prolonged Q-T interval, the following is to be noted. Our study began in November 1947 and continued until the end of February 1948. By November 15, 1948, all children but one with prolonged Q-T intervals were discharged from the hospital in good condition (“arrested”). One was transferred to another institution while still showing activity of the primary complex.

**Comment**

If prolongation of the Q-T interval is an indication of myocardial disease, then 29 per cent of our patients with “active” cases and 34 per of those with “inactive” cases had myocarditis—presumably tuberculous in origin, or, differently stated, 35 per cent of the total group (20 out of 59) were afflicted with tuberculous myocarditis. This is quite an exaggerated figure in the light of the evidence in the literature on the subject. Admittedly, tuberculous myocarditis is much more commonly observed in children than in adults, but even in children the incidence is only about 3.9 percent.8 The type of involvement reported in children has been chiefly that of miliary tubercles in the myocardium, probably attributable to the fact that in the great majority there was present generalized miliary tuberculosis.5 Since none of the children studied have evidence of miliary tuberculosis, on clinical grounds at least the suspicion that some of the children had tuberculous myocarditis would hardly be justified. None of the tracings presented other than Q-T abnormalities to suggest myocardial damage; no conduction difficulties were seen in any but one child to suggest nodular infiltration of the myocardium. In a previous publication it was shown that a prolonged P-R interval in patients with pulmonary tuberculosis does not necessarily indicate myocardial damage.6 The interstitial myocarditis observed in tuberculosis is ascribed to nontuberculous pulmonary infection by Roberts and Lisa.7 None of our patients had clinical evidences of the associated pathologic process described by these authors.

It thus appears fairly certain that the Q-T abnormalities observed in our group are not expressions of tuberculous myocarditis. Just what is its significance is at present not known. Perhaps it is a function of a nonspecific extracardiac factor which participates in disease in general, an expression of disturbed biochemistry which is known to influence this particular segment of the electrocardiogram.8—11

The nondependence of the Q-T interval on the height of the sedimentation rate or temperature has already been commented on.

Could the Q-T interval be a sensitive indicator in rheumatic heart disease specifically and not any other type of carditis? The electrocardiogram was never thought to possess such selective properties.
SUMMARY AND CONCLUSION

Fifty-nine cases of pulmonary and extrapulmonary tuberculosis in children were studied with particular reference to the Q-T interval in the electrocardiogram. Thirty-five per cent of the patients of this group presented prolongation of the Q-T interval with no corroborative evidence of myocardial involvement. The frequency of the prolonged Q-T interval was greater in the group of patients with inactive tuberculosis than in the patients showing evidences of tubercular activity.

Doubt is expressed as to the diagnostic or prognostic significance of the prolonged Q-T interval. It is apparent from this study that in evaluating the myocardial state in tuberculosis the Q-T segment of the electrocardiogram does not contribute a reliable criterion. Furthermore, our observations apparently are not in accord with the idea that prolongation of the electrical systole is a specific finding in children with active rheumatic heart disease.

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Circulation. 1950;1:1184-1187
doi: 10.1161/01.CIR.1.5.1184
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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the World Wide Web at:
http://circ.ahajournals.org/content/1/5/1184

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