THE Q-T INTERVAL IN RHEUMATIC FEVER

BY

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The prolongation of the Q-T interval in the electrocardiogram of patients suffering from valvular disease or rheumatic carditis was noticed by Berliner (1931) and by Drawe, Hafkesbring, and Ashman (1937). These observations were not considered to be of value for the diagnosis of cardiac involvement during or after an attack of acute rheumatic fever until Taran and Szilágyi (1947), studying a series of 50 cases, claimed that the duration of electrical systole was lengthened in all children whose electrocardiograms had been taken during an attack of acute rheumatic carditis.

Their report revived interest in the value of the measurement of the Q-T interval, and further papers have since been published: Abrahams (1949) found a prolonged Q-T interval in 90 of his 100 patients with active carditis, and agreed with Taran and Szilágyi (1947) that this measurement was of diagnostic value. However in Pokress’ and Goldberger’s (1949) series only 14 of the 50 patients (28%) with active rheumatic fever had a Q-T interval above the normal maximal value.

The results of the present series differ significantly from those of Taran and Szilágyi (1947) and of Abrahams (1949). This paper* is an attempt to explain this difference, and to assess the value of the measurement of the Q-T interval in rheumatic fever.

Methods

Goldberger’s (1948) nomogram was chosen to relate the measured Q-T interval to the heart rate. This nomogram expresses the Q-T interval as the ratio of the measured to the ideal interval for that heart rate. The ideal Q-T interval for each heart rate is estimated by the use of Bazett’s (1920) formula, and since this formula was used in the other reports, we were able to compare our results with those of the earlier workers.

All recordings were made with the child in a reclining posture, and after he had been in bed for at least 24 hours.

In approximately half the cases the apparatus used was the General Electric Company electrocardiographic machine, and in the rest the Elmquist Junior (General Radiological Company) machine.

Lead II was used for the measurements, unless the end of the T wave was ill defined, in which case the complexes in lead I or lead III were preferred. The measurements were made with a pair of compass dividers and a hand lens. Particular care was taken while measuring the Q-T interval to measure it from the beginning of the Q deviation to the point at which the T wave again became isoelectric. The Q-T and R-R intervals of at least five cardiac cycles were measured, and the Q-T ratio of each separate cycle was found on the nomogram and the average of these values estimated.

Material

One hundred and twelve electrocardiograms of 102 children, whose ages ranged from 3 1/2 to 14 years, were examined. All the children were in-patients at the Children’s Hospital, Sheffield, and at the Ash House Hospital School. Eighty-two had rheumatic fever at various stages, and the remaining 20 were controls. They were divided into the following groups.

Group I. These children had definite evidence of active rheumatic infection. Polyarthritis, marked lassitude, and anorexia were the main features of their present illness. The sedimentation rate estimated on admission by the micromethod was above 20 mm. in one hour, and remained elevated for at least six weeks, in the absence of other discoverable causes. At the time the electrocardiogram was recorded, polyarthritis had subsided in most of the children but lassitude, pallor, and anorexia persisted, while the sedimentation rate remained high. This group was further subdivided into two sub-groups, Ia and Ib. The children in sub-group Ia either had at the time of the recording, or developed within a short time after the recording and during the same attack of rheumatic fever, an apical systolic murmur of moderate or loud intensity (Levine’s (1933) grade III to grade VI), harsh in
Fig. 1.—Q-T ratios in children with rheumatic infection and in controls.
nature, long, and well conducted to the axilla, or an apical diastolic murmur, or both. These auscultatory findings were always confirmed by more than one observer. In none of these cases was a friction rub heard at any time.

The children in sub-group Ib neither had, nor subsequently developed during the same attack of rheumatic fever, any of the murmurs described above.

**Group II.** This consisted of children who were known to have had an attack of acute rheumatic fever with carditis, as defined by the criteria laid down for sub-group Ia, and in whom one or both of the murmurs described persisted. All clinical signs of activity had, however, subsided at the time of the recording. Only children whose weekly sedimentation rate had been below 10 mm. in one hour for at least two months were included in this group.

**Group III.** The controls were children of the same age group who were admitted for minor surgical operations, and had neither signs of infection nor abnormal cardiac signs.

The number of children and of recordings in each group is shown in Table 1.

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of Children</th>
<th>E.C.G. Recordings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ia. Active rheumatic fever; carditis definite</td>
<td>46</td>
<td>55</td>
</tr>
<tr>
<td>Ib. Active rheumatic fever; carditis doubtful</td>
<td>12</td>
<td>13</td>
</tr>
<tr>
<td>II. Inactive rheumatic heart disease</td>
<td>24</td>
<td>24</td>
</tr>
<tr>
<td>III. Normal controls</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Total</td>
<td>102</td>
<td>112</td>
</tr>
</tbody>
</table>

**Results**

**Individual Measurements.** In Fig. 1 the Q-T ratios for the four groups are presented. The lower horizontal line at 1·01 corresponds roughly to a Q-T interval of 0·405 corrected according to Bazett’s (1920) formula. It represents the upper limit of normal in Taran’s, and Szilagyi’s (1947) series. The upper horizontal line at 1·06 corresponds to a corrected Q-T interval of 0·422 which represents the upper limit of normal Q-T interval for children (Ashman and Hull, 1937). This upper limit of normal has been also adopted by Abrahams (1949), while Pokress and Goldberger (1949) place the upper limit of normal even higher, at 1·08.

It can be seen from Fig. 1 that the Q-T intervals of normal children were all below Ashman’s and Hull’s upper limit of normal, and this maximal normal value can therefore be accepted for the present study. Six recordings in Group Ia, two in Group Ib, and three in Group II showed a Q-T interval above the upper limit of normal.

**Statistical Comparison.** The figures of each group as a whole were compared with those of the other groups, and a difference was accepted as statistically significant when the possibility of its being due to chance was less than 5%. In these children in Group I of whom more than one recording was taken, the mean of the measurements in each child was used in the statistical comparison. There was no statistically significant difference between Group Ia and Group II, namely between children with active and inactive carditis, or between Group I as a whole (Ia and Ib) and Group II. Group I was not significantly different from the control Group III, while there was a significant difference between Groups II and III. Finally all the children with active rheumatic fever or inactive carditis taken together (Group I and II) had a Q-T interval significantly prolonged when compared to the Q-T interval of the 20 controls (Group III).

**Quiescence.** Five of the children in Group Ia eventually became inactive by our criteria. The Q-T intervals of two of these children were longer in the active stage.

**Discussion**

The value of the measurement of the Q-T interval in rheumatic fever must be assessed from two different angles. The first, of great practical importance, is whether the measurement helps in the diagnosis of active rheumatic carditis. The second, of more theoretical interest, is whether rheumatic infection exerts any influence at all on the duration of the electrical systole.

In only six of 55 recordings taken during an active rheumatic carditis (sub-group Ia) was the Q-T interval above the maximal normal limit. If to this number are added the children with rheumatic activity, but without definite cardiac involvement, the proportion of lengthened Q-T intervals becomes eight of 68 (11·8%). Three out of 24 children with inactive rheumatic carditis (Group II) had a prolonged Q-T interval (12%). These results suggest that in a case of rheumatic fever a Q-T interval above the normal limit indicates the presence of active rheumatic carditis, but this diagnostic help is available in only a small number of children.
It is evident also that the measurement of the 
Q-T interval does not help in deciding that rheumatic 
activity has ceased, since it was prolonged even 
in cases of inactive rheumatic heart disease.

It may be argued that in the three children in 
Group II with prolonged Q-T interval there was still 
latent rheumatic activity. This is unlikely, however, 
since these three children were allowed gradually 
to resume their normal life, and there was no 
evidence of reactivation of the rheumatic fever by 
the criteria defined.

These results are very different from those of 
Taran and Szilagyi (1947) and of Abrahams (1949). 
The former found that 100% of their cases with 
active rheumatic carditis had a Q-T interval longer 
than that found in their inactive ones. Unfortu-
nately they do not state in their paper the criteria 
for the diagnosis of active carditis. In another 
publication, however, the senior author (Taran, 
1947) states that one of the primary clinical criteria 
of active carditis is 'the disturbance in relationship 
of systole to diastole.' If such a criterion was 
indeed used for the selection of cases with active 
carditis, it is not surprising that all had a lengthened 
Q-T interval.

Abrahams (1949), on the other hand, defined 
more clearly the criteria for dividing his cases into 
active and inactive carditis, and these criteria are 
similar to those adopted in the present study. It is 
difficult, therefore, to determine why our findings 
are so different from his. In Abrahams' (1949) 
series the Q-T intervals are on the whole longer 
than our own, and it is unfortunate that in his paper 
 neither the number of normal subjects examined 
nor the measurements of their Q-T intervals are 
given. Even from Abrahams' (1949) data it does 
not appear that the Q-T interval may help in 
establishing cessation of rheumatic activity, since in 
five out of 12 cases of inactive carditis the Q-T 
interval was lengthened. It is true that it was 
suspected that these five cases were not really 
inactive, but the reasons for suspecting this inactivity 
are given for one case only. It seems, therefore, 
that although rheumatic infection may prolong the 
duration of electrical systole, the change is so 
inconstant and slight that it is not often of diagnostic 
value. Even when it is, it may indicate the presence 
of rheumatic carditis, but not whether the carditis 
is active or inactive.

Taran and Szilagyi (1947), and Abrahams (1949) 
have claimed that repeated measurements of the 
Q-T interval in the same child indicate a relationship 
between the duration of the electrical systole and 
the degree of rheumatic activity. The number of 
similar cases studied in the present series is too small 
to justify general conclusions. However, it must be 
noted that even among the electrocardiograms of 
five children, two did not demonstrate this relation-
ship.

Summary

The Q-T interval was measured in 112 electro-
cardiograms of 82 children with rheumatic fever and 
of 20 normal controls.

The Q-T interval was above the maximal normal 
limit in eight of 68 tracings taken during active 
rheumatic infection, in three of 24 tracings of 
children with inactive rheumatic heart disease, and 
in none of the tracings obtained from the controls.

There was a significant difference between the 
mean Q-T interval of all the children with active or 
inactive carditis and the control group.

It is concluded that rheumatic infection may 
prolong the duration of the electrical systole, but 
the degree of this change is such that it is of diagnostic 
value in only a few cases.

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