The Q-T Interval in Chronic Cor Pulmonale

By JAMES K. ALEXANDER, M.D., M. IRENE FERRER, M.D.,
REJANE M. HARVEY, M.D., AND ANDRE COURNAND, M.D.

In 13 patients in whom the diagnosis of chronic cor pulmonale was made after careful physiologic studies, the Q-T interval was found to be within normal limits regardless of the presence or absence of cardiac failure. The finding is in sharp contrast to the prolongation of this interval in the more common types of heart disease, as exemplified by a group of 14 patients with hypertensive and arteriosclerotic heart disease. Measurement of the Q-T interval therefore may be useful in differentiating pure chronic cor pulmonale from other types of heart disease with congestive failure.

ALTHOUGH there has been much consideration in the past given the duration of the Q-T interval in the electrocardiogram, more recently attention has been directed with renewed interest toward its possible diagnostic significance in various clinical conditions. It seems to be generally agreed that the Q-T interval represents the period of depolarization and repolarization of the ventricular muscle, and that normally there is variation in its length, dependent upon age, sex, and heart rate. The primary factors actually determining its duration in normal and pathologic states, presumably inherent in muscle metabolism, await further elucidation.

It is known, however, that the duration of the Q-T interval may be shortened by digitalis administration, hypocalcemia and vagal stimulation. It may be prolonged in association with changes in position, myocardial infarction, cardiac enlargement, congestive heart failure of varied etiology, hypopotassemia, hypocalcemia, and administration of certain drugs such as quinidine, atropine and adrenaline. The concept that the Q-T interval is often prolonged beyond the upper limit of normal with acute rheumatic carditis has recently been challenged, as has the contention that greater muscle mass causes lengthening. The relationship between the Q-T interval and the duration of mechanical systole is inconstant and the use of the term “electrical systole” to describe the Q-T interval seems, therefore, unjustified, particularly since the conduction phenomena associated with depolarization and repolarization are in no way analogous to actual mechanical contraction.

The present study was undertaken to determine what alterations in Q-T interval, if any, might be found in association with chronic cor pulmonale, since this latter has not been previously mentioned in the literature and in view of the changes observed in the duration of the Q-T interval in other types of heart disease.

Material and Methods

All 13 cases of chronic cor pulmonale reported in this study fulfilled the accepted criteria for the diagnosis, namely the presence of chronic pulmonary disease, evidence of right heart enlargement or failure, marked cyanosis, and no other demonstrable etiology of their heart disease. Most of these patients have been reported in a previous paper with physiologic studies to confirm the clinical diagnosis, and in all instances actual measurements indicated the presence of pulmonary insufficiency, pulmonary hypertension, arterial unsaturation and polycythemia. Three patients (L. P., L. B., and T. D.) had been in congestive failure recently but were not in failure at the time of study, and a fourth (J. McC.) had never been in failure. The remainder had clinical evidence of failure, and the
hypervolemia and elevated mean auricular and right ventricular end diastolic pressures characteristic of right heart decompensation. All patients had normal sinus rhythm or sinus tachycardia by electrocardiogram, and had received no digitalis or other cardiac drug prior to study.

Q-T measurements were made in the usual manner (from the beginning of the initial Q-T intervals were chosen for comparison with the patients having chronic cor pulmonale, had, in addition to the usual clinical findings of left ventricular or left and right heart failure, physiologic studies confirming the presence of these forms of decompensation according to previously published criteria.\(^9\) Again the rhythm was normal sinus, and no digitalis preparations or other drugs had been adminis-

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Heart Rate</th>
<th>Aver. Q-T (sec.)</th>
<th>Upper Limit of Normal Q-T (sec.)</th>
<th>K</th>
<th>Upper Limit of Normal for K</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. P.</td>
<td>M</td>
<td>96</td>
<td>0.332</td>
<td>0.344</td>
<td>0.421</td>
<td>0.392 0.400 0.433</td>
</tr>
<tr>
<td>L. P.</td>
<td>M</td>
<td>83</td>
<td>0.303</td>
<td>0.368</td>
<td>0.357</td>
<td>0.295 0.328 0.346</td>
</tr>
<tr>
<td>T. D.</td>
<td>M</td>
<td>93</td>
<td>0.300</td>
<td>0.351</td>
<td>0.375</td>
<td>0.338 0.341 0.381</td>
</tr>
<tr>
<td>B. B.</td>
<td>M</td>
<td>107</td>
<td>0.259</td>
<td>0.328</td>
<td>0.398</td>
<td>0.308 0.338 0.398</td>
</tr>
<tr>
<td>T. A.</td>
<td>M</td>
<td>100</td>
<td>0.295</td>
<td>0.338</td>
<td>0.381</td>
<td>0.308 0.338 0.398</td>
</tr>
<tr>
<td>J. B.</td>
<td>M</td>
<td>100</td>
<td>0.308</td>
<td>0.338</td>
<td>0.398</td>
<td>0.308 0.338 0.398</td>
</tr>
<tr>
<td>L. B.</td>
<td>M</td>
<td>85</td>
<td>0.338</td>
<td>0.363</td>
<td>0.404</td>
<td>0.308 0.338 0.398</td>
</tr>
<tr>
<td>F. C.</td>
<td>M</td>
<td>66</td>
<td>0.343</td>
<td>0.404</td>
<td>0.362</td>
<td>0.308 0.338 0.398</td>
</tr>
<tr>
<td>M. V.</td>
<td>M</td>
<td>59</td>
<td>0.390</td>
<td>0.425</td>
<td>0.387</td>
<td>0.308 0.338 0.398</td>
</tr>
<tr>
<td>J. McC.</td>
<td>M</td>
<td>110</td>
<td>0.285</td>
<td>0.325</td>
<td>0.388</td>
<td>0.308 0.338 0.398</td>
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<tr>
<td>F. E.</td>
<td>F</td>
<td>88</td>
<td>0.342</td>
<td>0.359</td>
<td>0.415</td>
<td>0.308 0.338 0.398</td>
</tr>
<tr>
<td>A. D.</td>
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<td>90</td>
<td>0.328</td>
<td>0.364</td>
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</tr>
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<td>B. B.</td>
<td>F</td>
<td>110</td>
<td>0.295</td>
<td>0.325</td>
<td>0.402</td>
<td>0.308 0.338 0.398</td>
</tr>
</tbody>
</table>

In this table "Aver. Q-T" indicates the actual measured average Q-T interval, and "K" the actual value of K calculated from Bazett's formula. "Upper Limit of Normal Q-T" is the upper limit of normal for duration of the Q-T interval at the given heart rate in each patient as cited by Ashman and Hull. "Upper Limit of Normal for K" refers to the upper limit for K for normal males and females according to the data of Bazett (B), Cheer and Li (CL), and Shipley and Hallaran (SH) respectively. See text.

deflection in the QRS complex to the end of the T wave at its return to the isoelectric line) in each lead of the twelve or fourteen lead electrocardiogram (standard leads I, II, III, aVR, aVL, aVF, V1 through V6, V5R, V6R) and these results averaged to give the "average Q-T" for the individual patient at his heart rate. There were fewer measurements in some instances due to the absence of clear demarcation of the T wave or technically unsatisfactory tracings, and an average value is reported for each patient. Cycle length was taken as the average R-R interval, measured as the distance between peaks of successive R waves, for 10 cycles. Calculation of K in Bazett's formula\(^{11}\) (Q-T = K \(\sqrt{\text{cycle length}}\)) was done by the slide rule method.\(^8\)

The 14 cases of hypertensive cardiovascular disease and arteriosclerotic heart disease whose Q-T measurement were done as described above.

**RESULTS**

The results of the Q-T measurements in 13 cases of cor pulmonale are summarized in table 1. It will be seen that in every instance the measured average Q-T duration for the given heart rate is below the upper limit of normal cited in the data of Ashman and Hull.\(^8\) The actual value of K is either below or at the normal upper limit in all instances except one using the criteria of Bazett\(^{11}\) and Cheer and Li,\(^7\) and in every instance, according to the criteria of Shipley and Hallaran.\(^21\) The average values for K in the 10 males (0.382) and three females (0.407) used in this study are both well below the upper limits of normal cited by all three sources. In males...
with chronic cor pulmonale, the average value of $K$ in the 6 patients with cardiac failure (0.382) is the same (0.381) as in the 4 patients (L. P., T. D., L. B., J. McC.) who were compensated.

For the purpose of comparison, the Q-T measurements in 14 cases of hypertensive cardiovascular disease and arteriosclerotic heart disease in failure are tabulated in table 2. Here the measured Q-T is greater than the upper limit of normal in all but 3 instances according to the data of Ashman and Hull. A further demonstration of this is shown in figure 1 where the measured Q-T interval is plotted against heart rate in the cases of chronic cor pulmonale and hypertensive cardiovascular disease and arteriosclerotic heart disease. The observed values are plotted in relation to the predicted upper limit of normal,\(^8\) which appears as a reference line. The values in chronic cor pulmonale are within normal limits and tend to fall well below those in hypertensive cardiovascular disease and arteriosclerotic heart disease, the majority of which are grouped above the upper limit of normal. The calculated values for $K$ in the patients with hypertensive and arteriosclerotic heart disease (table 2) are above the upper limit of normal in every instance, according to Bazett’s criteria, in all but 2 borderline instances, according to those of Cheer and Li, and in all instances but 4, according to those

### Table 2.—The Q-T Interval in Fourteen Patients with Hypertensive Cardiovascular Disease and Arteriosclerotic Heart Disease

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Type Failure</th>
<th>Heart Rate</th>
<th>Aver. Q-T (sec.)</th>
<th>Upper Limit of Normal Q-T (sec.)</th>
<th>K</th>
<th>Upper Limit of Normal for K</th>
</tr>
</thead>
<tbody>
<tr>
<td>N. P.</td>
<td>M</td>
<td>L &amp; R</td>
<td>100</td>
<td>0.308</td>
<td>0.338</td>
<td>0.398</td>
<td>0.392 0.400 0.433</td>
</tr>
<tr>
<td>C. G.</td>
<td>M</td>
<td>L &amp; R</td>
<td>120</td>
<td>0.308</td>
<td>0.310</td>
<td>0.436</td>
<td></td>
</tr>
<tr>
<td>M. R.</td>
<td>M</td>
<td>L &amp; R</td>
<td>107</td>
<td>0.340</td>
<td>0.325</td>
<td>0.455</td>
<td></td>
</tr>
<tr>
<td>J. B.</td>
<td>M</td>
<td>L</td>
<td>100</td>
<td>0.346</td>
<td>0.338</td>
<td>0.447</td>
<td></td>
</tr>
<tr>
<td>G. R.</td>
<td>M</td>
<td>L</td>
<td>130</td>
<td>0.336</td>
<td>0.294</td>
<td>0.496</td>
<td></td>
</tr>
<tr>
<td>P. A.</td>
<td>M</td>
<td>L &amp; R</td>
<td>73</td>
<td>0.400</td>
<td>0.384</td>
<td>0.442</td>
<td></td>
</tr>
<tr>
<td>T. B.</td>
<td>M</td>
<td>L</td>
<td>75</td>
<td>0.389</td>
<td>0.384</td>
<td>0.436</td>
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</tr>
<tr>
<td>J. M.</td>
<td>M</td>
<td>L &amp; R</td>
<td>115</td>
<td>0.330</td>
<td>0.317</td>
<td>0.457</td>
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<tr>
<td>V. B.</td>
<td>M</td>
<td>L</td>
<td>78</td>
<td>0.347</td>
<td>0.378</td>
<td>0.390</td>
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<tr>
<td>J. S.</td>
<td>M</td>
<td>L &amp; R</td>
<td>83</td>
<td>0.378</td>
<td>0.368</td>
<td>0.446</td>
<td></td>
</tr>
<tr>
<td>J. S.</td>
<td>M</td>
<td>L</td>
<td>83</td>
<td>0.394</td>
<td>0.368</td>
<td>0.465</td>
<td></td>
</tr>
<tr>
<td>S. F.</td>
<td>F</td>
<td>L &amp; R</td>
<td>65</td>
<td>0.430</td>
<td>0.418</td>
<td>0.448</td>
<td>0.440 0.421 0.456</td>
</tr>
<tr>
<td>I. J.</td>
<td>F</td>
<td>L</td>
<td>97</td>
<td>0.361</td>
<td>0.353</td>
<td>0.459</td>
<td></td>
</tr>
<tr>
<td>D. M.</td>
<td>F</td>
<td>L &amp; R</td>
<td>65</td>
<td>0.424</td>
<td>0.418</td>
<td>0.443</td>
<td></td>
</tr>
</tbody>
</table>

For symbols see table 1. "Type Failure" refers in this table to the presence of left-sided (L) or left and right-sided (L and R) congestive failure. See text.

fig. 1. The Q-T interval in patients with chronic cor pulmonale compared with patients with hypertensive and arteriosclerotic heart disease. The reference line here represents the predicted upper limit of normal for the measured Q-T interval in seconds at various heart rates, according to the data of Ashman and Hull. Closed circles represent the measured values for the Q-T interval in patients with chronic cor pulmonale and the crosses indicate the values in patients with hypertensive and arteriosclerotic heart disease.
of Shipley and Hallaran. In contrast to the results in cases of chronic cor pulmonale, the average values for K in males (0.434) and females (0.450) are either above or at the upper limits of normal cited by the three quoted sources. It may be of interest to note that the average value for K in males with left sided heart failure alone (0.449) appears to be somewhat higher than that in males with both left and right sided failure (0.439).

**DISCUSSION**

Although this report is based on a small series of cases, the diagnosis of chronic cor pulmonale was clearly established in each instance and well documented with physiologic studies. The data obtained are open to only one interpretation, namely that with regard to the four sources of normal values quoted, the Q-T interval in chronic cor pulmonale is within normal limits both in patients with or without congestive failure. In addition, the value of K in this series is the same regardless of cardiac decompensation.

On the other hand, as might be expected on the basis of previous similar studies, Q-T measurements in patients with hypertensive cardiovascular disease and arteriosclerotic heart disease in cardiac failure were found to be prolonged beyond the upper limit of normal in the majority of cases.

The explanation for the absence of Q-T interval prolongation in chronic cor pulmonale cannot be given with certainty at this time. However, the evidence accumulated to date in this laboratory indicates that the burden in chronic cor pulmonale is on the right heart with resultant hypertrophy and dilatation on that side, and sparing of the left heart. Since the levocardiogram is said to account for approximately 60 per cent of the total potential differences in the electrocardiogram, potentials originating entirely in the dextrocardiogram are therefore less readily manifest, and changes in the Q-T interval of the right heart, if present, might perhaps not be readily appreciated in the completed electrocardiogram.

The finding of a definite prolongation of the Q-T interval in the patients with hypertensive and arteriosclerotic heart disease is in accord with previous reports. The literature, however, contains no specific information as to the Q-T interval in patients with chronic cor pulmonale. The finding of a normal Q-T interval in such patients, as demonstrated by this study, is in sharp contrast to the findings in the more common forms of heart disease. Thus the duration of the Q-T interval may be a diagnostic aid in differentiating pure chronic cor pulmonale from other types of heart disease resulting in congestive failure. When chronic cor pulmonale exists in association with another form of cardiac disease, such as arteriosclerotic heart disease, the Q-T interval may be prolonged.

**SUMMARY AND CONCLUSIONS**

1. Measurements in 13 cases of chronic cor pulmonale with or without cardiac failure indicate that the Q-T interval is not prolonged in this condition.

2. Fourteen cases of hypertensive cardiovascular disease and arteriosclerotic heart disease in failure studied for comparison showed prolongation of the Q-T interval, as might be expected on the basis of previous reports.

3. It is suggested that measurements of the Q-T interval may be used as a diagnostic tool to aid in differentiating pure chronic cor pulmonale from other types of heart disease resulting in congestive failure.

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