
BY PAUL D. WHITE AND SEELEY G. MUDD
(From the Cardiac Clinic and Laboratory of the Massachusetts General Hospital, Boston)
(Received for publication October 3, 1928)

INTRODUCTION

The duration of systole, both absolute and relative to diastole, has long been a subject of interest to physiologists, but it has been almost wholly ignored by clinicians. In fact many physicians are not even aware that the duration of systole varies so much as it does with heart rate and that the first and second heart sounds are on some occasions twice as far apart in time as on other occasions. A considerable physiological literature relative to the duration of systole exists, but very little is found in the clinical literature. Fridericia (1) (2) and Miki (32) are the only writers who have discussed the subject in any detail from the clinical point of view, that is, as to the effects of various pathological conditions on the duration of systole. The one finding of common knowledge physiologically and known to the few clinicians who have been interested in the subject is that systole shortens appreciably with increasing pulse rates, but less rapidly than does diastole.

It has seemed to us worthwhile to gather more data on the effect of various clinical conditions on the duration of electrical systole to supplement the scanty information that we have at present. Hence we have made measurements of electrical systole in a large group of patients with various disease conditions as well as in a group of normal controls.

The duration of electrical systole we have measured from the electrocardiogram (the interval between the beginning of the Q wave
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and the end of the T wave) as will be recounted below. Although this interval has not coincided with the measurements made on many mechanical curves, it is possible and in fact probable that it represents more accurately the true duration of systole, provided good measurable curves are obtained, than does any mechanical record. At least this interval is a reliable index of the duration of systole and can be used in a study of the relative effects of various conditions with a high degree of confidence in its reliability for such comparisons.

LITERATURE

Fridericia (1) in 1920 published a paper on the duration of systole under normal conditions as measured by the electrocardiogram, which method he considered reasonably accurate. He referred to early measurements of systole both by mechanical records and by electrocardiogram. He concluded from his own study and from observations in the literature that "with rising pulse rates systole and diastole shorten but the latter more." In measuring the Q-T interval of the electrocardiogram, he found the Q or R onset usually sharp with an accuracy of 0.001 second. The end of T was accurate in suitable curves to 0.01 second. If the P wave fell on the T wave, measurement was, he thought, impossible. Variations in the leads were such that he always used lead II. Average errors were not over 0.005 second.

He carefully selected and studied 50 normal people mostly between 20 and 40 years old, 28 male and 22 female. He found the following variations of the duration of systole with pulse rate:

<table>
<thead>
<tr>
<th>Pulse rate</th>
<th>Number of cases</th>
<th>Average Q-T interval</th>
<th>Limits of Q-T interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>50-60</td>
<td>4</td>
<td>0.405</td>
<td>0.420-0.398</td>
</tr>
<tr>
<td>60-70</td>
<td>10</td>
<td>0.370</td>
<td>0.392-0.344</td>
</tr>
<tr>
<td>70-80</td>
<td>11</td>
<td>0.353</td>
<td>0.373-0.325</td>
</tr>
<tr>
<td>80-90</td>
<td>7</td>
<td>0.339</td>
<td>0.352-0.327</td>
</tr>
<tr>
<td>90-100</td>
<td>8</td>
<td>0.335</td>
<td>0.360-0.321</td>
</tr>
<tr>
<td>100-110</td>
<td>5</td>
<td>0.318</td>
<td>0.340-0.308</td>
</tr>
<tr>
<td>110-120</td>
<td>1</td>
<td>0.302</td>
<td></td>
</tr>
<tr>
<td>120-130</td>
<td>3</td>
<td>0.290</td>
<td>0.312-0.273</td>
</tr>
<tr>
<td>Over 130</td>
<td>1</td>
<td>0.280</td>
<td></td>
</tr>
</tbody>
</table>

From these data Fridericia constructed the following formula for the determination of the normal average duration of the Q-T interval of the electrocardiogram (systole): \( S = 8.22 \sqrt{\rho} \), in which \( S = \) Q-T interval, \( \rho = \) pulse period in hundredths of a second, and 8.22 = constant.
Fridericia also investigated the effects of various drugs and poisons on the duration of ventricular systole. Among other findings he reported that when the heart rate was increased by adrenalin, systole was shortened much less than when the tachycardia resulted from muscular work.

A later paper the same year (1920) by Fridericia (2) reported his findings in the study of the duration of systole in the electrocardiogram by the use of his formula under various abnormal conditions. He concluded that a deviation greater than 0.045 second from the expected duration of systole as determined by his formula was the outer limit of normal. Two hundred and eleven curves of 124 patients were studied with measurements made from lead II. He found two chief difficulties in measurement, first in arrhythmia, and the 30 cases showing arrhythmia he considered separately, and second in the presence of abnormalities of the T wave (absence, inversion, diphasic nature). He found that inversion of the T wave shortened systole.

Of 94 patients with normal cardiac mechanism, Fridericia found only 65 whose electrocardiograms were satisfactory for measurement. Of these 65 only 7 showed an abnormal duration of systole. The diagnoses in these seven cases were; mitral stenosis 2, aortic insufficiency 1, aortic stenosis 1, aortic and mitral insufficiency 1, myocarditis and scoliosis 1, and chronic nephritis with hypertension and uremia 1. Fridericia concluded that heart muscle weakness and increased work for the heart caused prolonged systole in these cases. However, hypertension, mitral stenosis, and aortic valve disease generally in this series gave normal Q-T intervals.

Of 30 patients with arrhythmia 5 cases of auricular fibrillation were unmeasurable and the T wave was negative in 9 more. In 15 cases with positive T waves all showed normal Q-T intervals except five, too long in four and too short in one. The cases with overlong Q-T intervals showed many extrasystoles in one, complete heart block in two and partial heart block in one. In one case of paroxysmal tachycardia the Q-T interval was found too short but normal in two others with the same mechanism.

Fridericia's papers are of considerable interest and confirm conclusively the observation that electrical systole shortens with increasing heart rates but less than does diastole. The application of his formula to clinical cases and the conclusions drawn at times from rather scanty material demanded further investigation of the subject.

Miki (32) added in 1922 clinical data to Fridericia's observations, but further study has seemed to us necessary to determine whether or not there is any practical application clinically for the determination of the duration of systole from the electrocardiogram.

Miki studied the electrocardiograms of 178 people and applied the three formulas of Fridericia (1), Bazett (30) and Lombard and Cope (26) to them all. There was a great variation in these cases between the various formulas, that of Lombard and Cope being least in agreement. Among the 178 cases there were one of aortic
stenois, 9 of aortic regurgitation, 10 of mitral regurgitation, 10 of mitral stenosis, 3 of pulmonic stenosis, 12 of combined valve lesions, 20 of left ventricular hypertrophy, 1 of right ventricular hypertrophy, 7 of cardiac dilatation, 7 of "myocarditis," 10 of exophthalmic goiter, 1 of myxedema, 21 of nervousness, 7 with normal hearts and 5 with bradycardia without block. In all this group there were only 13 cases which gave a value of the duration of the Q-T interval more than the limit allowed by Fridericia's formula and these 13 cases were not particularly significant except that most of them had rapid rates (exophthalmic goiter, exercise, nervousness). Of 21 cases of auriculo-ventricular block, 16 gave results longer than the calculated figure from Fridericia's formula while only 9 were greater than Bazett's formula allowed. Miki concluded that in dissociation the duration of systole may become abnormally long and that with damage to heart muscle there tends to be a shortening of systole rather than a lengthening, as Fridericia had believed.

Other workers, mainly physiologists, have as already stated, studied the duration of systole and references to their publications are listed at the end of this paper. It may merely be noted here that the first who made observations on the shortening of systole with increasing pulse rates from pulse tracings were Garrod (3, 4, 5, 6) in 1870 to 1875, Thurston (7) in 1876, Landois (8) in 1876, Waller (9) in 1887, Edgren (10) in 1889, Stockmann (11) in 1889, Kraus (12) in 1891, Lüderitz (13) in 1892, Einthoven and de Lint (14) in 1900, and Zuntz and Schumburg (15) in 1901. Since then there have been many papers published concerning the duration of systole as measured from pulse tracings or electrocardiograms, but there has been little of the clinical application of the measurement of systole.

METHOD OF PRESENT STUDY

During the past three years the electrocardiograms of 213 individuals, 50 normal and 163 abnormal, have been studied by us to determine the influence of various factors on the duration of electrical ventricular systole. Only good records with clearly defined Q-R-S and T waves were measured. Lead II was generally selected for measurement but if the ventricular deflections were higher and more clearly defined in leads I or III, either of these other leads was used. A comparison of the time intervals in the various leads was made in several cases and as a rule the lead with the highest T wave gave the best indication for measurement of the Q-T interval. Generally such a lead gave also somewhat longer Q-T intervals but usually within the limit of error allowed in measurement.

The duration of electrical systole was considered to be the time interval between the onset of the Q-R-S wave and the end of the T wave. The use of this measurement for the duration of systole has
already been discussed by a number of workers and we believe that even if the Q-T interval may not exactly indicate the duration of systole at least it is a satisfactory index of it and accurate for comparative studies. As we have already remarked we believe that this Q-T interval may actually record the duration of systole better than does any other measurement.

In each case the duration of the Q-T interval was measured for three or more successive heart cycles and then averaged; the heart rate was determined by measurements also of the T-Q intervals giving us thus the total cycle lengths. The Lucas comparator was used for all the measurements. Time intervals were figured in ten thousandths of a second but finally recorded in thousandths of a second. They are accurate we believe down to one-hundredth of a second. The electrocardiograms were made almost invariably in the sitting position, rarely in the recumbent position.

The results have been charted as seen in the figures. Because of the difficulty and dissatisfaction with the use of any formula, simple curves have been constructed to show the normal limits of the duration of electrical systole according to the chief factor influencing it, namely pulse rate. The findings in groups of cases representing a variety of different conditions have been plotted against these normal control curves. We believe that this method serves better than any formula to show when systole is abnormally long or short, and to demonstrate the fact in a simple graphic way. We have plotted in addition to our own 50 normal control cases, 190 normal cases from the literature (those giving satisfactory Q-T measurements), giving us thus a total of 240 normals for comparison with our abnormal cases.

RESULTS

1. Normal cases

1. Results at rest. In figure 1 is seen the chart of our 50 normal control cases. There is evident a definite relationship between duration of electrical systole in thousandths of a second marked off by horizontal coordinates, and heart rate in beats per minute marked off by vertical coordinates. Thus at rates between 60 and 70 the normal range of systole in our cases was from 0.400 to 0.314 second, at rates between 90 and 100 it was from 0.356 to 0.280, and at rates between
Fig. 1. Chart Giving the Duration of the Q-T Interval of the Electrocardiogram According to Pulse Rate in 50 Normal Subjects Including 20 Men, 15 Women, and 15 Children.

Each dot represents one case. Heart rate and cycle length are shown in horizontal coordinates and duration of the Q-T interval in thousands of a second in vertical coordinates. All the succeeding charts are similarly constructed.

The limits of measurement of these 50 normal cases are connected by curves giving a comet shaped track.
120 and 150 from 0.295 to 0.225 second. The limits of these measurements are joined, resulting in a comet shaped track, narrow at fast rates. These 50 normal cases included 20 men, 15 women and 15 children (both boys and girls). For this figure and those that follow the duration of the heart cycle is plotted horizontally as well as heart rate, being 2.0 seconds for a rate of 30, 1.0 second for a rate of 60, 0.6 second for a rate of 100, 0.5 second for a rate of 120, and 0.4 second for a rate of 150.

In figure 2 is seen the chart of the 190 normal cases from the literature (Fridericia, (1), Miki (32), Kraus and Nicolai (16), Seham (42), Bazett (30), Katz and Feil (36), and Fenn (35)). They include men, women and children. On this chart in dotted lines are superimposed the curves of figure 1. It will be seen that the limits in figure 2 are somewhat greater through most of the chart than those in figure 1. The coördinates represent as in figure 1 the duration of systole and heart rate. On all the charts to follow, these same coördinates are indicated and the combined limit curves of the 240 normal cases of figures 1 and 2 are superimposed so that at a glance one can tell the situation in a given condition. In the chart of the normal cases from the literature shown in figure 2, the duration of systole at heart rates of 40 to 60 varies from 0.480 to 0.308 second, at rates of 80 to 100 from 0.390 to 0.270 second, at rates of 120 to 130 from 0.312 to 0.230 second, and at rates of 150 to 160 from 0.240 to 0.220 second. Two cases at extremely fast heart rates (180 and 198) fall outside the projected limits, one above and one below.

2. Normal subjects in different positions. Five normal subjects were electrocardiographed in three positions—lying, sitting and standing. In every instance with one exception, the pulse rate rose as the position was changed from recumbent to sitting, and in each instance without exception, the pulse rate rose further on changing from the sitting to the standing position. As will be seen in figure 3 the Q-T interval measurements show that the duration of electrical systole varies exactly as does the pulse rate. The normal limit curves from figures 1 and 2 are charted on figure 3.

It would appear that the effect of gravity in cutting down the blood inflow volume in the right heart, as reported by Lombard and Cope (41), is not necessary in these five subjects discussed here to explain
Fig. 2. Chart giving the duration of the Q-T interval of 190 normal individuals published in the literature.

The outer limits of measurement of these cases are connected by solid lines and the limit lines of our own series of 50 normal cases are superimposed as interrupted lines. Two cases at very rapid rates are seen to fall outside these limit lines when projected.

In all figures to follow the combined outer limit lines of these 238 normal cases are charted and are used as an indication of normal or abnormal length of Q-T interval at varying heart rates.
FIG. 3. THE Q-T INTERVALS OF FIVE NORMAL SUBJECTS IN VARIOUS POSITIONS
A–E, in lying position, A′–E′ in sitting position, and A″–E″ in standing position
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Fig. 4. The Q-T intervals of ten normal individuals before and after exercise.
A, K, at rest; A', K', B', D', B'' after exercise.
Fig. 5. The Q-T intervals of five normal individuals before and after digitalization.

A-E, before digitalis, A'-E' after digitalis.
the variation in the duration of electrical systole. Heart rate alone as in the records from the larger series of normal subjects in the sitting position (fig. 1) may be accounted the chief, though doubtless, not the sole factor. Whatever part gravity may play in these five subjects is a minor one. However, it is quite possible that heart rate itself acts by its effect on the amount of blood entering the heart, duration of electrical systole being controlled by this content of blood rather than by the varying heart rate in itself. Other things being equal, the faster the heart rate the less blood enters the heart, the slower the rate the larger the blood volume in the ventricles. If this is so it would appear that gravity too, with change in position, should influence the duration of systole, but this influence is not apparent in the measurements of the present five subjects.

3. Normal cases after exercise. In figure 4 are charted the results of exercise on the duration of ventricular systole in 10 normal subjects (young men). First, the subjects were electrocardiographed at rest (A to K) and then immediately after exercise consisting of running up and down four flights of stairs (A’ to K’). Three subjects were recorded a second time after exercise (B'', C'', D'') and one was recorded a third time (B''').

It is obvious that all the measurements remained well within the normal limits as charted from figures 1 and 2.

4. The effect of digitalis on normal subjects. Five normal subjects were digitalized (to mild toxic effects) in the course of 7 to 10 days (2.0 to 3.0 grams of digitalis leaf by mouth), and electrocardiograms before digitalis and at the height of the effect of the drug were measured. As will be seen in figure 5, all measurements were well within normal limits permitting us to conclude that digitalis in the normal subject does not influence appreciably the duration of electrical systole as measured by the Q-T interval of the electrocardiogram.

II. Abnormal cases

We now turn to strictly pathological conditions and will consider first structural defects such as cardiac enlargement and valvular disease, then the effect of other conditions and diseases like hypertension per se and diabetes, and finally functional conditions like congestive failure and abnormal tachycardias, bradycardias, and
Fig. 6. The Q-T interval of ten patients with very large hearts without abnormal axis deviation

$F =$ four cases with auricular fibrillation also
Fig. 7. The Q-T intervals of nine patients with very large hearts and abnormal left axis deviation.
arrhythmia. There are included in these pathological groups a total of 163 cases.

1. **Structural.**
   A. **Enlargement of the heart without abnormal axis deviation.** Ten cases with marked cardiac enlargement but without abnormal axis deviation were studied. In some, the cause of the enlargement was unknown. In a few, hypertension or aortic regurgitation were factors. Six had normal rhythm and four had auricular fibrillation. The results are charted in figure 6 and the Q-T interval is within normal limits in every instance.

   We had thought that marked enlargement of the heart might cause prolongation of electrical systole. This proved not to be the case when the Q-T intervals of this group of cases were measured.

   B. **Enlargement of the heart with abnormal left axis deviation.** To supplement the cases of cardiac enlargement without abnormal axis deviation, nine more cases with enlarged heart and an abnormal degree of left axis deviation were studied. Hypertension and aortic regurgitation were the chief underlying factors. Only one of these nine patients showed a Q-T interval longer than the normal outer limits (fig. 7). One other case was on the upper border line of normal and one just within it. The others were well inside the normal limits.

   Thus of 19 cases with very large hearts, only one showed a duration of systole above the normal limits as measured by the Q-T interval. It may be said, therefore, that cardiac enlargement does not ordinarily cause an increase in the Q-T interval.

   C. **Aortic regurgitation.** Nine cases of aortic regurgitation, due to rheumatic or luetic etiology, were studied and are charted in figure 8. In all the duration of systole was normal.

   D. **Aortic stenosis.** Aortic stenosis, both in animal experimentation and in the clinic, has been reported as a cause of systolic lengthening (13, 20, 37, and 38). Figure 9 shows the results of the measurement of electrical systole of 6 cases of our own, one of which, very marked in degree, was confirmed by post mortem examination. All measurements fall within normal limits, including even the one case B with slight bundle branch block in addition.

   E. **Mitral stenosis.** Ten cases of mitral stenosis all showed Q-T intervals well within normal limits (fig. 10).

   F. **Congenital heart disease.** Eight cases of congenital cardiac
Figure 8. The Q-T Intervals of Ten Patients with Aortic Regurgitation
Fig. 9. The Q-T intervals of six patients with aortic stenosis; one case had also right bundle branch block.
Fig. 10. The Q-T Intervals of Ten Patients with Mitral Stenosis
FIG. 11. THE Q-T INTERVALS OF EIGHT PATIENTS WITH CONGENITAL HEART DISEASE

$D = \text{those with patent ductus arteriosus (3). } P = \text{those with pulmonic stenosis (5)}$
Fig. 12. The Q-T intervals of ten cases with uncomplicated essential hypertension.
Fig. 13. The Q-T Intervals of Five Cases with Uremia

A = 2 records of one case
**Fig. 14. The Q-T Intervals of Five Patients A–E with Varying Blood Serum Calcium Content**

The figures showing the amount of calcium (in milligrams per 100 cc. serum) in the blood are given below each dot representing a Q-T interval.
Fig. 15. The Q-T interval of five patients with diabetes mellitus
defects all showed normal duration of systole as measured by the Q-T interval. Five of the patients were diagnosed as having pulmonic stenosis (P) and three as having patent ductus arteriosus (D).

2. Extrinsic factors. A. Hypertension. Hypertension has at times past been found present when systole is prolonged (28, 35 and 37) but in ten cases that we have studied in which the single factor of hypertension entered, we found all with a Q-T interval within normal limits, though one was on the upper limit of normal. These cases showed little or no cardiac enlargement by physical examination or x-ray and no abnormal axis deviation by electrocardiogram.

B. Uremia. Five cases of uremia showed normal duration of electrical systole in all except one (fig. 13). On both of two separate occasions, this patient's Q-T interval was too long.

C. Blood calcium studies. As shown by figure 14, the content of calcium in the blood serum appears definitely to be a factor in controlling the duration of the Q-T interval, in almost as striking a manner as is the pulse rate. This relationship has already been pointed out by Carter and Andrus (34) and our findings are strongly confirmatory. Five cases have been studied by us from the standpoint of calcium content of the blood serum and the two cases (tetany) which showed marked diminution of the serum calcium (A with 4.0 mgm. and D with 5.0 mgm. per 100 cc. of blood) both showed prolongation of the Q-T interval beyond the normal, with a return to normal as the blood calcium rose.

D. Diabetes mellitus. Five cases of diabetes mellitus were studied by us, three of them during the stage of hyperglycemia; all showed Q-T intervals within normal limits (fig. 15).

E. Hyperthyroidism. The effect of hyperthyroidism was studied in eleven patients, in two of whom electrocardiograms were taken before and after correction of the hyperthyroidism by operation. Also in two of the cases, records were obtained during periods of auricular fibrillation as well as during normal rhythm. As will be seen in figure 16, all the measurements are within normal limits except two, one a case with a metabolic rate of plus 32 per cent and normal rhythm and the other after operation with a metabolic rate of -5 per cent and auricular fibrillation. This latter case showed during normal rhythm, normal Q-T intervals both before and after operation. All the other cases showed normal duration of electrical systole.
FIG. 16. THE Q-T INTERVALS OF ELEVEN PATIENTS WITH HYPERTHYROIDISM; IN TWO OF THESE CASES A AND C THE Q-T INTERVALS AFTER OPERATION AND CORRECTION OF THE HYPERTHYROIDISM ARE ALSO SHOWN (A', C'', C''')

B'', C'' = cases during auricular fibrillation. The figures below the dots give the basal metabolic rates
FIG. 17. THE Q-T INTERVALS OF FIVE CASES WITH HYPOTHYROIDISM A–E

The Q-T intervals of the same cases after thyroid therapy are also given (A'E'). The basal metabolic rates are shown by figures below the dots representing the Q-T intervals.
Fig. 19. The Q-T intervals of twelve patients with congestive failure; in three cases (A, B and C), the Q-T intervals after recovery are also shown (A', B' and C'). Auricular fibrillation additionally present in cases A and C.
F. Hypothyroidism. The effect of hypothyroidism on the duration of the Q-T interval was measured in five cases both before and after thyroid therapy. In every case, the findings were well within normal limits as seen in figure 17, although we had thought that a sluggish heart in myxedema might prolong electrical systole. Of course, the pulse rate was invariably slower during the hypothyroid stage, and in accord with the slower pulse rate, the Q-T interval was longer.

G. Acute infection. Five cases have been studied to determine whether or not acute infection affects the duration of the Q-T interval. Electrocardiograms were taken and measured both during the infection and after recovery. Pneumonia was the infection in two cases and tonsillitis and pharyngitis in the other three. The Q-T interval during the infection was generally short but in exact relationship to the degree of tachycardia and hence well within normal limits.

It is of considerable interest to observe that in two of the cases, the pulse rate was actually higher after the infection than during it, due to excitement and effort syndrome but nevertheless the Q-T intervals varied consistently as the heart rate.

3. Functional conditions. A. Congestive failure. To determine whether congestive failure affects the duration of electrical systole we studied twelve cases, three with auricular fibrillation and nine with normal rhythm. Three of these patients we investigated again after the congestive failure had disappeared under rest and digitalis. We have already noted that digitalis per se does not affect the Q-T interval. All the measurements both during and after congestive failure were within normal limits as shown in figure 19.

Heart muscle weakness and congestive failure have been mentioned in the literature as possible causes for prolongation of systole (2, 29) and also for shortening of systole (32, 37). Our study of the twelve cases reported above shows no definite effect either one way or the other.

B. Auricular paroxysmal tachycardia. Five cases of auricular paroxysmal tachycardia have been studied by us both during the paroxysms and also during normal rhythm. A little difficulty has been experienced at times in making the measurements but only those electrocardiograms allowing a reasonable degree of accuracy have been selected. As seen in figure 20, there is a tendency for the Q-T
Fig. 20. The Q-T Intervals of Five Patients During Auricular Paroxysmal Tachycardia (A'-E') and During Normal Rhythm (A-E)
FIG. 21. THE Q-T INTERVALS OF CASE DURING NORMAL RHYTHM (A) AND DURING VOLUNTARY ACCELERATION OF THE HEART BEAT
Fig. 22. The Q-T Intervals of Five Patients with Atricular Fibrillation (A-E) and of the Same Patient After Restoration to Normal Rhythm by Quinidine Sulphate (A-E).
Fig. 23. The Q-T intervals of two cases of ventricular paroxysmal tachycardia during the paroxysms (A', B') and during normal rhythm (A, B)
interval to be longer at the paroxysmal rates than would be expected on continuing the so-called normal limit curves. The slowest paroxysmal rate—148—was found with a Q-T interval within normal limits, but all four cases with rates over 180 showed relatively long Q-T intervals. Of course, here we have no normal controls for comparison.

C. Voluntary acceleration of the heart rate. One rare case of voluntary acceleration of the heart rate was studied and found to have durations of the Q-T interval perfectly normal at varying rates (fig. 21).

D. Auricular fibrillation with restoration of normal rhythm. The effect of auricular fibrillation on the duration of the Q-T interval was studied in five cases in whom normal rhythm was later restored by quinidine sulphate. A comparison of the Q-T interval during auricular fibrillation and during normal rhythm was made. The results are charted in figure 22. All measurements were normal except three which were prolonged; two of these measurements were from the same case (B B') during auricular fibrillation and during normal rhythm and that case had also right bundle branch block which will be discussed later; the third measurement was also delayed beyond the normal but occurred in a case only during a very rapid ventricular rate (164) in auricular fibrillation, the Q-T interval being within normal limits when the rhythm was normal and the rate slow (73). The other three cases showed entirely normal rhythm. It would seem then from these measurements and those of seven other cases with auricular fibrillation shown in figures 6 and 19, that auricular fibrillation in itself does not affect the Q-T interval, which may be prolonged, however, if there is a coincident bundle branch block or an extreme tachycardia. Auricular fibrillation has been reported in some cases in the literature with overlong Q-T intervals (2) and also with Q-T intervals shorter than normal (2, 31, 32, 36).

E. Ventricular paroxysmal tachycardia. Two cases of ventricular paroxysmal tachycardia were electrocardiographed during their paroxysms and during normal rhythm. The results are shown in figure 23. In one case the Q-T interval is much prolonged beyond the normal outer limit (B') and in the other case the Q-T interval falls also above this upper limit line if projected, but only a little above normal. In both cases the Q-T interval is well within normal limits during normal rhythm.
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Fig. 24. The Q-T Intervals of Atrial Premature Beats (A-E) and of Normal Beats (A-E) in Five Cases.
Fig. 25. The Q-T intervals of ventricular premature beats (A’-D’) and of normal beats (A-D) in four cases.
Fig. 27. The Q-T intervals of five patients with partial atrio-ventricular block. A-A', B-B', and C-C', represent the Q-T intervals in three of the cases at varying degrees of block and heart rate.

Duration of electrical systole in seconds.

Rate per minute (b.p.m.)
F. Auricular premature beats. A comparison has been made in five cases between the duration of the Q-T interval of an auricular premature beat without aberration and that of a normal beat (fig. 24). In one case the auricular premature beat Q-T interval was beyond the upper normal limit and in three other cases on or very close to it. Only one was well within normal limits. The “rate” for the premature beats was calculated from the interval between the preceding normal beat and the premature beat.

G. Ventricular premature beats. A similar study was made of four cases of ventricular premature beats. Figure 25 shows that in all of the four cases the Q-T interval of the premature beat was much prolonged beyond the normal limits for heart rates based on the interval between the preceding normal beat and the premature beat. This is what one would expect since the abnormal origin of the beat should cause a slower spread of the impulse and slower rise of intraventricular pressure than normally.

H. Sinoauricular bradycardia. Seven instances of sinoauricular bradycardia without heart disease were studied. All Q-T intervals were within normal limits except one which was slightly overlong (fig. 26).

I. Partial auriculo-ventricular block. Five cases of partial auriculo-ventricular block have been examined and are charted in figure 27. One was electrocardiographed during a period of high grade partial block at two different rates in each case. All measurements of electrical systole except the two of one of the cases are within normal limits though varying rather widely. The one case with prolonged Q-T interval during both fast and slow rates with block was a boy of six years with miliary tuberculosis.

J. Complete auriculo-ventricular block. Six cases of complete auriculo-ventricular block showed abnormally long Q-T intervals in all except two and those two were close to the upper edge of normal. However, three of these four case with overlong Q-T intervals had also bundle branch block and the Q-T intervals were extraordinarily prolonged, with one exception the longest that we have encountered (all over 0.6 second in duration). One of them with varying bundle branch block gave a measurement of the Q-T interval of over 0.7 second, the longest of our entire series and about 0.2 second above
Fig. 28. The Q-T intervals of six patients with complete auriculo-ventricular block. Cases A, B, and E had also bundle branch block.
Fig. 29. The Q-T Intervals of Six Patients with Bundle Branch Block.
Cases L, L had left bundle branch block, case L.C. left bundle branch block and complete auriculo-ventricular block, cases A right bundle branch block, and case A. intraventricular block of lesser grade.
Fig. 30. The Q-T intervals of two cases during atrioventricular nodal rhythm (A, B) and during normal rhythm (A, B).
Fig. 31. The Q-T Intervals of Five Patients with Low Voltage, One of Whom Had Also Complete Atrio-Ventricular Block.
the normal upper limits. Of the three cases of complete auriculo-ventricular block without bundle branch block only one had a Q-T interval beyond normal limits and that was close to the line (fig. 28).

K. Bundle branch block. Six further cases of bundle branch block (in addition to the three already noted under complete auriculo-ventricular block, and the one under aortic stenosis) have been studied. Two showed right branch block and in one of these the Q-T interval was just below and in the other just above the outer limit of normal. Three cases showed left bundle branch block; the Q-T interval in one of these was just within normal limits, one was on the border line itself, and one was very far beyond normal limits (nearly 0.7 second). In this last case, complete heart block was also present and so that instance should be added to the three already described with extremely long Q-T intervals under the heading of complete heart block. The sixth case showed a lesser grade of intraventricular block and its Q-T interval was barely within normal limits. However, all six cases were well above the normal average for duration of Q-T interval and near or beyond the upper normal limits. This finding is to be expected in view of the slow distribution of stimulus through the ventricles and inefficient onset of ventricular contraction (fig. 29).

L. Atrioventricular nodal rhythm. Two cases of the rare atrioventricular nodal rhythm were studied, one also during normal rhythm and the other during auricular fibrillation. Both showed normal measurements for the Q-T intervals in each instance (fig. 30).

M. Low voltage of electrocardiogram. Finally, five cases showing low voltage by electrocardiogram were studied. All showed Q-T intervals within normal limits except one with a very low pulse rate (38) due to complete heart block (fig. 31).

SUMMARY AND CONCLUSIONS

1. A study is here reported of the measurements of the Q-T interval made by us on carefully selected electrocardiographic plates with the help of the Lucas comparator in 213 individuals, of whom 50 were normal to act as controls (20 men, 15 women and 15 children), and 163 were abnormal subjects to illustrate the effect of various pathological conditions.

2. A brief review of the literature concerning the effect of various
factors on the duration of systole has been given, and references to papers listed in a bibliography.

3. The interval from the beginning of the Q-R-S complex to the end of the T wave in satisfactory electrocardiograms may be considered as indicative of the duration of electrical systole, and perhaps is a more accurate measurement of actual systolic duration than any other. The more accurately mechanical records of ventricular contraction are obtained, the more closely does the duration of mechanical systole approach that of electrical systole.

4. The prime factor influencing the duration of both mechanical systole and the Q-T interval of the electrocardiogram has been found almost invariably by previous workers to be heart rate. This we have confirmed in our electrocardiographic study. The faster the heart rate the shorter the duration of systole and of the Q-T interval, although at faster heart rates the relative proportion of the heart cycle made up by systole steadily increases, as shown by the slope of the curve in figure 1. Whether the amount of blood entering the heart at various rates is a controlling factor is not clear.

5. As a standard for judging the normal duration of electrical systole we have used a chart on which have been plotted according to heart rate and duration of the Q-T interval (coördinates) first the measurements of our 50 normal cases (fig. 1) and then those of 190 normal cases from the literature (fig. 2). The outer limits of these 240 normal case measurements have then been joined by curve lines and the resulting figure has been used for judgment as to variation from the normal of our 163 abnormal cases. This method has seemed to us considerably more satisfactory than the application of any formula (no suitable formula has been devised). Also our method has the advantage of rapid graphic illustration. Against the normal curves have been plotted the measurements of the Q-T interval in all of our 163 abnormal cases divided into groups according to the abnormal condition present.

6. For the 240 normal cases the outer limits of normal for the Q-T interval varied from 0.480 to 0.300 second at a heart rate of 50 and heart cycle of 1.2 second to 0.262 to 0.214 second at a heart rate of 150 and heart cycle of 0.4 second. There is thus at slower pulse rates a wide normal variation perhaps due to variations in diastolic filling of the heart. The curve (fig. 2) resembles somewhat a comet track.
7. In normal individuals, the Q-T interval remained normal with change in position, exercise and digitalization.

8. Of all the 163 abnormal cases, only two gave a duration of Q-T interval slightly shorter than normal and those were both cases with hyperthyroidism, one of whom also had auricular fibrillation and the other a well marked tachycardia. Both of these cases deviated but little from the normal (0.018 second in one and 0.025 second in the other). No case in the entire series of 163 abnormal individuals gave a duration of systole much shorter than normal.

9. A number of the 163 abnormal cases showed Q-T intervals longer than normal. Of these the most striking and uniform were four groups, first those cases with bundle branch block (six out of ten, the remaining four being on or close to the upper edge of normal), second those cases with very low blood serum calcium content (both of two cases), third the complexes of ventricular paroxysmal tachycardia (both of two cases) and of ventricular premature beats (all of four cases), and fourth four out of five cases of auricular paroxysmal tachycardia. Bundle branch block, ventricular premature beats and ventricular paroxysmal tachycardia are alike in the abnormal and slow distribution of the excitation wave through the entire ventricular musculature. The rates for the auricular paroxysmal tachycardia were so fast that we had no normal curves for comparison except by projection which is based on supposition only.

10. The slow pulse of complete heart block (seven of nine cases, four of which seven, however, had bundle branch block also and one low voltage), of one case of sinoauricular bradycardia (out of seven), and of one case of partial auriculoventricular block (out of five) was associated with an abnormally long Q-T interval. The longest intervals of all were found in four cases of combined bundle branch block and complete heart block and they were 0.718 second (at rate of 36), 0.668 second (at rate of 34), 0.638 second (at rate of 23) and 0.632 second (at rate of 45). These were the only Q-T intervals in the entire series of 213 cases which measured more than 0.6 second long.

11. A few other scattered cases showed Q-T intervals slightly longer than normal. They were one case (out of five) of uremia, one case (out of nineteen) with a very large heart, one case (out of five) of
auricular premature beats (three others were on or near the upper normal limit line), and one case of auricular fibrillation with a very rapid ventricular rate. Thus there were altogether of the 163 abnormal cases, 22 which showed overlong Q-T intervals and 18 of those showed either bundle branch block, very slow pulse rates, as in complete heart block, very fast pulse rates as in auricular or ventricular paroxysmal tachycardia, ventricular premature beat complexes, or too low a content of calcium in the blood serum.

12. Of pathological conditions, cardiac enlargement, aortic regurgitation, aortic stenosis, mitral stenosis, congenital cardiac defects hypertension, diabetes mellitus, hyperthyroidism, hypothyroidism, acute infection, congestive failure, auricular fibrillation, atrioventricular nodal rhythm and low voltage in themselves did not affect the duration of the Q-T interval in the series studied here.

13. The measurement of the duration of the Q-T interval of the electrocardiogram is apparently of little or no clinical value.

We take great pleasure in acknowledging the help that has been given to us in the measurements of the Q-T interval by Drs. Howard B. Sprague, T. Duckett Jones, J. Francis Kellogg, and Paul V. Ledbetter.

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