alone there was a recurrence within three months in three. This was regarded as an unsatisfactory result. Although one case out of 32 treated with oral penicillin and sulphadimidine suffered a recurrence in three months, it must be borne in mind that this group included by far the greatest number of severe cases. The satisfactory end-results in 93 cases of acute otitis media treated with chemotherapy alone are held to obviate the necessity for surgical intervention. The results of bacteriological examination in 11 cases indicate that the majority of cases are probably due to infection with penicillin-sensitive organisms, but that non-sensitive bacteria, particularly *Bact. coli*, form an important minority. It is held that the co-administration of sulphonamides with penicillin will effectively deal with most of these infections. This is particularly important in those cases (the majority) in which perforation of the ear-drum has not occurred and, as a result, the nature of the invading micro-organism cannot be ascertained. In the occasional instance in which a more specific antibiotic is required, combined therapy will stay the infection until the result of bacteriological examination is known.

Summary

The role of chemotherapy in acute otitis media is discussed and the advantages of combining penicillin and a sulphonamide are outlined. The synergistic action of these two drugs and the desirability of co-administration in mixed infections is stressed. The literature on oral penicillin administration is surveyed, and it is concluded that this is an effective and convenient method of administering the antibiotic.

The results in 93 cases of acute middle-ear infection are described. Of these, 61 were treated with either oral penicillin alone or oral penicillin and sulphadimidine, according to severity. Out of 34 severe cases treated in this manner, perforation of the tympanic membrane had already occurred in 19 before treatment was begun. The results were uniformly good, with no permanent sequelae or after-impairment of hearing. Healing of the ear-drum occurred in all cases in which perforation had occurred. The bacteriology in 11 cases showed a preponderance of infection with *Staph. aureus* and *Str. haemolyticus*. However, the infection was usually a mixed one, with *Bact. coli* and *Proteus* as the usual co-invaders.

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THE Q-T INTERVAL IN MYOCARDIAL INFARCTION

BY

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The Q-T interval of the electrocardiogram is measured from the beginning of the Q wave to the point where the T wave ends at the isoelectric line. It is an indication of the total time occupied by ventricular excitation and recovery. Recently, great interest has been evinced in this hitherto neglected feature of the electrocardiogram, and its variations have been reported in a variety of conditions.

A statement in a leading textbook of electrocardiology (Wolff, 1950) that "abnormal Q-T occurs regularly as a result of myocardial injury. If QRS is normal, the prolonged Q-T must signify a profound disturbance in the recovery process" aroused interest in this question, and, as a search of the literature of the subject was barren in its results, this particular study was embarked on.

Clinical Material and Methods

As this study was undertaken to evaluate the significance of the Q-T interval in myocardial infarction particularly, it was decided to rule out all possible variables. For this purpose all the normals which serve as controls were selected from the ages of 40 and above. It was also made absolutely certain that all the normals had no electrocardiographic abnormality. The group of cases of myocardial infarction selected was also carefully scrutinized to exclude any which showed any changes of rhythm, of ventricular hypertrophy, of bundle-branch block, or of hypertensive heart disease, or which demonstrated either erect or inverted U waves. Also, any patient who had had digitalis (Berliner, 1931) or who had been on a regime of mercurial diuretics was discarded.

A total of 600 normal tracings (400 males and 200 females) and 800 tracings of myocardial infarction were studied. All the tracings had been taken on the standard Cambridge string-galvanometer electrocardiogram, by the same individual, with the same standardization (1 mV=1 cm.). In the examination of each tracing a strict routine was adhered to, the tracings being examined under a powerful magnifying-glass. Any tracing which showed any abnormality in the shape or the duration of the QRS complex was discarded, as were also all tracings in which the end of the T wave was not clearly definable. Each Q-T interval was measured from the point at which the Q wave (in the absence of the Q wave, the R wave) left the isoelectric line to the point where the T wave after its excursion returned to the isoelectric line. Each R-R interval was measured from the peak of the R wave of one complex to the peak of the R wave of the next complex.

To calculate the Q-T and R-R intervals the following procedures were followed: (1) Any two Q-T and R-R intervals were measured in each of Leads II, aVF, V₂, and V₄, giving a total of eight separate values for both the Q-T and the R-R intervals. (2) The Q-T₀-Q-T interval corrected to the rate (Taran and Szilagyi, 1947) of each corresponding Q-T and R-R interval—was worked out as explained below. (3) The average of these eight Q-T₀'s was taken as the final Q-T₀ figure. This procedure was followed in each tracing throughout the 1,400 tracings studied.

Methods of Calculation of Q-T₀

Various formulae have been recommended from time to time for the correlation of the Q-T interval to the cardiac cycle. The one that is simplest and has stood the test in most studies on the Q-T interval is that formulated by Bazett (1920):

$$S = K \sqrt{C}, \text{ where } S = \text{systole} = (Q-T) \\ C = \text{cycle-length} = (R-R) \\ K = 0.37 \text{ second for males and } 0.40 \text{ second for females}$$

Taran and Szilagyi (1947) introduced a very important new conception—the Q-T₀—namely, the duration of the Q-T interval corrected for the cycle-length. By the use of this modification, Bazett's formula standardizes the Q-T interval to a heart rate of 60 a minute, or, in other words, to a cycle-length (R-R interval) of 1 second. Now, the formula can be expressed as:

$$Q-T_0 = \frac{Q-T}{\sqrt{R-R}}$$

To obviate tedious calculations involved by this formula, use was made of the slide-rule method (Fox *et al.*, 1950), based on Taran and Szilagyi's modification of Bazett's formula. After the first few calculations on the slide-rule, Kissin's nomogram (Kissin *et al.*, 1948) was used in conjunction with the slide-rule. This nomogram is based on Taran and Szilagyi's modification of Bazett's formula, and employs exactly the same principle as the slide-rule method. Since no differences were recorded in the use of either of these methods, after the first 200 readings, Kissin's nomogram was employed in all cases.

All figures obtained were statistically analysed by the formula $\sigma = \sqrt{\frac{\sum(x-\bar{x})^2}{n}}$ and found to be admissible.

Results

The results of the normal electrocardiographic tracings of the 400 males and 200 females, divided into age groups, are shown in Table I. In both the normal males and females

TABLE I.—Q-T₀ Plotted Against Age for Normal Males and Females (Aged 40 and Over)

Age Group	No. of Cases		Mean Q-T ₀ in Seconds		Q-T ₀ Range in Seconds	
	Male	Female	Male	Female	Male	Female
40-49 ..	124	97	0.398	0.415	0.392-0.404	0.409-0.421
50-59 ..	163	54	0.397	0.404	0.392-0.402	0.399-0.409
60+ ..	113	49	0.388	0.400	0.379-0.397	0.391-0.409
Total ..	400	200	0.394	0.406	0.387-0.401	0.399-0.413

there seems to be an inverse relationship between age and the duration of Q-T₀, but when the χ^2 test was applied no significant difference was found. Sex, however, definitely

TABLE II.—Comparison of Q-T₀ in Seconds in Normal Subjects Obtained by Various Authors

Author	Males	Females	Average Q-T ₀
(1) Bazett (1920) ..	0.370	0.400	0.385
(2) Dock (1931) ..	0.3841	0.3890	0.3885
(3) Cheer (1930) ..	0.3743	0.3877	0.3810
(4) Taran and Szilagyi (1947)	—	—	0.3924
(5) Alimurung <i>et al.</i> (1950)	0.403	0.400	0.4015
(6) Krasnoff (1950) ..	0.392	0.406	0.399
(7) Present study ..	0.394	0.406	0.400

In studies (1), (2), (3), and (5) "K" is used instead of "Q-T₀".

seems to play a part, the average Q-T₀ for normal males being 0.394 second, with a range of 0.387 to 0.401 second; and the average for females 0.406 second, with a range of 0.399 to 0.413 second.

These "control" readings are compared with those obtained by other authors (Table II). It will be noticed that almost all the figures approximate one another closely. Moreover, if it is appreciated that the studies of Taran and Szilagyi and of Alimurung *et al.* contained only children, further proof is added to the statistical contention already stated that Q-T₀ has no correlation with age.

Q-T₀ in Cases of Myocardial Infarction

Eight hundred electrocardiographic tracings following myocardial infarction were studied (496 of males, 304 of females). The procedure used in measuring the Q-T₀ was exactly as already outlined. The Q-T₀'s in males and females were plotted against the localization of the infarct, and this is shown at various stages of the infarct—on the first day, at the end of the first week, at the end of the first month, at the end of the sixth month, at the end of a year, and at varying periods after a year. Tables III and IV demonstrate the changes taking place in males and females respectively.

TABLE III.—Average Q-T₀ at Various Periods After Myocardial Infarction in Males, Plotted Against Localization of Infarct

Localization of Infarct	No.	Average Q-T ₀ in Seconds					
		1st Day	1st Week	1st Month	6th Month	1st Year	1 Year +
Antero-lateral	238	0.425 (39)	0.485 (35)	0.475 (60)	0.460 (64)	0.443 (13)	0.413 (27)
Posterior	192	0.430 (32)	0.430 (36)	0.417 (52)	0.416 (33)	0.443 (20)	0.413 (19)
Antero-septal	66	0.431 (19)	0.427 (16)	0.415 (20)	0.411 (11)	—	—

The figures in parentheses show the total number of tracings in each group.

TABLE IV.—Average Q-T₀ at Various Periods After Myocardial Infarction in Females, Plotted Against Localization of Infarct

Localization of Infarct	No.	Average Q-T ₀ in Seconds					
		1st Day	1st Week	1st Month	6th Month	1st Year	1 Year +
Antero-lateral	112	0.414 (30)	0.460 (34)	0.434 (35)	0.430 (6)	0.421 (7)	—
Posterior	108	0.470 (32)	0.465 (32)	0.450 (24)	0.435 (9)	0.418 (11)	—
Antero-septal	84	0.474 (17)	0.466 (21)	0.431 (25)	0.424 (8)	0.414 (13)	—

The figures in parentheses show the total number of tracings in each group.

It will be noticed that exactly the same pattern is followed in both the sexes. In antero-lateral infarctions, after a preliminary rise of the Q-T₀ interval on the first day, there is a further maximal rise at the end of the first week, after which it gradually regresses, but fails to reach normal levels. In the posterior and antero-septal infarctions the maximal rise is seen on the first day, and then the Q-T₀ interval regresses, until after a year it is nearing normal limits. Hence it is evident that the localization of the infarct has no direct bearing on the Q-T₀, this particular interval being always prolonged in the acute stages and gradually tending to return to normality with the healing of the infarct.

TABLE V.—Average Q-T₀ in Males and Females at Various Periods After Infarction

Period of Time Elapsed After Infarction	Average Q-T ₀ in Seconds			
	Males		Females	
	No.	Q-T ₀	No.	Q-T ₀
1 day ..	90	0.425	79	0.423
1 week ..	87	0.458	87	0.482
1 month ..	132	0.450	84	0.470
6 months ..	108	0.440	23	0.454
1 year ..	33	0.424	31	0.423
1 year + ..	46	0.414	—	—

Chart showing average Q-T_c in males and females at various periods after infarction.

Since the localization of the infarct does not play a significant part, but the stage of the infarct does, it was decided to tabulate the Q-T_c's against the period of time elapsed after infarction, irrespective of the localization. This is shown in Table V and the Chart.

Discussion

The Q-T interval of the electrocardiogram represents the period of electrical systole of the heart. Katz (1921) suggested that measuring the duration of systole in diseased hearts compared with normal hearts would give a method of determining the functional integrity of the myocardium. White and Mudd (1929) pointed out that the Q-T interval is prolonged in paroxysmal tachycardia or disorders causing a marked widening of the QRS complex, but not in conditions associated with structural defects. Dock (1931) did not regard the Q-T interval as a satisfactory index of cardiac function, since only one-third of patients with failing hearts have electrical systoles longer than the maximum in normal subjects of the same sex and age. Moreover, he found that the duration of the Q-T interval in hypertensives with left ventricular enlargement without failure is the same as in those who are badly decompensated.

In 1939 Ashman disagreed with the above authors, and showed that measurement of the electrical systole gives valuable information regarding the degree to which the myocardium is affected in cases of diphtheria or acute rheumatic carditis. Taran and Szilagyi (1947) confirmed this in cases of acute rheumatic carditis, though their results are vigorously contested (Alimurung *et al.*, 1950). Similar prolongation of the Q-T interval has been noted in cases of hypopotassemia (Bellet and Dyer, 1937; Stewart *et al.*, 1940; Brown *et al.*, 1944; Currens and Crawford, 1950); and in cases of hypocalcaemia (Carter and Andrus, 1922; Kellogg and Kerr, 1936; Barker *et al.*, 1937).

Katz (1921), Robb and Turman (1947), and Horwitz and Graybiel (1948) have shown that many physiological factors such as cellular metabolism, sex, and position may influence the Q-T interval. Berliner (1931), Bayley (1942),

Savilahti (1945), and Krasnoff (1950) have shown that prolongation of the Q-T interval occurs in hypertension, bundle-branch block, ventricular hypertrophy, etc. (pathological cardiac states). Hence it may be taken as fairly certain that ischaemia and injury of the myocardium have a bearing on the duration of the Q-T interval.

As all these various factors may be playing a part, it is difficult to estimate the effects of one or many of these possible factors on the prolongation of the Q-T interval. However, this study definitely supports previous work of Krasnoff (1950) that the Q-T interval is prolonged in the early stages of myocardial infarction, and gradually diminishes until, after about one year, average normal levels are approached. There are good grounds for believing that the prolongation of the Q-T interval is directly related to the myocardial damage, though the immediate mechanism remains unknown.

A good correlation exists between these results and the findings of Mallory *et al.* (1939), which demonstrated the changes in the cardiac musculature following acute myocardial infarction. These results are summarized in Table VI. It will be seen that necrosis of the musculature starts on the first day, and reaches a maximum at one week, when there is maximum dead and dying muscle. At the end of a month granulation tissue is present, and clearance of the dead muscle is being effected, and collagen has started to be laid down—the earliest signs of repair. At the end of six months repair is fairly well advanced, and at the end of a year there is maximum deposition of collagen.

The Chart shows that the changes in the Q-T_c interval closely follow this pattern. Thus the Q-T_c interval increases on the first day, reaches its maximum at the end of a week, when cardiac damage is at a maximum, and then gradually recedes as the process of repair sets in.

Hence the Q-T_c interval may prove to be of use in estimating the duration of rest in bed, so that activity is indulged in only when a firm process of repair has set in.

Summary

The Q-T interval is dependent on the cardiac rate. The average normal Q-T_c was found to be 0.394 second, with a range of 0.387–0.401 second for males, and 0.406 second, with a range of 0.399–0.413 second for females. The Q-T interval has no relation to age. It was found to be prolonged in cases of acute myocardial infarction reaching a maximum at the end of the week, gradually approaching normal limits again after about one year. A definite correlation is shown between the length of the Q-T_c and pathological changes in cardiac musculature of cases of acute myocardial infarction. The Q-T_c bears no relation to the localization of the infarct.

The methods of selection of cases, the choice of the formula used, and the methods of measurement are described in detail.

TABLE VI.—Pathological Changes in Cardiac Muscle Following Myocardial Infarction in Relation to Time-interval (Modified from Mallory *et al.*, 1939)

Time Interval	Necrotic Muscle	Polymorphs	Blood Vessels and Connective Tissue	Removal of Fibres	Pigmented Macrophages	Eosinophils	Plasma Cells and Lymph	Collagen
1 day	++	+	0	0	0	0	0	0
1 week	++++	+++	++	+	+	+	+	+
1 month	+	0	++++	++	++++	0	++++	++
6 months	0	0	++++	?	+	0	+	+++
1 year+	0	0	++++	0	+	0	0	++++

The relation of the Q-T interval to various physical, chemical, and pathological states is discussed.

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CHLOROQUINE IN THE TREATMENT OF AMOEBIC LIVER ABSCESS

BY

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The antimalarial action of the derivatives of the 4-aminoquinoline series was thoroughly established during the last war, and from among the series chloroquine (7-chloro-4-(4-diethylamino-1-methylbutylamino)-quinoline diphosphate) was found to be one of the most effective compounds, and to be of low toxicity. Observations revealed that the drug had a peculiar depot action, being stored in the liver in concentrations as high as 500 to 600 times the plasma level (Conan, 1948). This property of the drug, as well as its pronounced antiplasmodial activity, led to the testing of its possible efficacy in protozoan infections other than those caused by malaria plasmodia. Tests carried out in England and America immediately after the war showed that it had a pronounced action in *Entamoeba histolytica* infections of the liver that were responsible for hepatic amoebiasis and liver abscesses. These experimental findings now seem to be generally confirmed by clinical trials.

Conan (1949) used chloroquine successfully in seven cases of amoebic hepatitis and found that it was an effective chemotherapeutic agent for the treatment of amoebic infections of the liver, being at least as effective as emetine but apparently without the latter's toxicity. In the same year Manson-Bahr (1949) reported his experience with chloroquine in a case of *E. histolytica* liver abscess in a very advanced stage, which could not

be influenced either by emetine or by streptomycin and penicillin. Fever and other symptoms persisted in spite of the latter. He then resorted to treatment with chloroquine and observed a prompt recession of the symptoms. The temperature was soon brought down to normal, and healing of the abscess was claimed after a fortnight's treatment. Similar results were reported by Murgatroyd and Kent (1948) in their case of liver abscess, which had proved very resistant to emetine, di-iodohydroxy quinoline, streptomycin, penicillin, and local emetine lavages. More comprehensive work on the subject has been reported by Harinasuta (1950, 1951), working in Siam, where hepatic amoebiasis is of frequent occurrence. In his first paper he reported nine cases of amoebic liver abscess (two with pulmonary complications) successfully treated with chloroquine. Later he made a comparative study of the action of chloroquine and emetine in amoebic liver abscesses, putting 45 cases on chloroquine treatment exclusively and an equal number of cases on emetine. In conclusion, Harinasuta remarked: "In comparison with emetine, chloroquine appeared to possess as much curative value in the treatment of amoebic liver abscess. It might be somewhat inferior on the point of relapse rate; but in other respects it had its own advantages—e.g., peroral administration, tolerability, and low toxicity in general—so that it might be employed even in cases in which emetine was contra-indicated." Among the side-effects of chloroquine Harinasuta listed nausea, vomiting, mild diarrhoea, transient pruritus, disturbance of ocular accommodation, and also a slight fall in the blood pressure. He noted, however, that these side-effects were usually mild and insignificant and only transitory in nature.

Among the publications by Indian research workers reference may be made to those of Sharma *et al.* (1951) and of Subramaniam (1951). The former reported on two cases of amoebic liver abscess from Bangalore which proved resistant to treatment with emetine, vitamin B-complex preparations, sulphonamides, and penicillin. At the time of chloroquine therapy the patients appeared to be so gravely ill that their chances of recovery seemed to be extremely small. The authors concluded: "Our impression was that the administration of chloroquine marked a turning-point in an otherwise downward course. Our cases are in line with the striking results by chloroquine reported by others, and the drug marks a definite advance in the treatment of amoebic hepatitis, especially cases with abscess formation." Subramaniam used chloroquine diphosphate only in four cases of amoebic hepatitis, but claimed good results with this exclusive treatment. He stated that in all cases the temperature subsided within 24 to 48 hours, there was a marked lessening of the pain, and the patient felt better.

Present Series

I have used chloroquine diphosphate ("resochin") in a series of 11 cases of amoebic liver abscess, and the results obtained seem to confirm the observations of others that this drug has advantages in the treatment of extra-intestinal infections with *E. histolytica*.

The patients were admitted to the King Edward Memorial Hospital or to the P.G. Singhanee Hindu Hospital, and were kept there throughout the course of treatment. They were asked to return if there were any symptoms suggesting relapse, and each was given a postcard to be filled in by a doctor treating him for the complaint. Each patient also received another card with particulars of the previous history and treatment given.