Length of Cardiac Cycle and QT Interval During Exercise and Recovery in Athletes and Nonathletes (36440)

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It is well known that the QT interval often shortens with an increase in heart rate. Bazett (1) has shown that the duration of the ventricular complex may be estimated by the equation $QT = K$ (cycle length)$^{1/2}$. For men, he found the K was 0.37 sec at rest. After exercise the K is increased. He stated that it could be altered in the absence of a normal balance between vagal and sympathetic activity. Heinen and Loosen (2), using this formula, showed that during exercise the calculated QT shortens before the measured QT changes and that after exercise the QT is shorter than predicted. Yu *et al.* (3) state that the QT is prolonged during exercise as estimated by Bazett's equation and shortened during the first 3 min of recovery. Many other equations (see 4–6) have appeared for calculating this relationship.

Methods. These studies were made on two groups: (a) nonathletes (laboratory workers and medical students), and (b) athletes who came to Kuala Lumpur for a final period of training before participating in track events. The subjects were Malays, Indians and Chinese. No differences in results due to race were observed. The room in which the studies were made was maintained at 75°F. The humidity was usually about 60%.

The first measurements were made on a bicycle ergometer (Collins Pedal-Mode ergometer 4300) using work loads of 5 or 10 kgm/sec. Nonathletes (12 laboratory workers) first sat quietly on the bicycle ergometer for 3 min while ECGs were recorded (Glass P7) and while expired air was continuously sampled from a face mask through 1 mm polyethylene tubing having an opening between the nose and mouth. The CO_2 changes were recorded using an infrared analyzer and linearizer (Beckman LB1). The last second of the recorded expiration was considered alveolar air. The ECG leads were taken from the chest—one at V8 position on the back and the other at either V2 or V5 depending on which gave the best recordings. Usually no difference was found in the duration of QT using different leads, though occasionally this might amount to 0.01 sec. The measurements throughout the test were obtained from only one of the two leads.

Recordings were obtained during a period of 3 min at rest, followed by 3 min of exercise and finally 3 min of recovery. With the polygraph running at 100 mm/sec it was usually possible to measure the intervals of the ECG complex accurately to 5 msec. The values given are based on the average of measurements made on at least four cycles, two during expiration and two during inspiration. A calibration of the infrared analyzer using 5% CO_2 in oxygen was recorded once per minute. The error resulting from using dry CO_2 in oxygen as the standard was determined by making various mixtures of CO_2 in room air and analyzing them in a Haldane gas analyzer after they were passed through the infrared analyzer.

The subjects in the group tested on the bicycle ergometer later walked on a treadmill (Collins treadmill P-2000) at 3 mph up a 16% grade for 3 min. In addition, measurements were made on eight medical students who walked or ran on the treadmill with a 16% grade for 1 min at 3 mph and for successive minutes thereafter at 4, 5 and 6 mph. Recordings were also obtained during 6 min immediately following the exercise. Similar measurements were made on two groups of athletes who walked or ran up to 9 mph on the treadmill.

Results. At rest young subjects often show

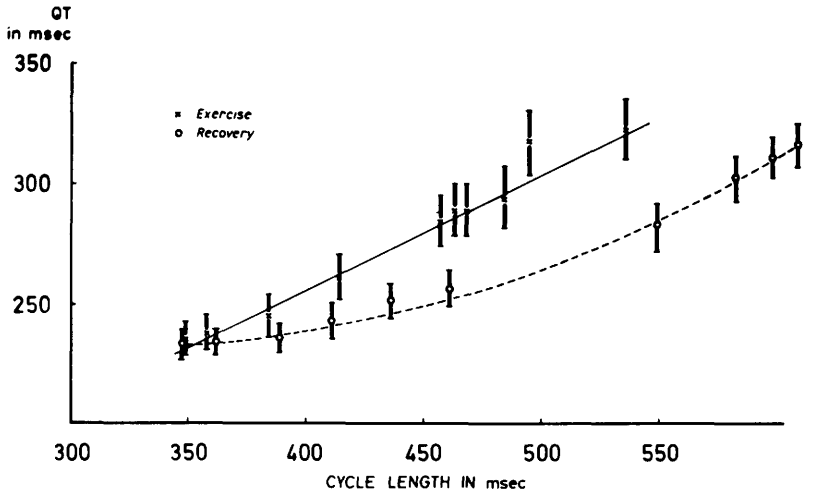

FIG. 1. The effects of exercise and recovery on the RR and QT intervals in athletes.

widely varying lengths of cardiac cycle which are related to the phase of respiration (sinus arrhythmia). These differences are believed to be due to changes in vagal activity (7) but since in man the vagal fibers do not extend into the ventricles (8) changes in ventricular activity, if they are brought about by the vagus, are due to atrial changes which affect the RR interval and only indirectly bring about changes in ventricular responses. In our subjects the duration of QT interval was not affected by the phase of respiration. In one resting subject the cycle length varied from 0.78 to 1.00 sec but the QT interval remained at 0.36. During rest, therefore, there was often no correlation between the RR and the QT intervals.

Relation between RR and QT intervals in exercise and recovery. Within 10 sec of the time exercise on the treadmill started the RR interval (cycle length) shortened in the different groups between 172 and 249 msec, whereas the QT decreased only 12–25 msec. These early QT changes were not significant but by the end of the first minute the QT interval had shortened 56–69 msec. A measurable shortening of the QT interval occurred 15 to 30 sec after the start of exercise. The rapid changes in cycle length at the beginning of exercise could occur as a result of decreased vagal tone or increased sympathico-adrenal activity but only the latter could also bring about changes in the length of ventricular systole.

Figure 1 and Table I show the relation between the length of RR and QT intervals in the first group of athletes during exercise and recovery. The equations for the best fit line for the period of exercise in all subjects are shown in Table II. The equation which best fits all of these results is QT = 70 msec +.47RR.

During recovery from exercise (as shown in Fig. 1) the cycle lengthens faster than the QT interval. The slopes and constants showing the relation between RR and QT intervals were quite different from those found in exercise. During the first minute of recovery the equations found for the various groups are shown in Table III.

The time required for the return of the QT interval to the resting level varies with the heart rate or QT length at the end of exercise. In those doing the heaviest work, the time required for the QT interval to return halfway to resting level (assuming the resting QT interval to be 350 msec) in the athletes with a mean heart rate of 172 beats at the end of exercise was 150 sec and for the students with heart rate of 186, was 204 sec. For the other untrained group with a light load of work (3 mph) and a heart rate of 151, half recovery took 75 sec. For 10 kgm of work with a heart rate of 151 the time was 75 sec and for 5 kgm with a heart rate of 108 it was 55 sec. Thus the recovery time for QT

TABLE I. Exercise on Treadmill with 16% Grade (1st group of 11 athletes).

	RR	QT	Alveolar pCO_2
Standing	785 ± 59	348 ± 13	40.6 ± 0.5
Exercise			
3 mph			
0– 9 sec	536 ± 24	323 ± 13	43.2 ± 0.5
10–19 sec	494 ± 20	318 ± 14	43.5 ± 0.6
20–29 sec	489 ± 20	294 ± 13	43.7 ± 0.7
30–39 sec	468 ± 20	289 ± 11	45.2 ± 0.7
40–49 sec	468 ± 23	289 ± 12	45.2 ± 0.8
50–59 sec	457 ± 22	284 ± 11	45.5 ± 0.7
4 mph			
2nd min	414 ± 18	262 ± 9	45.4 ± 1.1
5 mph			
3rd min	384 ± 13	245 ± 9	45.9 ± 1.0
6 mph			
4th min	358 ± 11	238 ± 7	46.3 ± 1.1
7 mph			
5th min	349 ± 9	236 ± 7	45.5 ± 1.0
Recovery			
0– 9 sec	348 ± 9	233 ± 7	49.3 ± 1.3
10–19 sec	362 ± 12	234 ± 5	52.3 ± 1.6
20–29 sec	389 ± 19	236 ± 6	50.7 ± 1.7
30–39 sec	411 ± 23	243 ± 7	50.1 ± 1.3
40–49 sec	436 ± 33	251 ± 8	48.8 ± 1.4
50–59 sec	461 ± 36	256 ± 8	47.0 ± 1.6
2nd min	549 ± 38	283 ± 10	44.9 ± 1.2
3rd min	582 ± 32	303 ± 9	42.3 ± 1.2
4th min	598 ± 29	311 ± 9	40.7 ± 0.9
5th min	609 ± 31	317 ± 10	40.6 ± 0.8

takes longer when the heart rates in exercise are faster. There is no evidence that the return of QT interval to normal is different in athletes than in nonathletes.

Heart rates in exercise and recovery. It was shown that the heart rate of athletes doing the same level of exercise is slower than that of nonathletes. At 6 mph, the students had a rate of 186 ± 3 while the athletes' rate was 156 ± 5. During recovery the heart rates of the athletes were halfway back to their resting level in 113 sec, whereas in the students it took 188 secs and yet they did a lighter load of work. The difference between the two groups would have been even greater if the students had not had such a fast preliminary standing heart rate, 92, compared with the athletes of 77. They may have been more excited before beginning the exercise than were the athletes.

PR interval during exercise. There was a shortening of the PR interval during exercise but this was very difficult to measure accurately. The PR interval at rest in laboratory workers averaged 143 msec at a cycle length of 873 msec and 124 msec at a cycle length of 453 msec. There were large individual differences and poor correlation between cycle length and PR interval.

Alveolar pCO_2 during rest exercise and recovery. The level of alveolar pCO_2 attained at the end of light, or heavy exercise was not measurably different. In the groups doing heavy exercise the alveolar pCO_2 at the end of exercise was 44.7 ± 1.1, 45.5 ± 1.0 and 47.4 ± 1.6 and for light exercise 47.4 ± 0.8. 48.2 ± 0.6 and 47.2 ± 0.6. Fifteen seconds after the exercise was over the athletes showed an increase in the alveolar pCO_2 over that at the end of exercise which averaged 6.8 and 7.9 mm in the two groups and was significant ($p > .01$). In the nonathletes doing heavy exercise there is a slight increase, 3.0 mm which was not significant. This increase in pCO_2 occurred at a time when the rate of respiration was slowing after exercise stopped. The return of the pCO_2 halfway to the resting level required in the athletes 90 sec and in medical students after the first period of exercise, 93 sec and after a second period, 120 sec. For the lighter work on the treadmill (laboratory personnel) the value was 70 sec while on the bicycle ergometer at 10 kgm/sec it was 55 sec and at 5 kgm, 44 sec. There is nothing immediately apparent in the alveolar pCO_2 measurements that sets the athletes apart from the nonathletes.

Discussion. The vagal nerves are very important in regulating resting heart rate. If the vagal endings are blocked by atropine, then the rate increases about 40 beats/min [Robinson *et al.* (9)]. The rapid increase in heart rate at the beginning of exercise is said to be due to release from vagal inhibition (10) and there is no reason to expect this change directly affects ventricular functions or brings about changes in QT interval (8). A decrease in vagal activity alone can increase the heart rate only to about 120 beats/min. In the heavy exercise studied, the faster rates can be explained by assuming increased sym-

TABLE II. Equations for Line of Best Fit Showing the Relation Between QT and RR Intervals During Exercise.

Treadmill			
Athletes, group 1	QT = 64 msec	+ 0.48 RR	(± 0.046)[a]
2	61	+ 0.50	(± 0.055)
Students	86	0.44	(± 0.080)
Lab. personnel	71	0.49	(± 0.046)
Bicycle ergometer			
Lab. personnel,			
10 kgm/sec	53	0.50	(± 0.100)
5 kgm/sec	82	0.40	(± 0.100)

[a] 95% confidence limit for slope.

pathico-adrenal activity. This is known to shorten ventricular systole as well as increase the heart rate (11). Although there is sometimes no correlation between the duration of the QT and mechanical systole (12), the changes in the cycle length and the QT in this study on exercise appeared to be associated. It is interesting to note that the constants found in the best fit curves for exercise (between 53 and 86 msec) are similar in duration to the QRS so that if measurements had been made from the end of the S to the end of the T, the equation perhaps might have been simplified to ST = 0.5 RR but the end of the S wave is often not sharply defined. The equation devised by Bazett (1) fits neither the exercise nor the recovery periods.

The difference in the relation between cycle length and QT interval in exercise and in early recovery suggests that after exercise there is a rapid increase in vagal tone together with slow decrease in the sympathico-adrenal activity. If the change were due entirely to decreased sympathetic activity one would expect the records to follow the exercise curve but in the reverse direction. In studies on dogs we have found that after vagotomy the equation showing the relation between RR and QT during the period when epinephrine is disappearing from the circulation is the same as that while epinephrine is being infused.

There may be many factors other than nervous or endocrine which affect the relation between the RR and QT intervals—possibly the temperature or pH of the environment of the cardiac cells. Yet for short periods during which our subjects exercised when external environmental temperatures were elevated from 75 to 95°F, we found no effect on the RR or QT intervals. There is also no evidence that the pCO_2 had any measurable effect on this relationship since the alveolar pCO_2 increased during exercise at a time when the RR and QT were shortening but immediately after exercise continued to increase when the RR but not the QT intervals

TABLE III. Equations for Line of Best Fit Showing the Relation Between QT and RR Intervals During the First Minute of Recovery From Exercise.

After treadmill exercise			
Athletes, group 1	QT = 156 msec	+ 0.22 RR	(± 0.02)[a]
2	164	0.21	(± 0.08)
Students	226	0.33	(± 0.10)
Lab. personnel	172	0.21	(± 0.02)
After bicycle ergometer exercise			
Lab. personnel,			
10 kgm/sec	162	0.23	(± 0.03)
5 kgm/sec	226	0.13	(± 0.02)

[a] 95% confidence limit for slope.

were lengthening.

Summary. Electrocardiograms obtained during exercise on a treadmill and on a bicycle ergometer have shown that the relation between the QT and RR intervals can be represented by the equation $QT = K + 0.5$ RR when K has a value of 70 msec. This equation was the same in trained and untrained subjects. During recovery from exercise the RR intervals lengthened much faster than the QT intervals.

The alveolar pCO_2 increased during the exercise while QT shortened. Immediately after exercise the alveolar pCO_2 continued to increase at a time when the QT was no longer shortening.

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