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Heart Rate and Catecholamine Contribution to QT Interval Shortening on Exercise

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Summary

Background: QT interval shortens with exercise. Some of this shortening is due to an increase in heart rate, and some is due to other effects of exercise, probably mostly neuroendocrine effects. Data from subjects with cardiac transplants have suggested that non-heart rate-related changes in QT interval on exercise are due to the effects of circulating catecholamines.

Hypothesis: We sought to determine whether changes in plasma catecholamine levels with exercise are an important contributor to non-heart rate-related QT interval shortening.

Methods: Subjects with DDD pacemakers were recruited. Subjects had QT intervals measured at rest, during a low fixed level exercise test designed to increase heart rate to about 110 beats/min, and, after resting, during pacing at a heart rate of 110 beats/min. Catecholamine levels were measured at each stage of the study.

Results: QT interval at rest was 420 ± 12 ms, during pacing 366 ± 16 ms, and on exercise 325 ± 14 ms. This then gave the proportion of QT interval shortening due to heart rate as 68.6 ± 9.3% of total QT shortening, with the range between 35 and 95.6%. There was no proportionality between the degree of QT interval shortening on exercise that was not due to increases in heart rate and changes in plasma catecholamine levels.

Conclusion: Two-thirds of exercise-induced QT interval shortening are due to an increase in heart rate, and one-third to other effects. Changes in plasma catecholamine levels on exercise were not closely related to changes in the QT interval on exercise.

Key words: QT interval, exercise, catecholamines, pacemaker

Introduction

A powerful influence on the QT interval is heart rate with, as is well known, QT interval shortening at higher heart rates.[1] Another important influence is the autonomic nervous system.[2] The autonomic changes associated with exercise shorten QT interval independent of heart rate.[3,4] The action of autonomic activity changes on the QT interval during exercise may be quite substantial.[4–6] In this study we set out to determine whether the non-heart rate-related neurohormonal influence was related to catecholamine status. In particular, some authors suggest that due to the scarcity of vagal innervation of the ventricle the effect of the vagus on the QT interval is minimal, and work on subjects with heart transplants suggests that the majority of the catecholamine influence on the QT interval is due to circulating catecholamines. We therefore chose to measure whether changes in circulating catecholamine levels were related to changes in the QT interval.[7,8]

Experiment Aims

This experiment was designed to answer the question as to whether heart rate or autonomic influences were more important in determining QT interval, and to determine their respective contributions. In addition, we sought to determine whether the non-heart rate-related QT interval shortening on exercise was related to changes in plasma levels of catecholamines.

Methods

The study included eight patients (six men, two women, age range 69–77 years, mean 73 years). Informed consent

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was obtained from the local ethical committee and from all patients. Patients with dual chamber pacemakers (DDD) implanted to treat high-grade atrioventricular block were studied. Those who were taking agents known to influence the QT interval were excluded. Drug exclusions included amiodarone, digoxin, beta blockers, and class I antiarrhythmic agents. There was no evidence of structural heart disease on clinical examination, and hearts were demonstrated as being normal on echocardiography.

Subjects sat on a bicycle ergometer for an initial 5-min stabilization period and were then randomly either paced in DDD mode or exercised for 10 min to increase their heart rate to ± 110 beats/min. Exercise-induced increases in heart rate were achieved by adjusting the bicycle workload appropriately. All patients had stable heart rates for at least 5 min before any measurements were made.

After a recovery interval of 2 h, subjects were crossed over to the other limb of the study and were either exercised or paced while on the bicycle to a heart rate of ± 110 beats/min.

The 10-min period was designed to be sufficiently long to allow for QT interval hysteresis. Pacing rate change was instantaneous, and exercise at a steady state workload produced very quick heart rate changes. Previous workers have suggested that with an instantaneous alteration in heart rate most QT interval changes have occurred by 2½ min in the step-up and 3 min in the step-down phase.[5] A period of 10 min was therefore chosen to allow full QT interval adaptation. The 2-h recovery period allowed any changes in catecholamine levels induced by exercise to subside.

Twelve-lead electrocardiograms (ECGs) were recorded on paper at 1 min intervals throughout the test, using a Marquette ECG machine, the CASE 15 (Marquette Electronics Inc., Milwaukee, Wis., USA). Standard gain (10 mm/mV) and paper speeds (25 mm/s) were used. QT interval was measured at least five times from standard lead II, using a magnifying graticule. The mean of these five values was then used.

Blood samples were collected from hand veins with the hand prewarmed at time −5, 0, 8 and 10 min of both the exercise protocol and the pacing protocol. The samples were kept in ice until they were separated by centrifugation at −20°C and the plasma stored at −20°C for later analysis of catecholamine levels.

Catecholamine levels (adrenaline and noradrenaline) were measured in our laboratory by high performance liquid chromatography in a previously validated setup.[9]

Statistics

Comparisons were performed using a paired *t*-test. Significance was accepted at the 5% level.

QT Terminology

Heart rate at rest, on exercise, and with pacing is designated as HR_{Rest}, $HR_{Exercise}$, and HR_{Pace}, respectively. The QT interval at rest, on exercise, and on pacing is referred to, respectively, as QT_{Rest}, $QT_{Exercise}$, and QT_{Pace}. In this study, QT interval shortening due to the effect of increasing heart rate is referred to as heart rate-related QT interval change, and this is determined by the equation

$$QT_{Heart\ rate} = QT_{Rest} - QT_{Pace}.$$

QT interval shortening due to exercise is defined as $QT_{Exercise} - QT_{Rest}$, determined at the same heart rate for exercise and pacing, and includes both the shortening due to an increase in heart rate and the shortening due to the autonomic changes of exercise.

Heart rate contribution to QT shortening was defined as QT shortening during pacing as a proportion of the total QT interval shortening on exercise and was obtained by the equation

$$QT[\%\ due\ to\ heart\ rate] = [QT_{Rest} - QT_{Pace}] / [QT_{Rest} - QT_{Exercise}] \times 100.$$

Results

QT Data

Data are presented as mean ± standard error. Indications for pacemaker implantation was syncope in five and presyncope in three patients. No patient had symptomatic coronary disease, and none was on any drugs known to affect the QT interval. There were no significant differences between the data obtained at −5 and 0 min, indicating baseline stability, and between 8 and at 10 min, except for very small differences in the QT interval during exercise.

Mean heart rates were, 65.5 ± 1.6 beats/min at rest, 112 ± 3.2 beats/min during exercise, and 107 ± 3.9 beats/min with pacing (p is nonsignificant for HR_{Pace} vs. $HR_{Exercise}$).

Uncorrected QT intervals, as shown in Figure 1, were 420 ± 12 ms at rest, 366 ± 16 ms during pacing, and 325 ± 14 ms on exercise ($p < 0.005$ for $QT_{Exercise}$ vs. QT_{Pace}).

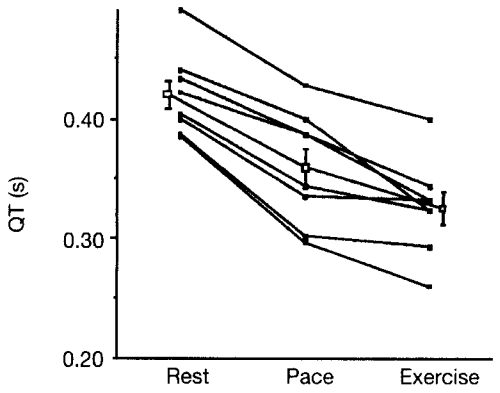

FIG. 1 Effect of exercise or pacing on the QT interval. Exercise shortens the QT interval significantly more than pacing ($p < 0.05$ by paired *t*-test).

TABLE I Noradrenaline levels with exercise/pacing

Time	−5	0	8	10 min
Exercise	489 ± 85	498 ± 98	1199 ± 188[a, b]	1563 ± 316[a, b]
Pacing	505 ± 81	500 ± 94	518 ± 88	560 ± 98

[a] = $p < 0.01$ vs. rest values.
[b] = $p < 0.05$ vs. pacing values.
Levels in pmol/l.

Although there were no significant differences within the group between the paced and the exercise heart rate, implying that it was valid to compare these QT intervals and to assume that heart rate as a variable had been removed, there were small differences, and it was possible that these may have affected the QT interval. Accordingly, we corrected the paced QT interval to the exercise heart rate by assuming a linear relationship between paced heart rate and the QT interval, thus obtaining the slope of the QT interval to heart rate and then correcting the paced QT interval to the exercise heart rate. Even with this correction there were still highly significant differences between the paced and exercise QT intervals with the corrected QT_{Pace} being 359 ± 17 ms ($p < 0.02$ $QT_{Exercise}$ vs. QT_{Pace}).

This then gave the proportion of QT interval shortening because of heart rate as 68.6 ± 9.3% of total QT shortening, with a range of 35 to 95.6%.

Catecholamine Data

There was no change in adrenaline levels with exercise (data not shown). Plasma noradrenaline levels (see Table I and Fig. 2), reached steady state within 8 min of exercise. Samples taken at 8 and 10 min showed no significant difference. Noradrenaline levels at rest preceding either phase or during pacing did not differ, as shown in Table I.

There was no correlation between the exercise-induced component of QT shortening and the absolute or relative increase in noradrenaline (Fig. 3). The small decrease in QT interval in the exercise limb between 8 and 10 min, when the heart rate did not alter further, did not correlate with any further changes in noradrenaline levels between 8 and 10 min.

Discussion

Though in this small study there was considerable variability, it appears that about two thirds of exercise-induced QT interval shortening is due to heart rate effects alone, and the remaining third is due to other exercise-related phenomena, probably neurohormonal. This remaining third is not closely related to resting or exercise plasma noradrenaline levels. Thus, with moderate levels of exercise consistent with day-to-day activities, heart rate contributes the majority of QT interval shortening. Whether this is true at all levels of exercise and for the full physiologic range of heart rates is not known.

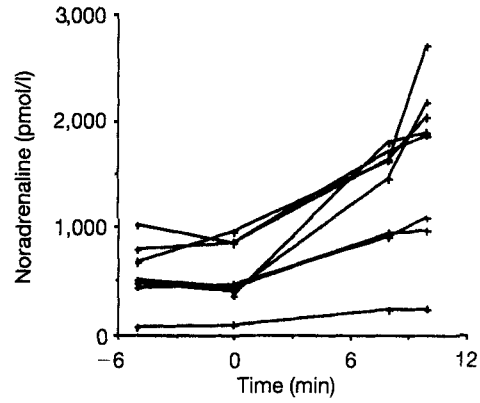

FIG. 2 Effect of exercise on noradrenaline levels. Exercise increases the noradrenaline levels substantially. The increase in noradrenaline is complete by 8 min and there is no further increase between 8 and 10 min.

This study found no relationship between the exercise-related QT interval shortening and changes in plasma levels of catecholamines. One explanation for these data may be that different subjects have receptor and second messenger systems that differ in their sensitivity to catecholamines, that is, that intrinsic cardiac sensitivity to catecholamines may vary between individuals. Furthermore, plasma levels of catecholamines are a poor reflector of cardiac levels of catecholamines, so this lack of a clear relationship does not preclude catecholamines having an important role in inducing QT interval shortening on exercise.[10] An alternative explanation is that catecholamines have no major importance in shortening the QT interval on exercise and that another factor is operating. Support for the relative unimportance of one arm of the

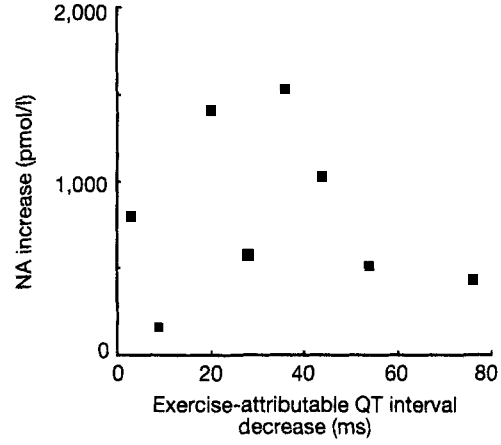

FIG. 3 Relationship between changes in plasma levels of noradrenaline (NA) and changes in QT interval with exercise. There is no relationship between the exercise-attributable decrease in QT interval (i.e., the decrease in QT interval is not attributable to changes in heart rate) and changes in NA levels.

catecholamine system comes from the finding that beta blockers, which lengthen QT interval at low heart rates and shorten QT interval at higher heart rates, have a neutral point (i.e., no effect of beta blockers on the QT interval) at about 120 beats/min.[11] One other possible mediator of QT interval shortening on exercise could be vagal withdrawal, and atropine has rather consistently been found to shorten QT interval.[2] The influence of the vagus was not measured in the current study. The influence of the autonomic nervous system on QT interval shortening during exercise could be explored further by examining the action of beta blockers and/or atropine in this pacemaker model.

Previous QT Interval Studies with Pacemakers

Pacemakers have been used since the early 1980s to dissect out the various influences during exercise on the QT interval.[12] Rickards' group studied subjects with fixed rate VVI pacemakers and determined that exercise had a QT-shortening effect independent of heart rate, and went on to use this effect to design the QT sensing rate-responsive pacemaker.[3] Fananpazir *et al.*, using fixed-rate VVI pacing and atrial synchronized ventricular pacing during exercise to determine the exercise and exercise plus heart rate contribution to QT interval shortening, thus derived the heart rate alone contribution to QT interval shortening.[4] This group thereby determined that the non-heart rate contribution to QT interval shortening on exercise varied from 25 to 84% between individuals. For the population as a whole, between a resting heart rate of 60 beats/min and an exercise heart rate of 120 beats/min, the heart rate contribution to QT interval shortening was 54%, which is a little less than was found in the study reported here. As the heart rate contribution to QT interval shortening was not directly measured in the Fananpazir study, there was an underlying assumption that the effect of catecholamines on QT interval did not depend on the absolute QT interval, and furthermore that the sympathetic response to exercise was the same with fixed-rate VVI pacing as with atrial synchronized ventricular pacing. However, it has been shown recently that there is a higher sympathetic activity during fixed-rate VVI pacing than during DDD pacing, and accordingly this may lead to excess QT interval shortening.[13, 14] This may, therefore, account for the Fananpazir finding that the non-heart rate component of QT interval shortening during exercise was higher than in the study presented here.

In a recent study using QT interval sensing pacemakers, a reasonable correlation was found between changes in noradrenaline on exercise and changes in heart rate, suggesting, unlike the findings in our study, that there is a relationship between QT interval changes on exercise and noradrenaline changes on exercise.[15] This study, however, did not directly measure the relationship between QT interval changes and catecholamine changes, and therefore it cannot be concluded that this study showed a direct relationship between changes in noradrenaline and in QT interval.

Subjects with pacemakers have been used to study the effects of the circadian rhythm on QT interval. Normal subjects with fixed-rate VVI pacemakers show QT interval lengthening at night, when vagal tone is known to be high and sympathetic tone low; heart transplant subjects have blunting of the usual diurnal variation in QT interval, and subjects with diabetes showed no diurnal QT interval variation.[16]

Other Influences on the QT Interval

There are multiple physiologic, pathologic, and drug influences on the QT interval. QT interval variability in normal subjects relates partly to unknown genetic factors, partly to age, with increasing age being associated with longer QT intervals, and partly to gender, with QT interval in men but not in women dropping after puberty by about 20 ms.[17–22] The circadian rhythm is a powerful influence on the QT interval, with prolongation at night, although the mechanism underlying nocturnal QT interval prolongation is not as yet clear.[16, 23, 24] Pathologic influences prolonging the QT interval include the rare genetic illness of hereditary QT interval prolongation.[25, 26] Nutritional status can have a powerful influence on the QT interval with starvation, such as that due to anorexia nervosa, prolonging the QT interval and refeeding shortening the QT interval.[27–30] Alcoholism, particularly when associated with histologic evidence of cirrhosis, is associated with prolongation of the QT interval.[31] A particularly powerful factor resulting in QT interval prolongation is left ventricular damage, with those with the greatest left ventricular damage likely having the longest QT intervals.[32, 33] It may be that the most common cause of acquired QT interval prolongation is that secondary to impaired left ventricular function. In some studies of patients with postmyocardial infarction, those with QT interval prolongation have been shown to be more prone to sudden cardiac death.[34–38] The effect of two drugs on the QT interval is interesting. First, amiodarone prolongs the QTc interval throughout the range of cycle lengths, with the degree of prolongation being partly related to the duration of therapy but not to the thyroid hormone status.[39–41] Those treated for ventricular arrhythmias who had the greatest degree of QT prolongation had the best long-term survival in some but not in all studies.[42, 43] The second drug to have an interesting effect on the QT interval is digoxin which, from observational studies, appears to shorten the QT interval by about 28 ms.[44]

Study Limitations

The study is small and therefore the results should be considered to be preliminary. As ventricular pacing results in QT interval prolongation due to slow intramyocardial conduction, it is difficult to know whether the data presented here are applicable to subjects with normal atrioventricular (AV) conduction and normal duration QRS complexes. We know of no paper in the literature that has compared the results of an intervention in those with normal AV conduction and then investigated the same intervention when subjects are ventricu-

larly paced, as would be needed to show that our results are applicable to those with normal AV conduction.

Whether the proportion of exercise-induced QT interval shortening that is due solely to increases in heart rate remains constant throughout exercise is not answered by these experiments. One might expect that with small increases in heart rate there are only small increases in plasma catecholamine levels, that accordingly a small proportion of the decrease in QT interval is due to non-heart rate-related phenomena, that with larger increases in heart rate there is a far greater increase in catecholamine levels, and that accordingly a larger proportion of QT interval shortening will be due to non-heart rate-related phenomena.

Conclusion

Two-thirds of exercise-induced QT interval shortening is due to heart rate, and the remaining one-third is not closely related to changes in plasma catecholamine levels.

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