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Research Article

Effect of Pre-Exercise Increase in Heart Rate on Subsequent Increase in Heart Rate During Different Stages and Maximal Heart Rate During Treadmill Testing in Normal Persons

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Abstract

There is no study on the effect of pre-exercise (standing from a supine position and subsequent hyperventilation) increase in heart rate on increase in heart rate during various stages of stress and maximal heart rate during treadmill testing in normal persons. We evaluated sixty-three normal individuals after strictly excluding various confounding factors. There was significant interindividual variability in pre-exercise increase in heart rate. Individuals who had an increase of more than forty beats per minute during the pre-exercise period had less increase/no increase or even decrease in heart rate during the first stage of the Bruce protocol. It was due to the cancellation of the exercise-induced increase in heart rate by settling down of pre-exercise increase in heart rate. The subsequent increase in heart rate during the second stage and the third stage was normal. The maximal heart rate attained by all individuals was also normal. An inadequate increase in heart rate during the first stage of Bruce protocol should be interpreted in the context of the increase in heart rate during the pre-exercise period.

Keywords: *Bruce Protocol; Coronary Artery Disease; Heart Rate; Hyperventilation; Exercise Electrocardiography; Prognosis; Treadmill Testing*

Introduction

Prior to exercise on the treadmill, electrocardiograms are recorded in a standing position and after hyperventilation to exclude wrong interpretation of electrocardiographic changes seen during exercise. Standing from the supine position as well as active hyperventilation increases heart rate [1,2]. We observed significant interindividual variation in the increase in heart rate from the supine position to the pre-exercise stage (including standing and active hyperventilation). The effect of the pre-exercise increase in heart rate on subsequent increases in heart rate during various stages of exercise and on maximal heart rate is not known. This is important because the magnitude of the increase in heart rate at different levels of exercise has been correlated with cardiovascular or overall mortality. Savonen., *et al*. [3] observed that blunted heart rate increase between 40 to 100% of maximal workload was associated with increased cardiovascular mortality. Leeper., *et al*. [4] observed that heart rate at one-third of the total exercise capacity significantly predicted both all-cause and cardiovascular risk. Other workers have observed that the inability to increase heart rate commensurate with the increase in workload is associated with increased overall or cardiovascular mortality [5-7]. However, none of the previous studies have considered the pre-

exercise increase in heart rate and its impact on the increase in heart rate during different stages of stress and on maximal heart rate. We, therefore, studied the effect of the pre-exercise increase in heart rate on subsequent increases in heart rate during different stages of exercise and on the percentage of age-predicted maximal heart rate attained at peak exercise in normal individuals.

Materials and Methods

Inclusion criteria

(i) Absence of any cardiovascular symptoms.

(ii) Normal clinical examination.

(iii) Normal resting twelve lead electrocardiogram.

(iv) Normal 2-dimensional and colour Doppler echocardiographic examination.

(v) No new electrocardiographic changes on standing and during active hyperventilation prior to exercise.

(vi) No clinical or electrocardiographic evidence of myocardial ischemia during exercise or recovery.

Exclusion criteria

(i) Patients with contraindications for exercise stress testing [8].

(ii) Conditions that could hamper exercise capacity e.g. debility, orthopaedic problems, haemoglobin concentration less than 10 gm% [9], left or right ventricular dysfunction [10] chronic pulmonary diseases, systemic or pulmonary hypertension.

(iii) Conditions that could affect the heart rate response to exercise e.g. autonomic neuropathy, paced ventricular rhythm, use of beta-blockers, diltiazem or verapamil.

(iv) Unexplained resting heart rate of less than 60 beats per minute. Such individuals could have sinus node dysfunction or increased parasympathetic activity that could affect the heart rate response to exercise.

(v) Conditions that could affect the correct interpretation of exercise electrocardiogram [11] e.g. intraventricular conduction defects, ST-segment or T wave changes in the resting electrocardiogram, presence of the preexcitation in the resting electrocardiogram, electrocardiographic evidence of left and/or right ventricular hypertrophy.

(vi) Development of any bradyarrhythmia, tachyarrhythmia or frequent premature beats during a treadmill test.

(vii) When any possibility of myocardial ischemia could not be excluded with confidence e.g. development of horizontal ST-segment depression of less than 1 mm, upsloping ST-segment depression, ST-segment depression localized to inferior leads with significantly downsloping P-Q segment, the new appearance of isolated shallow inversion of T waves inversion, increasing frequency of ventricular premature beats during exercise or unique appearance of ventricular premature beats during recovery.

Exercise test protocol

(i) Mason-Likar lead system [12] was used. All twelve leads were recorded simultaneously.

(ii) Bruce protocol [13] was followed.

(iii) Exhaustion rather than age-adjusted target heart rate was taken as the endpoint to achieve maximal heart rate and exclude any possibility of myocardial ischemia [14].

(iv) Exercise was terminated if there was the development of angina, any magnitude of ST-segment elevation, ST-segment depression of 1 mm, any bundle branch block, increasing frequency of premature ventricular contractions or inability to exercise from any cause [15].

(v) Ten seconds of the post-exercise cool-down walk was practised to avoid post-exertional dizziness or syncope due to a sudden reduction in venous return or a vagal response due to sudden slopping of exercise [16].

(vi) Recovery was usually recorded for six minutes. It was extended if indicated.

Sixty-three individuals (46 males and 17 females) qualified for the final analysis. Age ranged from 20 to 73 years (44.2 ± 8.09 years).

Evaluation of heart rate

Computer-derived heart rate was used for analysis. Heart rate was calculated manually if it was felt that there was some error in the computer evaluation of heart rate. Age-predicted maximal heart rate was calculated by the formula of Fox., *et al*. (220-age) [17].

Statistical analysis

Individuals were divided into three groups:

(i) Group A- Pre-exercise increase in heart rate of fewer than 30 beats per minute.

(ii) Group B- Pre-exercise increase in heart rate between 20 to 40 beats per minute.

(iii) Group C- Pre-exercise increase in heart rate of more than 40 beats per minute.

The difference between different groups was evaluated using an unpaired 't-test [18].

Results

Group A included 21 males and six females. Group B included 14 males and 11 females. Group C had 11 males. Individuals of group C were significantly younger ($P < 0.05$) than individuals of groups A and B (Table 1). The three groups were comparable ($P > 0.10$) regarding resting supine heart rate, resting supine systolic blood pressure and resting supine diastolic blood pressure (Table 1).

Groups	Group-A	Group-B	Group-C			
Pre-exercise position to start of	<20 bpm	<20-40 bpm	<40 bpm	P Value		
Sex M:F	21:6	14:11	11 males	A vs B	A vs C	B vs C
Age years (mean ± SD)	41.51 ± 11.56	40.88 ± 14.63	32.36 ± 18.93	> 0.10 (NS)	> 0.10 (NS)	< 0.05
Resting supine HR/bpm (mean ± SD)	75 ± 10.72	75.2 ± 9.94	72.92 ± 5.98	> 0.10 (NS)	> 0.10 (NS)	> 0.10 (NS)
Resting supine SBP HR/bpm (mean ± SD)	120.22 ± 10.27	122.5 ± 7.93	122.27 ± 8.76	> 0.10 (NS)	> 0.10 (NS)	> 0.10 (NS)
Resting supine DBP (mean ± SD)	83.48 ± 8.30	81.5 ± 6.19	85.09 ± 6.08	> 0.10 (NS)	> 0.10 (NS)	> 0.10 (NS)

Table 1: *Showing demographic variables in different groups.*

Abbreviation: bpm: Beats Per Minute; HR: Heart Rate; F: Female; M: Male; NS: Not Significant; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure.

Mean values of pre-exercise increase in heart rate increased significantly from group A to group B ($P < 0.001$) and further from group B to group C ($P < 0.001$) (Table 2). An increase in heart rate during the first stage of exercise showed a reciprocal change. There was no significant difference in the increase in heart rate between group A and B. However, the increase in heart rate in group C was significantly lower ($P < 0.001$). There was no significant difference in the increase in heart rate between different groups during the third stage ($P > 0.10$). During stage four, the increase in heart rate was lower in group A ($P = 0.05$) than in groups B and C. By the end of the exercise, all individuals who attained age-predicted maximal heart rate. There was no significant difference in the percentage of age-predicted maximal heart rate attained by different groups.

Groups	Group-A	Group-B	Group-C	P Value		
Increase in HR from the supine position to the start of exercise	< 20 bpm	< 20 - 40 bpm	< 40 bpm	A vs B	A vs C	B vs C
Increase in HR (mean ± SD) pre-exercise	13.97 ± 2.64	27.0 ± 4.99	47.0 ± 15.77	< 0.001 (VS)	< 0.001 (VS)	< 0.001 (VS)
Stage-1	26.03 ± 11.21	27.52 ± 14.90	-0.18 ± 14.10	> 0.10 (NS)	< 0.001 (VS)	< 0.001 (VS)
Stage-2	18.44 ± 6.48	18.6 ± 9.11	15.09 ± 14.57	> 0.10 (NS)	> 0.10 (NS)	> 0.10 (NS)
Stage-3	21.52 ± 16.96	18.08 ± 10.73	23.54 ± 19.30	> 0.10 (NS)	> 0.10 (NS)	> 0.10 (NS)
Stage -4	8.14 ± 9.02	14.0 ± 12.43	16.90 ± 15.08	0.05 (BLS)	> 0.05 (NS)	> 0.10 (NS)
Percent of age-predicted maximal heart rate	91.40 ± 6.03	94.29 ± 10.33	96.36 ± 8.68	> 0.10 (NS)	> 0.05 (NS)	> 0.10 (NS)

Table 2: *Increase in heart rate during different stages of stress during the treadmill test.*

Abbreviation: VS: Very Significant, NS: Not Significant, BLS: Borderline Significant.

Discussion

On standing, there is gravity-mediated pooling of about 300 to 800 ml of blood in the lower extremities and inferior mesenteric area. This result is a decrease in venous return and a resultant decrease in stroke volume. There is a transient fall in blood pressure which results in a reflex increase in sympathetic activity that causes peripheral vasoconstriction and an increase in heart rate [19]. Normally heart rate increases by 10 to 15 beats per minute [20]. Usually, the change in heart rate and blood pressure normalize in one minute [20]. However, the whole process is complex and involves several afferent and efferent neuronal pathways and nuclei in tractus solitarius and other areas of the medulla. The inability of this complex process to respond adequately can result in a greater increase in heart rate and the persistence of this increase over a longer period. This results in significant interindividual variation in heart rate response to standing from the supine position. Anxiety, inappropriate sinus tachycardia and postural orthostatic tachycardia syndrome [21] also contribute to significant interindividual variability.

Active hyperventilation also increases heart rate [22,23]. Release of excitatory neurotransmitters from the brain is probably responsible. Lowered arterial carbon dioxide concentration also results in splanchnic vasodilation via inhibition of the vasomotor centre. The resulting drop in venous return results in the reduced ventricular filling decreased stroke volume and a reflex increase in heart rate. An increase in heart rate depends on the rate, depth and duration of hyperventilation. This contributes to interindividual variation in heart rate response to hyperventilation.

Active hyperventilation soon after standing from a supine position results in an additive effect on an increase in heart rate. This increase in heart rate due to standing and subsequent active hyperventilation gradually settles down to the basal heart rate of the indi-

vidual. This period extends over the first stage of the treadmill test. The magnitude of settling down of the heart rate is proportional to the magnitude of the increase during standing and hyperventilation because the heart rate tends to revert to its basal state. As the exercise progresses during stage one, cardiac output increases to maintain an adequate blood supply to the exercising muscles. This occurs due to neural inputs from mechanoreceptors and chemoreceptors within the active skeletal muscles [24]. Initially the stroke volume increases but it quickly plateaus and the heart rate rises to maintain the progressive increase in cardiac output [25]. Heart rate at the end of the first stage of treadmill testing is, therefore, a sum total of a decline in heart rate that increased due to standing and hyperventilation on one side and an increase in heart rate due to exercise on the other side. This explains our observation that the group which had the maximal increase in heart rate during the pre-exercise period (group C) had the lowest heart rates at the end of the first stage of treadmill testing.

It is believed that a lower heart rate under a standard exercise load suggests better conditioning [26]. Our observations show that an abnormal increase in heart rate during the pre-exercise period can be another cause for a less-than-expected increase in heart rate during the first stage of treadmill testing using the Bruce protocol. Leeper., *et al*. [4] felt that a heart rate rise at one-third of the total exercise capacity significantly predicted both all causes and cardiovascular risk. They however did not consider the pre-exercise increase in heart rate. Our observations show that a less-than-expected increase in the heart rate by the end of the first stage of the treadmill test using the Bruce protocol can occur in normal persons showing have to increase of more than 40 beats per minute in the pre-exercise period.

We observed that increase in heart rate during the second and the third stage was similar in all three groups. This shows that the effect of increased heart rate during the pre-exercise period does not extend beyond the first stage of the Bruce protocol.

During the fourth stage increase in heart rate was significantly less in group A and was maximum in group C. This had no relation to the change in heart rate during the pre-exercise period. It was probably related to the age and gender composition of individuals in the different groups. Persons of group C were significantly younger than individuals in groups A and B. Further, group C had only males. During exercise initial increase in heart rate is due to the withdrawal of parasympathetic tone [27]. Subsequent increase in heart rate is due to increased sympathetic activity [27]. Responsiveness of the sino-atrial node to sympathetic stimulation declines with advancing age due to apoptosis of the sinoatrial node [28] and less calcium influx in the sinoatrial node [29]. Aerobic work capacity also declines with advancing age [30,31] due to age-related changes in skeletal and cardiac muscles [32,33]. All these factors lead to a gradual decrease in maximal heart rate with advancing age. Males have the greater aerobic capacity and, therefore, can take a greater workload and attain a greater heart rate [9,34,35].

There was no significant difference in the percentage of age-predicted maximal heart rate in different groups. This shows that a pre-exercise increase in heart rate did not affect the maximal heart rate attained by an individual. Pre-exercise increase in heart rate was compensated by less increase in heart rate during the first stage. Subsequently, the increase in heart rate was similar in the second and third stages.

Conclusion

Standing from a supine position and subsequent active hyperventilation increase heart rate prior to the start of exercise. The magnitude of this pre-exercise increase in heart rate shows significant interindividual variation. A significant increase in heart rate during the pre-exercise period (more than 40 beats per minute) is compensated by less increase/no increase or even decrease in heart rate during the first stage of Bruce protocol. Subsequently in the second and the third stage, the increase in heart rate is normal with the result that the percentage of age-predicted maximal heart rate is normal irrespective of the pre-exercise increase in heart rate. An increase in heart rate during the first stage of Bruce protocol should be interpreted in the context of the pre-exercise increase in heart rate.

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