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Association between heart rate variability and training response in sedentary middle-aged men

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Abstract The effect of exercise training on heart rate variability (HRV) and improvements in peak oxygen consumption ($\dot{V}O_{2\text{peak}}$) was examined in sedentary middle-aged men. The HRV and absolute and relative $\dot{V}O_{2\text{peak}}$ of training ($n = 19$) and control ($n = 15$) subjects were assessed before and after a 24-session moderate intensity exercise training programme. Results indicated that with exercise training there was a significantly increased absolute and relative $\dot{V}O_{2\text{peak}}$ ($P < 0.005$) for the training group (12% and 11% respectively) with no increase for the control group. The training group also displayed a significant reduction in resting heart rate; however, HRV remained unchanged. The trained subjects were further categorized into high ($n = 5$) and low ($n = 5$) HRV groups and changes in $\dot{V}O_{2\text{peak}}$ were compared. Improvements in both absolute and relative $\dot{V}O_{2\text{peak}}$ were significantly greater ($P > 0.005$) in the high HRV group (17% and 20% respectively) compared to the low HRV group (6% and 1% respectively). The groups did not differ in mean age, pretraining oxygen consumption, or resting heart rate. These results would seem to suggest that a short aerobic training programme does not alter HRV in middle-aged men. Individual differences in HRV, however, may be associated with $\dot{V}O_{2\text{peak}}$ response to aerobic training.

Key words Heart rate variability · Aerobic training · Peak oxygen consumption

Introduction

One of the most pronounced cardiovascular adaptations to aerobic exercise is a lowered resting heart rate (HR). For instance, it has been reported that trained runners often possess resting HR of less than 50 beats min$^{-1}$ (Costill 1986). Similarly, lower resting HR have been shown to occur in sedentary individuals after they have been exposed to aerobic training (Maciel et al. 1985). One mechanism which has been proposed to explain the relationship between training and resting bradycardia has been an increase in parasympathetic or vagal tone (Kenney 1985; Smith et al. 1987). However, support for the influence of increased vagal tone on the heart after training is equivocal.

For instance, results of some studies using pharmacological blockade (e.g. atropine) have indicated that trained compared to untrained subjects possess higher vagal tone (Ekblom et al. 1973; Smith et al. 1987). However, other blockade studies have not detected differences in vagal tone between these groups (Lewis et al. 1980; Maciel et al. 1985). Furthermore, studies using heart rate variability (HRV) as an indirect assessment of vagal tone have also produced equivocal results. For example, Maciel et al. (1985), Reiling and Seals (1988), and Furlan et al. (1993) have not found that trained individuals possessed greater influence of vagal tone on the heart, whereas De Meersman (1992) and Dixon et al. (1992) have.

Factors that may contribute to the inconsistency of these results are the variety and validity of methods used to assess vagal sensitivity, the predominant use of cross-sectional studies, and the use of young individuals as subjects who already possess average or above average levels of influence of vagal tone on the heart. For example, it is possible that training may not substantially influence parasympathetic outflow in individuals who already possess high vagal tone (e.g. young, healthy men). Thus, the existence of a maximum may offset the potential increase in vagal tone generated through aerobic training. Consequently, individuals possessing lower levels of vagal tone may show a greater resting vagal response to training. As vagal tone has been shown to decrease with age (Hellman...
and Stacey 1976) the training of older individuals may demonstrate greater changes in HRV.

A related issue to the exercise–parasympathetic influence relationship is whether pretraining vagal tone influences fitness improvement in response to aerobic training. For instance, some individuals show substantial increases in maximal oxygen consumption (\(\dot{V}O_{2\text{max}}\)) after training, whereas others show little improvement (Bouchard 1984). Past research examining this issue has focused on phenotype factors such as muscle fibre distribution (Costill et al. 1976), substrate availability (Dalrymple et al. 1973), and heart size (Personen et al. 1975). The association between pretraining HRV and \(\dot{V}O_{2\text{max}}\) response to training, however, has not been examined.

Therefore, the purpose of the present study was to assess the influence of aerobic training on HRV of middle-aged men undertaking an aerobic training programme. A second purpose was to examine the association between pretraining HRV and peak oxygen consumption (\(\dot{V}O_{2\text{peak}}\)) response to aerobic training.

### Methods

#### Subjects

Middle-aged, male sedentary subjects were randomly assigned to either a training (\(n = 25\)) or control group (\(n = 15\)). Table 1 shows subject characteristics. All the subjects were screened by a physician before undertaking a maximal exercise treadmill test. A total of 19 subjects completed the exercise programme of the original 25 subjects in this group. 1 subject demonstrated supraventricular tachycardia at maximal exercise, whereas the remaining 5 dropped out of the study because of scheduling conflicts and health problems. The subjects that completed the training programme were then categorized into high (\(n = 5\)) and low (\(n = 5\)) HRV groups (Table 3) based on their pretraining resting HRV.

#### Apparatus

**Electrocardiogram and respiration**

The electrocardiogram (ECG) and respiratory pattern were recorded using a Grass physiograph (model 7D) that was linked to an A-D converter (R.C. Electronics, model ISC-67) and a 386 PC computer collecting at 1,000 samples s\(^{-1}\). For ECG, two electrodes were positioned on the fifth rib either side of the chest and a third to the upper back to the right of the third vertebra of the thorax. Respiratory pattern and rate were assessed by recording the temperature of respired gas. A nasal thermistor (Grass model TCT 1R) that was attached to the main port of a plastic mask produced a signal in millivolts in response to the change in temperature of the respired gas.

**Peak oxygen consumption**

The subjects wore a Hans-Rudolph face mask while walking on a Quinton model Q65 treadmill. Oxygen consumption was recorded through standard open-circuit spirometry. Inspired ventilation was assessed by a Rayfield gasometer, that was interfaced to an Apple II computer. Expired gas was firstly channeled into a 7-l mixing chamber and then analysed using a calibrated Applied Electrochemistry S-3A oxygen analyser and a Beckman LB-3 carbon dioxide analyser. Outputs from the analysers, which were calibrated before each test, were continuously integrated to the computer using Rayfield REP200c software. Oxygen consumption was determined at 15-s intervals.

#### Experimental procedures

All procedures were approved by a Human Investigation Committee. The subjects were tested at the same time of day for both pre- and post-testing and were instructed to refrain from eating, smoking, and ingesting caffeine or alcohol for at least 3 h before the test.

**Maximal exercise capacity treadmill protocol**

The subjects reported to an exercise physiology laboratory and performed a \(\dot{V}O_{2\text{peak}}\) treadmill test (Balke protocol). The subjects walked on a treadmill at 3.3 mph and the gradient was increased by 1% every minute until the subject felt exhausted (Balke and Ware 1959).

**HRV assessment**

Prior to the maximal exercise test the subjects underwent resting HR and HRV assessment. The subjects completed a health questionnaire and then ECG electrodes, the mask containing the thermistor, and headphones were attached. These procedures took approximately 30 min. All explanations and instructions were tape-recorded, and the same tape used for pre- and post-test. The subjects were instructed to perform paced breathing (i.e. to breathe in and out continuously to a count of 4 s in and 4 s out) and they practised at the beginning of the session until they could produce a continuous breathing pattern with a consistent tidal volume. The voltage associated with this tidal volume was recorded and then monitored during the test to ensure that the subjects produced a voltage commensurate with the tidal volume they had recorded during the practice. During the test, the breathing pattern was also monitored and abnormal respiratory patterns (e.g. coughing, breath-holding) were excluded from the data analysis. All the subjects displayed similar breathing patterns before and after the training programme. The assessment period consisted of 15 min of quiet sitting during which ECG was recorded for 64 s beginning at min 5, 10, and 12, and for 90 s beginning at min 13.

#### Treatment

The intervention consisted of 24 supervised exercise sessions. The subjects exercised three times each week at an intensity of 60% of

### Table 1: Biometric data

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Mean SEM</th>
<th>Mean SEM</th>
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</thead>
<tbody>
<tr>
<td>Train (n = 19)</td>
<td>46.2 1.4</td>
<td>45.0 1.4</td>
</tr>
<tr>
<td>Untrain (n = 15)</td>
<td>178.6 1.19</td>
<td>179.6 1.9</td>
</tr>
<tr>
<td>Mass (kg) pretraining</td>
<td>87.1 1.4</td>
<td>83.9 2.4</td>
</tr>
<tr>
<td>Mass (kg) post-training</td>
<td>85.6 3.3</td>
<td>84.9 2.3</td>
</tr>
</tbody>
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