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Gender differences in parasympathetic reactivation during recovery from Wingate anaerobic test

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Abstract

Background and Purpose: *We wanted to investigate gender differences in parasympathetic reactivation from supramaximal exercise.*

Materials and Methods: *Parasympathetic reactivation from a Wingate anaerobic test was investigated in 16 male and 15 female volunteers. Heart rate recovery was assessed as the difference between peak exercise heart rate and heart rate recorded following 60 seconds of recovery (HRR_{60}). The time constant of the first 30 s post-exercise HR (T30) was determined as a negative reciprocal of the slope of the regression line. Another time constant decay (T) was obtained by fitting the 5 minute post-exercise HRR into a first-order exponential curve. Measures of heart rate variability (HRV) were used to describe the changes in autonomic cardiac regulation following exercise.*

Results: *Post exercise heart rate recovery was faster in male participants, demonstrated through HRR_{60} (29.5±8.9 vs. 23.4±9.8 seconds respectively) and T30 (292.4±88.7 vs. 409.2±138.3 seconds respectively), but the time constant of the exponential heart rate decay (T) did not differ between the two genders (140.4±55.7 in males and 130.3±49.7seconds in females). The present study demonstrated similar RMSSD, lnHF and HFnu at rest in male and female participants. The time course of $RMSSD_{30}$ recovery was impaired immediately after exercise. None of the observed vagal HRV indices have restored after five minutes of recovery following the 30-s Wingate test, but the post-exercise $lnHF_{2\text{-}5min}$ was significantly smaller in females (3.3±0.9 ms^2 in males vs. 2.5±1.0 ms^2 in females).*

Conclusion: *The immediate HRR and parasympathetic reactivation was affected by gender and was attenuated in female participants.*

INTRODUCTION

The period following strenuous exercise is thought to be particularly critical to the cardiovascular system *(1).* Sudden changes in cardiac autonomic regulation occur, that may alter cardiovascular homeostasis. The initial rapid decrease of heart rate after exercise cessation is mainly due to prompt restoration of vagal tone, with sympathetic withdrawal becoming more important later in recovery *(2).* To quantify parasympathetic reactivation after exercise, indices such as HR recovery (HRR) and heart rate variability (HRV) have been used *(2, 3, 4, 5, 6).* Vagal related indices such as root mean square of successive difference of the R-R intervals (RMSSD) and high frequency (HF) power of HRV are

widely used methods *(4)* but new parameters, like time-varying parasympathetic indices (root mean square of successive difference of the R-R intervals calculated on successive 30-s segments, $RMSSD_{30}$), have been proposed to describe the changes in autonomic cardiac regulation following exercise more thoroughly *(2)*.

The magnitude and time-course of vagal and sympathetic recovery depends on the preceding exercise intensity *(7)*. To study HRR most authors have investigated cardiodynamics following moderate or maximal aerobic activities *(2, 6, 8)*. Restoration of heart rate (HR) and HF power of HRV is slower and less complete after acute bouts of high intensity compared with long lasting low intensity exercise *(9, 10, 11)*. There are however fewer data on cardiovascular recovery following supramaximal dynamic exercise. It has been previously reported that 5 min after 30-s supramaximal exercise, stroke volume, HR and cardiac output were elevated whereas total peripheral resistance was reduced compared with pre-exercise values *(12)*. Studies assessing autonomic regulation during recovery from supramaximal exercise have reported suppressed HRV *(7, 13)*.

Several publications on gender-related differences in HRV indices provided conflicting results *(14, 15, 16, 17)*. Some report an overall increased autonomic activity in man relative to women with decreasing difference through aging *(15, 17)*; others point out the more favourable autonomic profile in females *(18)*. Most of the articles report pronounced sympathetic modulation in man compared with women *(14, 16)*. On the other hand, vagal tone seems to be more controversial. Some articles reveal no gender-specific differences *(14)*; some show augmented vagal modulation in females *(16, 18)*, others attenuated *(17)*. An overall increased autonomic drive is present in men compared with women, but more detailed assessment of autonomic, metabolic and cardiovascular response to exercise and recovery remains to be elucidated *(15)*.

The purpose of this study was to investigate the gender differences in parasympathetic reactivation from supramaximal exercise. We firstly hypothesize that short supramaximal exertion will alter cardiovascular autonomic regulation and result in incomplete vagal restoration after 5 minutes of recovery, and secondly that there are gender-related differences in immediate HRR and parasympathetic reactivation.

MATERIAL AND METHODS

Subjects

Sixteen sedentary male (age 20.6±1.1yrs, stature 183.3±7.6cm, body mass 76.7±8.7kg, BMI 22.9±2.5) and fifteen sedentary female participants (age 20.7±0.7yrs, stature 170.5±6.9cm, body mass 62.5±8.9kg, BMI 21.5±2.6) gave written informed consent to take part in the study. The participants were not engaged in any organized physical activity for the last six months before the start of the investigation. All subjects were in self-reported good health, without medications and had no medical histories from cardiovascular diseases. Female participants were tested in the midluteal phase of their cycles (18^{th}-24^{th} day). They also underwent a general physical examination to exclude any acute diseases and ailments of the cardiorespiratory and locomotor system. All procedures conformed to the Declaration of Helsinki and were approved by the local ethics committee.

Study design

Measurements were undertaken in a quiet room, air temperature ranging from 22 to 24 ºC, between 9 and 12h. Subjects were instructed to perform no strenuous exercise and to have a solid night's rest the day before the testing, nor to consume alcohol, caffeine or any psychoactive substances.

All participants were familiarized with the testing procedure prior to data collection. After five minutes of quiet rest in the seated upright position on the cycle ergometer, ECG was recorded for five minutes pre-exercise and immediately following a 30-s Wingate anaerobic test (WAnT). Participants performed a WAnT on a cycle ergometer (Wattbike cycle ergometer, Wattbike Ltd, Nottingham, UK). Prior to the exercise test, seat height was adjusted to accommodate the subject`s stature such that the knee would be slightly bent at maximal leg extension. Toe clips were used to ensure maximal safety throughout the testing session. Participants performed a 3-min warm-up session pedaling at a cadence of 80 rpm at a constant power output around 50 W. The warm-up included two to three brief (3-5 s) sprint bouts to maximal cycling speed at higher power outputs *(19)*. After 1-min rest participants were instructed to pedal at full speed in a standing position with the ergometer unloaded for 5s. Then the full breaking force was applied and a 30-s count started. Subjects were verbally encouraged by the researchers throughout the test. Peak power (PP) was measured as the highest power achieved at any 5-s stage of the test. Mean power (MP) was defined as the mean of power output vales throughout the 30-s period of the test. Peak heart rate (HRpeak) was obtained as the highest value of heart rate during the anaerobic test. Post-exercise HR (HRpost) was defined as the HR at the end of a five-minute recovery period.

Assessment of post-exercise heart rate recovery

Digital ECG (VNS-Spektr, Neurosoft, Ivanovo, Russian Federation) was recorded, during exercise and recovery. A sampling rate of 1000 Hz was chosen and recordings were transferred to a PC via a USB interface. The epochs gained from the V5 lead were saved in a computer for further analysis. All R-R intervals were edited

by visual inspection to exclude all the undesirable or ectopic beats. They were deleted with the post extra systolic beat and replaced automatically with interpolated adjacent R-R interval values.

HRR was assessed during the 5 minute period following submaximal cycle exercise by three methods. The first HRR index was defined as the absolute difference between the final HR at exercise completion and the HR recorded following 60 seconds of recovery (HRR_{60}) *(4).* The second HRR index was calculated using a semi-logarithmic regression technique *(20).* The natural logarithm of heart beats during the initial rapid HR decrease (for the first 30 s) was plotted against the elapsed time of recovery, and a linear regression analysis was applied. The time constant of the short-time post-exercise HR decay for the first 30 s (T30) was determined as a negative reciprocal of the slope of the regression line *(20).*

The third HRR index was the time constant decay obtained by fitting the 5 minute post-exercise HRR into a first-order exponential curve *(4, 5, 6).*

The resultant heart rates vs. time data were modeled with an iterative technique using MatLab to fit the following equation:

$HR = HR_0 + HR_\Delta e^{(-t/T)}$ where:

HR = heart rate

HR_0 = stabilized heart rate following exercise

HR_Δ = maximal heart rate – HR_0

t = time (s)

T = decay constant

Time-varying vagal-related HRV indices

Because the R-R interval changes in a curvilinear fashion over the initial 5 min of recovery, no standard short-term HRV measures can be applied. However shorter time intervals (15, 30 or 60 s) better approximate a linear change over time with superimposed oscillations. As Golberger assessed, the optimal duration for short segment analysis of a time-varying vagal-related indices is 30 s *(2).* The square root of the mean of the squares of differences between adjacent R-R intervals (RMSSD) was calculated for each of the 30 s segments of the 5 min recovery ($RMSSD_{30}$) (2).

Short-term resting HRV analysis

Heart rate variability analyses were performed on the last 3 min of the 5 min recovery period to ensure stability of the data *(4).* Vagal-related HRV indices like the square root of the mean of the squares of differences between adjacent R-R intervals ($RMSSD_{2\text{-}5min}$), the natural logarithm of high frequency power density ($lnHF_{2\text{-}5min}$) and the normalized HF power ($HFnu_{2\text{-}5min}$ calculated as HF/(LF+HF) ratio) were calculated for the 3 min recovery period (21).

Data analysis

The distribution of each variable was examined with the Lilliefors normality test. Homogeneity of variance was verified by the Levene`s test. Heart rate recovery measures (HRR_{60}, T) were analyzed for within- and between-group differences. Within-subjects differences among four recovery conditions were analyzed with t-test for dependent samples and between group differences with t-test for independent samples. All statistical analyses were carried out using MatLab 6 software (The Math Works Inc, Natick, MA, USA) and the Statistica 8.0 software package (Statistica, StatSoft®, Tulsa, USA)

RESULTS

Peak power was significantly greater in male (588.4±96.0 W, 7.7±1.5 W/kg) compared with female participants (356.7±48.2 W, 5.4±1.8 W/kg) ($p<0.001$). A similar difference was present in mean power between the male (860.8±192.6 W, 11.3±2.3 W/kg) and female subjects (512.0±61.8 W, 7.8±2.6 W/kg). Heart rate at rest in the upright seated position was lower in the male participants ($p<0.001$). The corresponding peak heart rate values at peak exercise were not significantly different between the two groups. Post-exercise heart rate recovery was faster in male participants demonstrated through HRR_{60} and T30, but the time constant of the exponential heart rate decay (T) did not differ between the two genders. Post-exercise heart rate recovery indices are presented in **Table 1**.

Short-term resting HRV measures pre- and post-exercise are presented in **Table 2**. The present study demonstrated similar RMSSD, lnHF and HFnu at rest in male and female participants. None of the observed vagal HRV indices have restored after five minutes of recovery following 30-s Wingate anaerobic test, but the

TABLE 1

Heart rate and heart rate recovery indexes of male and female participants.

	Male	Female	p
HRrest (beats/min)	90.5±15.7	93.1±14.0	<0.05
HRpeak (beats/min)	187.9±9.5	188.6±7.7	>0.05
HR post (beats/min)	112.0±6.2	117.9±9.9	<0.05
HRR60 (s)	29.5±8.9	23.4±9.8	<0.05
T30 (s)	292.4±88.7	409.2±138.3	<0.05
T (s)	140.4±55.7	130.3±49.7	>0.05

Values are means±SD. HRrest, hert rate at rest; HRpeak, peak heart rate; HRpost, post-exercise heart rate; HRR60, number of heart beats recovered in 60 s after exercise cessation; T30, time constant of short-time heart rate recovery for the first 30 s; T, time constant of HR decay during the 5-min recovery.

TABLE 2
Short-term vagal related resting HRV measures pre- and post-exercise.

	Male pre	Male post	Female pre	Female post
$RMSSD_{2\text{-}5min}$ (ms)	27.3±8.3	5.3±2.9*	22.1±14.0	3.8±1.8*
$lnHF_{2\text{-}5min}$ (ms^2)	6.3±0.6	3.3±0.9*	6.0±0.8	2.5±1.0*§
$HFnu_{2\text{-}5min}$ (ms^2)	25.0±10.8	24.1±9.3*	31.6±11.3	22.1±6.6*

Values are means±SD. RMSSD2-5min, root mean square of successive difference of R-R intervals; lnHF2-5min, high frequency power density of R-R intervals; HFnu2-5min, the normalized HF power. Subscript "2-5min" means calculated on the last 3 min of the 5 minute recovery periods. * p<0.05 vs. pre-exercise; § p<0.05 vs. male

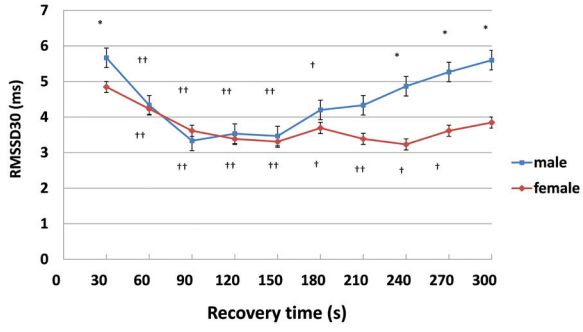

Figure 1. *Mean±SE. Root mean square of successive difference of the R-R intervals calculated on successive 30-s segments ($RMSSD_{30}$) during the 5-min recovery period, for male and female participants. * Significant difference vs. male participants (p<0.05). † Significant difference vs. $RMSSD_{30}$ at the end of exercise (p<0.05); †† significant difference vs. $RMSSD_{30}$ at the end of exercise (p<0.001)*

post-exercise $lnHF_{2\text{-}5min}$ was significantly smaller in females (3.3±0.9 ms^2 in males vs. 2.5±1.0 ms^2 in females).

The time course of $RMSSD_{30}$ recovery was impaired immediately after exercise as shown in **Figure 1**.

DISCUSSION

The 30-s Wingate Anaerobic Test (WAnT) measures short-term supramaximal intensity power output using a cycle ergometer. It was developed in the 1970s and is one of the most widely used anaerobic performance test *(19)*. The WAnT is a reliable assessment of the physiological responses to supramaximal exercise requiring the bulk of energy to be provided by the ATP-phosphagen (CP) energy system *(22)*. Our results revealed that male participants have larger anaerobic power than females which is in accordance with previous studies *(22, 23)*. As in the studies of Zouhal and colleagues, women developed lower absolute peak power than men during the Wingate test *(24)*. They explained this gender difference partly by differences in body mass and body composition. Hill suggests that actual gender differences in anaerobic capacity may be even greater than 35% *(25)*.

In agreement with our recent study *(3)*, the present study also suggests that resting heart rate values in the upright seated position were higher due to reflexly mediated orthostatic influences. Female participants presented significantly higher HR in comparison with their male counterparts. Similar observations were reported by Barantke *et al. (14)* in the supine position and Schouwenberg *et al. (26)* in the upright position. Nevertheless, the Wingate test appeared to induce a severe stress for both genders, as judged by high and equal peak pulses at the end of the exercise.

Post exercise heart rate recovery was faster in male participants demonstrated through HRR_{60} and T30, but the time constant of the exponential heart rate decay (T) did not differ between the two genders. Early recovery is dominated by parasympathetic reactivation, with sympathetic withdrawal becoming more important later in recovery *(2)*. This is consistent with the findings of Imai and colleagues *(20)* who used parasympathetic and sympathetic blockade in the experiments. They found that T30 was primary mediated by vagal activity, whereas Perini et al. suggested that the exponential HRR could not be explained only by the prompt vagal reactivation but also with the presence of sympathetic activity *(8, 20)*.

The present gender difference in T30 might suggest faster vagal restoration in male participants. Faster parasympathetic reactivation in males with similar time constant values compared with female participants might suggest that vagal restoration in males is influenced by higher postexercise sympathetic activity. The sympathoadrenal response to exercise is greatly influenced by exercise intensity *(24)*. High plasma epinephrine and norepinephrine concentrations have been observed after exercise of supramaximal intensity *(27, 28, 29)*. Exercise induced plasma epinephrine responses to supramaximal cycling were greater in males than females *(30)*. Gratas-Delamarche and collegues also observed higher plasma adrenaline concentration in men in response to strenuous exertion *(31)*. Some researcher found no gender differences in the catecholamine response to vigorous exercise, but they investigated trained man and women *(24, 32)*.

High concentration of epinephrine affects the cardiovascular system differently in men and premenopausal women, with β_1 and β_2 adrenergic activity prevailing in men (increased HR and reduced vascular tone) and α-adrenergic activity prevailing in women (increased systolic blood pressure) *(26)*. Miyamoto and colleagues showed that the dynamic HR response to vagal stimulation can be attenuated via activation of α-adrenergic receptors on the cardiac vagal nerve terminals by high plasma norepinephrine levels *(33)*. Although we did not measure catecholamine kinetics after exercise, we believe

that the slower vagal restoration in females might be partly attributed to gender differences in catecholamine effects on the cardiovascular system.

Parasympathetic reactivation, i. e. the time course of $RMSSD_{30}$ was highly impaired immediately after exercise. Values calculated after the first thirty seconds are in accordance with T30, revealing faster vagal restoration in male participants. After this period values in both genders dropped, but in male participants recovery was observed after the fourth minute. Research in the area of parasympathetic reactivation has revealed that the restoration of autonomic control of heart rate appears to be dependent on exercise intensity. HRR after moderate- to high exercise showed slower vagal restoration than after low-intensity exercise *(11).*

Buchheit compared three exercise routines with comparable net energy expenditure but with different participation of aerobic and anaerobic power generation *(4).* They revealed a significantly slower parasympathetic reactivation after repeated sprint exercise, with higher anaerobic contribution than that shown with submaximal continuous exercise. Wingate anaerobic test as an all-out exercise of supramaximal intensity over the 30-s course engages 84% of total ATP regeneration from anaerobic sources *(34).* Buchheit et al. argued that higher concentration of plasma metabolites plays an important role, though the influence of lactate accumulation on postexercise autonomic regulation has still not been directly evaluated *(4).* Indeed, short-term supramaximal anaerobic exercise has been associated with a substantial lactic acidosis *(29).* Metaboreceptors with sympathetic afferents might be activated by accumulated waste products during exercise possibly suggesting an alternative mechanism for sympathetic predominance and delayed restoration of vagal tone *(35).* Additionally, maintained sympathetic outflow evoked by the arterial baroreceptors' response to more vigorous and rapid changes in pressure after exercise might also delay parasympathetic reactivation as well *(36).*

The present study demonstrated similar RMSSD, lnHF and HFnu at rest in male and female participants. Several studies have reported gender differences regarding sympathetic and parasympathetic tone with conflicting results, while other studies failed to show significant difference whatsoever *(14, 15, 16, 17).* Barantke and colleagues reported no gender differences in absolute HF values, however HF in normalized units was significantly higher in female subjects *(14).*

None of the observed vagal HRV indices have restored after five minutes of recovery following 30-s Wingate anaerobic test, but the post-exercise $lnHF_{2\text{-}5min}$ was significantly smaller in females. This finding is in accordance with the impaired recovery of $RMSSD_{30}$ in female participants after five minutes of recovery. Thirty second supramaximal physical exertion greatly alerts the sympathovagal system. The magnitude and time-course of vagal and sympathetic recovery depends on the preceding exercise intensity *(7).* High intensity exercise induces prolonged vagal reactivation and HRV recovery, with progressive increase of high- and low-frequency HRV power indices that still might not reach resting values after 10 minutes *(9, 37),* 15 minutes *(11, 37, 38, 39),* 30 minutes *(10)* or even one hour *(11, 39).*

Mendonca and collegues reported that the cardiac autonomic function of women is more affected by supramaximal exercise than that of men *(18).* Hence the more suppressed lnHF after 5 minutes of recovery in females in our experiment is not surprising. Even in articles where female participants showed stronger vagal modulation, it was not reflected in higher baroreflex sensitivity *(14, 16).*

In conclusion, disturbance of cardiovascular homeostasis induced by 30-s supramaximal exertion led to an incomplete restoration of vagal tone manifested by depressed vagal-related HRV indices after 5 minutes of recovery. Immediate HRR and parasympathetic reactivation was attenuated in female compared with male participants. Future studies are warranted to investigate physiological mechanisms that will explain gender differences and delay in autonomic regulation following supramaximal exercise.

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