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Heart rate variability analysis during immediate recovery from exercise in overweight/obese healthy young adult females

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Background: Data on heart rate variability (HRV) changes during immediate recovery period after exercise in overweight/obese healthy young adult females are still inconclusive. The aim of this study was to evaluate the heart rate variability (HRV), heart rate recovery (HRR), and arterial blood pressure immediately after cessation of exercise in overweight/obese healthy young adult females.

Methods: This study was carried out in the laboratory of the Physiology Department, Faculty of Medicine, King Abdulaziz University. Fifty-five female students were classified into normal weight group and overweight/obese group. HRV, HRR, and systolic and diastolic blood pressures (SBP, DBP) were estimated at resting condition and immediately after cessation of exercise.

Results: During recovery, heart rate was significantly increased in overweight/obese group along with significant decrease in rMSSD (square root of the mean of the sum of the squares of differences between adjacent NN intervals) and HF (high-frequency power) compared with normal group. The recovery of heart rate, normalized HF, and normalized sympathovagal balance to their baseline values were significantly lowered in overweight/obese group. Both SBP and DBP were significantly lowered from their baseline values during recovery in normal group but unchanged in overweight/obese group.

Conclusions: This study provides evidence that overweight/obesity are associated with decreased HRR and delayed vagal reactivation, in addition to impaired postexercise hypotension early in the recovery period after exercise in healthy young adult females.

KEYWORDS

exercise, heart rate recovery, heart rate variability, overweight/obesity

1 | INTRODUCTION

Autonomic nervous system is an important regulator of homeostasis as it controls involuntary physiologic processes, including digestion, hormonal regulation, blood pressure, and heart rate (Czura & Tracey, 2005). During exercise and recovery, autonomic nervous system modulates cardiovascular function to satisfy the metabolic requirements of working muscles. Vagal withdrawal and sympathetic stimulation increase the heart rate during exercise. With a transition from dynamic exercise to inactive recovery, heart rate declines to the preexercise

value and is considered as heart rate recovery (HRR). HRR is primarily the result of return of vagal activity. Delayed HRR has a prognostic value in predicting ventricular arrhythmia and sudden cardiac death (Nishime, Cole, Blackstone, Pashkow, & Lauer, 2000).

Heart rate variability, the variation of the intervals between successive heart beats (RR intervals), is a standard noninvasive method for assessing the autonomic control on sinus node. Lower HRV is an indicator of autonomic dysfunction and has been related to several diseases such as diabetes, hypertension, and cardiovascular diseases (TaskForce, 1996).

Obesity is imposing a growing threat to world health, as 39% of adults in many countries are categorized as clinically overweight, with higher incidence in females (World Health Organization, 2014).

This work was carried out in the Physiology Department, Faculty of Medicine, King Abdulaziz University, Jeddah, Saudi Arabia.

While obesity is known to be highly associated with lower HRV or autonomic dysfunction under resting condition (Koenig et al., 2014), studies that investigate the influence of overweight/obesity on the HRV, HRR, and arterial blood pressure during immediate recovery period from acute exercise are limited particularly in healthy young adult females. In this context, this study was planned to evaluate the heart rate variability, heart rate recovery, and arterial blood pressure immediately after cessation of exercise in overweight/obese healthy young adult females.

2 | SUBJECTS AND METHODS

2.1 | Subjects

This study was carried out in the laboratory of the Physiology Department, Faculty of Medicine, King Abdulaziz University, Jeddah, Saudi Arabia. Fifty-five female students were recruited by advertisement in the Faculty of Medicine, King Abdulaziz University. Subjects were eligible for inclusion if they were female, healthy, and between 20–24 years old. Exclusion criteria were as follows: male; hypertension; cardiac, pulmonary, renal, or autoimmune diseases; regular exercise; smoking; consumption of alcohol; or any medications.

2.2 | Experimental protocol

The experimental design was approved by the Unit of Biomedical Ethics Research Committee in King Abdulaziz University, Faculty of Medicine. All females signed an informed consent to participate in the study. Data collection was carried out in the laboratory of the physiology department at room temperature between 21 and 25°C. The body weight and height were determined while the subjects wore light cloths and no shoes, using digital medical scale (DETECTO, 758C, Elk Grove Village, IL, USA). BMI was calculated by dividing the weight in kilograms by the square of the height in meters. Females were classified according to the WHO definitions (World Health Organization Geneva, 1999) into two groups: normal weight (BMI 18.5–24.99 kg/m², $n = 27$ females) and overweight/obese (BMI ≥ 25 kg/m², $n = 28$ females). After 20 min of resting and acclimatization in supine position, the baseline HRV and arterial blood pressure were recorded. Then, all females underwent 3-min step test. The step test was performed by stepping up and down a 12-inch bench, following a metronome adjusted at 96 beats/min which corresponded to 24 cycles/min for 3 min under program supervision. Then, a second measurement of arterial blood pressure and HRV were taken immediately after cessation of exercise.

2.2.1 | Measurement of arterial blood pressure

The systolic and diastolic blood pressures (SBP and DBP) were recorded on left upper arm by using an automatic device (Spot Vital Sign series 420; Welch Allyn Inc., Skaneateles Falls, NY, USA) according to WHO recommendations (World Health Organization, 1996). The average of two measurements was recorded.

2.2.2 | HRV measurements and analysis

Short-term ECG recording (3 min) was conducted by using a ML865 power lab 4/25T, ML 408 Dual Bio-Amp/Stimulator machine (AD Instruments Pty Ltd, Castle Hill, Australia) installed on IBM computer. The version 4.0 of the chart for windows software was used in the analysis. The time domain HRV parameters measured in this study were as follows: HR (beat/min; bpm): mean heart rate in the entire recording; rMSSD (ms): square root of the mean of the sum of the squares of differences between adjacent NN intervals. Spectral analysis was performed with Fast Fourier Transform technique. The frequency domain parameters measured herein were high-frequency component (HF, ms²): power frequency between 0.15 and 0.4 Hz; low-frequency component (LF, ms²): power frequency between 0.04 and 0.15 Hz. The sympathovagal balance (LF/HF) is the ratio of LF and HF. HF and LF were presented in absolute values of power and normalized units as well. Normalized LF and HF (nu LF and nu HF) were calculated as the relative value of each power component in proportion to the total power minus the VLF component. Normalized LF/HF (nu LF/HF) was estimated as the ratio of nu LF and nu HF (TaskForce, 1996).

2.2.3 | Recovery measurements

During the first 3 min of inactive recovery after cessation of exercise the heart rate, nu HF and nu LF/HF were monitored and the restoration toward the baseline (resting) value was calculated for each and defined as heart rate recovery (HRR), normalized high-frequency recovery (nuHFR), and normalized sympathovagal balance recovery (nu LF/HFR), respectively. They were calculated as percentage change of the value after exercise from their matched baseline value.

2.3 | Statistical analysis

All variables were presented as mean $\pm$ standard error of mean (SEM). The one-sample Kolmogorov-Smirnov test was used to test for normality of variables. Those variables that were not normally distributed were transformed and expressed in form of natural logarithms (ln). Unpaired *t*-test was done to determine the differences between groups. Paired *t*-test was used to estimate the differences within groups (for baseline versus values during recovery period); Pearson correlation analysis was used to investigate the relationship between BMI and the recovery measurements. *p* values $< .05$ were considered statistically significant. SPSS windows version 20 (SPSS Inc., Chicago, IL, USA) was used in the analysis.

3 | RESULTS

Anthropometric characteristics of the study subjects, expressed as mean $\pm$ SEM, are presented in Table 1.

Table 2 demonstrates a significant higher heart rate in the overweight/obese group than in normal group ($p < .001$) during recovery.

TABLE 1 Anthropometric characteristics of the study groups

	Normal weight (27)	Overweight/obese (28)
Age (years)	20.7 ± 0.29	21.55 ± 0.46
BW (kg)	51.19 ± 0.86	76.72 ± 1.67*
Height (cm)	157.4 ± 1.34	157.55 ± 1.15
BMI (kg/m ²)	20.79 ± 0.30	30.64 ± 0.41*

Results are presented as mean ± SEM. In parentheses is the number of observations in each group. BW, body weight; BMI, body mass index.

*Significance of difference by unpaired *t*-test (*p* < .05).

Also, during recovery, the parasympathetic indices ln HF, ln rMSSD (expressed in log-transformed form), and the normalized HF all were significantly lower in overweight/obese group than in normal group (*p* < .001, for all). Correspondingly, the normalized sympathovagal balance (nu LF/HF) showed a significant increase in overweight/obese group when compared with normal group (*p* < .01). In LF and nu LF did not differ significantly between the two study groups during the recovery period.

Systolic and diastolic blood pressures were significantly higher in overweight/obese group than in normal group (*p* < .001, for all) during recovery. Moreover, both were significantly lower in normal group (*p* < .01, *p* < .001 respectively) and unchanged in overweight/obese group when compared with their matched baseline values (Table 2).

The recovery measurements, HRR, nu HFR, and nu LF/HFR were significantly delayed in overweight/obese compared with the normal group (*p* < .01, for all) (Figure 1).

The correlation studies are presented in Figure 2 and show negative relationship between BMI and each of HRR, nu HFR, and nu LF/HF. The correlation was significant with HRR and nu HFR (*p* < .03, *p* < .02 respectively).

FIGURE 1 Recovery measurements in the study groups. Results are presented as mean ± SEM. HRR, heart rate recovery; nu HFR, normalized high-frequency recovery; nuLF/HFR, normalized sympathovagal balance recovery. *Significance of difference by unpaired *t*-test (*p* < .05)

4 | DISCUSSION

The major finding of this study was the decreased vagal activity, along with increase in heart rate in overweight/obese healthy young adult females during immediate recovery after exercise. Also, there were negative correlations between BMI and the recovery of each of heart rate, normalized high frequency, and normalized sympathovagal balance. Moreover, there was a lack of postexercise hypotension in overweight/obese females.

Our results showed a significant decrease in parasympathetic indices rMSSD and HF, in addition to a significant decrease in the recovery of HF (nu HFR) in overweight/obese group compared with normal group. This observation suggests slow vagal reactivation immediately after the exercise which mitigated the recovery of the heart rate to the baseline value. In support of this observation, the significant increase in nu LF/HF in overweight/obese group compared with normal group

TABLE 2 Baseline and recovery values of heart rate variability indices and systolic and diastolic blood pressures in the study groups

	Normal weight (27)		Overweight/obese (28)	
	Baseline	Recovery	Baseline	Recovery
HR (bpm)	76.23 ± 1.46	84.57 ± 1.53**	80.09 ± 1.30	95.31 ± 1.80***
ln HF (m ²)	3.68 ± 0.13	3.58 ± 0.12	3.14 ± 0.14*	2.79 ± 0.17***
nu HF(%)	48.58 ± 1.91	43.92 ± 2.57**	39.97 ± 2.72*	29.31 ± 1.98***
ln LF (m ²)	3.60 ± 0.17	3.32 ± 0.13	3.18 ± 0.13	3.12 ± 0.13
nu LF(%)	27.11 ± 2.00	25.13 ± 2.38	32.53 ± 3.40	29.81 ± 3.65
nu LF/HF	0.59 ± 0.06	0.63 ± 0.07**	1.01 ± 0.17*	1.26 ± 0.23***
ln rMSSD (ms)	2.22 ± 0.07	2.11 ± 0.07	1.84 ± 0.08*	1.75 ± 0.08*
SBP	109.7 ± 1.77	106.2 ± 1.26**	121.36 ± 2.33*	118.36 ± 2.13*
DBP	71.2 ± 1.49	68.6 ± 1.11**	76.9 ± 1.51*	76.1 ± 1.90*

Results are presented as mean ± SEM. In parentheses is the number of observations in each group. ln, natural logarithm; nu, normalized unit; HR, heart rate; HF, high-frequency power; LF, low-frequency power; LF/HF, sympathovagal balance; rMSSD, square root of the mean of the sum of the squares of differences between adjacent NN intervals; SBP, systolic blood pressure; DBP, diastolic blood pressure.

*Significance of difference by unpaired *t*-test (*p* < .05).

**Significance of difference by paired *t*-test (*p* < .05).

FIGURE 2 Correlation between body mass index (BMI) and recovery measurements; heart rate recovery, HRR; normalized high-frequency recovery, nu HFR; normalized sympathovagal balance recovery nuLF/HFR

confirms the shift of sympathovagal balance toward sympathetic overactivity and vagal withdrawal. Also, the significant negative correlation between the BMI and the HRR as well as the nu HFR recorded herein supports our suggestion.

Consistent with our finding, Dimkpa & Oji (2010) demonstrated negative correlations between HRR (measured in the first minute of recovery after cycle ergometer exercise) and BMI in healthy adults. However, Esco & Williford (2013) did not record any significant relation between BMI and HRV indices, which were recorded in the first minute of recovery in healthy young adult men. The dissimilarity in the intensity of protocol exercise used in our study and in the latter study can explain this discrepancy (Blain, Limberg, Mortensen, & Schrage, 2012).

The current results also revealed a significant higher systolic and diastolic blood pressure in overweight/obese group when compared with the normal group during the recovery period. Moreover, systolic and diastolic blood pressures were significantly decreased from their baseline values in normal group, while both were unchanged from their baseline values in overweight/obese group during recovery period. This finding suggests an early vascular dysfunction with reduced postexercise vasodilatation in concomitance with obesity. This is in line with the sympathovagal imbalance recorded in overweight/obese group, where the sympathetic dominance impaired the postexercise vasodilatation. In agreement with this finding, the adjustment of the total peripheral resistance from exercise to recovery was impaired in obese adults (Blain et al., 2012).

In fact, the present results showed that obesity also influenced the baseline HRV and arterial blood pressure. The overweight/obese group presented a significant lower baseline rMSSD, ln HF, and nu HF associated with significant higher baseline sympathovagal balance and systolic and diastolic blood pressures compared with normal group. Therefore, the delayed recovery of heart rate and vagal activity reported in overweight/obese group after exercise might be attributed to the adiposity-induced autonomic imbalance already existed at resting condition, in addition to altered cardiovascular autonomic response to exercise that has been reported in obese subjects by an earlier study (Dipla, Zafeiridis, Koidou, Geladas, & Vrabas, 2010). Moreover, obesity-related autonomic imbalance can be triggered by reflex mechanisms as decreased baroreceptor sensitivity and blunted baroreflex (Canale et al., 2013) by metabolic factors as insulin resistance and hyperinsulinemia (Straznicki et al., 2015) and by disturbed adipokine production from visceral fat with a mainly hyperleptinemia (Shi & Brooks, 2015) and hypoadiponectinemia (Boer-Martins et al., 2011).

5 | CONCLUSION

We demonstrated HRV, HRR, and arterial blood pressure immediately after cessation of exercise and found a major influence of overweight/obesity. The reactivation of vagal activity, the heart rate recovery, and the postexercise hypotension all were significantly mitigated in overweight/obese healthy young adult females.

5.1 | Limitation

There are few limitations concerning this study. The study participants were limited to only healthy young adult females, and therefore, we cannot generalize our conclusion to males, or subjects with different age. Also, our sample size was relatively small. We have not determined the type of obesity, since other studies reported a noticeable role of central obesity in cardiovascular autonomic dysfunction (Farah, Barros, Balagopal, & Ritti-Dias, 2014).

5.2 | Recommendation

HRV and HRR were modifiable indices, particularly when caloric restriction combined with exercise (de Jonge, Moreira, Martin, & Ravussin, 2010). Therefore, prevention of weight gain and weight loss regimens should be the major priorities for combating obesity associated autonomic dysfunction, hypertension, and their consequences in the future. Furthermore, future studies are recommended to analyze the influence the duration and intensity of exercise on autonomic nervous system activity in obese individuals.

CONFLICT OF INTEREST

All authors declare that there are no conflicts of interest.

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