ORIGINAL ARTICLE

The Effect of Submaximal Incremental Running Test on Heart Rate Variability in University-level Male Football Athletes

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ABSTRACT

Introduction: The purpose of this study was to investigate the effect of incremental running intensity on heart rate variability during recovery in university-level male football athletes. **Methods:** Twenty healthy males aged between 19-26 years old were randomly assigned to control (no running) and intervention (modified Bruce protocol running) groups. Analyses were performed at pre-exercise, 24, 48, and 72 h post-exercise. The low-frequency (LF) and high frequency (HF) in peak, absolute power and normalised unit (n.u.), as well as LF/HF ratio, were assessed. **Results:** Intervention group showed a reduced LF n.u. response (ES=1.69) whereas HF n.u. showed the opposite response (ES=1.51) at 24 h post-exercise. The intervention group showed a significant difference in the LF/HF ratio at 48 h post-exercise (p=0.002; ES=1.05). **Conclusion:** These findings demonstrated that incremental type of exercise influence both sympathetic and parasympathetic response for at least 48 h post-exercise.

Keywords: Heart rate variability, Spectral analysis, Modified Bruce protocol

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INTRODUCTION

Heart rate variability (HRV) monitoring is a non-invasive method for the investigation of cardiovascular autonomic function by measuring the variations in the successive heartbeats known as R-R intervals. The R-R intervals, explained by the time between two R waves, are used to interpret the autonomic nervous system (ANS) modulation input of the heart function. The HRV frequency-domain has been widely used to interpret the ANS response with the same precision as the electrocardiogram. The physiological correlates of using frequency-domain analysis has enhanced the understanding of the modulatory effect of vagal activities. A Task Force on HRV Standards of Measurement, Physiological Interpretation, and Clinical Use has identified certain frequency bands correlates with physiological adaptations (1). These are known as low-frequency (LF) band, measuring at 0.04-0.15 Hz, which reflects sympathetic activity and high-frequency (HF) band, measuring at 0.15-0.4 Hz, which reflects parasympathetic activity. The balance between sympathetic and parasympathetic activity can be determined by the ratio of LF to HF power (LF/HF).

Clinically, a lower HRV has been interpreted as an unfavorable prognosis for cardiovascular disease (2).

Increased high-frequency indices of HRV refers to the patients with transient myocardial ischemia, notably during coronary artery occlusion (3). In sports, the HRV is mostly used for monitoring training and recovery aids (4). During low-intensity exercise, an increased LF/HF ratio shows a sympathetic predominance while a decreased LF/HF ratio indicates parasympathetic predominance during high-intensity exercise compared to resting baseline (5). Athletes that apply strenuous exercise in training will cause an impaired parasympathetic activity and demonstrating a sympathetic predominance during the recovery (6). The utilisation of HRV as a training and monitoring instrument for overtraining, fatigue and recovery after a training process has become increasingly popular and earned merits in the sports fraternity (7).

Acute effect of exercise is associated with regulation in the ANS. The ANS is partially affected by multiple acute effects of exercise that were accrued rather than the adaptation of chronic exercise stress. Following 24 h after an acute submaximal exercise at 65% VO_2 peak, the heart is said to be more sensitive to ANS influence and adaptable to any subsequent stressors (8). However, the effect of acute exercise stress at a much higher intensity may prolong the recovery from exercise by delaying the restoration of vagal activities (9). Particularly for athletes who undergo rigorous training and fitness evaluation cycle, monitoring recovery has been a central process. A commonly used fitness evaluation, the incremental exercise test is a robust test to evaluate oxygen capacity. Incremental exercise-to-exhaustion or maximal ergometric testing is typically used in trained

and elite athletes. However, data from sub-maximal exercise ergometric testing are well accepted with diagnostic and prognostic values, often an advantage over maximal exercise due to medicolegal reasons (10). At submaximal exercise intensities, heart rate response to stress events is already highly visible and sensitive to cardiovascular regulation (11). One such specific test is the modified Bruce protocol used in individuals with limited exercise capacity. Modified Bruce protocol has been demonstrated to induce less physiological stress than its original protocol, hence perturbation in the ANS modulation may be lower (12). However, the goal of 85% of age-predicted maximum HR exercise testing in modified Bruce protocol meets the termination criteria for stress on the heart to induce myocardial ischemia (13). Exercise stimulates sympathetic drive and modifies the coronary vasomotor tone which affects the coronary blood flow (14). As exercise progresses above 85% of maximum HR, the risk of medical complications may increase. Sub-maximal tests are performed at shorter durations, with less stressful demands on the cardiovascular system. Hence, HRV can be routinely assessed in a large number of athletes, by providing HRV monitoring as an applicable fitness and recovery markers can assist in supporting informed decisions training and recovery aspects of team sports (15). We were interested in finding out the magnitude of ANS modulations over a few days in trained individuals following a submaximal incremental exercise protocol to better understand the duration of recovery associated with this running intensity. We hypothesised that submaximal incremental exercise will produce a sustained sympathetic predominance at post-exercise. Hence, this study was to assess the sympathetic and parasympathetic HRV indices following a submaximal incremental running test in university-level athletes.

MATERIALS AND METHODS

Participants
The sample size calculation using G*Power software version 3.1.9.2 (Universität Düsseldorf, Germany) was based on a previously conducted method which indicated a significant mean change in post-exercise HRV outcomes (frequency-domain), providing a relative effect size of 1.48 with the alpha of 0.05 and power of 80% (16). Twenty healthy males, aged between 19-26 years old, who volunteered and consented to this study were football players with at least a year of experience competing at university level football match, actively undergoing football training for at least three times per week and having submaximal volume of oxygen consumption (submax VO_2) between 30 – 40 % of VO_2max . The participants were excluded if they have any history of heart diseases, musculoskeletal injuries or on any medication. All participants were requested to abstain from caffeine intake and strenuous physical activity at least 48 h prior to- and post-72 h performing the experimental protocol . The protocol of this study

was conducted in accordance with the Declaration of Helsinki and approved by the Human Research Ethics Committee of Universiti Sains Malaysia (USM/JEPeM/18020148). Physical characteristics of the study participants are presented in Table I.

Table I: Physical characteristics of study participants

	Control (n=10)	Intervention (n=10)
Age (years)	22.9±1.2	23.4±1.1
Weight (kg)	62.6±6.7	70.0±9.2
Height (cm)	166.7±4.3	171.5±6.2
BMI (kg/m²)	22.6±1.8	23.2±1.9
BF (%)	21.2±4.1	23.8±2.5
FFM (kg)	50.2±4.6	53.3±3.7
Baseline HRrest (beats.min⁻¹)	65.1±10.4	68.6±7.0
Predicted HRmax (beats.min⁻¹)	197.1±1.2	196.6±1.1

Results in mean±SD. BMI: body mass index, BF: body fat, FFM: fat-free mass, HR: heart rate.

Protocol
Participants were randomly assigned in equal numbers to two groups, intervention (modified Bruce Protocol) and control (no running) groups using a block randomisation method. On the first visit, all participant's height (cm) and body weight (kg) were measured using a stadiometer incorporated weighing machine (Model 707, Seca Corporation, USA). Body fat percentage (% BF), fat-free mass (FFM) and body mass index (BMI) were measured using a body impedance analyser (TANITA, TBF-140 model). Then, all participants wore a heart rate (HR) monitor (Model V800, Polar Electro, Finland) around the wrist and an HR transmitter sensor (H1, Polar Electro, Finland) strapped around the chest before quietly lying supine for 5 min to record baseline (pre) HRV. During HRV recording, participants were were not permitted to move their body or talk at all but to breathe spontaneously in a relaxed position. The breathing rate of participants was not controlled. During the familiarisation session, the intervention group walked on the treadmill at incremental speeds and grade that will be similarly applied on the actual day while wearing an expired gas mouthpiece.

During the experimental days, intervention group participants performed the modified Bruce protocol on the treadmill (Model TMX 425CP, Full Vision Inc, USA). According to the previous method, the slope and the speed of the ramp were modified to increase at a lower workload than the standard Bruce protocol test to guarantee a progressive physical effort on the treadmill (17). Immediately after warm-up, the participants walked on treadmill by stages with speeds increased every 3-min, starting from 2.74, 4.02, 5.47, 6.76, 8.05, 8.85, 9.65, 10.46, 11.26 and 12.07 km.h⁻¹. The treadmill slope was increased 2% every 3-min, starting at 10% with 3-min duration at each stage. A total duration of 6-min warm-up was performed at a 2.7 km.h⁻¹, 0% slope for 3-min followed by an increase to 5% grade

for 3-min. The HR and volume of oxygen consumption (VO_2) (Parvo Medics Inc., Sandy, UT) were monitored continuously whereas ratings of perceived exertion (RPE) were monitored every 3 min intervals using the Borg's scale (18). The warm-up and cool-down intensities were set equivalent to the first two stages of the modified Bruce protocol. Participants were given ad libitum access to plain drinking water before the start of the protocol. The exercise was automatically stopped when the participants achieved 85% of the age and gender predicted maximum HR calculated using the equation by Roy and McCrory (19). The participants from the control group were requested to resume their normal daily activities and refrain from strenuous exercise.

All participants from both groups returned to the laboratory at 24, 48 and 72 h after the experiment day. During these periods, all participants were refrained from strenuous exercise. All heart rate data was recorded at 8.00 a.m according to procedures similar to the first visit. The HRV recorded were then synced with Polar Flow online tool (Polar Electro Oy) before extracted to Kubios HRV Software for analysis (20). An artefact correction threshold level ±0.25 s was employed for ectopic beats before the calculation of the following components: LF (0.04-0.15 Hz) and HF(0.15-0.4 Hz) in power, absolute and normalised units (n.u.); and LF/HF ratio of HRV.

Statistical Analysis

Statistical analysis was performed using Statistical Package for Social Sciences (IBM SPSS Statistics for Windows, Version 22, IBM Corp., USA). All data were tested for normality using the Shapiro-Wilk test. Independent student t-test was used to compare physiological data between groups. A two-way repeated-measures analysis of variance was used to compare between group and time (pre-exercise, 24 h post, 48 h post and 72 h post-exercise). Descriptive data were expressed as mean ± standard deviation (mean±SD). The magnitude of the effect size between groups was calculated and interpreted based on a previous study (21). Significance difference was set at $p<0.05$.

RESULTS

The changes in LF and HF components during pre, post-24 h, post-48 h and post-72 h exercise are presented in Table II and III. The incremental exercise caused a significant difference in LF n.u. (p=0.033; ES:1.69) and HF n.u. (p=0.013, ES:1.51) at 24 h post-exercise when compared to control. In the intervention group, the LF n.u. was reduced by 28.9% whereas the HF n.u. was increased by 26.0% at 24 h post-exercise compared to pre-exercise. As for HF peak and absolute power, no significant effects were observed in all measured time points. Significant differences were observed in the LF/HF ratio in the intervention group at pre-exercise (p=0.003; ES:1.49) and 48 h (p=0.002, ES:1.05) post-exercise (Table IV).

Table II: Mean±SD, ES, rating and p-value of LF components at pre, post-24 h, post-48 h and post-72 h

Frequency Domain	Groups	Mean ± SD			
		Pre	Post-24h	Post-48h	Post-72h
LF (Hz)	Control	0.09±0.03	0.08±0.03	0.07±0.03	0.07±0.02
	Intervention	0.09±0.03	0.17±0.09	0.11±0.09	0.08±0.02
LF (ms²)	Control	1652±799	1385±754	1313±490	1842±1201
	Intervention	3272±1913	1780±1262	2387±1791	3014±2127
LF (n.u)	Control	51.5±22.2	56.4±16.0	52.4±17.0	44.3±19.8
	Intervention	51.5±11.5	36.6±16.2	44.9±23.7	45.3±27.9

Frequency Domain	Groups	ES (95% CI), Rating, P-value			
		Pre	Post-24h	Post-48h	Post-72h
LF (Hz)	Control	0.13	1.65	0.76	0.44
	Intervention	(-0.02,0.04) Small p=0.650	(0.03,0.15) Large p=0.006*	(0.004,0.08) Medium p=0.031*	(-0.02, 0.07) Medium P=0.239
LF (ms²)	Control	0.99	0.20	1.40	0.73
	Intervention	(604,2576) Large p=0.003*	(-431,1082) Small p=0.379	(887,3607) Large p=0.003*	(-451,2795) Large p=0.146
LF (n.u)	Control	0.002	1.69	0.15	0.78
	Intervention	(-16.6, 16.6) Small p=0.999	(1.5,29.9) Large p=0.033*	(-18.9,15.9) Small p=0.861	(-12.9,27.4) Small p=0.459

LF: low-frequency; CI: confidence interval; ES: effect size; Hz: peak power; ms²: absolute power; n.u: normalised unit. The magnitude of the ES (rating): <0.2 trivial; 0.2–0.6 small; 0.6–1.2 moderate; > 1.2 large, *p < 0.05: Significance difference between groups.

Table III: Mean±SD, ES, rating and p-value of HF components at pre, post-24 h, post-48 h and post-72 h

Frequency Domain	Groups	Mean ± SD			
		Pre	Post-24h	Post-48h	Post-72h
HF (Hz)	Control	0.45±0.25	0.49±0.30	0.48±0.27	0.25±0.08
	Intervention	0.43±0.29	0.47±0.29	0.47±0.19	0.29±0.13
HF (ms²)	Control	1720±1434	1510±942	2440±1458	3010±2353
	Intervention	3210±2058	2629±1879	2897±2221	3288±1980
HF (n.u)	Control	54.3±23.4	43.4±15.9	51.5±22.5	55.5±19.8
	Intervention	50.3±11.1	63.4±16.2	64.0±17.8	61.6±19.7

Frequency Domain	Groups	ES (95% CI), Rating, P-value			
		Pre	Post-24h	Post-48h	Post-72h
HF (Hz)	Control	-0.94	-0.6	-0.30	1.43
	Intervention	(-0.28,0.23) Trivial p=0.836	(-0.29,0.26) Trivial p=0.893	(-0.23,0.21) Trivial p=0.944	(-0.07,0.14) Large p=0.460
HF (ms²)	Control	1.43	1.08	0.44	0.27
	Intervention	(-175,3157) Large p=0.076	(-278,2516) Large p=0.110	(-1308,2222) Small p=0.593	(-1765,2322) Small p=0.778
HF (n.u)	Control	0.30	1.51	1.00	0.46
	Intervention	(-13.2,21.2) Small p=0.632	(4.8,35.0) Large p=0.013*	(-6.5,31.6) Large p=0.183	(-12.5,24.7) Small p=0.498

HF: high-frequency, CI: confidence interval; ES: effect size; Hz: peak power; ms²: absolute power; n.u: normalised unit. The magnitude of the ES (rating): <0.2 trivial; 0.2–0.6 small; 0.6–1.2 moderate; > 1.2 large *p < 0.05: Significance difference between groups.

Table IV: Mean±SD, ES, rating and p-value of LF/HF ratio at pre, post-24 h, post-48 h and post-72 h

Frequency Domain	Groups	Mean ± SD			
		Pre	Post-24h	Post-48h	Post-72h
LF/HF Ratio	Control	1.79±1.33	1.60±0.97	1.36±0.84	1.10±1.01
	Intervention	1.88±2.91	0.88±0.98	1.48±2.09	1.85±2.77

Frequency Domain	Groups	ES (95% CI), Rating, P-value			
		Pre	Post-24h	Post-48h	Post-72h
LF/HF Ratio	Control	1.49 (1.19,4.85) Large p=0.003*	0.38 (-0.14,1.71) Small p=0.091	1.05 (0.86,3.39) Large p=0.002*	0.07 (-1.56,1.26) Small p=0.826
	Intervention				

LF/HF: low-to-high frequency; CI: confidence interval; ES: effect size; LF/HF: low-frequency-to-high-frequency. The magnitude of the ES (rating): <0.2 trivial; 0.2–0.6 small; 0.6–1.2 moderate; > 1.2 large, *p < 0.05: Significance difference between groups.

The volume of oxygen consumption and actual HR attained by the intervention group from the modified Bruce protocol are shown in Table V. The RPE measured at the end of running was 13.8 ± 2, which was rated as "Somewhat hard".

Table V. Oxygen consumption and heart rate data from the modified Bruce protocol of the intervention group (n=10)

	VO_2 (ml.kg^{-1}.min^{-1})	HR (beats.min^{-1})
Pre-exercise	4.26±1.7	77.90±13.3
Warm-up	9.07±1.9	90.70±12.9
85% HRmax	32.53±10.9	165.90±2.1
Active recovery (walking)	8.20±2.7	105.90±11.9

VO_2: Oxygen consumption, HR: heart rate, RPE: rate of perceived exertion. Results in mean±SD

DISCUSSION

The results obtained in this present study revealed that incremental exercise to 85% HRmax cause changes in the parasympathetic response up to 24 h post-exercise. Relative power estimated as HF n.u. was significantly increased whereas LF n.u. was significantly decreased at 24 h post-exercise. These results concur with previous data showing the main shift to parasympathetic activation after exercise (22). As peak and absolute values, our study showed the HF were not significantly affected up to 72 h post-exercise. Hence, our results suggest that the intensity of the exercise test and load were enough to maintain the dynamic autonomic regulation following submaximal exercise up to 72 h post-exercise. The sustained parasympathetic function can be influenced by endurance training. HRV modulation, due to vagal-tone, as a result of endurance training, has been associated with cardiorespiratory fitness (23). However, we did not correlate fitness with HRV values. Thus, it is not certain if the parasympathetic activation was due to the fitness level of our subjects. There may be a possibility that the full recovery of parasympathetic control to pre-exercise levels may have occurred immediately post-exercise irrespective of improved vagal tone (6). Rapid recovery of HRV can vary from 15 mins in sedentary subjects to an hour for a complete recovery following a moderate exercise intensity (2, 24).

As for LF power, our results showed significant increases at 24 h, and 48 h post-exercise with absolute power significantly increased at 48 h post-exercise. Previous study suggested that LF component of R-R interval variability reflects sympathetic activation (25). The LF is also associated with vagal tone, which is reflected by synchronous fluctuations in blood pressure and baroreflex activity (26). One of the possible adaptations to exercise is an increase in parasympathetic tone and thus an increase in the HF component (27). The higher the parasympathetic tone, and thus higher HRV, may be as a result of cardiovagal baroreflex sensitivity (28). This may suggests our submaximal protocol elicited temporary changes in the baroflex activity. However, as mentioned earlier, LF n.u. has also shown a significant decrease in our study, which was reflective of parasympathetic dominance at 24 h post-exercise. Although the reduced LF n.u. values in our study may seem to reflect diminished cardiac-vagal and sympathetic nerve activity possibly due to our subject's participation in regular physical training (29), the effect of intermittent type of training on reducing LF n.u. is still unclear and warrants further investigation. In multiple sprint interval exercise of supramaximal intensity and heavy endurance exercise or continuous fast running, the supine LF power was increased (30, 31). Regardless of physical fitness, these aforementioned studies, shows that a increased level of exercise intensity stimulates sympathetic activity that continues to dominate during recovery after stopping exercise, although parasympathetic activity has been re-activated (32).

Researchers consider LF relative power expressed in n.u. as a marker of sympathetic activity while LF power and absolute power as parameters that include both sympathetic as well as vagal influences (33). Thus, during recovery from exercise, LF peak and absolute power may be affected by the activities of increased breathing or respiratory sinus arrhythmia, occurring as a reaction to increased regulations of venous blood into the cardia due to the rising tidal pressure and volume in the lungs (34). At resting condition, LF was said to represent baroreflex activity and may be affected by increased breathing rate and mental activity as well (35, 36). This respiratory factor is particularly relevant to be controlled so that increased fitness from performing exercise can be accurately measured. Hence, we believe that the significant pre-exercise LF absolute values obtained in our study may be significantly affected by this respiratory factor prior to exercise in the intervention

group, causing the same significant changes in the pre-exercise LF/HF ratio as well. It is essential to note that reporting both absolute and relative values were to allow for direct comparisons between individuals in the same cohort (37) for practical and meaningful feedback, which is important in athletes undergoing the same training programme.

Parasympathetic activity in the LF and HF components was seen at post-exercise. However, when calculated as LF/HF ratio, sympathetic dominance was observed. The absolute and relative power of the frequency-domain HRV analysis may provide insights between several individuals, whereas the LF/HF ratio was to estimate a sympathovagal interaction (25). The presence of a significantly higher LF/HF ratio in the current study at 48 h post-exercise showed that the sympathetic activity appears to be maintained. Schmitt et al. (37) reported a higher LF/HF ratio in the supine position in the fatigued soccer players, showing a predominantly sympathetic activity. Our study demonstrated that incremental running to 85% HRmax has the potential to modify sympathetic activity after incremental running up to 48 h due to reduced vagal tone.

Previous data on HRV response to acute resistance type of exercise, the LF n.u. and HF indices returned to baseline values in 48 and 72 h respectively (38). Immediate post resistance exercise has been shown to reduce vagal tone as reflected in the HF n.u. index (39). Eventhough exercise such as resistance type of training is shorter in duration, the intensity of exercise is the most pertinent factor in modulating HRV. The parasympathetic activity (HF) typically decreased in exercise intensity above 50% peak VO_2 (40). In slightly trained individuals, the HF has been shown to decrease in response to increment in exercise intensities between 60% and 90% peak power output but remained unchanged beyond the 90% peak power output (41). Furthermore, prolonged and exhaustive cycling exercise can alter the HF components to gradually decrease in both athletes and non-athletes but not in moderate exercise (42). Stressful exercise conditions that lower the HF power should be avoided in ergometric exercise testing (43, 44). Previous data have shown that reduced HRV may indicate a risk of arrhythmias and sudden death after exercise (45). We agree that the reduction of parasympathetic activity and increases in sympathetic activity may show reduced recovery in athletes after intense exercise (46). Hence, sufficient rest should be provided after exercise of this nature.

Our study was limited by the breathing rate of the participant which we did not attempt to control. Although HRV was not affected by breathing during exercise, the breathing rate during resting can be a contributor to autonomic response (36). Considering the importance of breathing rate on recovery HRV recording in the 5 min epoch, whether during supine, standing or sitting, a metronomic breathing procedure with specific measurement conditions such as body posture must be reported to provide accurate assessment in future studies (44). Water intake, for which were given ad libitum in our study, has shown to enhance the immediate post-exercise vagal reactivation and sympathetic withdrawal (47). Although we measured VO_2 at 85% HRmax, we acknowledged that previous data found no correlation between VO_2 and recovery HRV (48). Hence, we did not attempt to determine whether maximal fitness (VO_2max) influenced the data but instead focused on the autonomic changes in the submaximal data. Future studies may include training load, or performance assessment and daily monitoring of individual HRV responses (49) including several days prior to intervention for better HRV interpretation.

CONCLUSION

Incremental running to 85% HRmax influenced the HRV autonomic response in football players for up to 48 h post-exercise. The parasympathetic activity was dominant at 24 h post-exercise when expressed as absolute and relative power. When expressed as LF/HF ratio, sympathetic activity was dominant at 48 h post-exercise. Acute submaximal exercise testing can modify autonomic response. The data may suggest HRV responses monitoring to be utilised for monitoring recovery in athletes from incremental exercise testing.

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