

Short-term recovery of heart rate variability and its relationship with blood pressure after different intensity treadmill exercise

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Research

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Abstract

Background : Heart rate variability (HRV) provides an opportunity to capture the tiny but early signs that may predict the future cardiovascular risk in healthy individuals and further, helps understand how well the cardiovascular autonomic system works. Aims of this study were to elucidate short-term recovery of HRV and its relationship with blood pressure recovery after different intensity treadmill exercise. **Methods :** Fifteen healthy participants performed four different conditions (REST; speed 6km/h; speed 8km/h; speed 10km/h), systolic and diastolic blood pressure per 30s (SBP, DBP) and 5-mins consecutive heart beats intervals were measured after each trial. Autonomic nervous regulation was evaluated using HRV time-frequency domain indices and heart rate asymmetry (HRA) indices. Each index was calculated using 5 mins electrocardiogram (ECG) series and consecutive 30-s windows in 5 mins. **Results :** the vagally related indices (RMSSD, pNN50 and HF) decreased and the indices representing overall variability (SDNN, LF) had different trends as intensity increasing. The sympathetic-vagal balance parameter LF/HF increased, too. HRV indices had strong correlations with DBP but weak with SBP. Meanwhile, heart rate asymmetry vanished after each trial. **Conclusions :** The findings suggested a vagal withdrawal as soon as the end of treadmill exercise. It could be concluded that sympathetic modulation was stronger as intensity increasing. During recovery period, DBP was mediated by vagal activation and sympathetic withdrawal. The diminished asymmetry in Poincaré plot was the result of sympathetic acceleration and vagal reduction.

Background

Heart rate variability (HRV) has been studied extensively across a variety of academic areas as it is thought to reflect the complex interaction between the autonomic nervous system and cardiovascular system [1]. Extracted from the electrocardiographic (ECG) signals, HRV provides a non-invasive insight into the interplay between sympathetic and vagal activities of the ANS [2, 3]. The normal variability is due to automatic neural regulation of heart and circulatory system, increased sympathetic activities or diminished vagal activities results in cardio-acceleration; on the contrary, low sympathetic activities or high vagal activities causes cardio-deceleration.

Short-term recovery of HRV during post-exercise period has been considered as an independent indicator for cardiovascular diseases [4]. It is widely believed that abrupt alterations in cardiovascular autonomic tone after dramatical exercise contribute to the increasing risk of cardiac death [5–7]. The result is mainly caused by sympathetic hyperactivity and reduced cardiac vagal tone after acute exercise [8]. Considering the profound influence of exercise on HRV, more and more researchers focus on the application of HRV in exercise. Parekh et al reported that high intensity exercise resulted in a greater shift in sympathetic-vagal balance compared to moderate exercise during post-exercise period [9]. Amano and Oliveira et al supported that monitoring parameters of HRV could be useful for tracking the time course of training adaptation in order to set optimal training loads which lead to improved performances [10–12]. Tulppo et al reported some indices of HRV increased with increasing exercise intensity [13]. The change of cardiovascular autonomic modulation at post-exercise is mainly triggered by parasympathetic reactivation

reduction, and it has important physiological and clinical significance as it is a potential factor of cardiovascular events following exercise [14, 15]. It's considered that three mechanisms are thought to be responsible for the neural cardiovascular modulation during exercise: the activation of higher brain centers, the reflex activity from chemo and mechanoreceptor as well as baroreceptor afferents [16–19]. Each mechanism activates neuronal circuits within the medulla and thus modulates the balance between sympathetic and vagal nerve. The influence of each mechanism on physiological parameters response to exercise depends on recruited muscle mass, muscle fiber type, exercise mode and exercise intensity [20]. Compared to low exercise intensity, high intensity would elicit a stronger chemoreflex response for blood flow and release of metabolites is limited.

The chemoreflex elevates blood pressure (BP) by a sympathetic vasoconstriction, also affects sympathetic modulation [21]. It is essential to evaluate the relationship between HRV recovery and BP recovery as combined HRV and BP metrics suggests cardiovascular autonomic short-term response to exercise. Unfortunately, few studies have evaluated the correlation between short-term HRV recovery and BP recovery at different intensity post-exercise applied to autonomic modulation.

Therefore, aim of the present study was to examine the cardiovascular autonomic response to different intensity exercises. Also, we identified the correlation between short-term HRV recovery and BP recovery following different treadmill exercises. This work largely focused on the short-term continuous BP recovery and mechanisms that controlled and regulated these outcomes. HRV indices were quantified by time-frequency domain analysis using 5 min ECG series, which was introduced in (2.4). Furthermore, we explored heart rate asymmetry (HRA) by Poincaré plot tool, which was to verified shift of sympathetic-vagal balance in different intensity treadmill exercise.

Results

HRV Recovery of time-domain indices

Time-domain HRV indices (SDNN, RMSSD and pNN50) with interval of 30 s were calculated in Fig. 2. All three indices tended to be stable in resting condition. At 6 km/h post-exercise, SDNN and pNN50 were observed obviously decreased compared to baseline of resting condition. Significant differences of the two indices were identified between resting condition and 6 km/h post-exercise. The recovery of RMSSD had a distinct float at 6 km/h post-exercise, and there was no significant difference from REST. After other two exercise trials, three indices went further declined in response to higher intensity. Significant differences of three indices were detected between 6 km/h and 8 km/h ,10 km/h post-exercise, but not between 8 km/h and 10 km/h trials.

HRV indices assessed by complete 5 min ECG series were summarized as mean $\pm$ standard deviation (SD) in Fig. 3. There were obvious decreased trends for indices as exercise intensity increased. No significant difference was noted only between REST and 6 km/h post-exercise, while among the others had significant differences.

Recovery of frequency-domain HRV indices

The results of frequency-domain HRV indices per 30 s were given in Fig. 4. Compared with REST, the values of LF and LF/HF ratio had no absolute change in 5 min, while HF got slightly decreased at 6 km/h 1 post-exercise. No significant differences were identified between these two trials. With exercise intensity increasing, LF and LF/HF ratio got distinctly augmented, and significant differences were found between REST and 8 km/h, 10 km/h trials. Moreover, at 8 km/h and 10 km/h post-exercise, the values of HF decreased compared to 6 km/h post-exercise, but no differences were identified among three trials for HF.

In Fig. 5, LF and LF/ HF ratio assessed by 5 min ECG series were observed increased as intensity increasing, accompanied with decrease in HF. For LF, there was no significant difference between 6 km/h and REST, 8 km/h post-exercise. Differences failed to reach statistical significance between REST and 6 km/h post-exercise for HF and LF/HF either ($p = 0.9380$, $p = 0.1076$). Among other trials differences were significant.

BP and its correlation with HRV indices

Simultaneous SBP and DBP were recorded in Fig. 6. Both SBP and DBP got declined trend and were back to resting condition in nearly 3.5 min during recovery period. Notably, different intensity exercise produced different cardiovascular response in DBP recovery. There was a slight reduction in DBP comparing to REST DBP following treadmill exercise, and the reduction trend was more obvious after lower intensity exercise.

Table 2 showed data for significant correlations between HRV indices and BP in different post-exercise periods. Time-domain SDNN, RMSSSD and pNN50 indices of HRV showed positive correlation with SBP only at 8 km/h post-exercise. Instead, at 10 km/h post-exercise, SBP was negatively correlated with frequency-domain LF and LF/HF indices and positive correlation with HF, no correlation with time-domain indices. For DBP, a positive correlation with time-domain SDNN, RMSSSD and pNN50 indices was observed after each trial, as well as a negative correlation with LF and LF/HF ratio. No correlation was found between DBP and HF at 8 km/h post-exercise, but showed a positive correlation during other recovery periods. Therefore, BP correlated with HRV indices at post-exercise period reflected cardiovascular autonomic activities.

Table 2
Spearman's correlation coefficients between BP and HRV indices

	SBP				DBP			
	rest	6 km/h	8 km/h	10 km/h	rest	6 km/h	8 km/h	10 km/h
SDNN(ms)	-	-	0.286**	-	-	0.478**	0.428**	0.278**
RMSSD(ms)	-0.374*	-	0.334**	-	-	0.455**	0.376**	0.405**
pNN50(%)	-0.315*	-	0.323**	-	-	0.438**	0.454**	0.342**
LF (ms ²)	-	-	-0.19*	-0.196*	-	-0.271**	-0.201*	-0.263**
HF (ms ²)	-0.321*	-	-	0.219*	-	0.242**	-	0.186*
LF/HF	0.282*	-	-	-0.182*	-	-0.263**	-0.205*	-0.255**
* represents p < 0.05; **represent p < 0.01.								

HRA at different intensity post-exercise

Visually, HRA is defined as imbalanced distribution of points above and below the line of identity of Poincaré plot. Figure 7 summarized short-term recovery of HRA indices over time in 5 min. All four indices stayed in a steady state in resting condition and were larger than 50%. At 6 km/h and 8 km/h post-exercise, these indices had normal fluctuations but did not change too much compared to REST. The indices showed no statistical differences between REST and these two trials. It was notable that indices got absolutely decreased during the first 30 s of recovery period. It was considered as the result of reactivation of vagal drive and a decrease in sympathetic drive that usually occurred immediately after exercise [41]. However, abrupt and obvious fluctuations of HRA indices were observed after the third trial. Indices went down to minimum at 3th min and then increased, but didn't recover to resting condition until 5 min at 10 km/h post-exercise. HRA indices were significantly different between this trial and others. Furthermore, Fig. 8 showed the mean $\pm$ SD of HRA indices assessed by 5 min ECG series. There was obvious difference between rest and exercise. Statistical differences were observed between resting condition and three trials, but not between the exercise trials.

Discussion

Cardiovascular autonomic response to different intensity exercise

The present study investigated the modulation of autonomic system at post-exercise period [42] via HRV indices calculated on consecutive 30-s windows. Meanwhile, the BP recovery was studied and the correlation between short-term recovery of HRV and BP at different exercise intensity was firstly investigated.

The results showed that cardiovascular autonomic modulation was altered in the recovery phase after different intensity exercise. Generally, RMSSD, pNN50 and HF were thought to reflect vagal modulation of sinoatrial (SA) node, while SDNN and LF marked both sympathetic and vagal nerve activities [43]. In recovery phase, sympathetic activity was more active after higher intensity exercise, which could be proved by increasing LF and LF/HF ratio. At the same time, the decreased RMSSD, pNN50 and HF demonstrated decreased vagal activity as exercise intensity increased. Our results were not entirely consistent with the previous reports, which suggested that the early recovery of HR was mainly mediated by sympathetic withdrawal and vagal reactivation [5, 44, 45]. It was reasonable to explain that vagal withdrawal was remained as soon as the end of exercise, which meant vagal influence on HR was stable in initial early recovery phase. Kannankeril et al examined that heart rate remained stable during 4–10 min in post-exercise phase [5]; Justin et al suggested HRV measured during 5–10 min of recovery would accurately portray vagal response instead of the first min of recovery [46]. The results in present study were in accordance with their researches.

The present findings suggested that the prognostic significance of delayed recovery was attributed to heightened sympathetic effects and attenuated recovery of parasympathetic effects. The similar variation trends and overall results of HRV indices between resting condition and at 6 km/h post-exercise suggested both sympathetic and vagal activation but sympathetic did not achieve dominance. Extreme changes of HRV indices after other two trials indicated that sympathetic nerve activity was greater and achieved dominance over vagal nerves after higher intensity exercise. Buchheit et al [42] and Fisher et al [47] had reported that high-intensity exercise increased contribution of glycolytic metabolism, and metabolites were observed by stimulating the sympathetic activity and delayed parasympathetic reactivation after exercise in their studies [48]. Furthermore, the LF/HF ratio, which was considered as an index of the balance between sympathetic and vagal activity, was increased as exercise intensity increased, indicating a possible shift toward a heightened sympathetic modulation. These findings would highlight further research about autonomic regulation after different intensity treadmill exercise.

The relationship between autonomic modulation and BP

BP was one of the most extensively studied variables evaluating recovery of cardiovascular system after exercise. The process was a dynamic period in which many physiological changes occurred. Treadmill exercise was considered as muscle mass exercise and would impose a volume load on cardiovascular system. Generally, magnitude of the increase in vascular conductance and reduction in vascular resistance was larger than elevations in cardiac output following dynamic exercise, which meant that vasodilation was the driver of BP recovery [49]. An interesting phenomenon was found this study: SBP returned to REST at the 4th min and remained stable after that, DBP recovered to REST at 3.5th mins but further went down to lower level after that, especially after lower-intensity exercise trial. This was mainly caused by venous return stemming from physical vasodilation of the vasculature [50].

Meanwhile, central command mechanism had been proposed to play an important role in cardiovascular regulation, it served as a feed-forward system that was related to post-exercise vasodilation [51]. That was why the relationship between HRV indices and BP appeared during recovery phase. The autonomic system

utilized chemo- and baroreceptors to monitor pressure as well as current metabolic status of the body, when chemo- and baroreceptor activities were integrated in the medulla, flow-pressure was constantly monitored and adjusted during exercise. Then, the change was fed back to medulla again, these receptors adjusted flow-pressure by altering HR and BP [52–55]. A positive correlation between DBP and SDNN, RMSSD, pNN50, HF indicated that DBP was regulated by vagal activation, while a negative correlation between DBP and LF meant reduction in DBP was also influenced by sympathetic withdrawal. The reason of the weak correlations between SBP and HRV indices needed further investigation. To summarize, autonomic system contributed to blood pressure regulation and further investigation about SBP was necessary to provide relevant and significant explanations. This finding gave a new insight into the interplay between BP regulation and cardiovascular autonomic activities in post-exercise period.

Autonomic modulation reflected by HRA at post-exercise

The nonlinear methods come from the fusion of topology and differential equation theory. As we know, HRV signals has non-linear dynamic characteristics, because heart rate is mainly controlled by the autonomic nervous system, while autonomic nervous activities are mainly affected by brain activity, which is a complex dynamic system containing multiple nonlinear oscillators. It is necessary to use nonlinear methods to analyze the dynamic characteristics of HRV.

Complementary to traditional time and frequency parameters, non-linear indices were able to identify the different patterns of autonomic regulation after different intensity exercise. HRA was described as a visible phenomenon, which quantified imbalance of heart rate acceleration and deceleration using a two-dimension Poincaré plot [39, 40, 56]. It was reported to mostly present in healthy participants under resting condition and decrease with pathology [57, 58]. Indeed, the asymmetry meant that sympathetic and vagal branches of ANS contributed unequally to heartbeat, the velocity and direction of shift changed from time to time in different exercise intensities. Previous studies suggested that elevated exercise intensity was crucial to cause imbalances of sympathetic-vagal activities. Figure 7 indicated that the higher exercise intensity, the greater the HRA indices fluctuation. The greater fluctuation was an indicator of acceleration contribution to magnitude asymmetry in elevated intensity. This was in accordance with the concept that the higher the intensity, the higher sympathetic activity in response to post-exercise excitatory stimulus, as we mentioned before. Furthermore, the results accessed by 5 min ECG series exhibited that HRA phenomenon disappeared at post-exercise period with indices (AI, PI, GI, SI) approaching 50%. This verified the acceleration of sympathetic modulation and reduction in vagal activity at post-exercise period. Guzik et al had reported that the HRA might be related to the response of the baroreflex to increase or decrease the blood pressure [39]. However, the inner mechanism between HRA and BP was unclear and remained to be investigated.

Limitations in this work included exclusion of younger and older subjects of any age range, so the findings could not be precisely extrapolated for these people. In addition, we performed the relationship between HRV and BP after different intensity treadmill exercise, which should be tested for different exercise modes. Previous investigators have demonstrated that cardiac vagal tone and vascular system had great different response to resistance exercise versus endurance exercise [50, 22]. We are intrigued to do more

research in the future work to provide additional information understanding the mechanism involved after different exercise modes.

Conclusions

In conclusion, the present study preliminarily investigated the cardiovascular autonomic responses to different intensity exercise. The results showed that participants had more augmented modulation of sympathetic tone as intensity increased and achieved dominance in high-intensity exercise. Also, the correlation between HRV indices calculated on consecutive 30-s windows and BP recovery indicated that the decrease of DBP was associated with vagal activation and sympathetic withdrawal. The diminished HRA in a Poincaré plot after exercise was influenced by acceleration of sympathetic modulation and reduction in vagal activity.

Methods

Participants

Table 1 gives the basic characteristics of all participants. Fifteen young participants were invited to take part in this study, including 10 males and 5 females. All participants were physically healthy and none of them had cardiovascular disease history and took medication. One week before the experiment, participants were informed not to smoke or take any exhaustive exercise and any alcohol or caffeine was not allowed for three days prior to the experiment. All participants gave written informed consent to participate in this study.

Table 1
Participant characteristics

Selection factor	Values
Total number (M, F)	15 (10, 5)
Age (years)	22.3 ± 1.2
Height (cm)	174.0 ± 8.4
Body mass (kg)	68.4 ± 9.5
BMI (kg/m ²)	22.5 ± 1.5

Experimental protocols

The study was performed in neutral environment as external factors (i.e., noise, temperature, light) could exert influences on HRV indices and caused sympathetic predominance [3]. Participants took three treadmill exercises: speed 6 km/h; speed 8 km/h; speed 10 km/h. All trials were tested on one day to minimize confounding effects on ANS from daily activities and diurnal influences. Kevin et al found that

cardiovascular autonomic system was not fully regained to baseline within 30 min of endurance exercise [22]. Parekh et al [9] and Terziotti et al [23] reported that HR remained elevated 30 min and full autonomic recovery may take more than 1 hour after treadmill exercise [15]. Basing on the fact that each trial lasted only 3 min in our study, participants were required to take a 2-hour break to ensure autonomic nervous system could recover to REST. It was believed that effects on autonomic modulation for next trial were excluded.

Upon arrival, participants were required to sit quietly to get relaxed. Then 5-mins data of ECG series were collected in sitting position while the participants remained at resting condition. Meanwhile, brachial BP was collected simultaneously during this period. The participants were then directed to run on a treadmill for each trial. Another 5-mins ECG series were collected immediately after each trial, as well as the simultaneous BP. The experiment procedure was showed in Fig. 1.

Data acquisition system

In the present study, we chose Power-lab/16sp system (Castle Hill AD Instrument, Australia, 2002), which could record, display and analyze ECG signal by combining software and hardware. The ECG signal was recorded at 1 kHz with a pre-amplification and filtered by a 0.13 Hz high-pass filter and a 100 Hz low-pass filter. Brachial BP was measured by using OMEON HEM-7211. Sphygmomanometer was calibrated before the experiment and after each exercise trial. Sampling frequency was set at 1 kHz. After eliminating abnormal R wave peaks, all R wave peaks corresponding to sinus rhythm were detected and NN intervals were calculated.

Assessment via HRV analysis

The prognostic value of HRV has encouraged the development of quantifiable interpretations of HRV by time and frequency-domain analysis and nonlinear methods [24]. As a measure of the fluctuation of heartbeat intervals, HRV is a reliable and quantitative marker of cardiovascular autonomic system activity. Short-term HRV analysis, which was used in this study, has been proved to overcome high non-stationarities problem in long-term HRV analysis [25, 26] and is less influenced by recording conditions and suitable for study of short time ANS response [27, 28]. HRV analysis is a well-recognized tool for estimating cardiac autonomic function [3]. In this study, all HRV indices were calculated using whole 5 min ECG series and at intervals of 30 s in 5 min.

1. Time and frequency-domain analysis

Time domain analysis is a direct method for statistical and geometric analysis of the collected NN intervals. Time domain indices include:(a) the standard deviation of all NN intervals (SDNN) that reflects the overall autonomic modulation;(b) the root mean square of standard deviation between adjacent NN intervals (RMSSD) and pNN50 which means the percentage of heart beats with the difference of adjacent NN intervals greater than 50 ms in all NN intervals [29], both of them reflect the vagal modulation associated with respiratory sinus arrhythmia.

Frequency domain analysis can provide changes of the activity level and the equilibrium of sympathetic and vagal nerve and is especially suitable for short- time HRV analysis. Fast Fourier transform was used to determine the power spectrum of HRV. The power spectrum was then divided into four parts. It has been generally accepted that high-frequency components (HF: 0.15–0.4 Hz) are mediated entirely by vagal nerve; the low frequency component(LF), which fluctuates between 0.04 and 0.15 Hz, is regulated by pressure receptors of sympathetic and vagal tension; while the very-low frequency (VLF) is probably due to slow mechanisms of regulation such as humoral and thermoregulatory factors [1, 30]; there is no clear physiological explanation about the ultra-low frequency(ULF) so far. The VLF and ULF component were not addressed in the present study. The ratio of LF to HF power is usually used to estimate sympathy-vagal balance. An increased ratio identified a sympathetic predominance during less intensity exercise, while a decreased ratio meant a vagal predominance during relatively more intense exercise [31, 32]. All time and frequency-domain indices were calculated at intervals of 30 s in 5 min.

2. HRA of Poincaré plot

Poincaré plot is one of the quantitative visual tools which gives information on long- or short-term HRV. It is physiologically asymmetric with respect to the line of identity (L: $y = x$), and the asymmetry usually shifts in the case of physiological change [33–36]. The conventional indices, SD1 and SD2, which are the standard deviations of data in the X 'and Y' directions in the rotated coordinate system, have no essential difference with time-domain indices. That is why we choose HRA indices to assess cardiovascular autonomic modulation in the present study. Many indices have been proposed to assess HRA. Area index (AI) is defined as the cumulative area of the sectors corresponding to the points that are located above the line of identity divided by the cumulative area of sectors corresponding to all points in the Poincaré plot, which is a new index proposed by Yan et al in 2017 [37]. It can be calculated by

$$AI = \frac{\sum_{i=1}^l |S_i|}{\sum_{i=1}^m |S_i|} \times 100\% \quad (1)$$

Other indexes include Porta's index (PI), Guzik's index (GI) and Slope index (SI) which are respectively defined as the number, the distance and the phase angle of points below L divided by total number, distance and phase angle of points in Poincaré plot except those located on the line [38–40]. Specifically,

$$PI = \frac{a}{m} \times 100\% \quad (2)$$

$$GI = \frac{\sum_{i=1}^l Di}{\sum_{i=1}^m Di} \times 100\% \quad (3)$$

$$SI = \frac{\sum_{i=1}^l |R\theta_i|}{\sum_{i=1}^m |R\theta_i|} \times 100\% \quad (4)$$

where a is the number of points below L , m is the total number of points except those on L in (2). D_i means the distance of point P_i to L in (3). $R\theta_i$ is the phase angle of the i -th point in (4).

Statistical analysis

Normal distribution of each index was assessed applying the Kolmogorov-Smirnov test revealing a normal distribution for all HRV indices. The overall indices calculated using complete 5 min ECG series were summarized as mean $\pm$ standard deviation (SD). The between-trial difference (i.e., rest vs 6 km/h, rest vs 8 km/h, rest vs 10 km/h, 6 km/h vs 8 km/h, 6 km/h vs 10 km/h, 8 km/h vs 10 km/h) of time and frequency-domain HRV indices was analyzed by repeated-measures two-way ANOVA. Spearman's correlation test was employed to verify the relationship between short-term HRV recovery and systolic, diastolic BP recovery. Statistical significance was accepted at $p < 0.05$. All statistical analyses were performed using the SPSS software (version 24, IBM, New York, USA).

Abbreviations

HRV: Heart rate variability

SBP: systolic blood pressure

DBP: diastolic blood pressure

ECG: electrocardiogram

ANS: autonomic nerves system

HRA: heart rate asymmetry

RMSSD: root-mean-square of difference-value of adjacent RR interval

SDNN: standard deviation of NN intervals

pNN50: The percentage of adjacent NN difference >50 ms in total sinus beats

HF: high frequency component

LF: low frequency component

VLF: very-low frequency

ULF: ultra-low frequency

AI: area index

PI: Porta's index

GI: Guzik's index

SI: Slope index

SD: standard deviation

Declarations

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

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Contributions

Liang Wu and Ping Shi: proposed the idea and designed the method; Jiang Shao and Anan Li performed experiments and analyzed the data. They made discussions and composed the manuscript together with Hongliu Yu and Yang Liu. All authors read and approved the final manuscript.

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Ethics declarations

Ethics approval and consent to participate

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. The study was approved by the ethics committee of University of Shanghai for Science and Technology, Shanghai, China (Ref. No. 2013-9010-14YZ091).

Consent for publication

All authors gave their consent for publication.

Competing interests

The authors declare that they have no competing interests.

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Figures

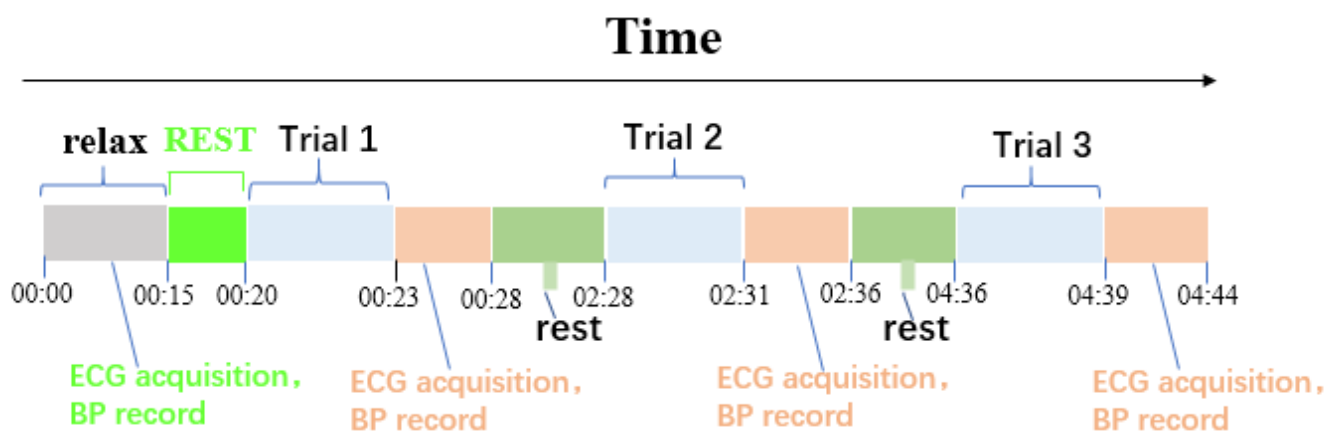


Figure 1

Timeline in mins of this experiment.

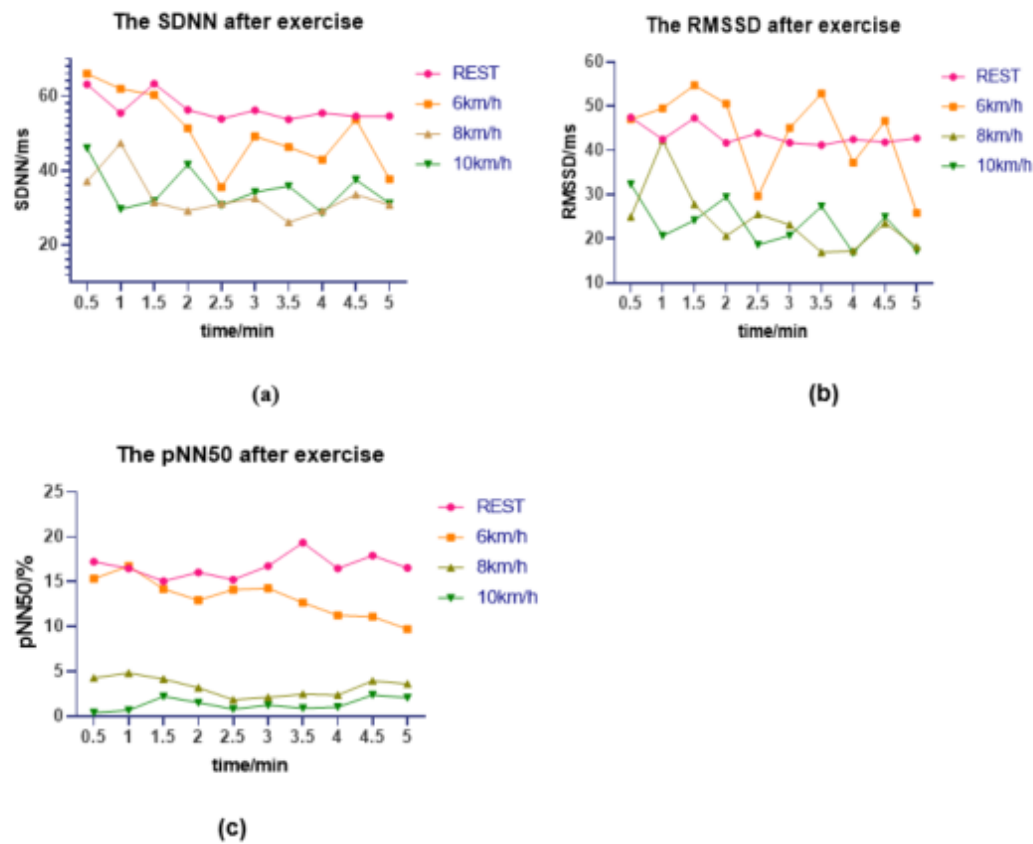


Figure 2

Time-domain indices per 30s at different intensity post-exercise. (a): SDNN;(b): RMSSD;(c): pNN50.

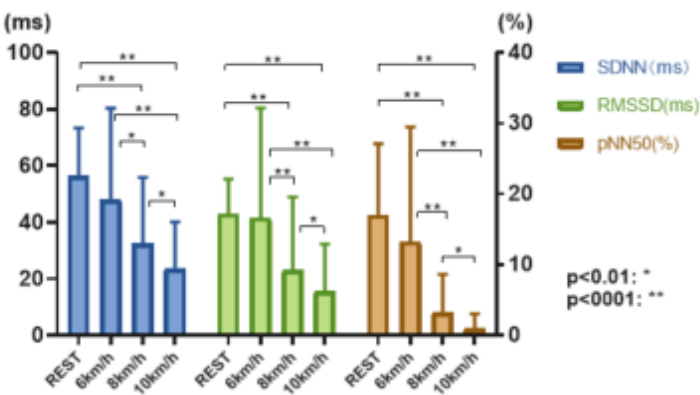


Figure 3

Time domain indices assessed by 5 mins ECG series in different trials.

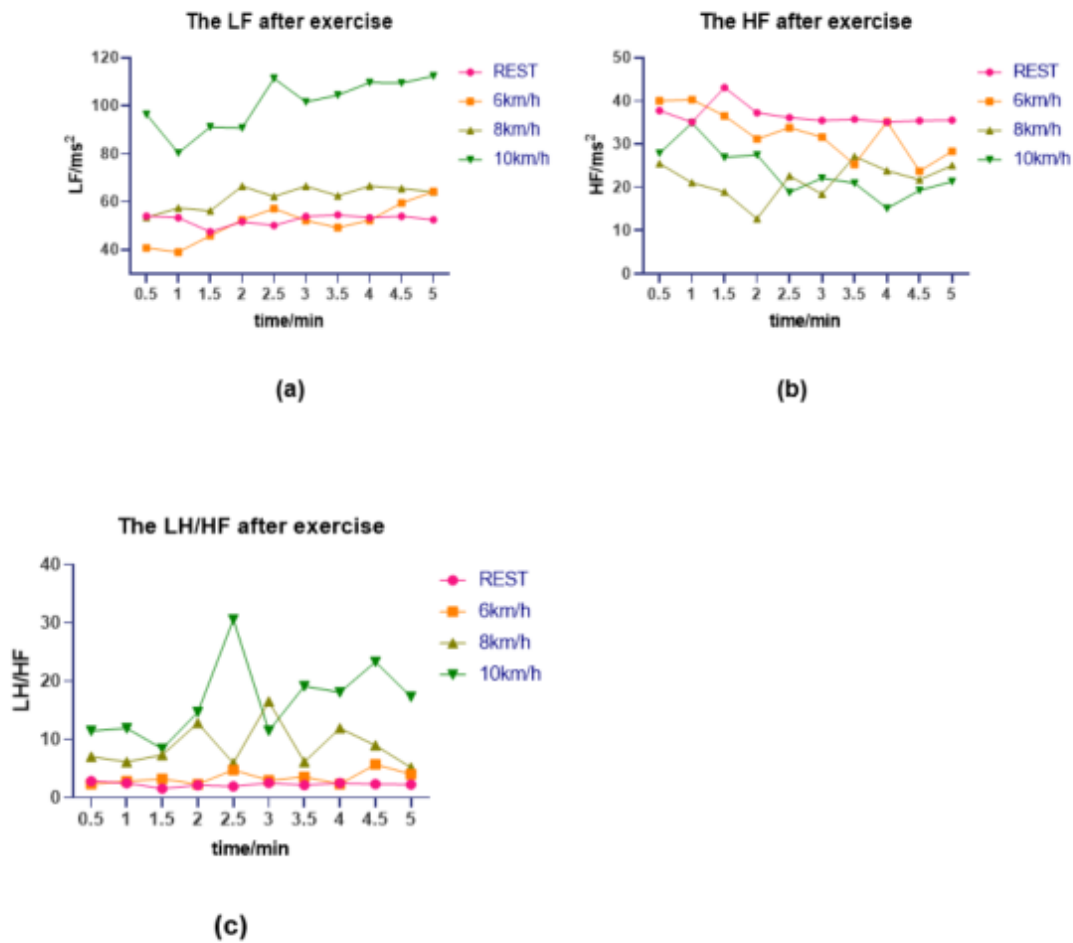


Figure 4

Frequency-domain indices per 30s at different intensity post-exercise. (a): LF;(b): HF; (c): LF/HF.

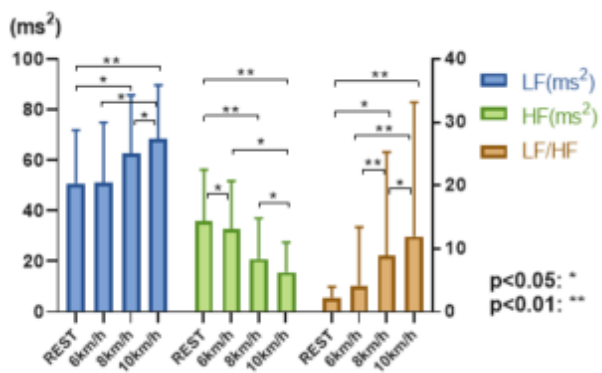


Figure 5

Frequency domain indices assessed by 5 mins ECG series in different trials.

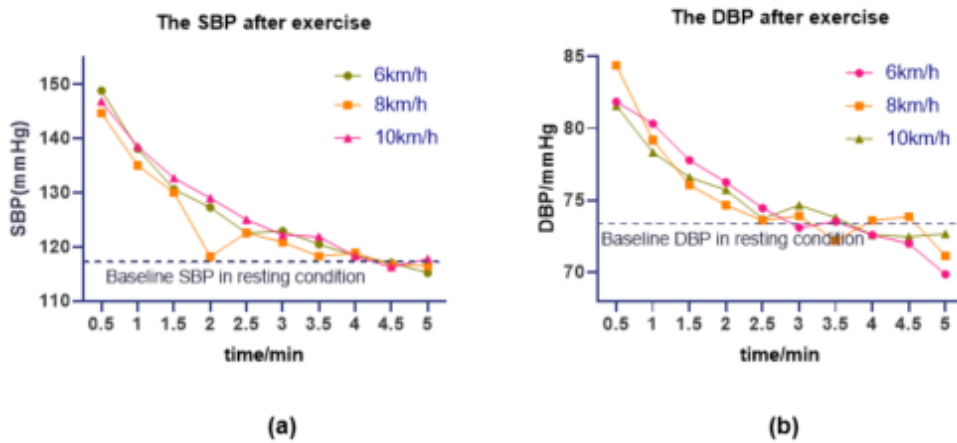


Figure 6

SBP and DBP at different intensity post-exercise. (a): SBP; (b): DBP.

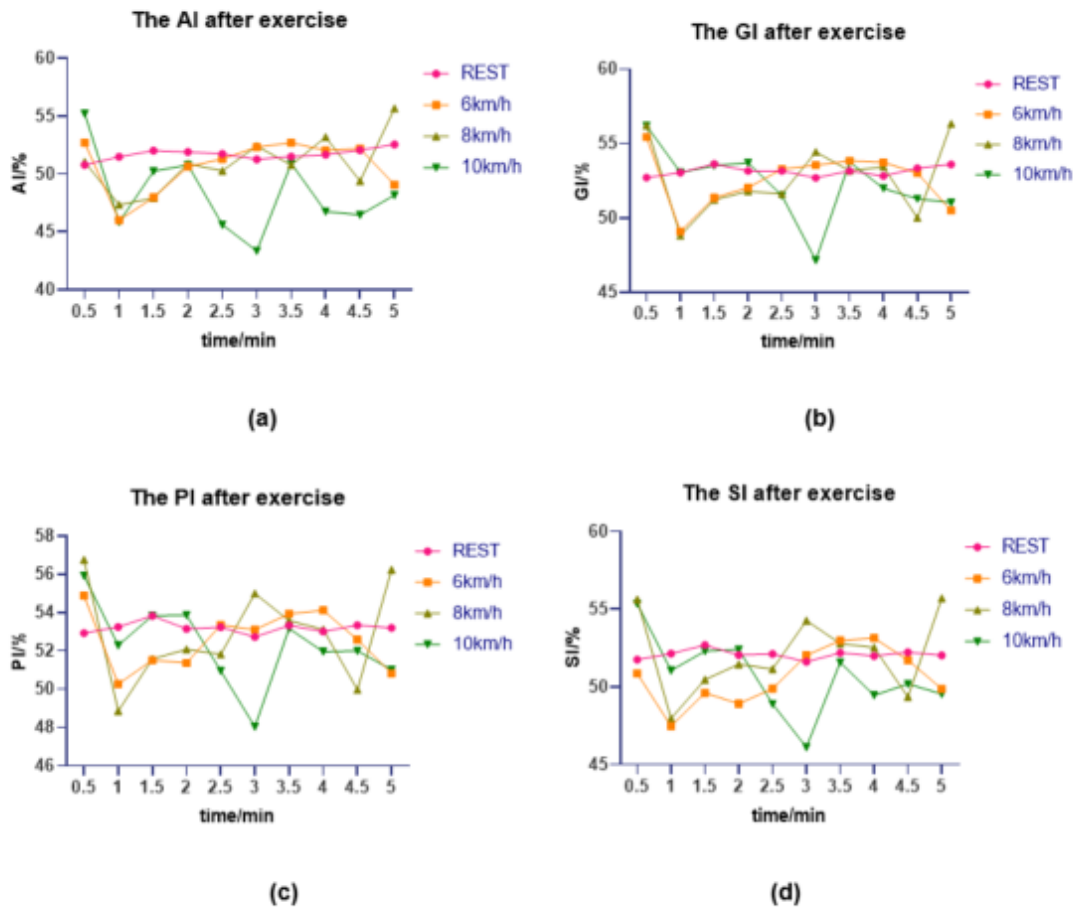


Figure 7

HRA indices per 30s at different intensity post-exercise. (a): The change of AI. (b): The change of GI. (c): The change of PI. (d): The change of SI.

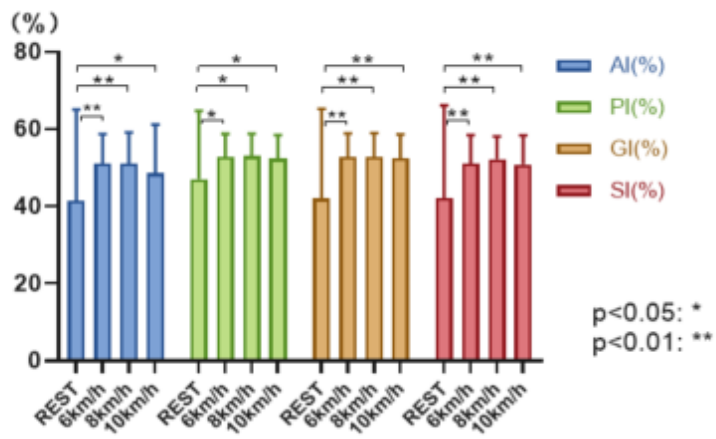


Figure 8

HRA indices assessed by 5 mins ECG series in different trials.