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Detraining effect on cardiac autonomic response to an all-out sprint exercise in trained adolescent swimmers

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Abstract

Purpose: This study aimed to investigate the effects of a 5-week training cessation period in the cardiac autonomic response after a 50 m swimming time-trial test. **Methods:** Twenty trained and highly trained adolescent swimmers (17.1 ± 2.7 years) performed a 50 m front crawl all-out before (Visit 1) and after a 5-week training cessation period (Visit 2). After the warm-up, heart rate variability (HRV) was recorded in a seated position using a Polar RS800CX heart rate monitor during the 10 min before (pre-exercise) and immediately after the 50 m front crawl all-out test (post-exercise). Two-way analysis of variance (Time × Visit) and analysis of covariance were conducted to compute the effect of the 50 m all-out-test in vagal-related HRV parameters (mean R-R, SDNN, RMSSD, pNN50, and HF) with Bonferroni post-hoc test. **Results:** All the HRV parameters had a Time main effect ($p<0.05$), showing a reduction after the 50 m in both Visits ($p<0.05$). Visit main effect was exhibited in all the variables ($p<0.05$), the pre- and post-exercise Mean R-R, LnSDNN, LnRMSSD, and LnHF values declined after the training cessation ($p<0.05$). LnpNN50 pre-exercise values were reduced in Visit 2 compared to Visit 1 ($p<0.05$). Only Mean R-R was further reduced in response to the test in Visit 2 than Visit 1 ($p<0.05$). **Conclusions:** After 5-week of training cessation, all the pre- and post-exercise vagal-related HRV parameters evidenced a reduction, suggesting an impairment in swimmers' physical status. Coaches should be cautious with the training loads at the start of the season.

Keywords: anaerobic exercise, autonomic nervous system, heart rate variability, parasympathetic activity, swimming performance.

Introduction

Heart rate variability (HRV) is a non-invasive and effective tool to monitor cardiac autonomic balance (i.e., the combined influence of sympathetic and parasympathetic branches of the autonomic nervous system on sinoatrial node).[1] HRV is the variation in time between consecutive heart beats or R-R peaks,[2] and is widely expressed using different parameters in time- and frequency-domains. HRV parameters can be easily assessed using portable devices as for example heart rate monitors.[3] Thus, these devices allow the assessment of HRV responses to diverse stimuli as exercise, neurophysiological changes during different athletes' training moments,[4,5] and also the training status of athletes,[1] offering data in "real-time" that facilitate decision making for controlling the training load,[6] among others.

The HRV at rest is currently used as a training control method, being a useful tool to evaluate the athletes' adaptation state.[7,8] For instance, recently, Pla et al. [9] showed how changing the volume in the training zones induces a different response in resting HRV parameters. Nevertheless, the "reactivity hypothesis" suggests that investigating responses to a stressor (e.g., exercise) may be a valuable monitoring approach in high-performance settings.[1] Indeed, the HRV response to acute exercise can be modulated with training. For instance, Yamamoto et al.[10] observed how after 6 weeks of endurance training, the parasympathetic activity, measured after 40 min cycling, increased. Similar results were observed after only 4-week of high-intensity interval training (HIIT), 14 well-trained adolescent swimmers presented an increase in parasympathetic modulation (i.e., higher values on vagal-related HRV parameters) after the same HIIT test session.[11]

The HRV response to exercise has been mostly studied in endurance events due to the association between cardiac autonomic response and aerobic fitness.[12] Nevertheless, it has also been observed an association between cardiac autonomic function and sprint performance.[13] Opposite to the outcomes observed for endurance athletes, a lower vagal tone in rest was positively associated with the 50 m best seasonal time (i.e., the lower the vagal tone the higher the performance).[13] Therefore, this contrast in the responses obtained in endurance and sprint athletes should be better explored to emphasize the importance of assessing HRV responses in sprint athletes. On the other hand, at the end of each season, most swimmers usually experience a 4-6 weeks interruption to their training programs, which commonly produces a reduction or cessation of their habitual day-to-day physical activity. Although this stoppage is necessary to enhance the athletes' physiological and mental recovery, is common that during this training cessation period they experience a physiological phenomenon called *detraining.*[14]

Detraining is commonly manifested as a partial or complete loss of training-induced anatomical, physiological, and performance adaptations.[14] This training cessation impairs the cardiovascular function in response to exercise, arising 5 – 10% the athletes' maximum heart rate (HR) for the same effort or evoking an important reduction in the vagally-mediated HRV parameters in high-level endurance runners.[15] However, these impairments have been mainly studied in endurance events.[14,16] Thus, due to the scarce knowledge about the impact of detraining in sprint swimming events, conclusive evidence concerning cause-and-effect relationships between sprint swimming performance and cardiac autonomic response is still required. Hence, it is of interest to test the effects of detraining on swimmers' HRV response to a sprint event. Therefore, this study aimed to investigate the differences between changes in HRV parameters induced by an all-out effort (50 m) after a 5-week training cessation period.

Methods

Participants

Twenty adolescent trained and highly-trained swimmers,[17] 13 males and 7 females participated in the current study. At beginning of the study, the swimmers had a mean age of 17.1 ± 2.7 years, 172.4 ± 8.2 cm of height; 62.9 ± 9.1 kg of body mass; body mass index (BMI) of 21.1 ± 2.2 kg/m^2; and 50 m front crawl *Fédération Internationale de Natation* (FINA) points of 494 ± 76 (Performance Level 4[18]). Similar anthropometrics values were observed during the second testing (172.6 ± 8.0 cm, 63.1 ± 8.8 kg, and 21.4 ± 2.2 kg/m^2). Swimmers had more than 5 years of competitive experience and trained 6 swimming sessions per week in the same squad under the direction of the same coach. The protocol, benefits, and risks associated with participation in the study were explained to each subject or their parents (e.g., swimmers under 18 years) before they provided written consent to participate. The study was conducted according to the code of ethics of the World Medical Association (Declaration of Helsinki, 2013), and the protocol was approved by the University Ethics Committee (project code: ANONYMITY).

Design

This data is part of a more comprehensive longitudinal single cohort study performed before and after 5-week off-season period.[19] Participants were engaged into 2 testing sessions (hereinafter *Visit*), at the end of the week prior to the last peak-performance of the competitive season (Visit 1) and before the beginning of the first week of training of the next competitive season (Visit 2). To avoid a possible "learning effect", before Visit 1, swimmers performed a "familiarization session" with the all-out test. Of note, before the beginning of the study, all swimmers followed the training program set by their coach.

In both Visits (Visits 1 and 2), swimmers were tested on the same time of the day to avoid possible biases due to circadian variation.[20] They were instructed to refrain from intense exercise and/or vigorous physical activity and to abstain from stimulant beverages consumption (during both the day before each Visit and within the Visit). The evaluation protocol took place in a 25 m swimming pool (25 m length × 16.5 m width with 27.3ºC, 29.4ºC, and 52% of water and ambient air temperature and relative humidity in the Visit 1, and 27.4ºC, 28.9ºC, and 54% in the Visit 2).

Methodology

On each testing Visit (**Figure 1**), swimmers arrived at the research center and anthropometric measurements were performed. The height and body mass were measured using a stadiometer/scale respectively (Seca 799, Hamburg, Germany). BMI was calculated as body mass (kg) / height^2 (m). On both Visits, the measurements were conducted by the same researcher with participants being barefoot and wearing the same swimsuit.

[Please insert Figure 1 near here]

Swimmers performed the same standardized warm-up[21] on both Visits, which complies the recommendations for active warm-up prior to competitive swimming, ensuring swimmers' readiness to give their maximum effort.[22] The warm-up was as follows: i) 300 m (100 m usual breathing, 100 m breathing every five strokes, 100 m usual breathing); ii) 4 × 100 m (2 × [25 m flutter kick + 25 m increased stroke length]) on 1:50 min; iii) 8 × 50 m (2 × 50 m drill; 2 × 50 m building up swimming speed; and 4 × [25 m race pace + 25 m easy]) on 1:00; and, iv) 100 m easy. Then, 10 min after the warm-up, swimmers performed a 50 m front crawl all-out (time-trial with dive start).

On both Visits, the R-R signal (HRV) was monitored after the warm-up. HRV was recorded in resting during the 10 min before (pre-exercise) and immediately after the 50 m front crawl all-out test (post-exercise) using a Polar RS800CX heart monitor (Polar Electro Oy Inc., Kempele, Finland) at a sampling frequency of 1,000 Hz[23,24]. Importantly, swimmers were assessed in the same position on both Visits – i.e., seated on the floor next to the lane with the back supported and legs bent. Moreover, they were asked to remain relaxed, to breathe normally, and not speak or move during the HRV evaluation.[25]

The HRV data was processed using Kubios software (v.3.0.0 [free version], HRV analysis, University of Eastern Finland).[26] Briefly, the "best" 5 minutes period (out of 10 min) that presented a normal (i.e., Gaussian) R-R distribution and no large R-R interval outliers –that could be identified as signal artefacts– was manually selected by trained researchers (A.P.F. and J.M.A.A) from the whole HRV recording). For more details about these and other relevant methodological aspects considered for the HRV signal processing see [25–27]. Finally, the "medium" Kubios filter was applied to interpolate artefacts in the R-R signal as previously proposed.[26]

From the filtered R-R signal the mean of the time interval between two consecutive R waves (Mean R-R) was obtained. Then, time- and frequency-domain vagal-related HRV parameters were calculated. In the time-domain, the standard deviation of all normal R-R intervals (SDNN), the squared root of the mean of the sum of the squares of successive normal R-R interval differences (RMSSD), the percentage number of pairs of adjacent normal R-R intervals differing by more than 50 milliseconds in the entire recording (pNN50), were derived using the abovementioned Kubios software. In the frequency-domain, from the spectral analyses using the non-parametric Fast Fourier Transformation algorithm (FFT), the power in the high frequency (HF: 0.15–0.4 Hz) was derived. Of note, in resting short-term recordings RMSSD, SDNN, pNN50, and HF are interpreted as indicators of vagal/parasympathetic activity.[2,28]

Statistical analysis

The normality of all distributions was verified using Shapiro-Wilk. Variables that exhibited normal distribution were presented as mean and standard deviation (mean ± SD). HRV parameters did not exhibit normal distribution and were presented as median and interquartile range. For analytic purposes, those variables that did not exhibit a normal distribution were transformed by computing the natural logarithm (ln). A two-way repeated measures analysis of variance (ANOVA) was performed for each HRV parameter. Bonferroni post-hoc tests were used and effect size was expressed as partial eta squared (η_p^2). Paired sample t test was used to compare 50 m time between Visit 1 and Visit 2. The analysis of the effects of the detraining (i.e., the interaction analyses) was tested using analysis of covariance (ANCOVA), with vagal-related HRV parameters as dependent variables in separate models, Visit (Visit 1 *vs.* Visit 2) as a fixed factor, and the pre-exercise values as a covariate (i.e., controlling the impact of pre-exercise values over post-exercise values). Statistical procedures were conducted using SPSS v.24.0 (IBM, Chicago, IL, USA). Figures were created using the GraphPad Prism v.8 (GraphPad Software, San Diego, California, USA). The significance level was set at $p \leq 0.05$.

Results

Swimming performance was impaired after the off-season (Visit 1: 28.64 ± 1.86 s; Visit 2: 29.37 ± 2.30 s; mean difference: -0.73 s; 95% confidence interval: -1.06, -0.38; $p < 0.001$). Additional information regarding participants' cardiac characteristics are presented in **Supplementary Material 1**.

There was a Time (i.e., pre- and post-exercise) main effect in Mean R-R ($p < 0.001$; $\eta_p^2 = 0.54$), LnSDNN ($p = 0.001$; $\eta_p^2 = 0.47$), LnRMSSD ($p < 0.001$; $\eta_p^2 = 0.52$), LnpNN50 ($p = 0.002$; $\eta_p^2 = 0.39$), and LnHF ($p = 0.001$; $\eta_p^2 = 0.46$). Lower post-exercise values compared to pre-exercise values were observed in both Visits in all the HRV parameters (**Figure 2**).

There was a Visit (i.e., Visit 1 and Visit 2) main effect in Mean R-R ($p < 0.001$; $\eta_p^2 = 0.83$), LnSDNN ($p < 0.001$; $\eta_p^2 = 0.81$), LnRMSSD ($p = 0.004$; $\eta_p^2 = 0.36$), LnpNN50 ($p = 0.035$; $\eta_p^2 = 0.21$), and LnHF ($p < 0.001$; $\eta_p^2 = 0.78$). Pre- and post-exercise values were lower in Mean R-R (**Figure 2A)**, LnSDNN (**Figure 2B**), LnRMSSD (**Figure 2C**), and LnHF (**Figure 2E**) in Visit 2 compared to Visit 1. LnpNN50 showed a reduction in pre-exercise values after the training cessation, however, post-exercise values did not change significantly (**Figure 2D**).

[Please insert Figure 2 near here]

ANCOVA showed a higher reduction in Mean R-R (i.e., Time and Visit interaction) in response to the 50 m front crawl all-out in Visit 2 compared to Visit 1 ($p = 0.005$, $\eta_p^2 = 0.019$). No other significant interaction between Time and Visit was observed (LnSDNN: $p = 0.393$, $\eta_p^2 = 0.020$; LnRMSSD: $p = 0.413$, $\eta_p^2 = 0.018$; LnpNN50: $p = 0.425$, $\eta_p^2 = 0.017$; and LnHF: $p = 0.674$, $\eta_p^2 = 0.005$).

Discussion

Our study aimed to investigate the differences between changes in vagal-related HRV parameters induced by an all-out effort (50 m) after the summer (5-week period) compared to before the summer (i.e., the detraining effect on HRV response to exercise). The results showed a greater reduction in mean R-R following the 50 m time-trial test after the off-season. Moreover, results showed a Visit main effect, being pre- and post-exercise vagal-related HRV parameters reduced after the training cessation period (i.e., in Visit 2 compared to Visit 1), suggesting an impairment in swimmers' fitness. These results were indeed corroborated by the swimmers' performance impairment.

Together with the swimming performance impairment, the training cessation evoked greater Mean R-R reduction in Visit 2 compared to Visit 1, suggesting that the decrease experienced after the 50 m all-out was different between visits. The greater Mean R-R reduction is in line with the higher HR obtained in response to the 50 m after the training cessation,[19] considering that these variables are negatively correlated.[25] However, none of the vagal-related HRV parameters were influenced in response to the exercise by the detraining. The post-exercise parasympathetic response is mainly dependent on the exercise intensity.[29] Thus, the similar reduction in HRV parameters in Visit 1 and 2 could be, at least in part, explained by the fact that the intensity was maximal in both Visits. Furthermore, our results showed a Visit main effect, with pre- and post-exercise values being lower in Visit 2 compared to Visit 1 (**Figure 2**). Since the study conditions were similar in both visits (i.e., same warm-up, intensity, and specific demands of the test), this reduction in pre- and post-exercise vagal-related HRV parameters after the off-season suggests a deterioration of the swimmers' physical status, which might negatively affect their ability to recover.[11,30] The physical fitness is, indeed, another factor that also influences the cardiac parasympathetic response.[29] However, the deterioration in physical fitness observed after 5-week of training cessation might not influence the vagal-related HRV parameters producing a further reduction after the off-season.

In this regard, the impact of detraining on cardiac autonomic function has been reported in endurance runners.[15] The aforementioned study showed a reduction of LnRMSSD at rest, however, contrary to our results, the post-exercise (5 min running at 9 km/h) LnRMSSD did not exhibit a significant reduction after 4-week training cessation period.[15] The main difference between studies is the intensity of the test, which is the main factor inducing changes in HRV.[29] It is possible that 5 min running at 9 km/h was not intense enough to show the changes evoked by the detraining.[15] Furthermore, the duration of the HRV recording was different (3 *vs.* 5 min), which could directly influence the quantification of some of the HRV parameters.[25]

Contrary to what has been observed in endurance athletes, Merati et al.[13] observed a positive association between the vagal tone (NN50 and pNN50) and 50 m freestyle time (i.e., the lower the vagal tone, the better the performance in 50 m freestyle) in sprint athletes. According to this relationship, one would expect that the reduction in vagal-related parameters observed after the off-season were coupled with an enhancement in 50 m performance; however, 50 m front crawl performance deteriorated after the off-season. In this regard, the autonomic modulation is affected by the type of training conducted, with parasympathetic predominance during high-volume and low-intensity training and sympathetic predominance during phases of low-volume and high-intensity training.[9] Hence, considering the differences in training sessions between endurance and sprinters,[9,31,32] and the importance of rapidly depressing cardiac vagal outflow and increasing cardiac output in sprint events,[13] it could be expected that lower vagal tone might be related with better performance. However, in this case, the lower parasympathetic activity observed after the off-season was induced by the detraining. Changes in HRV parameters were likely as a consequence of plasma volume reduction, which, together with a lack of aerobic stimulation, has been associated with lower parasympathetic activity.[9] Moreover, the reduction of plasma volume induces a reduction in stroke volume.[14] To counterbalance the effect of reduced stroke volume on cardiac output, HR increases.[14,33] However, the rise of HR is not sufficient to counteract the reduction in stroke volume (i.e., it evokes a reduction in cardiac output),[14] which, in line with our results, negatively affects performance.[14]

On the other hand, our results showed a Time main effect, exhibiting a reduction in all the HRV parameters (**Figure 2**) after the exercise in both Visits (i.e., lower post- than pre-exercise values in both visits). This was expected as, due to the exercise, substantial cardiovascular adjustments must occur to meet the demand of working muscles. This was in line with previous studies showing an extremely large suppression of cardiac parasympathetic activity following exercise, especially after high-intensity exercises. The HRV restoration to pre-exercise values after exercise differs between athletes with different training statuses (e.g., highly trained *vs.* inactive),[29] however, it is unknown whether a short training cessation period might evoke a delay in the HRV restoration to pre-exercise values in trained and highly-trained swimmers. Thus, future studies should analyze if the detraining might impact the return of HRV to pre-exercise values.

It is important to consider the position in which HRV was measured when comparing our results with future studies. Since the posture in which the assessments are conducted affects the parasympathetic activity,[25,34] different results might be obtained when measuring upright or supine. Finally, certain limitations should be acknowledged: 1) basal HRV was not assessed (i.e., before warm-up) due to equipment and time availability. However, the HRV assessment was performed after the warm-up to isolate the effect of the 50 m, otherwise, we would not know if the final result would be influenced by the test or the warm-up; 2) The exact time of awakening was not recorded. However, swimmers kept their habits during the evaluation sessions, and the tests were performed in the morning during their training schedule. Hence, we assume they should have woken up at the same time every day.

Practical applications

The recovery process, even after a single bout of exercise, involves the integration of several systems and physiological functions. Changes in the autonomic nervous system, indirectly assessed through HRV parameters, may reflect exercise-induced changes in physical status. The reduction in pre- and post-exercise vagal-related HRV parameters observed in our study could suggest that, even a single bout of exercise, an exercise cessation period may have a negative impact during the training phase, as swimmers would need more time to recover within (and presumably between) swimming session. Hence, caution should be taken with training loads at the start of the season, since the worse physical status could negatively affect the training adaptations. HRV is thus a non-invasive, inexpensive, and easy-to-use tool that coaches could use to monitor swimmers' condition and get the most out of them.

Conclusion

In conclusion, after only 5-week of training cessation, swimmers evidenced a reduction in pre- and post-exercise vagal-related HRV parameters. These reductions for the same all-out exercise suggests an impairment in swimmers' physical status.

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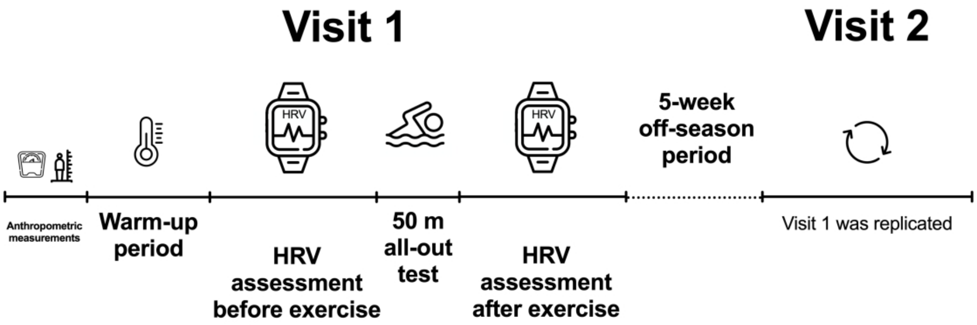
Figures captions

Figure 1. Study design and evaluations carried out. Heart rate variability (HRV) was evaluated after the warm-up and after the 50 m front crawl all-out test.

Figure 2. Changes in heart rate variability (HRV) parameters after the 5-week training cessation. Panel A correspond to Mean R-R; Panel B correspond to LnSDNN; Panel C correspond to LnRMSSD; Panel D correspond to LnpNN50; Panel E correspond to LnHF. Abbreviations: Ln, natural logarithm; SDNN, standard deviation of R-R intervals; RMSSD, square root of the mean squared differences between successive R-R intervals; pNN50, the percentage number of pairs of adjacent normal R-R intervals differing by more than 50 milliseconds (ms) in the entire recording; HF, power in the high frequency (0.15–0.4 Hertz [Hz]); PRE_V1: before off-season pre-exercise values; PRE_V2: after off-season pre-exercise values; POST_V1: before off-season post-exercise values; POST_V2 after off-season post-exercise values; #Time main effect; *Visit main effect (Bonferroni post-hoc test).

Supplementary Material 1. Cardiac characteristics of the swimmers' before and after the 5-week off-season (i.e., Visits 1 and 2, respectively).



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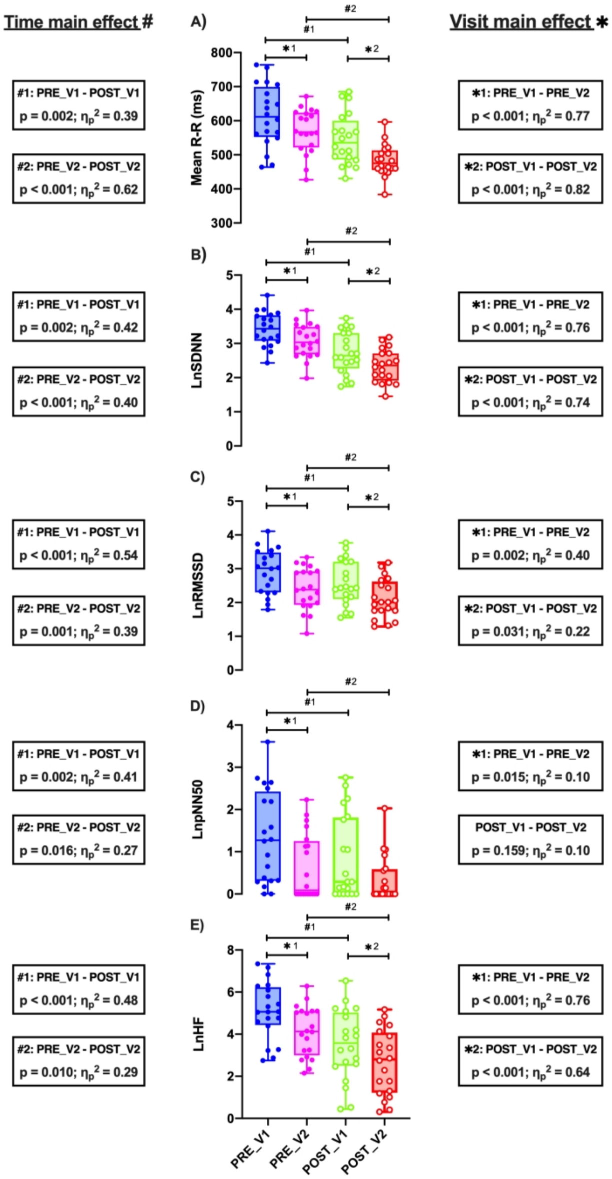
300x101mm (300 x 300 DPI)



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Supplementary Material 1. Cardiac characteristics of the swimmers' before and after the 5-week off-season (i.e., Visits 1 and 2, respectively).

Variable	Visit 1		Visit 2	
	Pre-exercise	Post-exercise	Pre-exercise	Post-exercise
Mean HR (bpm)	99 ± 15	111 ± 15	106 ± 13	125 ± 12
Mean R-R (ms)	615.11 ± 89.90	545.54 ± 74.71	570.12 ± 64.60	483.30 ± 46.31
SDNN (ms)	30.87 [24.07]	14.07 [17.35]	20.65 [18.11]	10.55 [8.16]
RMSSD (ms)	20.39 [22.53]	11.35 [16.52]	10.81 [11.66]	7.48 [8.01]
pNN50 (%)	3.55 [9.99]	1.33 [5.14]	1.10 [2.50]	1.00 [0.79]
FFT HF (ms^2)	157.94 [427.90]	36.85 [140.62]	62.13 [143.74]	16.53 [55.52]

Data presented as mean ± standard deviation. Median [IQR: interquartil range] are presented for heart rate variability parameters as these variables presented skewed distributions. HR: heart rate; R-R: interval between two consecutive R waves; NN: Normal RR intervals SDNN: the mean standard deviation of all normal RR intervals; RMSSD: the square root of the mean of the sum of the squares of differences between adjacent NN intervals; pNN50: percentage (%); FFT: Fast Fourier Transformation; HF: high frequency band (0.15 – 0.4 Hz) index of parasympathetic activity.