

**ENERGY DRINK BEFORE EXERCISE DID NOT AFFECT AUTONOMIC
RECOVERY FOLLOWING MODERATE AEROBIC EXERCISE: A CROSSOVER,
RANDOMIZED AND CONTROLLED TRIAL**

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ABSTRACT

Introduction: Energy drink (ED) intake could initiate physiological changes owing to its stimulant characteristics and, it improves endurance and athletic performance. We evaluated the acute effects of ED on autonomic heart rate (HR) control during recovery after a session of submaximal aerobic exercise. **Method:** The study was completed by submitting 29 healthy males between 18 and 30 years old to three conventions: (A) Maximum exercise test by the adapted Bruce protocol; (B) Placebo protocol (PP) - water intake 15 minutes prior to exercise, rest in dorsal decubitus for 15 minutes followed by 5 minutes of treadmill running at 1% inclination, initial speed of 5 km/h for 5 minutes 25 minutes with 60% of the velocity consistent to the maximum oxygen consumption (VO₂max), and finally 60 minutes of recovery at rest in the supine position; (C) Experimental protocol (PE) - similar to PP previously, but with ED intake 15 minutes before physical exercise. The time, frequency and geometric indexes of HR variability (HRV) were inspected before and after exercise. **Results:** There was a significant ($p < 0.05$, $< 5\%$) effect on the HRV index (HR-nu and ms^2 , LF-nu and ms^2 , LF/HF, SD1, SDNN and RMSSD), indicating a reduction in HRV in the first 5 minutes after exercise in both protocols (PP and PE). Yet, no protocol interaction was detected, suggesting no effect of ED on HRV throughout recovery after submaximal aerobic exercise. **Conclusion:** There was no significant effect of ED on the autonomic control of HR in the recovery phase after submaximal aerobic exercise.

Keywords: Energy drink; Autonomic Nervous System; Aerobic exercise; Recovery; Nonlinear dynamics.

INTRODUCTION

Energetic drinks (ED) are characterized by a non-alcoholic component constituted by ingredients that raise the physical and psychological temperament [1]. They are usually formulated of caffeine, 2-aminoethanesulfonic acid and citric acid. ED are available in commercial outlets and consumed typically by adolescents. It is alleged that the use of caffeine may cause advantageous physiological changes in exercise performance [2-4].

Alternatively, 2-aminoethanesulfonic acid (taurine) is recognized as an antiarrhythmic agent due to its involvement in the regulation of cation transport and it could circumvent several types of arrhythmias [5, 6]. So, it is vital to realize that persistent and isolated use of 2-aminoethanesulfonic acid as stated by the scientific literature diminishes the incidence of ventricular fibrillation, specifically in studies on rats [7]. ED are primarily consumed in the anticipation of the stimulating effect of caffeine, which is then able to mobilize energy, improve endurance and physical performance [8].

Administration of 2.5mg/kg ED prior to exercise activated positive responses in exercise performance without inducing any collateral cardiological effects [9]. Wiklund *et al.* [10] studied the interaction of ED and the autonomic nervous system (ANS) during exercise in healthy young subjects (five men and five women aged between 19 and 30 years old) who sustained maximal effort on an ergometric bicycle for 30 minutes after the ingestion of 0.75mg ED. Concluded by electrocardiographic analysis, there were no cardiac arrhythmias and the recovery of the ANS following exercise was slower when the participants ingested ED [10].

In this way, it is consistent to assume that the behavior of the ANS should be suitably evaluated by heart rate (HR) variability (HRV). For this purpose, HRV has been studied by several researchers as a simple, reliable and non-invasive technique characterized by the

fluctuations of the intervals between consecutive heart beats (RR intervals), effectively inferring the behavior of the ANS aside several physiological or pathological conditions [11, 12]. It is vital to emphasize that the ANS is fundamental for the maintenance of homeostasis and responsible for the varied actions and reactions in the different organs and physiological systems, together at rest and for the duration of exercise [11]. Previous studies have supported the relevance of HRV to deliver autonomic dysfunction and related syndromes [13-17].

Based on the ANS status controlling the human metabolism, physical exercise and ED consumption; this study aimed to assess the influence of ED ingestion on autonomic HR modulation during recovery from aerobic exercise.

METHODS

CONSORT Statement

This study followed the CONSORT (Consolidated Standards of Reporting Trials) statement. Our investigation provides details of the study population and settings; subject selection (inclusion or exclusion criteria); methods of randomization; efficacy and safety procedures. The study design and statistical procedures have been described. We included particulars concerning trial design, participants, interventions, outcomes, sample size, randomization and statistical methods.

Trial Design

This was a crossover, randomized and controlled trial performed at the Department of Medicine, Federal University of Sao Paulo, Sao Paulo, SP, Brazil. The project was registered in the ClinicalTrials.gov (Protocol number NCT02917889) and is accessible online (<https://clinicaltrials.gov/ct2/show/NCT02917889>).

Participants

The project was permitted by the Research Ethics Committee of the Federal University of São Paulo (Resolution 466/12 of the National Health Council of 10/10/1996, Protocol number: CEP-2200/11). A total of 35 subjects, aged between 18 and 30 years, who were healthy and physically active consistent with International Physical Activity Questionnaire (IPAQ), were selected at a university in the city of Presidente Prudente -SP, Brazil [19].

The timetable of events were standardized between 17.30 and 21.30, with the intention of reducing the possible influences of the circadian variation on the constraints evaluated. The temperature was maintained between 23°C and 24 °C and humidity between 60% and 70%.

The study excluded individuals that presented the following characteristics: patients with cardiovascular, orthopedic, respiratory, neurological, smoking, alcohol, obese or other pathological conditions; subjects who took pharmacotherapies that influenced cardiac activity or were sedentary consistent with the IPAQ [19], those that had at no time consumed an energy drink or those who declined to sign free and informed consent [20].

Initial Assessment

The subjects responded to an anamnesis to identify behaviors, physical conditions and pathologies that could influence them in the study. IPAQ before the protocols were taken, the sample was assessed by body weight via a digital scale (Plenna, TIN 00139 MAXIMA, Brazil), height was measured via a stadiometer (ES 2020 - Sanny, Brazil) and then, the Body Mass Index (BMI) was calculated, Next, we started the physical assessment phase and the determination of Vo2max.

Interventions

The process involved three randomized protocols performed in three different sessions, with a minimum interval of 48 hours between them, in order to allow the participants an adequate recovery time:

1) Determination of oxygen consumption (VO_{2max}) via the maximum effort test - the trial was completed on treadmill (TPEE) by means of the Bruce protocol for the following; use of 60% of the speed matching the VO_{2max} for the load applied [21, 22], and evaluation with and without ED consumption. The expired gases analysis was finished via the Quark PFT commercial system (Comend, Rome, Italy), which attained the VO_{2peak} recognized to be the highest VO_{2max} reached throughout the test.

2) Placebo protocol (PP): at this stage the subjects consumed 200 ml of water, then continued at rest for 15 minutes in the supine position, followed by 30 minutes of aerobic exercise on treadmill, for 5 minutes at a velocity of 5.0 km/h and 25 minutes with 60% of the velocity corresponding to the VO_{2max} + 1% inclination, and lastly, 60 minutes of recovery in the supine position.

3) Experimental protocol (PE): in this stage the subjects accomplished the same procedure as the PP, but with different ED intake (45 calories, 11.2 g carbohydrate, 80 mg sodium, 32 mg caffeine, Taurine 400 mg, Niacin 4.6 mg, Pantothenic Acid 2 mg, Vitamin B6 0.5 mg, Vitamin B12 0.4 mg, Glucuronolactone 240 mg, inositol 20 mg) 15 minutes prior to onset [9, 23).

All protocols were finalized on the same treadmill (TPEE; Inbrasport ATL 2000, Brazil) and conducted by the same researcher.

Measures were taken to safeguard hydration. This was to circumvent problems, as dehydration could affect the results. For instance, reduced endurance performance, decreased psychological and cognitive performance through exercise [24, 25], increases in body

temperature, HR and systolic volume reduction [26], and rarely sudden death because of functional modifications [27),

Hydration

The American College of Sports Medicine (ACSM) [24] published in 2007 sanctions on fluid replacement that indicate intervention before, during and after physical exercise, with the purpose of keeping the subject as hydrated as possible. It is suggested that the individuals consume in the region of 500 ml of fluids in the two hours previous to the exercise routines, to promote acceptable levels of hydration and enough time for excretion of excess ingested water. Consequently, the subjects were instructed to consume 500 ml of water 2 hours before the protocol initiation.

The test was completed on a treadmill (TPEE; Inbrasport ATL 2000, Brazil) via the Bruce protocol. Verbal encouragement was required to acquire a physical effort close to the maximum. The test was terminated by the subjects' exhaustion or clinical and/or electrocardiographic changes that prevented the continuity of the test. During the test implementation, the HR monitor Polar RS800CX (Polar Electro, Finland) and the independent perception of effort (PSE) via the Borg Scale for perceived pain and exertion were monitored in the final phases of each stage. For the exercise test, subjects should reach 90% of the estimated maximum heart rate ($220 - \text{age}$) [28].

At the 5th, 10th and 15th minutes of rest, HR, systolic blood pressure (SBP), diastolic blood pressure (DBP) were logged. After these measurements the subjects completed physical exercise on a treadmill with an intensity of 5 km/hour + 1% inclination in the initial 5 minutes for warming-up, followed by 25 minutes with an intensity equal to 60% of the velocity consistent to the VO_2max with a matching slope. Throughout this phase, in the 15th and 30th minutes new HR measurements were taken.

HRV indices were completed at the following moments, Rest 10-15 minutes pre-exercise, (then, recovery post-treadmill exercise) Rec1 0-5 minutes, Rec2 5-10 minutes, Rec3 15-20 minutes, Rec4 25 -30 minutes, Rec5 35-40 minutes, Rec6 45-50 minutes, Rec7 55-60 minutes.

HRV analysis

After an explanation of the techniques required for data collection, a sling strap was located on the distal third of the sternum with the Polar RS800CX heart rate receiver, a valid device for heart rate analysis, beats and application of this data to study HRV [29].

During HRV analysis, the PE groups' pattern of behavior was logged beat-to-beat. Equally, for the PP group. We selected 256 consecutive stable RR intervals and completed digital and manual filtration to eliminate premature ectopic beats and artifacts, and only series with more than 95% of sinus beats were included in the study [30].

For a description of HRV, we recommend consultation of the following publications [31-33].

For the analysis of HRV; linear methods studied in the time domain (SDNN and RMSSD) and frequency (LF, HF and LF/HF in normalized units-nu and ms^2) and geometric methods (triangular index and TINN) [11, 34,35].

Kubios[®] HRV analysis software package (Kubios[®] HRV version 2.0, University of Kuopio, Finland) was enforced to calculate these indexes.

Randomization

The subjects and the researchers were not informed about the order of the procedures. An investigator who did not participate in the study completed the random allocation sequence, enrolled the participants and assigned participants to their appropriate interventions.

Sample Size Calculation

For sample size definition, the sample calculation was made based on the investigation bearing in mind the RMSSD index of the PP as a variable [18]. The extent of significant differences assumed was 11ms, in consideration of a standard deviation of 16.2 ms, with alpha risk of 5% and beta of 80%. The sample size resulted in 28 subjects in both protocols.

Data Analysis

Normality of quantitative data was determined via the Shapiro-Wilk test. Based on these results, the Student's t-test for unpaired data (normal distribution data) or the Mann-Whitney test (non-normal data distribution) was required to compare the variables between the experimental and placebo protocols at the same phase of analysis. The contrast between the moments of the same protocol was attained via repeated measurements analysis of variance (ANOVA) followed by the Bonferroni test for parametric data or, the Friedman test followed by the Dunn test for non-parametric data. Differences in all statistical tests were considered significant at the level $p < 0.05$ (or, $< 5\%$). Assessments were made using Minitab Software (Minitab version 13.20, PA., USA).

RESULTS

The anthropometric features of the 29 volunteers as well as the responses obtained in the maximal effort test are defined in Table 1.

HF absolute units index: In PP it was detected that HF significantly reduced in the recovery phases in Rec 1 ($p = 0.01$) compared to rest before exercise (Table 2). In PE a reduction of this index was observed in Rec1 ($p = 0.001$) and Rec2 ($p = 0.05$) in relation to rest (Table 3).

HF normalized units: In PP it was noticed that HF significantly reduced in the recovery phases in Rec 1 compared to rest before exercise ($p = 0.05$) (Table 2).

LF/HF index: In both groups, there was a significant decrease in recovery phases in Rec 1 compared to rest before exercise (Table 2 & 3). In the PE, there was a more distinct reduction of this index at rest ($p = 0.001$).

LF index absolute units: In PP, there was a significant reduction in recovery phases in Rec 1 compared to rest before exercise ($p = 0.01$) (Table 2). In the PE, a significantly greater reduction of this index was detected in Rec1 regarding rest ($p = 0.001$) (Table 3).

LF index in normalized units: In both groups, no significant statistical deviations were observed in the investigated index ($p > 0.05$) (Table 2 & 3).

SD1 index: In PP, there was a significant reduction in recovery phases in Rec 1 ($p = 0.001$) and Rec2 ($p = 0.01$) compared to rest before exercise (Table 2). In the PE, a significant reduction of this index was detected in Rec1 ($p = 0.001$) and Rec2 ($p = 0.01$) in relation to rest (Table 3). Yet, there was no difference between the groups ($p > 0.05$).

SD2 index: In both groups, no significant statistical changes were detected for the index evaluated ($p > 0.05$) (Table 2 & 3).

RMSSD index: In PP, there was a significant reduction in recovery phases in Rec 1 ($p = 0.001$) and Rec 2 ($p = 0.05$) compared to rest before exercise (Table 2). In the PE, a significant lessening of this index was observed in Rec1 ($p = 0.001$) and Rec2 ($p = 0.05$) in relation to rest (Table 3). Yet, there was no difference between the groups ($p > 0.05$).

SDNN index: In PP, there was a significant decrease in recovery phases in Rec 1 compared to rest before exercise ($p = 0.05$) (Table 2). In the PE, no significant reduction of this index was detected ($p > 0.05$) (Table 3).

DISCUSSION

Our study was commenced to better comprehend the possible effect of ED on HR autonomic control during recovery from exercise. The main results indicate that ED consumed before aerobic submaximal effort did not affect HRV indices during recovery from exercise.

Considering the clinical relevance of studying the physiological effects of ED on the organism, the research literature has considered the effects of ED on the ANS during exercise [9, 36]. Yet, the only study that estimated the heart rate dynamics during the recovery phase after exercise following ED consumption and absorption was unclear.

In a scientific literature review, amid the studies that tested the impact of ED on recovery after exercise, this study is the first to offer inclusion criteria, familiarity with ED, accompanied by IPAQ and VO_2 max by direct measures. So, we attempted to sanction the reliability of the protocols and to encounter the initial proposal by including only males to eliminate effects of sex hormones on HRV, thus distorting the statistical outcomes.

In this study design, the principal result is that ED did not significantly influence the recovery of HRV following a session of exercise. The time and spectral HRV analysis presented a decrease of HRV in the recovery phase after exercise without interaction between the protocols, indicating a lack of significant effect of the ED.

The absence of changes between control and ED protocols throughout this study can be explained by the exercise load enforced during the protocols. Also, physical exercise with intensity greater than 30% can promote dominance of the sympathetic nervous system to prepare the body for exercise. As a result, it would, in this situation, increase the release of catecholamines [37]. In this way, it is understood that ED did not stimulate further release of catecholamines to slow HR autonomic recovery.

In a previous study with similar aims, Nelson *et al.* [38] applied maximum exercise test on a stationary bicycle and evaluated cardiorespiratory capacity directly through VO₂ max in 15 subjects (7 males and 8 females). The study was split into 3 phases on different days: in the first, the load was determined, on the second and third days the subjects submitted to exercise on the same exercise bicycle for 15 minutes at 30% of the value reached in the maximum test followed by 15 minutes in 60% of the maximum value, increasing the loading according to ventilatory threshold to 80%. Next, it was increased to 100% until fatigue. The researchers' emphasized the increase in HR because of ED but there was no significant change in HRV in response to ED.

Our results partly support the above-mentioned results probably due to primordial differences. The inclusion of females and males in the same protocol and type of exercise are factors that would influence the muscular load. The ED taken was of a mixed composition for the current study. In addition, it was not reported if the partakers were habitual ED drinkers, conditions that could lessen the acute effects of ED [38].

Another protocol [9] studied 15 female students with a mean age of 21 years split into three groups (placebo, energy drink 1.25 mg/kg and energy drink 2.5 mg/kg). The cardiorespiratory capacity was measured via the Bruce protocol and the exercise was completed on a treadmill starting with 5 minutes of warming-up, then progressively reaching 80% of the VO₂max. Timing was scheduled and exercise was terminated when the subject reached voluntary fatigue. The researchers did not observe changes in the HRV indexes. Yet, it was detected that consumption of 2.5 mg/kg ED improved the performance of the exercises.

In the study directed by An *et.al.* [9], the ED consumption routine was unreported. Similarly, the key difference was the exercise timing with 6 minutes at high intensity.

The relevance of ED use in daily life is reinforced specifically for high performance athletes. There is evidence that ED consumption promotes increased blood pressure and potentially increases risks for developing heart disease [36], including cardiac arrhythmias and sudden death [39, 40]. So, it is vital to study the undesirable effects, specifically when linked to the practice of physical exercise.

This study promotes a concept that requires highlighting. The study's population periodically consumed ED, ANS stimulating beverages for instance tea, coffee, hot chocolate and caffeinated soft drinks daily. The participants' physical fitness levels could have prejudiced the results, as they offered high VO_2 max values ($\sim 47,66$ mL/kg/.min) [41]. Therefore, we cannot extrapolate our results to subjects under different situations. We did not evaluate non-linear HRV analysis. For example, entropy, symbolic analysis and recurrence analysis may provide different discriminative power related to time and frequency domain. We encourage further studies in this perspective.

As this is a new study, we stress the necessity to develop further studies in an attempt to examine ED in distinct populations, with different ages and genders. Thus, we encourage forthcoming research to explore the outcomes on persons with cardiovascular or metabolic disorders, to discover preventative strategies to reduce the risk of health problems.

CONCLUSION

ED absorption before aerobic exercise did not significantly influence the recovery of heart rhythm autonomic control after undertaking moderate-intensity aerobic exercise.

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TABLES LEGEND

Table 1. Mean values, and standard deviations, minimum and maximum values of the anthropometric variables and the maximum stress test.

Table 2 – Illustrates average and standard deviation of the indexes before aerobic exercise and in the diverse phases of recovery following exercise in PP.

Table 3 – Illustrates average and standard deviation of the indexes before aerobic exercise and in the diverse phases of recovery following exercise in PE.

Table 1. Mean values, and standard deviations, minimum and maximum values of the anthropometric variables and the maximum stress test.

Variables	Mean \pm DP	Minimum-Maximum
Age (years)	24,44 \pm 2,84	[19 – 29]
Height(m)	1,79 \pm 7,96	[1,69 – 1,94]
Weight(kg)	84,52 \pm 13,70	[58 – 113]
BMI (kg/m²)	26,01 \pm 3,43	[23,28 – 36,70]
VO2max(ml/kg/min)	47,66,00 \pm 1,79	[98,62 – 60,98]
HR peak (bpm)	189,37 \pm 10,24	[175 – 214]
60% HR peak (bpm)	113,89 \pm 6,13	[102 –125]

BMI = body mass index; kg = kg; m = meter; VO2 max = maximum oxygen consumption; ml = milliliters; min = minute; HR = heart rate; bpm = beats per minute.

Table 2 – Illustrates average and standard deviation of the indexes before aerobic exercise and in the diverse phases of recovery following exercise in PP.

Variables	Rest (10-15min)	Rec (0-5min)	Rec (5-10min)	Rec (15-20min)	Rec (25-30min)	Rec (35-40min)	Rec (45-50min)	Rec (55-60min)
SDDN	70.11 ± 6.57	101.69 ± .37*	62.05 ± 21.80	68.67 ± 28.76	79.69 ± 46.03	75.97 ± 27.39	79.45 ± 30.27	84.33 ± 31.11
RMSSD	51.3±33.19	34.8 ±14.67*	46.9± 22.23*	47.3± 25.37	52.0± 29.41	51± 29.80	48.4±26.66	50.5± 27.11
LF(ms²)	1014± 1116.16	1357 ± 990.34	996±1043.43	1331± 1248.64	1141± 1978.72	1759± 1258.42	1690±1537.05	2068±1243.86
HF (ms²)	841± 1455.85	505±443.25*	618±796.5648*	594± 1002.29	873± 1375.09	904± 1203.23	844± 901.25	766± 1073.06
LF(nu)	54.6± 19.36	73.8± 12.97*	67.2± 19.81	62.5± 17.25	61.4± 21.44	65.2±17.07	68.1± 15.37	66.2± 16.68
HF(nu)	43.0 ± 19.88	26.0 ± 12.54*	38.3 ± 19.46	31.7 ± 17.35	36.3 ± 20.94	34.7 ± 17.03	31.8 ± 15.29	33.7 ± 19.20
LF/HF	1.323± 1.21	2.662± 2.79*	2.050± 1.79	2.151± 1.19	1.755± 1.80	1.879± 1.27	2.141± 1.22	1.964± 2.27
SD1	38.5± 23.70	25.8± 12.16*	33.5± 15.41*	33.5± 19.67	37.2± 21.50	38.4± 21.35	37.7± 19.46	40.8±20.63
SD2	86.8±34.39	129.4±59.46	74.2±29.84	82.1±38.73	81.9±58.46	92.7±37.28	96.7±43.42	99.8±39.37

*Value with statistical difference in relation to rest (ANOVA for repeated measurements followed by the Bonferroni test, $p < 0.001$). Rec: Recovery post exercise; Rest: 10-15 minutes pre-exercise; Rec1: 0-5 minutes; Rec2: 5-10 minutes; Rec3: 5-20 minutes; Rec4: 25-30 minutes; Rec5: 35-40 minutes; Rec6: 45-50 minutes; Rec7: 55-60 minutes; nu: standard unit; ms²: absolute unity; SDDN: standard deviation of all normal RR intervals; RMSSD: square root of the mean square of differences between adjacent normal RR intervals; LF: low frequency; HF: high frequency; LF/HF: ratio low frequency/high frequency; SDI: standard deviation of the instantaneous variability of the beat-to-beat heart rate; SD2: standard deviation of long-term continuous RR interval variability.

Table 3 – Illustrates average and standard deviation of the indexes before aerobic exercise and in the diverse phases of recovery following exercise in PE.

Variables	Rest (10-15min)	Rec (0-5min)	Rec (5-10min)	Rec (15-20min)	Rec (25-30min)	Rec (35-40min)	Rec (45-50min)	Rec (55-60min)
SDDN	70.65±27.05	63.17± 28.97	59.73± 25.10	64.19±29.24	77.43±39.73	87.99±46.59	89.85±36.89	86.25±36.47
RMSSD	58.88±33.17	20.51± 10.09*	6.58±30.70*	52.92±33.33	59.25±35.83	58.68±35.32	58.5±33.02	58.56±35.67
LF(ms²)	1404.85± 1190.67	1308.33± 1284.21	1180.92± 1361.60	1383.96± 1478.45	1414.77± 1592.54	2052.96± 2035.45	2336± 2120.74	2217.07± 2099.54
HF (ms²)	1385.37± 1499.36	281.77± 392.02*	963.59± 1565.14	1175.33±1 414.32	1444.59± 1612.45	1289.70± 1329.11	1296.33± 1489.34	1452.25± 1870.13
LF(nu)	55.09±20.00	81.05±12.83*	60.41±20.63	59.34±16.74	55.35±18.68	63.03±16.15	65.89±15.49	65.59±15.82
HF(nu)	44.78±20.02	18.88±12.83*	39.38±20.52	40.50±16.78	44.54± 18.65	36.86± 16.15	34.02±15.45	34.34±15.80
LF/HF	1.79±1.51	6.11±4.45*	2.61±2.59	1.97±1.41	1.76±1.46	2.22±1.42	2.59±1.65	2.79±2.38
SD1	44.24±23.91	19.07±17.04*	35.04±23.80*	40.49±26.27	44.66±26.66	43.56±25.10	44.40±25.46	44.34±25.17
SD2	89.10±33.23	89.23±39.63	76.85±30.90	81.87±36.58	99.68±52.54	115.58±63.71	117.98±49.84	114.08±47.35

*Value with statistical difference in relation to rest (ANOVA for repeated measurements followed by the Bonferroni test, $p < 0.001$). Rec: Recovery post exercise; Rest: 10-15 minutes pre-exercise; Rec1: 0-5 minutes; Rec2: 5-10 minutes; Rec3: 5-20 minutes; Rec4: 25-30 minutes; Rec5: 35-40 minutes; Rec6: 45-50 minutes; Rec7: 55-60 minutes; Nu: standard unit; ms²: absolute unity; SDNN: standard deviation of all normal RR intervals; RMSSD: square root of the mean square of differences between adjacent normal RR intervals; LF: low frequency; HF: high frequency; LF/HF: ratio low frequency/high frequency; SDI: standard deviation of the instantaneous variability of the beat-to-beat heart rate; SD2: standard deviation of long-term continuous RR interval variability.