Acta Medica Mediterranea, 2016, 32: 1683

THE CHANGES ON THE HRV AFTER A WINGATE ANAEROBIC TEST IN DIFFERENT SIMULATED ALTITUDES IN HEALTHY, PHYSICALLY-ACTIVE ADULTS

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ABSTRACT

Introduction: *The purpose of the present study was to examine the effects of single Wingate anaerobic test on heart rate variability (HRV) parameters at 162 m, 1015 m, 2146 m, and 3085 m of altitudes, and to understand how normobaric hypoxia affect the autonomic nervous system.*

Material and methods: *A total number of 21 (10 male, 11 female) physically active adult college students participated in this study voluntarily. After the informed consent forms were filled out by all participants, and their body composition was measured in the first visit, subjects visited the laboratory four more times with the 2 days of intervals. In every visit a participant's HRV measurement was collected after the hypoxia mask was placed on his/her face, and participant was taken to Wingate Anaerobic Test with the mask. Immediately after the Wingate Anaerobic Test, the HRV was collected again. This procedure was applied at 162 m, 1015 m, 2146 m, and 3085 m of altitude randomly.*

Results: *Despite the fact that all HRV values were significantly different in the comparisons of pre- and post-test results, no change was found in any of SDNN, SDSD, RMSSD, TP, HF, LF/HF, HF, HFnu, LF, LFnu, and VLF parameters depending on the different altitudes. We also found no change on anaerobic power levels.*

Conclusion: *In accordance with the present findings of the study it could be concluded that to exposure to normobaric hypoxia at 162 m, 1015 m, 2146 m, and 3085 m had no additional effect on both autonomic nervous system determined by using HRV measurement and anaerobic performance.*

Keywords: *Heart rate variability, altitude, Wingate test, physically active.*

DOI: 10.19193/0393-6384_2016_5_150

Received *April 30, 2016;* ***Accepted*** *July 02, 2016*

Introduction

Exercises at altitude make more rapid and greater physiological changes than those at sea level. This is because altitude hypoxia leads to exercise-induced physiological changes such as total blood volume, hemoglobin, number of red blood cell, mitochondrial concentration and muscle enzyme activity changes(1). Sincethe Mexico City Olympic Games were heldin 1968, the high altitude training (HAT) methods have had a substantial place in the area of training sciences to enhance the performance of athletes(2). Researchers tried live high-train highprocedure to gain the positive consequences of altitude training extensively(2, 3, 4, 5).

Nevertheless, due to the fact that to bring athletes to high altitude, and keep them there at least a couple of weeks(6) take a long time and cost too much, new approaches have been found. To provide live high-train low/high or live low-train high (also known as intermittent hypoxic training) environments at the sea level, some special altitude chambers and/or devices in laboratories have been utilized(7, 8, 9).

To date, the big majority of high altitude studies examined the performance effects of different training methods at different altitudes, and numerous studies reported beneficial effects of these procedures(10, 11, 12), whereas some others did not(7, 13).

The alterations and adaptations observed as a result of high altitude training are mostly considered to be depended to autonomic nervous system(14, 15, 16). Heart rate variability (HRV) is a non-invasive method to evaluate the autonomous heart functions(17),and has been used in the field of sports sciences to investigate chronic effects of exercise and acute effects of physical activity(18) by examining parasympathetic and sympathetic influences on heart. The successful adaptation of ANS to HAT has a critical importance not only for the expectation of increased performance of an athlete but also the health of an athlete. Therefore, researchers have also focused the relationship between HRV and HAT in recent years. Menz et al. (2016) found decreased HR results at submaximal load after a training program in hypoxia (at 4500 m) and demonstrated a decrease on sympathetic activity(19).

The effects of acute hypoxia on HRV were investigated by Zhang et al. (2014). Researchers found significant changes on some frequency-domain HRV parameters which indicated a decrease on sympathetic activity at hypoxia (3000 m, and 4000 m) by comparison with sea level(20). Taralov et al. (2015) reported that the sympathetic activity was maintained during hypoxia, and the decreased vagal activity increased right after hypoxic exposure in healthy men(16). Another studies also observed increased sympathetic activity after exposure of acute hypoxia(15, 21-24).

Nevertheless, none of the research mentioned above has sought the HRV responses after a single, maximum anaerobic work load at different altitudes. In the present study, therefore, we aimed to investigate the acute effects of a Wingate anaerobic test on heart rate variability parameters at 162 m, 1015 m, 2146 m, and 3085 m in healthy, physically-active adults. These measurements would provide us to compare the mean differences of HRV values obtained at different altitudes, and understand the mechanism of HRV after a single maximum work load in normobaric hypoxia.

Methods

Participants

A total number of 21 (10 male, 11 female) physically active adult college students participatedin this study voluntarily. The participants composed of healthy students who used to exercise minimum 3 days a week, one hour per session for at least 6 months prior to the studyregularly. The mean age of participants was 19.81 ± 1.25 year, body weight 66.27 ± 13.12 kg, body height 171.67 ± 8.64 cm, and percent body fat 20.69 ± 4.93 %.

Procedures

First,the informed consent forms were filled out by all participants, and their body composition was measured in this first visit. All participants visited the laboratory 4 more times with the 2 days of intervals. In every visit a participantwas lain down, then the hypoxia mask was placed on his or her face, and the HRV measurement was collected. After HRV measurement was finished, participant was stood up and taken to Wingate Anaerobic Test (WAnT) ergometer with the mask on his/her face, and the test was started with standardized warm up and resting procedure. Immediately after the WAnT, the participant was lain down, and the HRV measurementwas performed again. The altitude of the hypoxia (162 m, 1015 m, 2146 m, and 3085 m) was determined for each visit randomly, and participants were blinded to the altitude condition. The participants were told not to drink alcohol, and do exercise for at least 24 hours prior to the each testing day, and also advised to maintain their normal drinking and eating habits. Throughout all these measurements the humidity was set under 60 %, and air temperature was kept between 20-22 C° in the laboratory.

Body composition

Body weightand percent body fat were measured with Avis 333plus (Korea) analyzer and Holtain branded stadiotmeter with 1-mm distance was used to measure body heights (Holtain, U.K.).

HRV analysis in normobaric Hypoxia

A portable hypoxic generator (Everest Summit II, Hypoxia, NY, USA) was used to provide the normobaric hypoxic ambient. The mask of the generator was placed on the participant’s face for five minutes in the supine position. The altitudes used in the present study were 162 m, 1015 m, 2146 m, and 3085 m. After finishing the resting period, the HRV measurement of four different altitude implementations wascollectedby using a device with model OmegaWave 800 (OW, Oregon, USA).

This measurement took approximately 5 minutes and allowed to have some time- and frequency-domain HRV parameters. The measurements were obtained in position of supine on the litter while the participants were wearing only shorts.Three of the 7 electrodes which were used during measurement were thoracic Wilson electrodes and 4 of them were tarsale Limb electrodes. During these measurements subjects were warned not to speak and move. The parameters derived from HRV measurement include; heart rate (HR), standard deviation of all NN intervals (SDNN), standard deviation of successive differences between adjoining normal cycles (SDSD), square root of the mean of the sum of the squares of differences between adjacent NN intervals (RMSSD), total power (TP), LF and HF ratio (LF/HF), high frequency (HF), normalized high frequency (HFnu), low frequency (LF), normalized low frequency (LFnu), and very low frequency (VLF).

HRV is a functional indicator to understand the state of the autonomic nervous system, and a better ANS is correlated with increased performance level(25). The balance of the sympathetic and parasympathetic nervous systems controls the heart rate(26). Sympathovagal balance is evaluated by LF/HF ratio, which is the one of frequency-domain analyses of heart rate variability. HF components are related to the cardiac parasympathetic activity, whereas LF components are relevant to both sympathetic and parasympathetic activity(22, 27).

An increased TP is related with increased parasympathetic activity. As for the other frequency-domain analyses, VLF is much less described in terms of the physiological evidence. Furthermore, measurements of VLF, LF, and HF components are usually performed in absolute values of power, but LF and HF may also be normalized (LFnu) (HFnu) as normalization leads to reduce the effect of the changes in total power on the values of LF and HF components(28). Unlike the frequency-domain parameters, the increase in all the time-domain parameters of HRV is related an increased parasympathetic activity.

Wingate anaerobic power test

The Wingate Anaerobic Power Test (WAnT) was conducted by using a cycle ergometer (Ergomedic 894 E Peak Bike, Monark, SWEDEN) according to the procedures suggested by Inbar et al.(29).

According to this, athletes warmed up for 4 minutes at between 60-80 rpm with two 3-second loads at 1:30 and 2.30 minutes, and had another 4 minutes for resting period. Subsequently, the athletes were encouraged verbally to show their maximal limits during the WAnT protocol. The parameters derived from the software were peak power (PP), relative peak power (RPP), average power (AP), relative average power (RAP), minimum power (MP), relative minimum power (RMP), and power drop (PD).

Statistical analysis

SPSS 20 (SPSS Inc., Chicago, IL, USA) was used for statistical data analyses in the research. Firstly, the distribution of data was tested by Shapiro Wilk test. After confirming the normality, the differences of HRV parameters and the outcomes of WAnT were compared by Variance Analysis in Repetitive Measurements or Friedman tests. In order to determine which group had different data in variance analysis in which Friedman Test was used, the analysis was performed with Wilcoxon Test. To analyze the pre- and post-test HRV results Paired Sample T-test or Wilcoxon Test was utilized according to the normality. Alpha value was accepted as 0.05 for all of the statistical analyses.

Results

The results of WAnT values and their mean differences according to different altitudes are shown in Table 1.

Parameters	162 m	1015 m	2146 m	3085 m	*p*_
PP (W)	695.15 ± 233.59	693.53 ± 251.85	670.93 ± 225.21	687.54 ± 240.34	0.326
RPP (W/kg)	10.29 ± 1.65	10.19± 1.81	9.92 ± 1.55	10.14 ± 1.71	0.305
AP (W)	473.24 ± 157.25	471.37 ± 160.18	466.16 ± 153.56	464.39 ± 160.40	0.164
RAP(W/kg)	7.00 ± 0.98	6.96 ± 1.05	6.89±0.94	6.85 ± 1.05	0.142
MP (W)	266.72 ± 103.02	250.87 ± 89.78	237.83 ± 81.56	247.92 ± 85.15	0.107
RMP (W/kg)	3.91 ± 0.85	3.73 ± 0.80	3.58 ± 0.88	3.67 ± .65	0.338
PD (%)	61.64 ± 7.65	63.14 ± 6.68	63.11± 10.00	63.59 ± 4.77	0.662

Table 1: WAnT values and their mean differences of all participants.

PP: Peak power; RPP: Relative peak power; AP: Average power, RAP: Relative average power; MP: Minimum power; RMP: Relative minimum power; PD: Power drop

According to the findings shown in Table 1 it could be seen that exposure to hypoxia at different altitudes had no effect in any of WAnT parameters.

In Table 2 the differences and their mean comparisons of the time- and frequency-domain HRV parameters derived from pre- and post-WAnT test are shown for all participants.

Parameters	162 m	1015 m	2146 m	3085 m	*p*_
HR (beat/min)	46.48 ± 11.53†	39.29 ± 11.62†	38.00 ± 10.20†	40.48 ± 11.78†	0.020*
SDNN	-38.33 ± 32.59†	-35.62 ± 29.43†	-37.33± 35.84†	-34.67 ± 26.57†	0.787
SDSD	-74.57 ± 56.08†	-64.05 ± 46.47†	-69.81 ± 62.91†	-60.86 ± 37.37†	0.184
RMSSD	-58.76 ± 43.94†	-50.14 ± 35.30†	-54.62 ± 49.58†	-48.24 ± 28.36†	0.264
TP	-2040.95 ± 2254.31†	-1816.81 ± 2566.17†	-2122.14 ± 3134.59†	-1629.52 ± 1789.49†	0.119
LF/HF	2.14± 1.74†	1.76 ± 1.89†	2.37 ± 2.56†	2.48 ± 2.64†	0.353
HF	-1388.67 ± 1986.78†	-1010.76 ± 1418.29†	-1496.24 ± 2688.56†	-1067.33 ± 1421.14†	0.640
HFnu	-30.13 ± 15.83†	-24.30 ± 13.44†	-25.98 ± 12.08†	-21.33 ± 12.97†	0.104
LF	-558.57 ± 417.81†	-705.67 ± 1122.60†	-539.24 ± 478.72†	-491.43 ± 375.78†	0.383
LFnu	30.15 ± 15.82†	25.25 ± 13.08†	25.96 ± 12.08†	24.92 ± 13.50†	0.611
VLF	-97.86 ± 43.07†	-101.05 ± 101.66†	-86.52 ± 49.54†	-114.10 ± 78.15†	0.312

Table 2: The differences and their mean comparisons of HRV parameters derived from pre- and post-WAnT test throughout normobaric hypoxia at different altitudes.

**p<0.05 among altitudes, †p< 0.01 between pre- and post-tests.*

HR: Heart rate; SDNN: Standard deviation of all NN intervals; SDSD: Standard deviation of successive differences between adjoining normal cycles; RMSSD: Square root of the mean of the sum of the squares of differences between adjacent NN intervals; TP: Total power; LF/HF: LF and HF ratio; HF: High frequency; HFnu: Normalized HF; LF: Low frequency; LFnu: Normalized LF; VLF: Very low frequency

The only significant change observed in Table 2 was HR among the altitudes. HR value at162 m altitude was significantly higher than 1015 m, 2146 m, and 3085 m altitudes ($p= 0.046$, $p= 0.003$, and $p= 0.030$ respectively).In addition to that, when the pre- and post-test results were examined, significant differences can be seen in all of the variables ($p< 0.01$).

Discussion and conclusion

The purpose of the study was to understand the acute adaptations of the autonomic nervous system after a maximal anaerobic load by using HRV values at different altitudes (162 m, 1015 m, 2146 m, and 3085 m) in healthy, physically-active adults. We, therefore, compared the differences of the time- and frequency-domain parameters of HRV which were derived from pre- and post- Wingate test. The results of the present study showed that there was no change in any of SDNN, SDSD, RMSSD, TP, HF, LF/HF, HF, HFnu, LF, LFnu, and VLF parameters depending on the altitudes.

The only significant change was found in heart rate value. The difference of heart rate was higher at 162 m. Apart from this, exercise effects were found significantly different between pre- and post-test results ($p< 0.01$). In a study investigating the effects of acute exposure to simulated altitude on heart rate variability during exercise at 500 m, 1500 m, 2500 m, and 3500 m within 2 h of exposure to that altitude, the effects of exercise intensity were found significant at all altitude levels in accordance with the present study results; but the altitude effects were found only during exercise at 3500 m(30).

Another study, conducted on sedentary and endurance-trained female subjects in order to determine the physiological responses of during maximal exercise at different levels of acute hypoxia (sea level, 1000 m, 2500 m, and 4500 m), showed that HRmax was lower at every altitude in both groups compared to sea-level measurements(31). When the changes of HR to high altitudewere examined, incompatible responses can be seen. Thus, the significant difference on HR seen at 162 m could be occurred coincidentally.

High altitude training and its effects on autonomic nervous system by using HRV measurement has been widely investigated in the recent years. Even though it is generally accepted in the literature that hypoxia has substantial effects on autonomic nervous system by causing increased sympathetic activity and decreased vagal activity(15, 21-24), the findings of the present study indicate opposite results.

A very similar study to the current was done by Al Haddah et al. (2012). Researchers investigated the acute effects of 20-second supramaximal running test in normobaric hypoxia (2400 m) and normobaric normoxia in healthy males.

Findings showed no difference in running speed and also maximal heart rate in both of the conditions. They also found no significant difference in HRV values (HR, LnRMSSD, LnHF, HFpeak, LnLF/HF, mRR) by comparison of hypoxia with normoxia. The reason not to find any relationship with increased altitude and impaired HRV in both of the studies could be depended on the type of the activity performed during hypoxia(15).

Studies with the significant effects on HRV after hypoxia were done by using submaximal or intermittent workload(15, 22), or without a workload(23, 32, 33). Povea et al. (2005) reported that TP, LF, LF/HF values decreased in acute hypoxia (1200 m) significantly more by comparison to normoxia in athletes(22). Al Haddah et al. (2012) found in the same study mentioned above that heart rate recovery decreases after a submaximal work load (lasted 5 min) at 2400 m of altitude(15). Studies were conducted not only on athletes but also on people with chronic diseases. Botek et al. (2015) found significantly decreased vagal activity after exposure to high altitude at 6200 m in healthy, male sports science students(32). Similarly, Iwasaki et al. (2006) reported that acute exposure to mild hypoxia (from 21 % to 15 % of inhaled oxygen) cause increased sympathetic activity(23). Limberg et al. (2015) investigated the effects of hypoxia on HRV values in people who had type 1 diabetes mellitus, and found worse impairments on RMSSD, SDNN and mean NN interval values during the hypoxia in this population(33). With the difference of the present researchthese studies did not expose the subjects to a work load at altitude.

Another outcome of the study was observing the acute anaerobic performance at different simulated altitudes. We found no change in any of the WAnT results depending on normobaric hypoxia. Even though it is accepted that long term exposure to hypoxia affects both aerobic and anaerobic metabolism(34), no significant change reported in many of the studies investigating the acute effects of hypoxia on anaerobic power. For instance, Oguri et al. (2008) stated that acute hypoxia (approximately 2000 m) does not affect the performance of supramaximal exercise such as 30 seconds Wingate test neither in athletes nor in untrained adults(35). Friedmann et al. (2007) supported that there is no influence of the acute hypoxia on anaerobic capacity(36). They concluded that anaerobic capacity is not significantly affected by acute exposure to moderate hypoxia (about 2500 m) in endurance-trained athletes. Additionally, normobaric hypoxia (2400 m) had no effect on 20-second supramaximal running speed(15). The results derived from the present study show a high consistency to these studies.

In accordance with the findings of the current study it could be inferred that to exposure a supramaximal load in normobaric hypoxia has no additional acute effects on both the level of anaerobic performance and HRV values except HR. HRV is a very sensitive indicator of autonomic nervous system, and can be affected even by the type of the exercise. For instance, Weippert et al. (2014) showed decreased SDNN, LF, and HF during isometric exercise when compared with dynamic in healthy men(37). It is known that HRV parameters are also influenced by the breathing rhythm(28).

In this present study we did not evaluate the breathing rhythm. Therefore, more research involving breathing rhythm measurements, and with different exercise types are needed to understand the mechanism of the autonomic nervous system when exposure to high altitude.

References

1) Fox L, Bowers RW, Foss ML. *The physiological basis of physical education and athletics*. Fourth edition. Florida: Saunders Company. 1988.
2) Goods PSR, Dawson BT, Landers GJ, Gore CJ, *Peeling P. Effect of different simulated altitudes on repeat-sprint performance in team-sport athletes*. Int J Sports Physiol Perform 2014; 9: *857-862*.
3) Humberstone-Gough CE, Saunders PU, Bonetti DL, Stephans S, Bullock N, Anson JM, Gore CJ. *Comparison of live high: train low altitude and intermittent hypoxic exposure*. J Sports Sci Med 2013; 12: *394-401*.
4) Brocherie F, Millet GP, Hauser A, Steiner T, Rysman J, Wehrlin JP, Girard O. *'Live high-train low and high' hypoxic training improves team-sport performance*. Med Sci Sports Exerc 2015; 47 (10): *2140-2149*.
5) Hamlin MJ, Hopkins WG, Hollings SC. *Effects of altitude on performance of elite track-and-field athletes*. Int J Sports Physiol Perform 2015; 10: *881-887*.
6) Bonetti DL, Hopkins WG. *Sea-level exercise performance following adaptation to hypoxia: a meta-analysis*. Sports Med 2009; 39: *107-127*.
7) Hamlin MJ, Marshall HC, Hellemans J, Ainslie PN, Anglem N. *Effect of intermittent hypoxic training on 20 km time trial and 30 s anaerobic performance*. Scand J Med Sci Sports 2010; 20: 651-661.
8) Aughey RJ, Buchheit M, Garvican-Lewis LA, Roach GD, Sargent C, Billaut F, Varley MC, Bourdon PC, Gore CG. *Yin and Yang, or peas in a pod? Individual-sport versus team-sport athletes and altitude training*. Br J Sports Med 2013; 47: *1150-1154*.
9) DiPasquale DM, Strangman GE, Harris NS, Muza SR. *Acute mountain sickness, hypoxia, hypobaria and exercise duration each affect heart rate*. Int J Sports Med 2015; 36: *609-614*.
10) Geiser J, Vogt M, Billeter R, Zuleger C, Belforti F, Hoppeler H. *Training high living low: changes of aerobic performance and muscle structure with training at simulated altitude*. Int J Sports Med 2001; 22 (8): *579-585*.

11) Dufour SP,Ponsot E, Zoll J, Doutreleau S, Lonsdorfer-Wolf E, Geny B, Lampert E, Flück M, Hoppeler H, Billat V, Mettauer B, Richard R, Lonsdorfer J. Exercise training in normobaric hypoxia in endurance runners, I: improvement in aerobic performance capacity. J Appl Phsiol 2006; 100 (4): 1238-1248.

12) Czuba M, Waskievicz Z, ZaJac A, Poprzecki S, Cholewa J, Roczniok R. *The effects of intermittent hypoxic training on aerobic capacity and endurance performance in cyclists*. J SportsSci Med 2011; 10 (1): *175-183*.

13) Morton JP and Cable NT. *Effects of intermittent hypoxic training on aerobic and anaerobic performance*. Ergonomics 2005; 48: *1536-1546*.

14) Akselrod S, Barak Y, Ben-Dov Y, Keselbrener L, Baharav A. *Estimation of autonomic response based on individually determined time axis*. Auton Neurosci 2001; 90: *13-23*.

15) Al Haddah H, Mendez-Villanueva A, Bourdon PC, Buchheit M. *Effect of acute hypoxia on post-exercise parasympathetic reactivation in healthy men*. Front Physiol 2012; 3: *289*.

16) Taralov Z, Terziyski K, Dimov P, Marinov B, Tarvainen MP, Perini R, Kostianev S. *Assessment of the acute impact of normobaric hypoxia as a part of an intermittent hypoxic training on heart rate variability*. The Czech Society of Cardiology 2015; 57: *e251-e256*.

17) Myllymaki T, Rusko H, Syvaoja H, Juuti T, Kinnunen ML, Kyrolainen H. *Effects of exercise intensity and duration on nocturnal heart rate variability and sleep quality*. Eur J Appl Physiol 2012; 112: *801-809*.

18) Hnidawei MA, Mjall M, Zayed Z. *The upper limit of physiological cardiac hypertrophy in elite male athletes*. Am J Appl Sci 2010; 7 (10): 1327-1333.

19) Menz V, Semch M, Mosbach F, Burtsher M. *Cardiorespiratory effects of one-legged high-intensity interval training in normoxia and hypoxia: a pilot study*. J Sports Sci Med 2016; 15: *208-213*.

20) Zhang D, She J, Zhang Z, Yu M. *Effects of acute hypoxia on heart rate variability, sample entropy and cardiorespiratory phase synchronization*. Biomed Eng Online 2014; 11: 13: *73*.

21) Sevre K, Bendz B, Hanko E, Nakstad AR, Hauge A, Kasin JI, Lefrandt JD, Smit AJ, Eide I, Rostrup M. *Reduced autonomic activity during stepwise exposure to high altitude*. Acta Physiol Scand 2001; 173: *404-417*.

22) Povea C, Schimitt L, Bruqniaux J, Nicolet G, Richalet JP, Fouillot JP. *Effects of intermittent hypoxia on heart rate variability during rest and exercise*. High Alt Med Biol 2005; 6 (3): *215-225*.

23) Iwasaki K, Oqawa Y, Aoki K, Saitoh T, Otsubo A, Shibata S. *Cardiovascular regulation response during stepwise decreases from 21 % to 15 % inhaled oxygen*. Aviat Space Environ Med 2006; 77 (10): *1015-1019*.

24) Mairer K, Wille M, Grander W, Burtsher M. *Effects of exercise and hypoxia on heart rate variability and acute mountain sickness*. Int J Sports Med 2013; 34: *700-706*.

25) Cataldo A, Zangla D, Cerasola D, Vallone V, Russo G, Lo Presti R, Traina M. *Influences of baseline heart rate variability on repeated sprint performance in young* soccer players. J Sports Med Phys Fitness 2016; 56 (4): *491-496*.

26) Acharya UR, Joseph KP, Kannathal N, Lim CM, Suri JS. *Heart rate variability: a review*. Med Biol Eng Comput 2006; 44 (12): *1031-1051*.

27) Farnelli CCJ, Kayser B, Binzoni T, Cerretelli P, Girardier L. *Autonomic nervous control of heart rate at altitude (5050 m)*. Eur J Appl Physiol Occup Physiol 1994; 69 (6): *502-507*.

28) Task-Force 1996. *Heart rate variability. Standard of measurement, physiological interpretation, and clinical use*. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Eur Heart J 1996; 17: *354-381*.

29) Inbar O, Bar-Or O, Skinner JS. *The Wingate anaerobic test*. Champaign: Human Kinetics. 1996.

30) Yamamoto Y, Hoshikawa Y, Miyashita M. *Effects of acute exposure to simulated altitude on heart rate variability during exercise*. J ApplPhysiol 1996; 81 (3): *1223-1229*.

31) Woorons X, Mollard P, Lamberto C, Letournel M, Richalet JP. *Effect of acute hypoxia on maximal exercise in trained and sedentary women*. Med Sci Sports Exerc 2005; 37 (1): *147-154*.

32) Botek M, Krejci J, De Smet S, Gaba A, McKune AJ. *Heart rate variability and arterial oxygen saturation response during extreme normobaric hypoxia*. Auton Neurosci 2015; 190: *40-45*.

33) Limberg JK, Dube S, Kuijpers M, Farni KE, Basu A, Rizza RA, Curry TB, Basu R, Joyner MJ. *Effect of hypoxia on heart rate variability and baroreflex sensitivity during hypoglycemia in type 1 diabetes mellitus*. Clin Auton Res 2015; 25: *243-250*.

34) Cerretelli P. *Muscle energetics and ultrastructure in chronic hypoxia*. Respiration 1992; 59 (2): *24-29*.

35) Oguri K, Fujimoto H, Sugimori H, Miyamoto K, Tachi T, Nagasaki S, Kato Y, Matsuoka T. *Pronounced muscle deoxygenation during supramaximal exercise under simulated hypoxia in sprint athletes*. J Sports Sci Med 2008; 7 (4): *512-519*.

36) Friedmann B, FreseF, Menold E, &Bärtsch P. *Effects of acute moderate hypoxia on anaerobic capacity in endurance-trained runners*. EurJ Appl Physiol 2007; 101 (1): *67-73*.

37) Weippert M, Behrens M, Rieger A, Behrens K. *Sample entropy and traditional measures of heart rate dynamics reveal different modes of cardiovascular control during low intensity exercise*. Entropy 2014; 16: *5698-5711*.

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