RESEARCH NOTE

Influence of Training Models at 3,900-m Altitude on the Physiological Response and Performance of a Professional Wheelchair Athlete: A Case Study

Santiago Sanz-Quinto,[1] Raúl López-Grueso,[1] Gabriel Brizuela,[2] Andrew A. Flatt,[3] and Manuel Moya-Ramón[1,4]

[1]Sports Research Center, Miguel Hernandez University, Elche, Spain; [2]Department of Physical and Sports Education, University of Valencia, Valencia, Spain; [3]Department of Health Science and Kinesiology, Georgia Southern University, Savannah, Georgia; and [4]Department of Health Psychology, Miguel Hernandez University, Elche, Institute for Health and Biomedical Research (ISABIAL-FISABIO Foundation), Alicante, Spain

Abstract

Sanz-Quinto, S, López-Grueso, R, Brizuela, G, Flatt, AA, and Moya-Ramón, M. Influence of training models at 3,900-m altitude on the physiological response and performance of a professional wheelchair athlete: A case study. *J Strength Cond Res* 33(6): 1714–1722, 2019—This case study compared the effects of two training camps using flexible planning (FP) vs. inflexible planning (IP) at 3,860-m altitude on physiological and performance responses of an elite marathon wheelchair athlete with Charcot-Marie-Tooth disease (CMT). During IP, the athlete completed preplanned training sessions. During FP, training was adjusted based on vagally mediated heart rate variability (HRV) with specific sessions being performed when a reference HRV value was attained. The camp phases were baseline in normoxia (B_N), baseline in hypoxia (B_H), specific training weeks 1–4 (W1, W2, W3, W4), and Post-camp (Post). Outcome measures included the root mean square of successive R-R interval differences (rMSSD), resting heart rate (HR_{rest}), oxygen saturation (SO_2), diastolic blood pressure and systolic blood pressure, power output and a 3,000-m test. A greater impairment of normalized rMSSD (B_N) was shown in IP during B_H (57.30 ± 2.38% vs. 72.94 ± 11.59%, $p = 0.004$), W2 (63.99 ± 10.32% vs. 81.65 ± 8.87%, $p = 0.005$), and W4 (46.11 ± 8.61% vs. 59.35 ± 6.81%, $p = 0.008$). At Post, only in FP was rMSSD restored (104.47 ± 35.80%). Relative changes were shown in power output (+3 W in IP vs. +6 W in FP) and 3,000-m test (−7s in IP vs. −16s in FP). This case study demonstrated that FP resulted in less suppression and faster restoration of rMSSD and more positive changes in performance than IP in an elite wheelchair marathoner with CMT.

Key Words hypoxia, heart rate variability, autonomic nervous system, paralympic, marathon

Introduction

It was first demonstrated in 2003 through peroneal microneurography that during acclimatization to high-altitude hypoxia, sympathetic overactivity occurs in lowlanders (19). More recently, Lundby et al. (25) reported the same sympathoexcitation in lifelong highlanders at 4,100 m and that their muscle sympathetic nerve activity burst frequency was twice the level recorded in a group of highlanders at sea level. Slightly lower activity was shown in highlanders compared with lowlanders after 50 days of exposure (chronic hypoxia). Noninvasive studies have shown altered autonomic heart rate regulation with altitude and endurance training (13,21) but their results are not in agreement with the increase in parasympathetic activity observed at altitude using invasive measures (6).

Higher resting heart rate variability (HRV) has been associated with better fitness (35) and increased exercise performance (9). In addition, successful adaptation to an endurance training program is reflected in an increase in several time- and frequency-domain indices of HRV (20) along with less day-to-day fluctuations (represented by the coefficient of variation, CV) (16). By contrast, lower HRV values have been related to maladaptive training responses (4,34). Recently, to maximize adaptations and avoid the risk of overreaching, a new training approach, using a flexible training program model (FP) has emerged where daily training loads are adjusted based on a reference value (RV) of postwaking, vagally mediated HRV (22,36). For example, a group of recreational endurance runners after an FP based

Address correspondence to Santiago Manuel Moya-Ramón, mmoya@umh.es.

33(6)/1714–1722

Journal of Strength and Conditioning Research

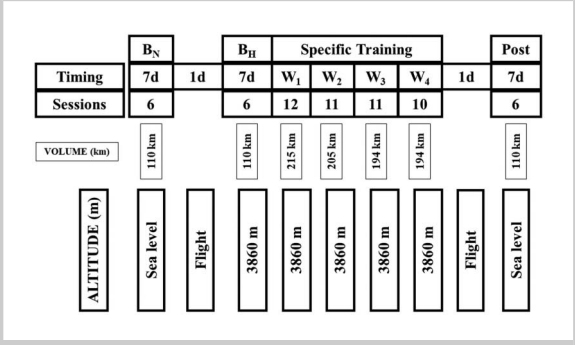

Figure 1. Inflexible planning (IP) experimental design. B_N = baseline in normoxia; B_H = baseline in hypoxia; W(1,2,3,4) = first, second, third, and fourth weeks, where specific sessions were performed after acclimatization phase; Post = returning sea level week. In W2, PM session on Friday was canceled due to feeling exhausted (5 days after day off). In W3, PM session on Monday was canceled due to heavy rain. In W4, PM sessions on Friday and Saturday were canceled due to feeling exhausted (5 and 6 days after day off). In W4, PM session on Monday was canceled due to heavy rain. Number of interval sessions: W1 #2–W2 #2–W3 #2–W4 #2. Number of sessions ~65% $\dot{V}O_2$max ~2-hour workouts W1 #2–W2 #2–W3 #2–W4 #2.

on HRV improved 3,000-m running performance, whereas no improvement was reported for the group after an inflexible planning (IP) (preplanned) program (36). Although HRV has been evaluated in athletes during periods of standardized training under hypoxic conditions (29), it remains unclear whether HRV-guided training at altitude would offer any advantages over standardized training. In a recent study with elite Nordic skiers who slept in normobaric hypoxia (F_iO_2 = 15.0%), only the HRV-guided training group improved roller-ski performance, whereas oxygen uptake

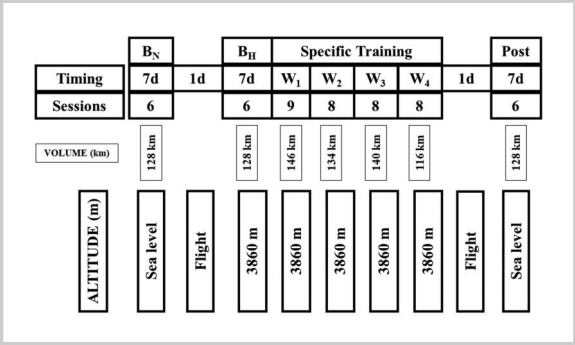

Figure 2. Flexible planning (FP) experimental design. B_N = baseline in normoxia; B_H = baseline in hypoxia; W ($_{1,2,3,4}$), first, second, third, and fourth weeks, where specific sessions were performed after acclimatization phase; Post = returning sea level week. In W_4, PM session on Monday was canceled due to heavy rain. Number of interval sessions: W_1 #2 (1 × "A" + 1 × "C"), W_2 #3 (2 × "A" + 1 × "C"), W_3 #2 (1 × "C" + 1 × "A"), and W_4 #2 (1 × "C"). Number of sessions ~65% $\dot{V}O_2$max ~2-hour workouts W_1 #1 (1 × "B"), W_2 #1 (1 × "B"), W_3 #2 (2 × "B"), and W_4 #0.

was improved by both hypoxic groups after the intervention (30).

Increased diastolic blood pressure (DP) at altitude has been associated with a sympathetic response as a way to compensate for hypoxia-induced peripheral vasodilation (25). However, the same research did not report changes in systolic blood pressure (SP). It is unknown whether marathon wheelchair athletes demonstrate a faster acclimatization to a hypoxic environment than able-bodied marathoners. Wheelchair athletes have less active muscles during propulsion (12) and the energy cost of running depends on anthropometric features such as the length of the muscles involved (18). Moreover, despite a similar heart rate in elite wheelchair marathoners (1) and elite able-bodied marathoners (3), oxygen uptake is quite lower in wheelchair athletes (1). Thus, oxygen status may be differently affected among individuals with a higher level of muscular atrophy in upper extremities, as in the current case study where the participant was diagnosed with Charcot-Marie-Tooth disease (CMT). Charcot-Marie-Tooth disease is the most common hereditary peripheral neuropathy, affecting up to 30 per 100,000 people worldwide (2). Charcot-Marie-Tooth disease totally affects distal muscle function and partially affects proximal function. Although muscle atrophy is associated with CMT, respiratory and cardiac system responses are not disturbed. Only one study has evaluated the effect of endurance training on HRV indices among individuals with CMT, finding that 12 weeks of interval training increased cardiac-parasympathetic activity (15).

With increasing altitude, there is a progressive decrease in peak heart rate (24), where 5 sea-level lowlanders performed maximal efforts under hypobaric hypoxia conditions, corresponding to altitudes of 3,300, 4,300, 5,300, and 6,300 m above sea level. Reduction in peak heart rate is approximately 1 $b \cdot min^{-1}$ for every 7 mm Hg decrease in barometric pressure below 530 mm Hg. Although mechanical stress in wheelchair racing seems to be lower than in running, cardiovascular strain is higher when comparing arm and leg exercise (11), whereas oxygen uptake is lower (1). Furthermore, resting heart rate range in sedentary individuals with CMT population seems to be the same as general population (15).

This case study compared the physiological and performance effects of 2 different training programs (IP and FP) conducted in the Peruvian Andes (3,860 m), over 2 consecutive years in an elite wheelchair marathoner with CMT. The primary objective was to determine which model facilitated better physiological and performance adaptations.

Methods

Experimental Approach to the Problem

A single-subject case study featuring an elite wheelchair marathoner with CMT was conducted to determine the effects of 2 different training models (i.e., IP vs. FP) at altitude on physiological and performance responses.

Table 1. Weekly mileage and strength volume and intensity in FP and IP.*

		Mileage (km) for IP and FP			
	Training program	W_1 (km)	W_2 (km)	W_3 (km)	W_4 (km)
60% $\dot{V}O_2$max	IP	126.8	120.8	110.8	105.8
	FP	96.1	76.7	61.9	104.8
65% $\dot{V}O_2$max	IP	68.0	68	62.0	68.0
	FP	30.0	30.4	58.4	
70–75% $\dot{V}O_2$max	IP	20.0	16.0	20.0	20.0
	FP	20.0	28.0	20.0	12.0
Overall	IP	214.8	204.8	192.8	193.8
	FP	146.65	135.6	140.6	116.8
Sessions	IP	12	11	11	10
	FP	9	8	8	8

	Resistance training for IP and FP			
Exercises	% RM	Sets	Reps	Recovery (s)
Bench press, close grip dumbbell press, seated military press, seated cable row	80	4	8	150

*FP = flexible planning; IP = inflexible planning; W(1,2,3,4) = first, second, third, and fourth weeks of specific sessions, after acclimatization phase; RM = repetition maximum.

Training camps at altitude were repeated on consecutive years at the same time and location in preparation for the competitive season. Cardiac-autonomic activity (vagally mediated HRV and resting heart rate, HR_{rest}), oxygen saturation (SO_2), and blood pressure were measured daily throughout the following periods: sea-level baseline, altitude baseline, 4 weeks of training at altitude, and 1-week post-training at sea level. Performance tests to evaluate power output on an ergometer and aerobic power in a 3,000 m test were conducted before and after the training camps. Changes in physiological and performance parameters were assessed within and between camps. Relationships between physiological parameters were quantified.

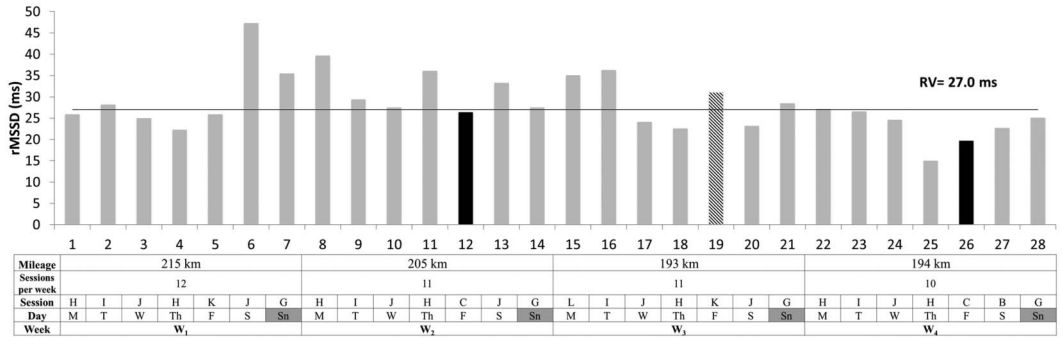

Figure 3. Heart rate variability during specific training weeks at 3,900 meters in IP. $W_{1,2,3,4}$ = specific training weeks in hypoxia; rMSSD = square root of the mean of the squares of the successive differences between adjacent NNs; RV = rMSSD value chosen to perform specific sessions in FP; Session B = 2 hours at the aerobic threshold; Session C = 6 × 2,000 m recovery reps 120; Session G = rest; Session H = 10 km below aerobic threshold + gym session +16 km below aerobic threshold in the afternoon; Session I = 20 × 400 m recovery reps 75 seconds + 16 km below aerobic threshold in the afternoon; Session J = 2 hours at the aerobic threshold +12 km below the aerobic threshold in the afternoon; Session K = 6 × 2,000 m recovery reps 120 seconds + 12 km below the aerobic threshold in the afternoon; FP = flexible planning; IP = inflexible planning. Columns in bold represent days where athlete was not able to complete afternoon session due to feeling exhausted in IP. Columns with bars represent days where heavy rain forced the athlete to cancel or modify a session.

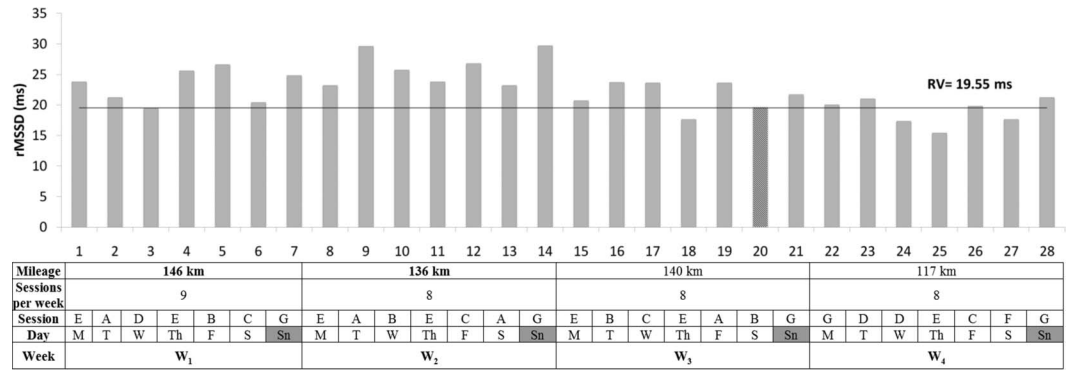

Figure 4. Heart rate variability during specific training weeks at 3,900 m in FP. $W_{1,2,3,4}$ = specific training weeks in hypoxia; Post = returning sea level week; rMSSD = square root of the mean of the squares of the successive differences between adjacent NNs; RV = rMSSD value chosen to perform specific sessions in FP; Session A = 20 × 400 m recovery reps 75 seconds; Session B = 2 hours at the aerobic threshold; Session C = 6 × 2,000 m recovery reps 120 seconds; Session D = 20 km below aerobic threshold in the morning +16 km below the aerobic threshold in the afternoon; Session E = 16 km below aerobic threshold in the morning + gym session in the afternoon; Session F = 20 km below aerobic threshold in the morning; Session G = rest; FP = flexible planning. Columns with bars represent days where heavy rain force athlete to cancel or modify a session.

Subjects

One professional male wheelchair athlete (mean ± *SD*: age = 36 years; height = 1.76 m; body mass = 50.0±0.81 kg; power output at second lactate threshold = 62 W) with CMT participated in this case study. This athlete was a silver medalist at the 2000 and 2004 Paralympic Games; a former world record holder in his division (T52, quadriplegics) in 800 m (116 seconds) and 1,500 m (216 seconds); a world record holder in 5,000 m (757 seconds) and half marathon (3,028 seconds); possesses the fourth best ever time in marathon (6,125 seconds); and holds a record of 106 victories in road events. He has accumulated more than 10 years of altitude training experience, has performed both altitude models, Live-High-Train-High and Live-High-Train-Low (23) and had been exposed to more than 8,000 hours of normobaric hypoxia.

After being informed of the requirements and risks associated with their involvement in this study, the participant provided written informed consent to be a research subject in this case study. All procedures were approved by the Ethics Research Committee of the University Miguel Hernández (Elche, Spain).

Procedures

The 2 training camps (IP and FP) each lasted 5 weeks in duration and were completed over 2 successive years (January and February 2015–2016) during the spring marathon preparatory cycle (Figures 1 and 2).

Inflexible Model. In the IP camp, the participant completed every preplanned workout. Camps were divided into 4 periods: 1-week at sea level as baseline in normoxia (B_N), a 1-week acclimatization phase as baseline in hypoxia (B_H), 4 specific training weeks (W_1, W_2, W_3 and W_4), and 1 week back at sea level (Post). Heart rate variability, SO_2, and resting HR_{rest} were collected daily.

B_N training was similar during B_H for both IP and FP camps. The first 2 days of B_H involved passive rest to minimize jet lag and acute mountain sickness symptoms caused by the long trip from Spain to Peru (time difference of 6 hours) and the change in altitude. Two daily training sessions were performed from Wednesday to Friday at an intensity <the aerobic threshold (i.e., "jog"). The morning and afternoon sessions were 20 and 16 km, respectively, with a 20-km jog also performed on Saturday morning. From W_1 to W_4 during IP, 12 sessions per week (≈200 km every 6 consecutive days) were performed, whereas Sundays were reserved for passive rest. (Table 1). Two resistance sessions were performed on Mondays and Thursdays (Figure 2), and 2 interval sessions were performed on a plateau at 4,090 m altitude on Tuesdays (20 × 400 m, recovery repetitions: 75 seconds) and Fridays (6 × 2 km, recovery repetitions: 120 seconds). Two-hour sessions at the aerobic threshold were performed on Wednesdays and Saturdays.

Flexible Planning. During B_H, the HRV reference value was determined in both models but was only used to guide specific sessions (36) in FP throughout W_1–W_4. One *SD* below the mean of the root mean square of successive differences (rMSSD) throughout B_H was chosen as the RV (36). Accordingly, the RV calculated for IP was 27.0 ms and for FP was 19.55 ms.

From W_1 to W_4, the training was fixed on Mondays and Thursdays, with morning sessions involving a 16-km jog and the afternoon sessions involving resistance training (Table 1). Sundays were passive rest.

If the RV was reached (rMSSD ≥19.55 ms), a specific session was performed in the morning, followed by an evening off. The specific sessions were: A (20 × 400 m on

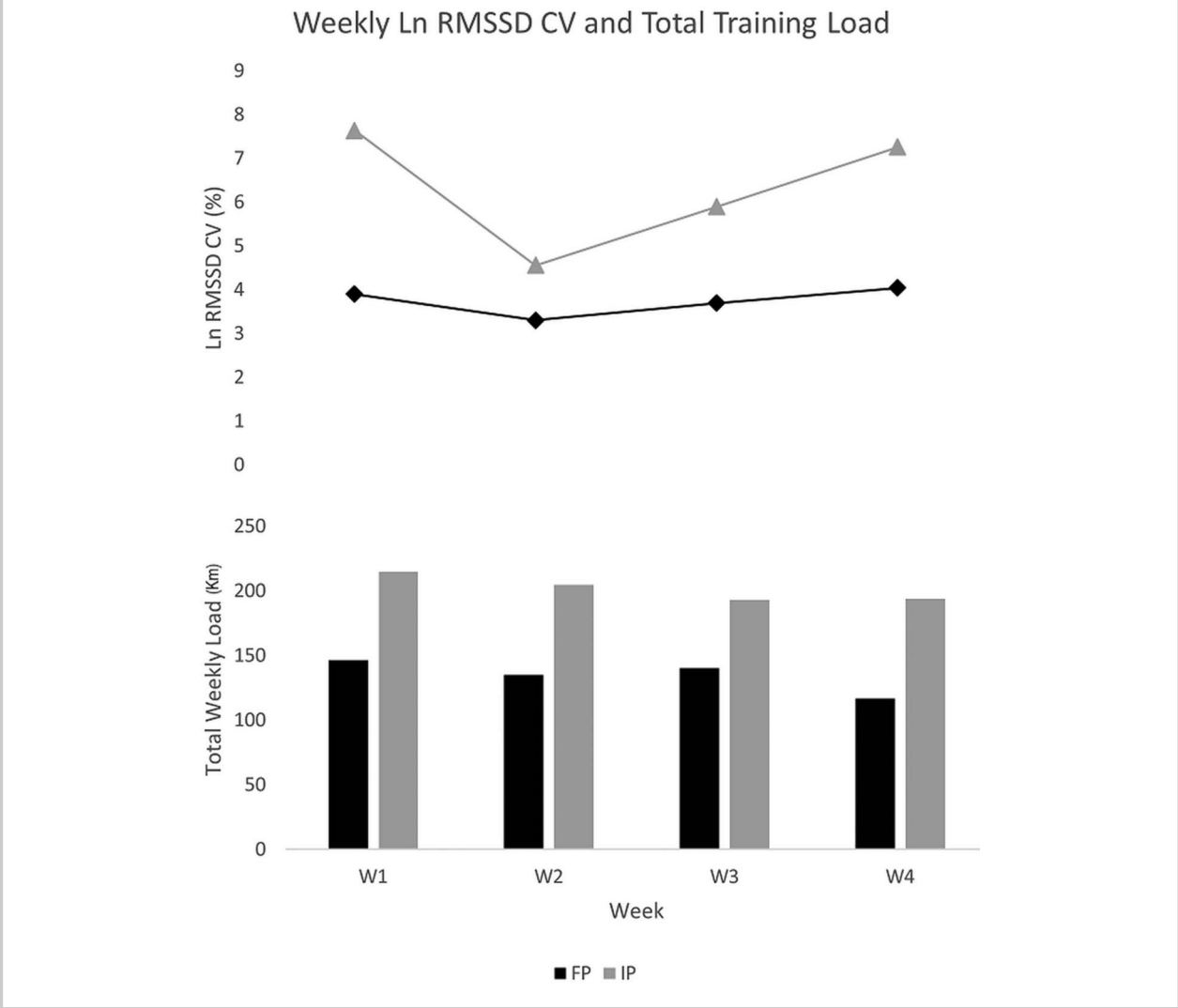

Figure 5. Weekly CV RMSSD and total training load for specific training weeks. Ln RMSSD CV = change in the CV of log-transformed root mean square of successive R-R intervals from FP (gray line and bars) and IP (black line and bars). Total weekly volume in kilometers from FP and IP. FP = flexible planning; IP = inflexible planning.

a plateau at 4,090 m, recovery repetitions: 75 seconds), B (2 hours at the aerobic threshold), and C (6 × 2 km on a plateau at 4,090 m, recovery repetitions: 120 seconds). The order to perform specific sessions was always in sequence (e.g., A, B, C). If the RV was <than 19.55 ms, 2 easy workouts were performed (morning, 20-km "jog" and afternoon, 16-km "jog"). The specific sequence of sessions would not be affected by the end of a week. For details of daily training in IP and FP, see Figures 3 and 4.

Resistance training structure and exercises are described in Table 1. Heart rate variability was recorded daily in the supine position after waking, bladder emptying, and in a fasted state. A metronome was used during HRV recordings to control for respiration rate (15 breaths·min^{-1}). A heart rate monitor (Polar RSCX 800; Kempele, Finland) was used to record R-R intervals. Filtering, correction, and detrending were applied to avoid ectopic beats. The HRV parameters were calculated with Kubios HRV 2.0 (Kuopio, Finland, 2008) analyzing the last 5 minutes of a 10-minute recording (5,33). The rMSSD was selected as the main index to assess HRV (7,8). Raw rMSSD values expressed in ms can be seen in Figures 3 and 4. The weekly CV of rMSSD was calculated for W_1–W_4 (Figure 5) as an indicator of training adaptation (16). The HR_{rest} was calculated as the HR average of the last 5 minutes of the R-R interval recording.

The SO_2 was measured with a finger pulse oximeter (Colson 650 2100; Frouard, France) in a seated position on awaking. The brachial blood pressure was measured (only in FP)

Table 2. Time- and frequency-domain indices of HRV, SO_2, and HR_{rest} before, during, and after both training programs.*†

Training program	Variable (mean ± SD)	B_N 1	B_H 2	W_1 3	W_2 4	W_3 5	W_4 6	Post 7
IP	Δ rMSSD	100	57.30 ± 2.38‡	61.14 ± 17.65‡§	63.99 ± 10.32‡§	58.43 ± 11.54‡§	46.11 ± 8.61‡§¶#	88.94 ± 25.34
FP		100	72.94 ± 11.59††	72.58 ± 8.74	81.65 ± 8.87††	67.51 ± 7.38‡	59.35 ± 6.81‡§¶††	104.47 ± 35.80**
IP	SO_2 (%)	98.86 ± 0.12	86.29 ± 0.53	88.74 ± 0.90	90.33 ± 0.53§‖	92.78 ± 1.07§‖	92.06 ± 1.35§‖	98.17 ± 0.32§‖¶#**
FP		98.64 ± 0.14	88.31 ± 2.46	91.19 ± 0.76§††	91.92 ± 0.82§††	92.35 ± 1.14§‖	92.64 ± 1.12§	98.08 ± 0.26§‖¶#**
IP	Δ HR_{rest}	100	130.49 ± 8.10‡	131.83 ± 6.98‡	128.12 ± 7.58‡	124.64 ± 6.71‡	123.80 ± 5.93‡	96.20 ± 4.23§‖¶#**
FP		100	130.17 ± 5.93‡	134.07 ± 3.92‡	130.06 ± 3.18‡	128.70 ± 6.84‡	127.87 ± 6.51‡	96.64 ± 8.99§‖¶#**

*HRV = heart rate variability; B_N = baseline in normoxia at 16 m; B_H = baseline in hypoxia at 3,860 m; W_1 = first week of specific training; W_2 = second week of specific training; W_3 = third week of specific training; W_4 = fourth week of specific training; Post = values after altitude training camp at 16 m altitude; IP = inflexible planning; FP = flexible planning; rMSSD = root mean square of successive R-R interval differences.
†The values (less SO_2) are normalized in percentages (Δ) with reference to the baseline in normoxia (B_N) at 16 m altitude.
‡Differences from B_N ($p < 0.01$).
§Differences from B_H ($p < 0.01$).
‖Differences from W_1 ($p < 0.01$).
¶Differences from W_2 ($p < 0.01$).
#Differences from W_3 ($p < 0.01$).
**Differences from W_4 ($p < 0.01$).
††Differences between groups ($p < 0.01$).

in a seated position, with the validated (Omron HEM-705CP) oscillometric sphygmomanometer. Measurements were made in triplicate and averaged. Both SP and DP were recorded.

Laboratory Test. Four days before B_N (Pre-4) and 11 days after altitude camp (Post11), an incremental test was performed on a specific wheelchair ergometer, where steady conditions were maintained (temperature 22–24° C, humidity 73–75%). The protocol (as described by Polo-Rubio) (27) included a 20-minute warm-up period at constant power (20 W). Then, the athlete started an incremental test at a brake power of 6 W, maintaining a stroke frequency between 90 and 100 strokes·min^{-1} and increasing the power by 3 W every 60 seconds until the athlete's heart rate passed 170 b·min^{-1} (just over his marathon pace intensity). Power output was considered as the ergometer braking power during the last completed step of the test. A heart rate monitor was included to measure heart rate. Due to the invasiveness of wearing a gas analyzer device during wheelchair propulsion, oxygen consumption ($\dot{V}O_2$) was not assessed.

3,000 m Test. Three days before B_N (Pre_{-3}) and 12 days after altitude camp ($Post_{12}$), a 3,000 m test was performed on a 200-m indoor track. After a warm-up of 6 kilometers plus 80 m strides, the test started from a static position. Conditions for all track tests were: temperature = 18.3 ± 2.1° C and humidity = 74–79%. The reason for choosing the $Post_{12}$ as the day to perform the 3,000-m test was because the athlete had his greatest marathon performance (Oita Marathon, 1 hour 43 minutes 46 seconds and Chicago Marathon, 1 hour 46 minutes and 13 seconds, both still quadriplegic division course records and both set in 2007) 12 days after arrival from altitude, after completing camps at 2,320-m altitude.

Statistical Analyses

The distribution of each variable was examined using the Kolmogorov-Smirnov normality test. Natural logarithm transformations (Ln) were applied to rMSSD. All data (except SO_2, SP, and DP) were normalized in percentages with reference to the baseline in normoxia (Δ) and presented as a mean ± *SD*. A repeated-measures ANOVA was performed for all the variables, including the factor TIME with levels B_N, B_H, W_1, W_2, W_3, W_4, and Post. A post hoc least significant difference (LSD) multiple range test determined differences between factor levels. Pearson's correlation coefficients were calculated for the rMSSD, SO_2, HR_{rest}, SP, and DP variables. Statistical significance was set at alpha = 0.05. Statistical analyses were performed using SPSS version 22.0 (SPSS, Inc., Chicago, IL, USA) software.

Results

Results for rMSSD, SO_2, HR_{rest}, SP, and DP can be viewed in Table 2. In IP, B_N rMSSD was significantly greater than all

altitude periods but not Post. Greater hypoxemia was observed in B_H ($p = 0.001$) compared with B_N. From B_H to Post, SO_2 values increased each week.

Increasing HR_{rest} was observed after exposure to altitude ($p = 0.001$). A negative correlation was found between HR_{rest} and SO_2 ($r = -0.43$; $p = 0.0188$).

In FP, B_N rMSSD was signficantly greater than all altitude periods but was not different from Post (Table 2). A strong correlation was found between rMSSD and SO_2 ($r = 0.54$; $p = 0.001$).

Greater hypoxemia was shown in B_H with SO_2 values being significantly lower ($p = 0.001$) than in B_N (Table 2). From B_H to Post, SO_2 values increased each week. HR_{rest} increased significantly ($p = 0.001$) from B_N to B_H. Post HR_{rest} was significantly lower ($p = 0.001$) than altitude values. A negative correlation was found between HR_{rest} and SO_2 ($r = -0.83$; $p = 0.001$).

We observed increased DP comparing all altitude periods with normoxic conditions ($p = 0.001$), whereas SP did not differ from prealtitude to acclimatization ($p > 0.05$).

Between-camp analysis revealed that rMSSD during FP was significantly greater than IP at B_H ($p = 0.004$), W_2 ($p = 0.005$), and W_4 ($p = 0.008$).

SO_2 was higher in FP at B_H ($p = 0.049$), W_1 ($p = 0.001$), and W_2 ($p = 0.001$), suggesting a faster recovery of this variable.

The ergometer test in IP showed relative changes in power output (46 W at Pre_{-4} vs. 49 W at $Post_{11}$), whereas in FP, change was 44 W at Pre_{-4} vs. 50 W at $Post_{11}$ ($p = 0.001$).

Both models reduced time set in 3,000-m test; however, due to the magnitude of the change, it cannot be considered a significant improvement. (IP $Pre_{-3} = 470$ seconds vs. IP $Post_{12} = 463$ seconds; FP $Pre_{-3} = 472$ seconds vs. FP $Post_{12} = 456$ seconds).

Discussion

This case study compared the effects of 2 different training models (IP and FP) on cardiac-autonomic activity and performance responses of an elite marathon wheelchair athlete with CMT. The primary objectives were to determine which model facilitated more desirable physiological and performance responses.

During IP, significant reductions in rMSSD (42.7%) were observed at B_H relative to B_N; however, it cannot be interpreted as a reduction in vagal activity because it has been reported that in acclimatization, there is an increase in both parasympathetic (6) using a vagal blockade and sympathetic activity (25) with peroneal microneurography. In line with our findings, one study demonstrated an 88% reduction in high frequency (HF) power (a frequency-domain index of HRV) during the first 2 days at 4,350 m altitude, improving only to within 54% of baseline HF after 6 days (13). However, we must be cautious with these results as Lundby et al. (25) did not report differences in muscle sympathetic nerve activity in acute exposure to hypobaric hypoxia (F_IO_2 of 0.12) in lowlanders compared with sea level. The rMSSD remained >10% below B_N values at Post, suggesting that the alteration of HRV remained after altitude exposure.

The athlete may have experienced greater suppression of rMSSD during IP than FP for several reasons. For example, total training loads at altitude were ~40% greater during IP compared with FP. Increased training loads have been shown to cause larger reductions along with greater daily fluctuation in rMSSD (i.e., higher rMSSDcv) concurrent with decrements in perceived fatigue and muscle soreness (17). The participant demonstrated consistently larger reductions in rMSSD relative to baseline (Table 2) and thus a higher rMSSDcv during each training week at altitude during IP (Figure 5). Moreover, the athlete was unable to complete prescribed sessions on at least 2 occasions during IP due to feelings of exhaustion (Figure 3). Finally, rMSSD remained >10% below B_N in IP when training loads were reduced at near-sea level (i.e., Post), which may reflect persisting effects of fatigue and inadequate recovery.

A significant decrease in SO_2 (14.71%) was shown in B_H. Hypoxemia below 95% SO_2 has been associated with impaired $\dot{V}O_2max$ (14). A slight increase until W_3 was shown, reflecting acclimatization to altitude (36).

Similar drops in SO_2 were shown in FP and IP; however, it was restored faster in FP. Until W_2, there could be a greater impairment of cardiorespiratory function in IP, as well as a greater impairment of ability to adapt to the acute hypoxia phase (28).

Lower SO_2 in B_H in IP could be induced by a lower ventilatory response (31), a phenomenon related to acute mountain sickness. Sympathoexcitation due to a decrease in SO_2 has been observed under hypoxic conditions (26).

The HR_{rest} showed similar increases in FP and IP (~17 $b \cdot min^{-1}$) from normoxia to hypoxia, a response reported recently at 3,454 m (32). There was a greater decreasing pattern in IP that might be explained by the greater training loads in IP (200 km per week) compared with FP (140 km per week) and more sessions at the aerobic threshold (8 vs. 4).

We found significant increases in both SP and DP. Although Lundby et al. (25) did not find differences in SP, more extenuated circumstances in our experiment might be the reason for a greater increase in this sympathetic marker. In fact, once specific training began, we found SP to be significantly higher than normoxic conditions (not observed in acclimatization). A phenomenon defined as a sympathetic response to hypoxia is an increase in DP (25), which was observed in the current case study, possibly to increase vasodilation (25).

Both training camps generated positive relative changes in power output and 3,000 m times. The greater improvement in FP is in agreement with a recent study from Schmitt et al.(30), where only Nordic skiers guided by HRV improved roller-ski performance ($-2.7 \pm 3.6\%$) 21 days after hypobaric hypoxia intervention. Our results conflict with

those of Buskirk et al. (10), where well-trained runners did not improve endurance performance after more than 40-day training at 4,000 m altitude. Our athlete was able to perform 2,000 m repetitions at 4,090 m in an average of 310 seconds, decreasing his performance compared with sea level by around 3%. At the same altitude, well-trained runners decreased performance 20–24% in 1,609 m or 3,218 m, respectively.

This study showed that an FP model guided by HRV induced less suppression and faster restoration of rMSSD and lower $rMSSD_{CV}$ compared with the IP training model. Both models showed an increase in performance after altitude exposure, but greater enhancement was observed after the FP model, despite administration of lower training loads. HRV-guided FP may therefore be a useful training method for maintaining training loads within the recovery capacity of the athlete at altitude.

Practical Applications

This case study suggests that HRV is a convenient, noninvasive, physiological marker that can be used to help autoregulate training loads in wheelchair marathoners. Individuals may be able to limit the magnitude of autonomic nervous system imbalance associated with living and training at altitude by using HRV-guided training in favor of inflexible, preplanned training. This method may facilitate smaller reductions and less fluctuation in indices such as rMSSD and $rMSSD_{CV}$ in addition to inducing less fatigue and greater endurance performance improvements from a lower training load.

Acknowledgments

The authors thank the athlete who volunteered for this case study.

References

1. Asayama, K, Nakamura, Y, Ogata, H, Hatada, K, Okuma, H, and Deguchi, Y. Physical fitness of paraplegics in full wheelchair marathon racing. *Paraplegia* 23: 277–287, 1985.
2. Banchs, I, Casasnovas, C, Albertí, A, De Jorge, L, Povedano, M, Montero, J, et al. Diagnosis of Charcot-Marie-Tooth Disease. *J Biomed Biotechnol*: 1–10, 2009.
3. Billat, VL, Demarle, A, Slawinski, J, Paiva, M, and Koralsztein, JP. Physical and training characteristics of top-class marathon runners. *Med Sci Sports Exerc* 33: 2089–2097, 2001.
4. Bosquet, L, Merkari, S, Arvisais, D, and Aubert, AE. Is heart rate a convenient tool to monitor over-reaching? A systematic review of the literature. *Br J Sports Med* 42: 709–714, 2008.
5. Bourdillon, N, Schmitt, L, Yazdani, S, Vesin, JM, and Millet, GP. Minimal window duration for accurate HRV in athletes. *Front Neurosci* 11: 456, 2017.
6. Boushel, R, Calbet, JA, Radegran, G, Sondergaard, H, Wagner, PD, and Saltin, B. Parasympathetic neural activity accounts for the lowering of exercise heart rate at high altitude. *Circulation* 104: 1785–1791, 2001.
7. Buchheit, M, Papelier, Y, Laursen, PB, and Ahmaidi, S. Noninvasive assessment of cardiac parasympathetic function: Postexercise heart rate recovery or heart rate variability?. *Am J Physiol Heart Circ Physiol* 293: H8–H10, 2007.
8. Buchheit, M, Chivot, A, Parouty, J, Mercier, D, Al Haddad, H, Laursen, PB, et al. Monitoring endurance running performance using cardiac parasympathetic function. *Eur J Appl Physiol* 108: 1153–1167, 2010.
9. Buchheit, M, Simpson, MB, Al Haddad, H, Bourdon, PC, and Mendez-Villanueva, A. Monitoring changes in physical performance with heart rate measures in young soccer players. *Eur J Appl Physiol* 112: 711–723, 2012.
10. Buskirk, ER, Kollias, J, Akers, RF, Prokop, EK, and Reategui, EP. Maximal performance at altitude and on return from altitude in conditioned runners. *J Appl Physiol* 23: 259–266, 1967.
11. Calbet, JA, Gonzalez-Alonso, J, Helge, JW, Sondergaard, H, Munch-Andersen, T, Saltin, B, et al. Central and peripheral hemodynamics in exercising humans: Leg vs. arm exercise. *Scand J Med Sci Sports* 25: 144–157, 2015.
12. Chow, JW, Millikan, TK, Carlton, LG, Morse, MI, and Chae, WS. Biomechanical comparison of two racing wheelchair propulsion techniques. *Med Sci Sports Exerc* 33: 476–484, 2001.
13. Cornolo, J, Mollard, P, Brugniaux, JV, Robach, P, and Richalet, JP. Autonomic control of the cardiovascular system during acclimatization to high altitude: Effects of sildenafil. *J Appl Physiol* 97: 935–940, 2004.
14. Dempsey, JA and Wagner, PD. Exercise-induced arterial hypoxemia. *J Appl Physiol* 87: 1997–2006, 1999.
15. El Mhandi, L, Pichot, V, Calmels, P, Gautheron, V, Roche, F, and Féasson, L. Exercise training improves autonomic profiles in patients with Charcot–Marie–Tooth disease. *Muscle Nerve* 44: 732–736, 2011.
16. Flatt, AA and Esco, MR. Evaluating individual training adaptation with smartphone-derived heart rate variability in a collegiate female soccer team. *J Strength Cond Res* 30: 378–385, 2016.
17. Flatt, AA, Hornikel, B, and Esco, MR. Heart rate variability and psychometric responses to overload and tapering in collegiate sprint-swimmers. *J Sci Med Sport* 20: 606–610, 2017.
18. Fletcher, JR and MacIntosh, BR. Running economy from a muscle energetics perspective. *Front Physiol* 8: 433, 2017.
19. Hansen, J and Sander, M. Sympathetic neural overactivity in healthy humans after prolonged exposure to hypobaric hypoxia. *J Physiol* 546: 921–929, 2003.
20. Hynynen, ES, Uusitalo, A, Konttinen, N, and Rusko, H. Heart rate variability during night sleep and after awakening in overtrained athletes. *Med Sci Sports Exerc* 38: 313–317, 2006.
21. Kanai, M, Nishihara, F, Shiga, T, Shimada, H, and Saito, S. Alterations in autonomic nervous control of heart rate among tourists at 2700 and 3700m above sea level. *Wilderness Environ Med* 12: 8–12, 2001.
22. Kiviniemi, AM, Hautala, AJ, Kinnunen, H, and Tulppo, MP. Endurance training guided individually by daily heart rate variability measurements. *Eur J Appl Physiol* 101: 743–751, 2007.
23. Levine, BD and Stray-Gundersen, J. "Living high-training low": Effect of moderate-altitude acclimatization with low-altitude training on performance. *J Appl Physiol* 83: 102–112, 1997.
24. Lundby, C, Araoz, M, and van Hall, G. Peak heart rate decreases with increasing severity of acute hypoxia. *High Alt Med Biol* 2: 369–376, 2001.
25. Lundby, C, Calbet, JA, van Hall, G, Saltin, B, and Sander, M. Sustained sympathetic activity in altitude acclimatizing lowlanders and high-altitude natives. *Scand J Med Sci Sports* 28: 854–861, 2018.
26. Macoun, T, Botek, M, Krejcî, J, and McKune, AJ. Vagal activity and oxygen saturation response to hypoxia: Effects of aerobic fitness and rating of hypoxia tolerance. *Acta Gymnica* 47: 112–121, 2017.

27. Polo-Rubio, M. *Influence of biomechanical and physiological variables on the athletic performance of wheelchair athletes. Optimization of performance of a world-class athlete [PhD Thesis]*: University of Valencia, Valencia, Spain, 2007.

28. Roach, RC, Greene, ER, Schoene, RB, and Hackett, P. Arterial oxygen saturation for prediction of acute mountain sickness. *Aviat Space Environ Med* 69: 1182–1185, 1998.

29. Schmitt, L, Hellard, P, Millet, GP, Roels, B, Richalet, JP, and Fouillot, JP. Heart rate variability and performance at two different altitudes in well-trained swimmers. *Int J Sports Med* 27: 226–231, 2006.

30. Schmitt, L, Willis, SJ, Fardel, A, Coulmy, N, and Millet, GP. Live high-train low guided by daily heart rate variability in elite Nordic-skiers. *Eur J Appl Physiol* 118: 419–428, 2018.

31. Schoene, RB, Lahiri, S, Hackett, PH, Peters, RM, Milledge, JS, Pizzo, CJ, et al. Relationship of hypoxic ventilatory response to exercise performance on Mount Everest. *J Appl Physiol* 56: 1478–1483, 1984.

32. Siebenmann, C, Rasmussen, P, Hug, M, Keiser, S, Fück, D, Fisher, JP, et al. Parasympathetic withdrawal increases heart rate after two weeks at 3,454 m altitude. *J Physiol* 595: 1619–1626, 2016.

33. Tarvainen, MP, Ranta-Aho, PO, and Karjalainen, PA. An advanced detrending method with application to HRV analysis. *IEEE Trans Biomed Eng* 49: 172–175, 2002.

34. Uusitalo, AL, Uusitalo, AJ, and Rusko, HK. Heart rate and blood pressure variability during heavy training and overtraining in the female athlete. *Int J Sports Med* 21: 45–53, 2000.

35. Vesterinen, V, Häkkinen, K, Hynynen, E, Mikkola, J, Hokka, L, and Nummela, A. Heart rate variability in prediction of individual adaptation to endurance training in recreational endurance runners. *Scand J Med Sci Sports* 23: 171–180, 2013.

36. Vesterinen, V, Nummela, A, Heikura, I, Laine, T, Hynynen, E, Botella, J, et al. Individual endurance training prescription with heart rate variability. *Med Sci Sports Exerc* 48: 1347–1355, 2016.