Case report

Monitoring Heart Rate Variability Before and After a Marathon in an Elite Wheelchair Athlete: A Case Study

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Abstract

The purpose of this study was to analyze heart rate variability (HRV) oscillations before and after a marathon which involved trans-meridian air travel and substantial time zone differences in a professional wheelchair athlete with Charcot-Marie-Tooth (CMT) disease. The natural logarithm of the root mean square difference between adjacent normal R-R intervals (Ln rMSSD) was measured daily on the days before, including and following the race. Relative to baseline, small (-3.8 - -4.6%) reductions in LnRMSSD were observed following relocation and on race-day, indicating only minor effects of travel on cardiac-autonomic activity. On the morning following the marathon, a 23.1% reduction in Ln rMSSD was observed, which returned to baseline by 48 h. The race time set by the athlete was the world-leading time in his class. This case study showed that Ln rMSSD responses to marathon in an elite wheelchair athlete with CMT was similar to those previously reported among unrestricted endurance athletes.

Key words: Autonomic nervous system, athletics, paralympic, cardiac autonomic modulations.

Introduction

The marathon is a physiologically demanding endurance racing event (Billat et al., 2001). While 42195 m completion times of elite runners are ~130 minutes, top wheelchair athletes (i.e. "wheelers") can finish the race within 90 minutes (for those with no upper-extremity impairments, sport classes T53-54) and 115 minutes (for those with trunk and arm impairments, T51-52). Even though mechanical stress in wheelchair racing seems to be lower than in running, cardiovascular load is similar while oxygen transport demands are quite lower (Asayama et al., 1985), possibly due to less muscle mass involved in wheelers than in runners (Fletcher and MacIntosh, 2017). To the best of our knowledge, only one previous study (Asayama et al., 1985) has analyzed heart rate (HR) dynamics in top-finisher marathon wheelchair athletes. The participants from this study maintained a high mean HR $(171.6 \pm 20.5 \text{ beats} \cdot \text{min}^{-1})$ continuously throughout the race. In contrast, a group of elite runners with a personal best marathon time of ≤ 2 hours and 11 minutes had a mean HR of 167 ± 5 beats min⁻¹ during a 10 km test at marathon pace (Billat et al., 2001).

Classical physiological measurements of the function of the cardiovascular system, such as mean HR, are currently being complemented by new signal analyses

-i.e., the heart rate variability (HRV)- that provides more precise information on the autonomic control of HR (Stanley et al., 2013). The most commonly used parasympathetic index is the natural logarithm (ln) of the root mean square differences between adjacent normal R-R intervals (Ln rMSSD) as it seems to be more reliable than other parasympathetic indices such as the high frequency (HF) component of R-R interval variability (Plews et al., 2013). Furthermore. Ln rMSSD has been used in studies of elite athletes (Plews et al. 2012). Ln rMSSD has been shown to display a coefficient of variation (CV) of 5 - 7% in elite endurance athletes and ~10% in recreational runners (Plews et al., 2014). Less day-to-day Ln rMSSD fluctuations (represented by CV) has been associated with more favorable adaptations to training among athletes (Flatt and Esco, 2016).

After strenuous training sessions, HR can be under sympathetic dominance, whilst recovery can be highlighted by the return of parasympathetic modulation to baseline (Brown and Weir, 2001). For example, 48 hours after strenuous exercise like a marathon, there have been reported signs of sympathetic activation in runners (Daniłowicz-Szymanowicz et al., 2005). Additionally, immediately after the completion of a half marathon, elevated sympathetic cardiac drive was shown (Dalla-Vecchia et al., 2014). In a study by Hynynen et al. (2010), a decrease in vagal-related markers (rMSSD and HF) the night after completing a marathon were observed relative to values obtained following only moderate exercise.

Consequently, in elite sport, the assessment of autonomic activity using different indices of HRV have been used for different purposes such as: a) to determine the cardiac regulation during different phases of training, including tapering in disciplines such as rowing (Iellamo et al., 2002) or triathlon (Stanley et al., 2015); b) determine the timing to prescribe intense training sessions when HRV reaches a reference value, considered as the optimal freshness condition for the athlete (Kiviniemi et al., 2007; Vesterinen et al., 2016); and c) to understand the physiological disturbance caused by training load near sea-level (Ornelas et al., 2017) or under stressful environmental conditions such as at altitude (Sanz-Quinto et al., in press). Another variable which could impair autonomic control of HR is trans-meridian air travel with substantial time zone differences (Tatehishi et al., 2002), which is a common circumstance in elite sports competition.

To date, there are no studies with elite wheelchair marathon athletes that have reported the pre-post-race autonomic activity. The current case study reports the daily HRV responses to an eastward trans-meridian flight (Alicante, Spain to Oita, Japan, 8 hour time difference) and a marathon in an elite wheelchair athlete affected by the Charcot-Marie-Tooth disease (CMT).

Methods

Subject

The athlete who participated in this case study was a 35year-old male professional wheelchair marathon athlete with CMT, class T52 by World Para Athletics. CMT is the most common hereditary peripheral neuropathy, affecting up to 30 per 100000 people worldwide (Banchs et al., 2009). CMT totally affects distal muscle function and partially affects proximal function (Banchs et al., 2009). The athlete was a highly accomplished competitor with a silver medal at the 2000 and 2004 Paralympic Games and 106 victories in road events, including Boston, Chicago, London and Oita Marathons. His main descriptive features are: height = 1.76 m; body mass = 52 kg; power output at second lactate threshold = 61 W; heart rate at second lactate threshold = $166 \text{ beats} \cdot \text{min}^{-1}$; training 8000 km per year; former world record holder in his sport class in 800 m (116 s), 1500 m (216 s), 5000m (757 s), half marathon (3028 s) and fourth best-ever time in marathon (6125 s). This study was set up at an international road race where his finishing time (6481 s) was ranked as the world best season time in the sport class T52 at the International Paralympic Committee Athletes ranking.

The participant provided written informed consent to be a research subject in this case study. All the procedures were approved by the Ethics Research Committee of the University Miguel Hernández (Elche, Spain).

Study design

Ten days before the race date (RD) an incremental test was performed on a specific wheelchair ergometer where steady conditions were maintained (temperature 22-24 °C, humidity 73-75 %). The protocol described by Polo-Rubio (2007) included a 20 min warm-up period at constant power (20 W). The athlete started the incremental test at a brake power of 6 W, maintaining a stroke frequency between 90 and 100 strokes min⁻¹, increasing the power by 3 W every 60 s until the athlete could not maintain that frequency. Power output was considered as the ergometer braking power during the last completed step of the test. The same HR monitor used in the marathon was used to register HR and a telemetry system (K4 b², COSMED, Rome, Italy) was used during wheelchair propulsion to measure O₂ uptake and CO₂ production. For calculating the second ventilatory threshold (Vt2), the recommendations by Chicharro et al. (1997) were followed. The Vt2 was estimated when the athlete generated 61 W, the O₂ uptake was 51 ml \cdot kg⁻¹ \cdot min⁻¹ and the HR reached 166 beats min⁻¹ ¹. In the last step, where the athlete was able to maintain the projected stroke frequency, he generated a power of 67 W, and the VO_{2max} was 57 ml·kg·min⁻¹, reaching 176 beats min⁻¹ at that intensity.

Six days before (RD_{-6,-5,-4,-3,-2,-1}) the marathon day in Oita, Japan and two days after racing (RD_{+1,+2}), the day-today HRV upon awakening in the supine position and after bladder-emptying were measured. For HR recordings (Polar RSCX 800, Kempele, Finland), a metronome was used to ensure breathing was consistent (15 breaths min ⁻¹). Detection and correction were applied to the R-R intervals to remove ectopic beats (Tarvainen et al., 2002). The Ln rMSSD was calculated using Kubios HRV 2.0 (Kuopio, Finland, 2008), analysing the last five minutes of each 10min record (Tarvainen et al., 2002). Only natural logarithm (Ln) values were used in the statistical analyses. The rMSSD were chosen as the main vagal HRV index.

For HR recording during exercise, the same-monitor for morning HRV recordings was used. During the race, a GPS (Polar, G3 GPS sensor, Polar ElectroOy, Kempele, Finland) was used to measure speed and record split times every 5000 m (0-5, 5-10, 10-15, 15-20, 20-25, 25-30, 30-35, 35-40) and the last 2195 m (40-42.2).

The athlete flew from Spain (GMT + 1) to Japan (GMT +9) immediately after morning HRV assessment on RD₋₃, so no measurements were taken on RD₋₂ because the trip took almost thirty hours. (Figure 1).

The Japanese Association of Athletics Federations officially approved the marathon course in Oita city. Race conditions were dry with temperatures between 24-26 °C, 77% humidity and $1.39 \text{ m} \cdot \text{s}^{-1}$ winds. The course was almost flat, with start and finish lines at the same altitude, so the only up and down course oscillations were short bridges.

During the 6481 s that the race lasted for the athlete of this study, some nutritional recommendations were followed to avoid a performance decline and optimize HR response (James et al., 2017). A solution of 1250 ml with 70 g of carbohydrate (CHO) was consumed at a comfortable rate, adhering to the minimum rate of 100 ml every 10 min to avoid dehydration (Dennis and Noakes, 1997). Therefore, the athlete's drinking-rate was 116 ml every 10 min. Because the participant did not report gastrointestinal issues in his career with the use of CHO gels (Pfeiffer et al., 2009), at the 31 km mark he consumed a 42 g CHO gel (Glucose + Fructose) (Jentjens et al., 2004). The total CHO rate was 1.03 g·min⁻¹ within the recommended range (Burke et al., 2001).

Statistical analysis

Ln rMSSD was averaged across all days pre-travel (RD-6 – RD-2) to serve as baseline (BL). The smallest worthwhile change (SWC) in Ln rMSSD was determined as \pm 0.5 of the BL standard deviation (Hopkins et al., 2009; Plews et al., 2012). Thus, all Ln rMSSD values obtained post-travel were compared to BL thresholds.

The distribution of marathon-derived exercise HR was examined using the Kolmogorov-Smirnov normality test. A repeated measures ANOVA was carried out for HR variable, including the factor, Race Split, into levels 0-5, 5-10, 10-15, 15-20, 20-25, 25-30, 30-35, 35-40 and 40-42.2. A post hoc LSD multiple range tests determined differences between factor levels. Statistical significance was set at p < 0.05, and all the analyses were conducted using the Statistical Package for Social Sciences (SPSS v. 22, Inc., Chicago, IL, USA).

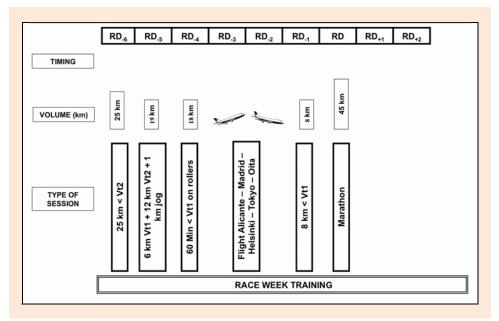


Figure 1. Race week training program.

Results

The average speed of the race was 6.51 m·s⁻¹, while the 5 km segment average of time was 770 s, being both very steady throughout the race. The 25 to 30 km segment was the slowest (6.37 m·s⁻¹), while the fastest was from 5 to 10 km (6.84 m · s⁻¹). The average time every 5 km was 770 s and it was very steady (see Table 1).

Differences between first half marathon (start line to 21096 m) average HR and the second half (21096 to 42195 m) were found ($163 \pm 6 \text{ vs } 167 \pm 6, p < 0.001$). In the first half of the race, the average HR was slightly lower than the mean HR during the full race (165 ± 7), while the second half was slightly higher.

The athlete covered the first half of the race in 53 min and 22 s (3202 s) and the second one in 54 min and 39 s (3279 s). From the start of the race to the 30 km mark, the athlete raced at an intensity slightly below the HR at Vt2 (166 beats \cdot min⁻¹), while from 30 km to the finish, line he raced at an intensity slightly higher than the HR at Vt2 (see Table 1).

The time set by the athlete was the world leading time in his class and 6^{Th} best-ever time in his division in the Oita International wheelchair marathon.

Ln rMSSD values can be viewed in Figure 2. Compared to BL, Ln rMSSD negatively exceeded the SWC (- 4.6%) on the first day post-travel (RD₋₁) and on RD (-3.8%). A greater reduction in Ln rMSSD (-23.1%) was observed one day post-race (RD₊₁). Ln rMSSD returned to within BL at two-days post-race (RD₊₂).

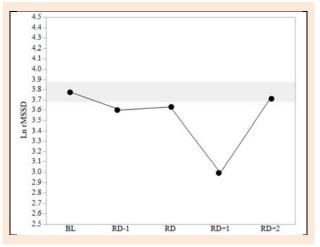


Figure 2. Baseline and daily natural logarithm of the root mean square differences between adjacent normal R-R intervals (Ln rMSSD). BL = baseline; RD-1 = one day before race day; RD = race day; RD+1 = one day post-race; RD+2 = two days post-race. The shaded gray area represents the smallest worthwhile change in Ln rMSSD.

Table 1. Heart Rate, speed and time during the race.									
Km	0-5	5-10	10-15	15-20	20-25	25-30	30-35	35-40	40-42.2
HR	161±5	164±6*	162±6†	165±6*‡	163±7*	163±5*§	169±7*†‡§	170±5*†‡§	170±4*†‡§
SDvt2	- 5.9	- 2.3	- 3.8	- 1.4	- 2.6	- 3.1	+ 3.1	+ 4.1	+ 4.4
Speed	6.43	6.84	6.46	6.69	6.46	6.37	6.40	6.64	6.42
SDMRS	08	+.33	05	+.18	05	14	11	+.13	09
Time	779	732	774	747	777	786	782	755	348
SDMRT	+9	- 38	+ 4	- 23	+ 7	+ 16	+12	- 15	+ 23

HR (beats \cdot min⁻¹), average heart rate in every 5 km segment; SD_{vt2} (beats \cdot min⁻¹), standard deviation of HR over ventilatory threshold of athlete; speed (m \cdot s⁻¹), average speed in every 5 km segment; SD_{MRS} (m \cdot s⁻¹), standard deviation of the speed of every segment over the mean race speed; Time (s), time of each 5 km segment; SD_{MRT} (s), standard deviation of time of each 5 km segment over the mean race time every 5 km.

* Differences from 0-5 (p<.001); † Differences from 5-10 (p<.001); ‡ Differences from 10-15 (p<.001); § Differences from 15-20 (p<.001); \parallel Differences from 20-25 (p<.001); ¶ Differences from 25-30 (p<.001); ** Differences from 30-35 (p<.001); †† Differences from 35-40 (p<.001); ‡‡ Differences from 40-42.2 (p<.001).

Discussion

The aim of this study was to assess Ln rMSSD oscillations before and after travel and a marathon in an elite wheelchair marathon athlete with CMT. The main finding was that Ln rMSSD showed small reductions (-3.8 - -4.6%) relative to baseline following travel, before the race. Ln rMSSD was further reduced (-23%) following the marathon for one day and subsequently returned to baseline two days post-race.

Heart rate

The exercise HR was very similar from the starting line to 30 km at which point the athlete started to lead the race and his HR exceeded the Vt2 intensity (166 beats \cdot min⁻¹). Even though temperature was high at that point of the race (24 °C), hydration status was maintained throughout the race and thus thermoregulation likely did not influence cardio-vascular responses as it has been recently demonstrated (James et al., 2017).

No differences were found regarding the cardiovascular response from top wheelchair athletes who were tested in the same marathon (Asayama et al., 1985) and elite marathon runners (Billat et al., 2001). Even though we did not measure the oxygen uptake during the race, the athlete analyzed in this study exhibited higher values (51 ml·kg·min⁻¹) at marathon intensity (Vt2 = 166 beats·min⁻¹ and power output of 61 W) in a lab test than the athletes tested by Asayama et al. (1985) but lower values than elite runners (Billat et al., 2001).

Speed and time

Speed was steady during the whole race with little variation in the mean race speed ($6.51 \text{ m} \cdot \text{s}^{-1}$) for each segment. The second segment was the fastest with an increment of 0.33 $\text{m} \cdot \text{s}^{-1}$ compared to the mean race speed and the sixth segment was the slowest where the speed was 0.14 m $\cdot \text{s}^{-1}$ under the mean race score. These results are in concordance with Haney and Mercer (2011) who concluded that slower marathon finishers had greater variability of pace compared with faster marathon finishers.

Time for every segment was very similar. It should be noted that in the last segment, entry into the stadium changed the surface of the track compared to the road and could influence the rolling resistance. This explanation has been tested before in cycling (Bertucci et al., 2013), decreasing the speed and increasing the time in this part of the race. Furthermore, the athlete stopped pushing the chair during last 20 m which is another factor that also may have affected this segment time.

HRV

Pre-race Ln rMSSD: Baseline Ln rMSSD (3.77 ± 0.19) was similar to that of a Paralympic swimmer with an undisclosed neuromuscular condition (Edmonds et al., 2014) and to that of recreational endurance runners (Plews et al., 2014). The coefficient of variation of baseline Ln rMSSD (5%) was consistent with that of elite endurance athletes during tapering (Plews et al., 2014).

Ln rMSSD decreased relative to BL upon arrival to Japan. This response has been observed in a case study of

an elite male decathlete who experienced reduced vagallymediated HRV relative to BL, following eastward travel across 6 time-zones (Botek et al., 2009). A previous study involving a team of junior rowers observed reductions in vagally-mediated HRV only after three days of relocation across 5 time-zones (Dransitin, 2008). Recent research has demonstrated that individual responses to air travel are related to both fitness and body composition (Oliveira-Silva et al., 2016). This may explain why the highly fit and lean athlete in the current case study only experienced small reductions in Ln rMSSD in response to relocation.

Reduced Ln rMSSD in the days preceding competition have been attributed to pre-competitive excitement (Morales et al., 2013) or to hemodynamic changes such as a reduction in plasma volume due to reduced training volume from tapering (Plews et al., 2013). The athlete in the current study only performed one workout (below the first ventilatory threshold) in the 3 days before the race due to travel and relocation. A decrease in Ln rMSSD on RD has also been observed in an elite triathlete across five competitions and was associated with an optimal competition performance status (Stanley et al., 2015). It is difficult to determine if the reduced Ln rMSSD on RD-1 and RD were a result of stress from travel (Dransitin, 2008; Botek et al., 2009), effects of temporary training cessation (Plews et al., 2013) or precompetitive anxiety (Morales et al., 2013). However, since the athlete performed very well in the race, the small decrements in Ln rMSSD before the race were unlikely fatigue-related (Hynynen et al., 2006).

A 23% reduction in Ln rMSSD relative to baseline was observed on the day after the race, indicating suppressed vagal modulation. Reduced cardiac-parasympathetic activity for ~24 hours in response to prolonged endurance exercise has been previously observed following a 75 km cross-country ski race (Hautala et al., 2001) and a marathon run (Hynynen et al., 2010). Additionally, a sixmonth study found that recreational marathon athletes showed a substantial decrease in vagally-mediated HRV when an internal load model (i.e. Training Impulse -TRIMP-) reached the highest values (Manzi et al., 2009). Further, a study with seven middle-distance runners showed a decrease of 41% in vagal related markers of HRV during three-weeks of heavy training (Pichot et al., 2000). Collectively, it appears that the wheelchair athlete in the current case study with CMT demonstrated similar cardiacautonomic responses to prolonged endurance exercise as compared with unrestricted endurance athletes (Stanley et al., 2013).

Conclusion

Small reductions in Ln rMSSD were observed following relocation, indicating only minor effects from travel. A much larger reduction in Ln rMSSD was observed one day post-race, which returned to baseline by 48 hours. The race time set by the athlete was the world-leading time in his class. The cardiac-autonomic response to marathon competition observed in this elite wheelchair athlete with CMT was similar to previous findings among unrestricted endurance athletes.

Acknowledgements

The authors wish to thank the athlete who volunteered for this case study. The experiments comply with the current laws of Spain and Japan. Authors declare no conflicts of interest in this research.

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Key points

- Monitoring cardiac-autonomic activity leading up to a race and in response to travel may be useful for objectively monitoring the athlete's condition for performance of the marathon.
- The intensity maintained by the athlete in the race, compared with a pre-race incremental test, can help coaches to optimize tapering in future events.
- A substantial reduction in Ln rMSSD was observed the morning after a marathon and subsequently returned to baseline by 48 h in an elite wheelchair athlete with Charcot-Marie-Tooth disease, which is consistent with responses reported among unrestricted endurance athletes.

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